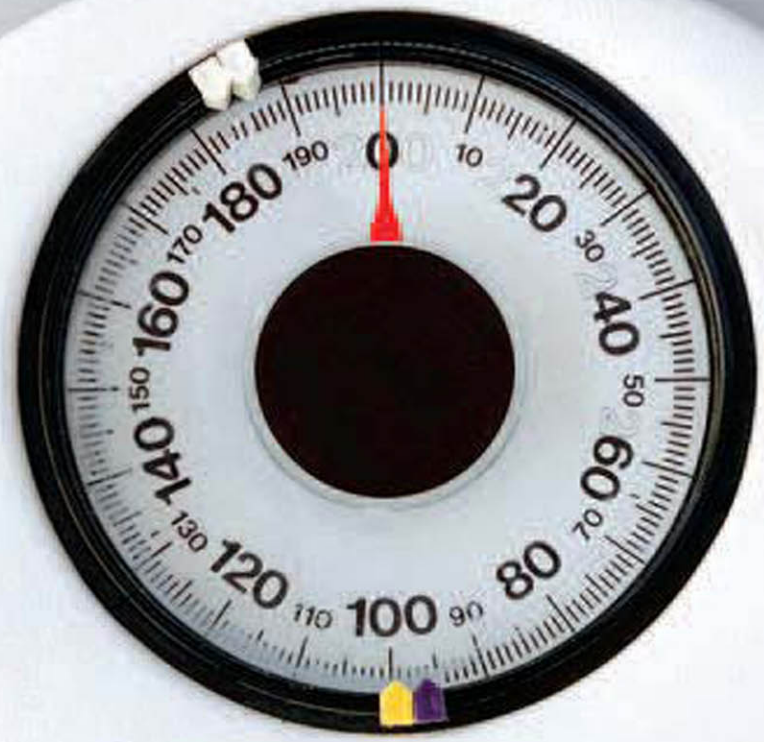

Encyclopedia of OBESITY

Kathleen Keller
GENERAL EDITOR

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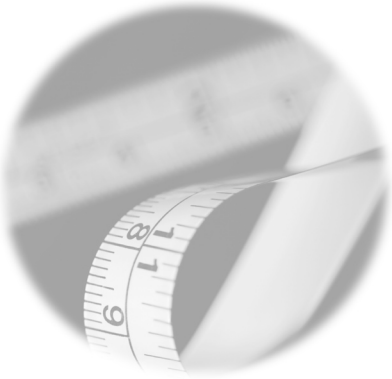

KATHLEEN KELLER
GENERAL EDITOR

A SAGE Reference Publication

SAGE Publications
Los Angeles • London • New Delhi • Singapore

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55 City Road
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Mathura Road, New Delhi 110 044
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SAGE Publications Asia-Pacific Pte. Ltd.
33 Pekin Street #02-01
Far East Square
Singapore 048763

Library of Congress Cataloging-in-Publication Data

Encyclopedia of obesity / Kathleen Keller, general editor.
p. cm.
Includes bibliographical references and index.
ISBN 978-1-4129-5238-5 (cloth)
1. Obesity--Encyclopedias. I. Keller, Kathleen.

RC628.E53 2008
362.196'398003--dc22 2007044491

This book is printed on acid-free paper.
08 09 10 11 12 10 9 8 7 6 5 4 3 2 1

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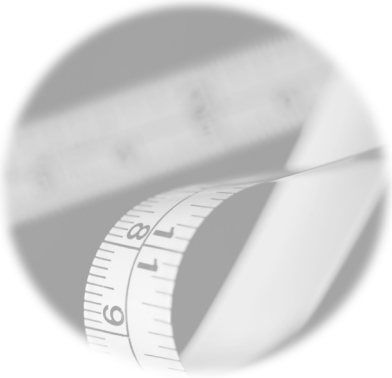
ENCYCLOPEDIA OF

OBESITY



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Encyclopedia of Obesity

About the General Editor

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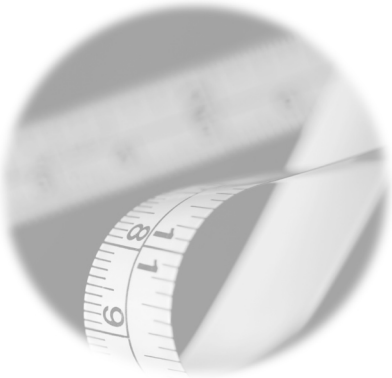
Research Associate, New York Obesity Research Center
Assistant Professor, Columbia University Institute of Human Nutrition

Kathleen Keller received her doctoral degree in Nutritional Sciences at Rutgers University in 2002, and her thesis focused on the genetics of taste perception, eating, and body weight in young children. She completed a three-year post-doctoral training fellowship at the New York Obesity Research Center where she specialized in childhood obesity research studies. In 2005, Keller received funding from the National Institutes of Health to continue research in the area of taste genetics and obesity in children, and she continues to work on this research at present. Most of her studies take place in the Child Taste and Eating Laboratory at St. Luke's Roosevelt Hospital in New York City, where she is currently serving as the Director.

While actively conducting research in childhood obesity, Keller has continued a number of mentoring, teaching, and writing activities. She is an Assistant Professor in the Institute of Human Nutrition at Columbia University, and an Assistant Adjunct Lecturer in the Department of Health Sciences at Brooklyn College. She regularly mentors high school research students, many of whom have gone on to win national recognition for research conducted in her laboratory. Further, Keller writes monthly executive summaries for a Columbia University seminar series on Appetitive Behavior. Through these varied teaching and writing responsibilities, she has gained an appreciation for the complexities of the obesity epidemic and the broad interests of researchers who work in this important field.



Introduction

IN MEDICAL TERMS, obesity is defined simply as an excess amount of adipose or fat tissue. The terms *adipose* and *obesity* both have their origins in Latin. Adipose stems from *adeps*, meaning fat, while obesity comes from the Latin—*obesus*—which, in turn, is a contraction of two Latin words, *ob* and *edere*, meaning to devour or eat away. At the most basic level, obesity is a disease of imbalance. Energy (in the form of calories) that is coming into the body outweighs the energy that is expended.

Historically, the disease has been known in every society, and can be evidenced as far back as the Ancient Egyptians. Hippocrates, the so called father of medicine, made one of the first accounts of the risks posed by being obese, and his suggestions for treatment were not that far removed from diets of today. He prescribed hard labor, sleeping on a hard bed, eating only once per day, eating fatty food for greater satiation, and walking naked as long as possible! Certainly, his patients would have lost weight when following that regimen, however, the practicality of implementing that medical advice today is questionable. The critical issue, however, is why obesity, despite origins in the ancient world, a simple cause and treatment, is one of the most salient threats to the welfare of our present society.

In recent years, the public's general interest and alarm in the obesity epidemic has become apparent. It is no longer possible to avoid the issue: newspapers, the evening news, health magazines, the fashion industry, television and movies, all contribute to the public's both interest, and inevitable saturation from the issues surrounding this epidemic. Cultural analysts would not be far off in stating that our current society is obsessed with both food, and the consequences of an excess of food—obesity. For a society that relies on the mass media for much of its education, the genuine fear of many health and medical professionals is that the general public will receive incorrect messages. As someone who works in the field, I often find myself biting my tongue at family functions or social gatherings, simply to prevent getting involved in arguments about the latest diet fads (most of which are ineffective). However, I also understand the public's deep frustrations over the state of medically purveyed obesity treatments—because there is no magic pill or treatment to cure and eradicate the disease. The best medical advice is to reduce your caloric intake by eating a variety of healthy foods and increase the amount of physical activity you get. Seems quite simple, but still, the majority of us will struggle to maintain a healthy weight, and this struggle is one we must fight for the rest of our lives.

The current interest and obsession with both food and obesity in present society is a predictable reaction to the recent increases in the occurrence of this disease. Over the past three to four decades, the prevalence of obesity in the United States has been consis-

tently on the rise, and depending on what resource you consult, the number of obese adults is estimated at anywhere between 30 and 35percent. If you expand this definition to include overweight (a less severe condition of excess body weight), those numbers skyrocket to between 60 and 65 percent. Further, according to most recent data from the National Health and Nutrition Examination Survey (NHANES), one of the primary surveys designed to study the health and nutrition status of adults and children in the United States, it is estimated that between 16 to 18 percent of children and adolescents are obese, while over one-third are overweight. Clearly, it is no wonder why some experts have suggested that researchers no longer ask the question "What causes people to become obese?" and that a better question might be, "How do some people remain lean in an environment of excess?"

The cut-offs for obesity are defined using reference standards for Body Mass Index, or BMI. BMI is a unit of weight that is adjusted for height, and is thought to be a good estimation of the amount of body fat one has. Depending on where BMI lies, cut-offs for lean, overweight, and obese have been defined. These classifications were set by expert committees who assessed some increased risk of morbidity and mortality with increasing BMI. But these categories are not without controversy, and even the scientific research community is at odds for defining the exact contribution that excess weight makes toward reducing life expectancies. The fact that obesity lowers quality of life is less controversial though, and it is well documented that even losing modest amounts of weight can improve health parameters and improve overall well-being.

While the contribution of obesity to increasing mortality risk in adults can be debated, there is growing concern that today's youths will be the ones to pay most dearly for this epidemic. If estimates continue as projected, the current generation of children will grow up to become the most obese in history. More importantly, it is expected that today's youths will actually be the first to have shorter life expectancies than their parents, a fact mainly attributed to early deaths due to obesity-related diseases. A clear example of this can be seen with Type 2 diabetes, the type of diabetes that results because the body can no longer produce enough insulin to meet the body's needs. Obesity exacerbates the progression of this illness, and over the course of 10 to 20 years, eventually the body becomes completely resistant to the effects of insulin. Until recently, this disease was rarely seen in children. Juvenile onset diabetes, now called Type 1 diabetes, was the form of this disease seen in children. Currently, it is estimated that over half of new diabetes cases in children are now Type 2. Once known as "adult-onset diabetes", Type 2 diabetes can now be seen in children as young as 2 years of age. The combination of the right genetic and environmental conditions has made for astoundingly rapid increases in the development of this disease in a population where it was once nonexistent.

The statistics are undoubtedly alarming. While awareness of the problem is arguably at an all time high, the general public and most health professionals are still unaware as to how to treat obesity, both on an individual basis and at the level of public policy. Furthermore, the perception amongst both the lay public and many health professionals is that obesity is largely due to a lack of will power and occurs in individuals of weak constitution. Thus, we blame the obese individual for his or her condition. Persons who suffer from the illness are made to pay for two seats on an airplane. They are rejected for health and life insurance policies. They are stigmatized in the mass media, in the workplace, and even by the medical professionals who treat them. Rarely has a medical condition been encountered where such ambiguity exists over how to treat the illness, and where on many occasions, the burden and blame is put on the individual. Therefore, the most important reason for this text is to provide education and awareness to the public that obesity is a complex, multi-faceted disease with biological, environmental, and socially mediated causes and consequences.

While several experts in the field recently debated the relationship between obesity and increased mortality, there is little argument for the direct role that being overweight plays on reducing quality of life. The burdens due to obesity, both individually and on a population level, are many. During childhood, obesity is particularly debilitating, and these children are often teased relentlessly and bullied by their classmates. Obesity is also associated with a range of health problems, such as Type 2 diabetes, cardiovascular disease, certain cancers, osteoarthritis, sleep apnea, hypertension, and many others. Thus, shorter life expectancies in obese patients are most likely attributed to the wide range of afflictions that coincide with this condition. Even if life expectancy is not shortened, the quality of those years for an obese individual might be lower, as they are prone to suffer both physically

and cognitively. As a society, we all pay this burden through higher health care costs, which in 2005 were estimated at $75 billion annually. The cost to treat both obesity and obesity related illnesses contributes to nearly 10 percent of the total medical expenditure in the United States.

One of the most important questions to ask is why we, as a society, continue to "expand," and similarly on an individual level, why is it so difficult to maintain a healthy weight. This question will be the focus of many of the topics explored in this encyclopedia. The answer is complex, in that despite the fact that obesity is simply a disease of energy imbalance, the exact reasons for this imbalance are vast, and in some cases unknown. Variations in biology between one human and another have blessed some with a perpetually lean frame, while others continually battle to keep their waist lines down.

Currently, we know of nearly 250 genes that can be altered, with outcomes affecting body weight regulation, and thus risk for obesity. Perhaps more pervading, though, is the current environment in developed countries that seems to encourage energy consumption. There are few places in the world where one can travel and not be within quick access to multiple fast restaurants and convenience stores. Economic conditions are such that high fat, high carbohydrate foods cost much less per calorie than do more healthful fruits and vegetables. To complicate matters, most U.S. cities and suburbs are not designed to promote safe walking or bicycling, but rather favor the convenience of automobile travel. Moreover, this generation of children are faced with multiple media sources, including television, computers, and ever-appealing video games.

Taken together, these environmental conveniences that signify growth and development of society, favor over-consumption, while simultaneously reducing the energy required to perform daily activities. Our genes, some of which were selected for during times of famine and food scarcity, are now faced with survival in a time of unparalleled food surplus. Because we evolved when food environments were much different than they are today, we continue to be much more efficient at holding onto calories than we are at expending them. All of these effects, in concert, have contributed to the current obesity epidemic.

THE ENCYCLOPEDIA

The purpose of this two-volume encyclopedia is to catalog entries from a variety of expert contributors from a vast array of obesity-related disciplines, including molecular biology, psychology, medicine, public health and policy, food science, environmental health, pharmaceuticals, physiology, endocrinology, and many others to summarize pertinent topics in obesity, and related health conditions. Approximately 475 entries are included that address the broad scope of the disease, from molecular and genetic causes, to the treatment of this disease at the public policy level. The editors have chosen topics that capture the current climate of obesity research, while still addressing and defining the core concepts related to this disease. Additionally, we have relied on experts from the medical and scientific research community to provide the majority of entries. In many cases, entries are written by pioneering or premiere researchers in the topic at hand. Our intention in this text is to create a reliable, accurate, and thorough resource for information about the obesity epidemic. Scholars, educators, researchers, physicians, and the general public should be able to access this resource for current, factual details on topics related to obesity.

This encyclopedia is targeted at a variety of audiences, from scholars to the general public. It is intended to serve as a general and nontechnical resource for students, teachers, and researchers who wish to understand the development of obesity as it prevails in the developing and under-developed nations.

Scholars who are conducting research will be able to access hundreds of topics related to obesity, all within a single text. This text should be a helpful reference to enrich current knowledge, or to investigate new topics of interest. Because obesity is such a pervading illness, the editors are dedicated to exploring the broad scope of topics related to obesity, in a user friendly manner that a variety of science and non-science disciplines can access.

In determining which topics to include, the editors first defined 15 to 20 major themes to address. Once the overall themes were established, we developed a list of potential topics to discuss within each theme. As a peer-review of these topics, we accepted feedback from many of the invited contributors, and adapted, added, or deleted many topics based on this review.

The final list is intended to be a broad overview of the relevant issues related to obesity, its etiology, consequences, treatment, outcomes, and policy implications. It is, however, in no way an exhaustive list. Thus, this resource is intended to be a starting place for learning about obesity, with additional references

provided at the end of each topic for additional research.

A quick perusal of these volumes will alert the reader to several obvious details about the text. First, the scope of obesity is extremely broad. A number of the articles address specific genes, proteins, receptors, or hormones that are involved with the development of obesity. Obviously, it would not be feasible, or practical, to write entries on each of the genes or proteins associated with obesity. Thus, the editors and contributors attempted to capture the "highlights" of this genetic research, with general headings on the genetics of obesity addressing this literature on a broad spectrum.

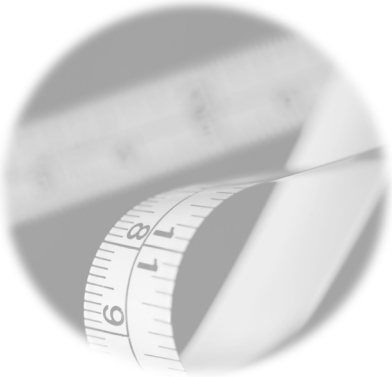
Second, the reader will notice that the layout of the text is much like that of a standard reference encyclopedia. This convenient organization will assist readers most when they have a specific idea of the term they wish to investigate, however, for readers who do not know the specific search term, each article is cross-referenced with other article headings. Third, as in most encyclopedias, there is some repetition in the material in these texts. For example, the topic of eating disorders is addressed in a number of sub-topics, ("Disordered Eating," "Eating Disorders and Athletes," "Eating Disorders and School Children," and "Eating Disorders and Gender"). Each entry will have a slightly different focus, but some major concepts might be reviewed in multiple entries. This redundancy is considered by the editors to be a positive attribute, since inevitably the themes that are repeated are ones that should be considered "take home messages."

MOVING FORWARD IN AN EVER-EXPANDING WORLD

In 2002, two teenagers from New York City filed a lawsuit against McDonalds alleging that daily exposure to the fast food giant's meals caused them to become obese and face lifelong health problems. Two years later, a documentary called "Super Size Me" followed Morgan Spurlock over 30 days of consuming McDonald's super sized entrees for three meals a day. Over the course of this time, he faced a range of unpleasant side effects, and medical consequences, the most logical conclusion being that McDonald's caused these medical nightmares. As a third insult, Eric Schlosser's 2001 book *Fast Food Nation* was made into a movie in 2006, which made light of the dark side of the fast food industry—one that is interested only in the bottom line and cares little about the quality of food and health of consumers. These examples of what seemed to be common themes in the twenty-first century shed light on the mindset of many individuals within the field. As prominent Yale psychologist and weight loss expert Kelly D. Brownell describes, many feel as though we are living in a "toxic environment." Undoubtedly, the politics of food and food acquisition in the United States and developing countries have made conditions ideal for the development of widespread obesity. The future of this epidemic will likely see more attempts to strike against this system, and to fix problems at the policy level, so that on an individual basis, living a healthier lifestyle is feasible and encouraged. While smaller changes, such as taxation of "empty calorie" foods like sodas and candies in some states, have already occurred, it is unlikely that the fast food industry will be made to pay for these costs. None of the many health-related lawsuits against these industries have been successful, and in 2004, the House banned future fast food lawsuits.

Despite limited success in striking against fast food industries, many other changes in the environment to favor healthier lifestyles are occurring. In 2006, the Board of Health in New York City voted to make it the first major city to ban the use of trans fats in restaurants by 2008. Following the largely successful ban on smoking in this city, the outlawing of trans fats was another step in the right direction. While trans fats are probably no more likely to make you obese than saturated or unsaturated fats, they have been linked to other health problems, such as coronary heart disease. Other major cities have passed similar laws. In addition, there are countless other examples of local and state initiatives to encourage more healthful lifestyles, by encouraging better access to parks, improving and building walking paths, reinstating physical activity in schools, improving choices of school lunches, and a variety of other examples, some of which will be highlighted in this text. While this does not eliminate the responsibility on the individual, it does help procure an environment that facilitates access to healthful food and safe activity for all, regardless of what neighborhood, city, state, or country. Consequently, these policy changes over the past few years appear to be moving us in the right direction. With continued government interest and increased personal awareness, the ability to reverse the effects of this disease are well within reach.

Kathleen Keller
General Editor



Reader's Guide

This list is provided to assist readers in finding articles related by category or theme.

BIOLOGICAL OR GENETIC CONTRIBUTORS TO OBESITY

Adipocytes
Adiponectin
Adrenergic Receptors
Agouti and Agouti Related Protein
Animal Models of Obesity
Animal QTLs (Quantitative Trait Locus)
Bardet-Biedl Syndromes
Cannabinoid Receptor
CD36 and FAT (Fatty Acid Transporters)
Cholecystokinin (CCK)
Cortisol
Cushing Syndrome
Cytokines
Db/Db Mouse
Dopamine Receptor
Down's Syndrome
Epistatic Effects of Genes on Obesity
Estrogen-Related Receptor
Familial Lipodystrophies
Fatty Acid Transport Proteins
Genetic Taste Factors
Ghrelin
Glucagon Receptor
Glucocorticoids
Glucokinase
G-Protein Coupled Receptors
Growth Hormone
HDL Receptors
Histamine Receptor
Hormone Sensitive Lipase
Human QTLs
Hypothyroidism
Insulin and Insulin Resistance
Insulin-Like Growth Factors
Interleukins
Intrauterine Growth Restriction
LDL Receptors
Leptin
Leptin Receptors
Lipoprotein Lipase
Low Birth Weight
Melanocortins
Mendelian Disorders Related to Obesity
Metabolic Rate
Monogenic Effects that Result in Obesity
Neuropeptides
NPY (Neuropeptide Y)
Obesity and the Immune System
Obesity Gene Map
Ob/Ob Mouse
Opioid Receptor
Perilipins

Binge Eating
Body Dysmorphic Disorder
Body Image
Bulimia Nervosa
Childhood Onset Eating Disorders
Cognitive-Behavioral Therapy
Depression
Dieting: Good or Bad?
Disinhibited Eating
DSM-IV
Eating Disorders and Athletes
Eating Disorders and Gender
Eating Disorders and Obesity
Eating Disorders in School Children
EDNOS
Families of Eating Disorder Patients
Feminist Perspective and Body Image Disorders
Genetic Influences on Eating Disorders
Hunger
Neurotransmitters
Night Eating Syndrome
Physiological Aspects of Anorexia
Physiological Aspects of Bulimia
Prevalence of Disordered Eating
Sexual Abuse and Eating Disorders
Treatment Centers for Eating Disorders
Weight Cycling and Yo-Yo Dieting

ENVIRONMENTAL CONTRIBUTORS TO OBESITY

Accessibility of Foods
Advertising of Foods to Children
Children's Television Programming
Economics of Food
Energy Density
Fast Foods
Food Advertising
Food Labeling
Governmental Subsidizing of Energy Dense Foods
Inaccessibility of Exercise
Increased Reliance on Automobiles
Increasing Portion Sizes
Palatability
Parental and Home Environments
Safe Play Opportunities for Children
School Lunch Programs
Schools and Obesity
Sodas and Soft Drinks
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Supersizing
Television
Toxic Environment

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Colon Cancer
Congestive Heart Failure
Depression
Elevated Cholesterol
Fatty Liver
Fertility
Fitness
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High-Density Lipoproteins
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Menstrual Problems
Mortality and Obesity
Osteoarthritis
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Ovarian Cancer
Ovarian Cysts
Overall Diet Quality
Polycystic Ovary Disease
Respiratory Problems
Sexual Health
Sleep Apnea
Stroke
Type 2 Diabetes
Urinary Incontinence in Severe Obesity in Women
Uterine Cancers

MEDICAL TREATMENTS FOR OBESITY

American Medical Association
American Obesity Association
Amphetamines
Caffeine
Cost of Medical Obesity Treatments
Dexatrim
Dieting: Good or Bad?

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OBESITY AND ETHNICITY/RACE

OBESITY AND THE BRAIN OR OBESITY AND BEHAVIOR

Compulsive Overeating
Depression
Disordered Eating
Eating Disorders in School Children
External Controls
Loneliness
Night Eating Syndrome
Obsessive Compulsive Disorder
Psychiatric Medicine and Obesity
Self-Esteem and Obesity
Stress
Suicidality
Well-Being

SOCIETAL INFLUENCES AND OUTCOMES OF OBESITY

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Appearance
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Breastfeeding vs. Formula Feeding
Built Environments
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Carbohydrate and Protein Intake
Computers and the Media
Eating Out in the United States
Fat Acceptance
Fat Intake
Flavor Learning
Food Advertising and Obesity
Food Guide Pyramid
Food Intake Patterns
Food Labeling
Food Preferences
Governmental Policy and Obesity
Income Level and Obesity
Nutrition Education
Obesity and Academic Performance
Obesity and Drug Use
Obesity and Sports
Obesity and the Media
Obesity in Schools
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Physical Activity Patterns in the Obese
Smoking
Soda and Soft Drink Intake
Stereotypes and Obesity
Supersizing
Variety of Foods and Obesity
Virtual Environments
Weight Discrimination
Women and Dieting
Western Diet

WOMEN AND OBESITY

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Body Image
Breast Cancer
Breastfeeding
Colon Cancer
Coronary Heart Disease in Women
Early Onset Menarche and Obesity in Women
Economic Disparities among Obesity in Women
Endometrial and Uterine Cancers
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Maternal Influences on Child Feeding
Menopause and Obesity
Morbid Obesity in Women
Obese Women and Social Stigmatization
Polycystic Ovary Disease
Pregnancy
Prevalence of Obesity in U.S. Women
Self-Esteem in Obese Women
Support Groups for Obese Women
Waist-to-Hip Ratio
Women and Diabetes
Women and Dieting

WORLDWIDE PREVALENCE OF OBESITY

Africa, North
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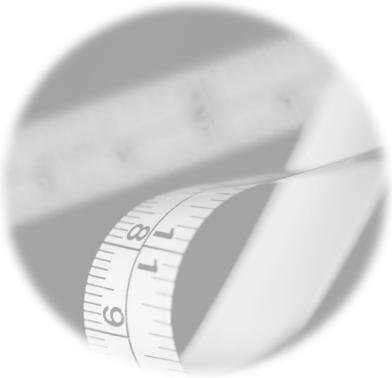
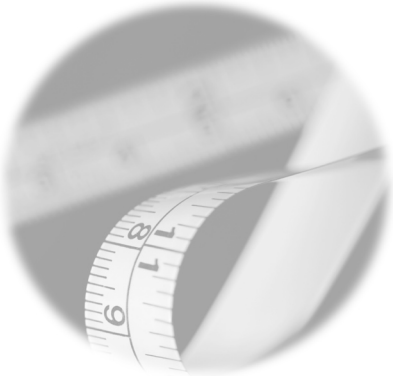
Asia, Central
Asia, East
Asia, South
Asia, Southeast
Australia and Pacific
Central America and Caribbean
Europe, Eastern
Europe, Western
Middle East
North America
South America
World Patterns



List of Articles



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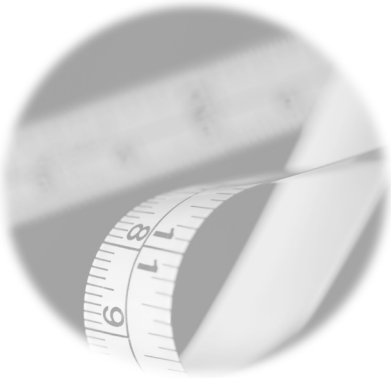

Body Mass Index

Body Mass Index (BMI), also known as the Quetelet Index (see next page), is a measure combining the weight and height of an individual. Body mass index can be calculated by dividing the weight by the square of the height.

Although invented in the mid 1800s by Belgian Adolphe Quetelet, BMI is currently used as a simple clinical tool for health care professionals to determine a patients potential risk for disease and call for intervention. However, BMI has been recently and incorrectly as an "absolute" tool for medical diagnosis and care rather than an suggestive indicator of risk.

BMI is used to classify individuals into categories. The most common definitions used clinically in the United States for BMI categories include Underweight (BMI<18.5), Ideal (BMI 18.5–25), Overweight (BMI 25–30), Obese (BMI 30–40) and Morbidly Obese (BMI over 40). According to the U.S. National Health and Nutrition Examination Survey in 1994, 59 percent of American men and 49 percent of women have BMIs over 25 and therefore categorized as either overweight or obese.

BMI categories have important correlations with disease risk. Higher BMI values are associated with cardiovascular disease and higher morbidity and mortality rates for almost any disease. According to some studies, a BMI of 30 and greater translates to an increase risk of death from any cause by 50 to 150 percent. Extremely low BMI values, below the ideal category, may indicate malnutrition or an eating disorder. Although a correlation may exist, researchers are also quick to acknowledge that BMI is not entirely accurate in predicting the risk of cardiovascular disease and death because it does not distinguish body muscle and fat. However, when used appropriately, categorization of an individual using the BMI can indicate to a healthcare provider that some intervention may be needed.

Jonathan Tan

To use the table, find the appropriate height in the left-hand column labeled Height. Move across to a given weight (in pounds). The number at the top of the column is the BMI at that height and weight. Pounds have been rounded off.

Body Mass Index Table

	Normal						Overweight					Obese										Extreme Obesity															
BMI	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**	**29**	**30**	**31**	**32**	**33**	**34**	**35**	**36**	**37**	**38**	**39**	**40**	**41**	**42**	**43**	**44**	**45**	**46**	**47**	**48**	**49**	**50**	**51**	**52**	**53**	**54**
Height (inches)	**Body Weight (pounds)**																																			
58	91	96	100	105	110	115	119	124	129	134	138	143	148	153	158	162	167	172	177	181	186	191	196	201	205	210	215	220	224	229	234	239	244	248	253	258
59	94	99	104	109	114	119	124	128	133	138	143	148	153	158	163	168	173	178	183	188	193	198	203	208	212	217	222	227	232	237	242	247	252	257	262	267
60	97	102	107	112	118	123	128	133	138	143	148	153	158	163	168	174	179	184	189	194	199	204	209	215	220	225	230	235	240	245	250	255	261	266	271	276
61	100	106	111	116	122	127	132	137	143	148	153	158	164	169	174	180	185	190	195	201	206	211	217	222	227	232	238	243	248	254	259	264	269	275	280	285
62	104	109	115	120	126	131	136	142	147	153	158	164	169	175	180	186	191	196	202	207	213	218	224	229	235	240	246	251	256	262	267	273	278	284	289	295
63	107	113	118	124	130	135	141	146	152	158	163	169	175	180	186	191	197	203	208	214	220	225	231	237	242	248	254	259	265	270	278	282	287	293	299	304
64	110	116	122	128	134	140	145	151	157	163	169	174	180	186	192	197	204	209	215	221	227	232	238	244	250	256	262	267	273	279	285	291	296	302	308	314
65	114	120	126	132	138	144	150	156	162	168	174	180	186	192	198	204	210	216	222	228	234	240	246	252	258	264	270	276	282	288	294	300	306	312	318	324
66	118	124	130	136	142	148	155	161	167	173	179	186	192	198	204	210	216	223	229	235	241	247	253	260	266	272	278	284	291	297	303	309	315	322	328	334
67	121	127	134	140	146	153	159	166	172	178	185	191	198	204	211	217	223	230	236	242	249	255	261	268	274	280	287	293	299	306	312	319	325	331	338	344
68	125	131	138	144	151	158	164	171	177	184	190	197	203	210	216	223	230	236	243	249	256	262	269	276	282	289	295	302	308	315	322	328	335	341	348	354
69	128	135	142	149	155	162	169	176	182	189	196	203	209	216	223	230	236	243	250	257	263	270	277	284	291	297	304	311	318	324	331	338	345	351	358	365
70	132	139	146	153	160	167	174	181	188	195	202	209	216	222	229	236	243	250	257	264	271	278	285	292	299	306	313	320	327	334	341	348	355	362	369	376
71	136	143	150	157	165	172	179	186	193	200	208	215	222	229	236	243	250	257	265	272	279	286	293	301	308	315	322	329	338	343	351	358	365	372	379	386
72	140	147	154	162	169	177	184	191	199	206	213	221	228	235	242	250	258	265	272	279	287	294	302	309	316	324	331	338	346	353	361	368	375	383	390	397
73	144	151	159	166	174	182	189	197	204	212	219	227	235	242	250	257	265	272	280	288	295	302	310	318	325	333	340	348	355	363	371	378	386	393	401	408
74	148	155	163	171	179	186	194	202	210	218	225	233	241	249	256	264	272	280	287	295	303	311	319	326	334	342	350	358	365	373	381	389	396	404	412	420
75	152	160	168	176	184	192	200	208	216	224	232	240	248	256	264	272	279	287	295	303	311	319	327	335	343	351	359	367	375	383	391	399	407	415	423	431
76	156	164	172	180	189	197	205	213	221	230	238	246	254	263	271	279	287	295	304	312	320	328	336	344	353	361	369	377	385	394	402	410	418	426	435	443

Source: Adapted from *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report.*

Accessibility of Foods

IF OBESITY DEPENDS to some extent on the overeating of food, then removing access to that food should logically lead to a reduction in cases of obesity. On the other hand, easy access to foods and intensive marketing efforts to promote frequent overconsumption can increase the prevalence of obesity. Of course, the principal issue with accessibility to food globally is the lack of food security faced by many millions of people. Although the green revolution of the 1970s led to the massive increase in the productivity of rice and wheat strains, alleviating problems of endemic starvation for millions, there continue to be many problems for those vulnerable peoples whose lives are threatened by climate change and environmental degradation. The inevitable added pressure of irrigation water needs have intensified concerns over the depletion of clean water sources for some of the very poorest people.

However, in terms of obesity, accessibility implies a positive correlation between the presence of food and the overindulgence in eating. The human body and human societies have spent centuries or millennia evolving to the relative presence or scarcity of foods and devoting resources to ensuring the availability of some fresh, or at least edible food for as long as possible. This has led to the salting and drying of meats, fish, and fruit; the pickling of vegetables; and the various technologies of smoking, preserving, and bottling. In countries in which naturally occurring spices and herbs are few (as in Britain and much of northern Europe), this promoted the enormously high prices of spice trade commodities and possibly stimulated the drinking of wine or beer to vary the endlessly salty taste of food for the majority of the year. In other countries, it has provided the foods that are now the staples and delicacies of their respective cuisines; *kimchi* in Korea is one notable example of this.

For the majority of people the majority of the time, food was very basic and involved a measure of the staple carbohydrate mixed with a small amount of vegetable or protein. Societies fortunate enough to live in climates in which fruit grows year-round have a natural alternative source of energy and, in premodern years, those people tended to be stronger and healthier than those from colder climes. However, as agricultural productivity has improved and economic development has led to increased incomes for many millions of people around the world, it has become possible for people to eat the food once reserved for feasts or other special occasions on a regular basis, perhaps even on a daily basis. This has led to an imbalance with the ways in which human bodies have evolved in order to deal with the intake of calories. The sudden, massive, and regular increase in calories has led to numerous problems with obesity from people whose bodies cannot cope with this unprecedented

diet. This is a relatively recent development: in most of the Western world there was widespread threat of famine as recently as the years following World War II (post-1945).

Globalization, too, has had an impact on accessibility of food and its relationship with obesity. First, food from around the world is now available in many places year-round. Those foods may have originally been designed as special feast foods or else as part of a specialized local diet. Taken from their context and perhaps adapted to local tastes by the addition of sweet or salty additives, these foods can cause rapid weight gain in bodies unadapted to them. The significant increase in obesity in many of the developing countries of East Asia may be attributed at least in part to this phenomenon, as Western foods (including dairy foods and chocolate-based confectionery) have been introduced in great quantities in societies in which they were previously almost entirely unknown.

Second, globalization also spreads different ideas and concepts of how other people around the world live. The accessibility of other cultures brings with it both the foods of other cultures and the encouragement to try these new foods. The massive amount of marketing money behind the internationalization of American fast-food chains underscores the extent to which it is possible to change eating behavior through these means.

A third implication of globalization is that it changes eating patterns. Partly through providing evidence of alternative lifestyles in which the historical nuclear or extended family ate the same food at the same time, and partly through creating different types of working life and lifestyle, globalization has changed the ways in which societies deal with food and eating. There has been a tendency to cause people to work longer, to eat at their desks, to graze throughout the waking day rather than at set times and to favor speed and taste rather than quality and nutrition.

These factors have been supported by the incredibly successful development of creation and distribution chains. In conjunction with the global phenomenon of urbanization, this has made possible a lifestyle in which obesity is a prevalent risk, whatever the attractions of that lifestyle. Habits and customs designed to enable the body to maximize the calorific value of foods in a time when food security was in short supply are now largely counterproductive among people to whom food availability is never a problem. The pace of change even seems to be accelerating in developed countries in which people are increasingly time-poor and, hence, tempted to search for short-term solutions for feeding and gratification which in the long-term are destructive to their health. Whether it is feasible to reverse these tendencies completely is currently unknown. However, local initiatives in some parts of the world have achieved encouraging results by promoting consumption of foods produced within the surrounding area, treating such foods in traditional ways, and encouraging people to enjoy the foods at a much slower pace. In addition to many other effects, this movement has the benefit generally of increasing the quality of ingredients and, hence, making dishes much more palatable than they have been traditionally considered. Greater and more effective attempts at educating people in how to shop, cook, and eat food would also yield beneficial results. While this is occurring in countries like the United Kingdom, there is still a long way to go.

SEE ALSO: Access to Nutritious Foods; Hunger.

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Access to Nutritious Foods

THE ENVIRONMENT STRONGLY influences food purchasing and consumption behaviors. The ability to obtain nutritious and affordable food is of particular importance to individuals of low socioeconomic status (SES), who must stretch limited resources to meet food and other vital needs. Yet, low SES individuals are disproportionately impacted by poor food access and high rates of obesity. Low SES individuals confront many barriers to the procurement of healthy food, including living in areas that are termed "food desserts." This term refers to areas in which it is dif-

ficult to obtain a variety of healthy foods at affordable prices. Reasons for this difficulty include an absence of supermarkets, limited healthy food available at local markets, and inadequate transportation.

ABSENCE OF SUPERMARKETS

Low SES individuals disproportionately live in areas with fewer large supermarkets than their higher income counterparts. Middle- and upper-income neighborhoods have more than twice as many supermarkets per capita than low-income neighborhoods. Residents of low SES neighborhoods are more reliant on smaller shops, convenience stores, and bodegas to acquire food. As such, compared to people living in higher-income areas, residents of low-income urban neighborhoods have limited access to high-quality food, enjoy fewer options in the variety of goods that are available to them, and pay higher prices for the groceries that are available.

LIMITED AVAILABILITY OF HEALTHY FOODS

Food availability in grocery stores is related to the diets of residents in the area of the store. Small corner stores typically do not sell the great a breadth of foods as do larger supermarkets, and available food choices tend to be more costly. Because of slower stock turnover, corner stores tend to stock more nonperishable foods than do larger supermarkets. These items require little sales experience to sell, and do not spoil or need care. As such, individuals residing in areas where corner stores provide the only available food choices may be confronted with the inability to access healthy foods, because those foods are not on their store shelves or are not affordable choices.

INADEQUATE TRANSPORTATION

Residents of many low SES communities lack access to a supermarket within a reasonable walking distance of their homes. This group may also experience difficulty traveling to distant shopping facilities because of a lack of private transportation. Without cars, residents must depend on public transit, taxis, or friends to travel to supermarkets outside of their neighborhoods. Even with sufficient public transportation, transporting large, heavy bags of groceries can be difficult on buses or subways. Taxi services are costly and friends are often unavailable or unreliable. Therefore, absence of proximate supermarkets often represents an inability to purchase nutritious, affordable food, particularly fresh fruits, vegetables, and other perishables. Healthy diets are compromised, contributing to a high prevalence of obesity.

The availability of nutritious foods is directly related to the number of supermarkets in the area of people's homes.

READY AVAILABILITY OF FAST FOOD

While the abundance of fast-food restaurants in low SES neighborhoods may provide a source of convenient and relatively cheap, tasty food, these establishments typically do not offer nutritious foods such as fresh fruit and vegetables. Instead, a typical fast-food meal is very calorie dense. Individuals who eat at fast-food restaurants tend to consume more fats, sugars, and carbohydrates and fewer fruits and nonstarchy vegetables than individuals who do not eat fast food.

FEDERAL NUTRITION PROGRAMS

Federal nutrition programs can increase access to high-quality, nutritious foods and improve nutrition

education for limited-resource families. Programs, such as food stamps and child nutrition initiatives support access to nutritious foods through supermarkets and farmers' markets in low SES neighborhoods. Yet, in many low SES neighborhoods, food stamp recipients are still at risk for food insecurity, defined as a "lack of access to enough food to fully meet basic needs at all times," due to the lack of such foods at local markets. Moreover, many individuals and families who are eligible for food stamps are not enrolled in the program.

SEE ALSO: Food Insecurity and Obesity; Hunger; Obesity and Socioeconomic Status.

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Acomplia

ACOMPLIA IS THE trade name of rimonabant (generic name), which has been approved in the European Union (EU), Argentina, and Mexico for the treatment of obesity. As of March 2007, rimonabant (the proposed U.S. trade name is Zimulti) is under review by the Food and Drug Administration (FDA) and it is not yet available in the United States.

Rimonabant is the first inverse agonist discovered for the cannabinoid-1 receptor (CB1R). Although rimonabant binds to the CB1R, it is not a cannabinoid-like compound and bears no structural resemblance to cannabinoids.

In cell-based in vitro assays, rimonabant inhibits the intrinsic activity of CB1R and produces an effect opposite that of an agonist; hence, rimonabant is classified as an inverse agonist. It also inhibits the binding of cannabinoid agonists and endocannabinoids to the CB1R, so it is sometimes referred to as an antagonist. In experimental animal studies, rimonabant does not exhibit cannabimimetic properties and it inhibits many agonist-induced effects. In rodent studies, rimonabant can cause significant weight loss or reduction of weight gain.

In clinical trials involving obese patients, rimonabant has been shown to cause weight loss and reduction of waist size. Beneficial secondary end points have also been demonstrated, including increased high-density lipoprotein cholesterol (HDL-C) and improved glycemic control.

Rimonabant at 20 mg is generally well tolerated. As described in the Summary of Product Characteristics approved by the EU regulatory agency, the most common adverse reactions resulting in discontinuation include nausea, mood alteration with depressive symptoms, anxiety, and dizziness.

SEE ALSO: Cannabinoid System; G-Protein Coupled Receptor.

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Addictive Behaviors

ADDICTION IS A psychiatric term that encompasses not only the uncontrolled use of drugs and the associated physical and psychological dependence to the substance, but also the social consequences of use. *Addictive behavior* is a term applied to a varying group of activities that share a common characteristic: they all overtake the time and attention of an in-

Addiction to substances create a vicious cycle of use, depression, lowered self-esteem, and further use.

dividual to the exclusion of other responsibilities. The emphasis on "to the exclusion of other responsibilities" is an important one, because there are examples of exclusive attention that are not considered addictive or undesirable, such as meditation and prayer as practiced by members of religious communities.

Common behaviors that can become addictive include use of illegal drugs, drinking of alcohol, smoking of tobacco products, gambling, shopping, sex, and exercising. Some people also consider excessive food intake to be an addiction. The negative social consequences associated with addictions proceed from characteristics common to most of the behaviors listed above, namely: (1) craving (the obsessive thinking and desire for the substance or activity even in the absence of cues that indicate the availability of the substance); (2) compulsive seeking for the substance or activity even in the presence of harm; (3) loss of control over the activity even when professing conscious desire to stop; (4) physical and/or psychological withdrawal symptoms after stopping the activity or substance usage; (5) lack of awareness of activities while engaged in substance use or other addictive activities; (6) denial of the detrimental effects of their actions toward substance use or activities; and (7) denial of substance use and behaviors and secretive substance use and behaviors. These characteristics are usually accompanied by feelings of low self-esteem and depression that create a vicious cycle of use, depression, lowered self-esteem, and more use.

While all addictive behaviors share common characteristics, they all differ in the relative risk of addiction through use or participation. Among the common abused drugs, relative rates of addiction from use are as follows: nicotine, 1:2; heroin, 1:3; cocaine, 1:5; marijuana, 1:9; and alcohol, 1:10. Behavior addiction risks are much lower, in the range of 1:100. The risk of addiction estimates the number of people who will become addicted among all people who engage in the particular activity; it does not address why a particular person will become addicted to one substance or activity versus another, or identify what substance or activity might be a successful alternative to divert the individual from his or her addiction.

SEE ALSO: Carbohydrate "Addictions"; Food "Addictions."

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Adipocytes

ADIPOCYTES ARE THE cells that constitute the adipose tissue, more commonly known as fat cells. Adipocytes have many functions in the body. Their primary function is as the storage site for triglycerides, the primary form in which the body stores fatty acids. Adipocytes can either use these stored fatty acids when energy is needed (during starvation), or they can release fatty acids into the body so that

other organs, such as the heart and muscle, can use fatty acids when they need energy.

Adipocytes have many other functions. For example, fat tissue plays an important role in insulating the body and providing cushioning around many organs. Another newly discovered role is that adipocytes have endocrine-like properties, meaning the adiopocytes can produce hormones (sometimes referred to adipokines) that affect the actions of other parts of the body. Leptin, the first adipokine isolated, was discovered 15 years ago. Leptin is believed to play a role in regulating a person's food intake and energy use. Other adipokines include adiponectin, resistin, TNF-alpha, and many interleukins. Many of their functions are still being clarified and there are likely many yet-to-be-discovered adipokines.

There are two different types of adipocytes in mammals: brown and white, a reference to the appearance of these cells under a microscope. Brown adipocytes have a greater number of mitochondria, more blood vessels, and uncoupling proteins (UCP) surrounding them than do white adipocytes. Brown adipocytes can generate heat while white adipocytes cannot. Adult humans have very few brown adipocytes, while infant humans have relatively many brown adipocytes. It is hypothesized that the brown adipocytes help regulate body temperature and provide energy for a developing child. Once development has ceased, brown adipocytes function diminishes and white adipocytes become the primary type of adipocytes in the body. Mammals that hibernate, such as bears, have many brown adipocytes, as these cells help normalize bottom temperature at the end of hibernation.

When a person loses weight, his or her adipocytes become smaller. This is because he or she is using up the stored fat within the cell. However, these adipocytes will still remain present in the body. This means once a person consumes extra calories, the adipocytes will be ready to store the excess fatty acids again. This may be one reason why it is so difficult for dieters to maintain their weight loss.

SEE ALSO: Adiponectin; Fatty Acid Transport Proteins.

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Adiponectin

THIS ENTRY DESCRIBES the adipose tissue hormone, adiponectin, its physical characteristics, and the variations with body fatness, gender, insulin sensitivity, and cardiovascular disease, highlighting possible links between its circulating size and form and metabolic and antiinflammatory actions.

Adiponectin, also known as Acrp30 or AdipoQ, is a hormone almost exclusively produced by adipose tissue. It is the most abundant adipose-derived circulating protein, being measured at high concentration (2–20 ug/ml), with typically higher levels in females than males. Obese people have lower levels of adiponectin. These levels increase after weight loss and correlate (negatively) better with the amount of visceral (rather than subcutaneous) fat. Lower levels are also found in insulin resistance, Type 2 diabetes, and cardiovascular disease. The protein may bind directly to molecules that cause inflammation, reducing their harmful potential, or may act to improve glucose tolerance and insulin sensitivity. It does this by binding to two distinct receptors widely present in the body, especially in skeletal muscle and the liver. Receptor binding leads to activation (by phosphorylation) of an enzyme called AMP-activated protein kinase, which in turn causes metabolic changes in cells, restoring energy balance. These changes include the increased uptake and use of glucose and the oxidation of fatty acids, which reduces fat content in the tissue. Insulin-sensitizing drugs such as thiazolidinediones increase circulating levels of adiponectin. This improves insulin sensitivity and may lessen atherosclerosis through anti-inflammatory actions on macrophages and foam cells.

The basic subunit is a single protein chain (a monomer), but the circulating forms are a trimer formed by the self-association of three monomers, a hexamer, and higher molecular weight (HMW) species. These are stable in circulation, and each has different meta-

bolic activities. High levels of HMW species are repeatedly associated positively with insulin sensitivity. Post-translational modification to the protein, including addition of sugar groups to amino acids in a collagen-like tail region, are crucial to the formation of HMW species, and to their insulin-sensitizing activity. However, the exact mechanisms controlling how adiponectin activates its receptors are unclear.

Mutant forms of the molecule which are unable to form HMW species have been found in some patients with diabetes, while mice in which the gene producing adiponectin has been destroyed tend to show insulin resistance, glucose intolerance, and blood vessels susceptible to damage. This is consistent with the postulated effect of adiponectin as a protective factor from atherosclerosis. Polymorphisms (inherited variations) of the genes for adiponectin and its receptors have also been associated with insulin resistance in humans.

SEE ALSO: Adipocytes; Inflammation; Insulin; Type 2 Diabetes.

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Adrenergic Receptors

ADRENERGIC RECEPTORS, ALSO referred to as adrenoceptors, are a class of G-protein coupled receptors that are targeted by catecholamines. These receptors, found in peripheral tissue, initiate a "fight-or-flight" sympathetic nervous response when bound to their endogenous ligands. Specifically, adrenergic receptors are targeted by the catecholamines epinephrine and norepinephrine (also known as adrenaline and noradrenaline, respectively). When the adrenal medulla releases these catecholamines in response to stress, the agonist-adrenergic receptor complex initiates responses including pupil dilation, energy mobilization, blood diversion toward skeletal muscle, sweating, increased blood pressure, and increased heart rate.

There are several subtypes of adrenergic receptors. The two major classes include the alpha-adrenergic (α) and beta-adrenergic (β) receptors. α-adrenergic receptors bind norepinephrine and epinephrine. β-adrenergic receptors link to G-proteins and initiate cAMP second messenger cascades when activated by catecholamine binding. Mediated by adenyl cyclase, β-receptor complexes work to increase the intracellular concentration of cAMP secondary messengers.

Adrenergic drugs stimulate the sympathetic nervous system directly by binding to an adrenergic receptor, or indirectly by stimulating norepinephrine release. These drugs are commonly used as therapy against bronchial asthma, cardiac arrest, and allergic reactions, but also hold great pharmacologic potential as appetite suppressants and obesity drugs. If one considers that weight control is the delicate balance between central and peripheral factors, it is easy to see why adrenergic receptors are a current, exciting pharmacologic target for obesity. While satiety and appetite are regulated by the central nervous system, energy mobilization is a function of the peripheral nervous system. The relatively new discovery of adrenergic receptors that control food cravings and energy expenditure suggests that obesity can be targeted both centrally and peripherally by the mediation of these receptors.

The most commonly investigated adrenergic receptors for obesity include the beta-3-adrenergic receptor and the 5-HT2C receptors. The β-3-adrenergic receptor, located in adipose tissue, regulates lipolysis and thermogenesis. It is now thought that this receptor plays a crucial role in weight control in humans, and a genetic mutation in the receptor has been identified to cause morbid obesity and type 2 diabetes. This genetic receptor mutation is associated with the hereditary obesity seen in the Pima Indians of Arizona. The stimulation of 5-HT2C receptors has been shown to decrease food consumption, and is the target of the popular prescription appetite suppressant sibutramine (Meridia®). Meridia stimulates 5-HT2C

receptors by blocking the reuptake of norepinephrine and serotonin, the major ligands for this adrenergic receptor.

SEE ALSO: Drug Targets that Decrease Food Intake/Appetite; Pima Indians; Sibutramine (Meridia), Type 2 Diabetes.

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Advertising

OVER THE PAST decade, political momentum has grown for strong action to tackle childhood obesity and there is an emerging consensus that regulation of food advertising to children is both necessary and achievable. Creating an environment in which children in the United States grow up healthy should be a priority for the nation. Yet the prevailing pattern of food and beverage marketing to children in America represents, at best a missed opportunity, and at worst, a direct threat to the health prospects of the next generation. Children's and their families' dietary and related health patterns are shaped by factors that include their biological affinities, culture and values, economic status, physical and social environments, and their commercial media environments.

Among these environments, none have more rapidly assumed central socializing roles among children and youth than the media. With the growth in the variety and the penetration of the media has come a parallel growth with their use for marketing, including the marketing of food and beverage products. Although advertising may contribute to dietary changes in America, the evidence does not support the notion that it is one of the major contributors to obesity.

ADVERTISING AND MARKETING

It has been estimated that the average child currently views more than 40,000 commercials on television each year, a sharp increase from 20,000 in the 1970s. Furthermore, an accumulated body of research reveals that more than 50 percent of television advertisements directed at children promote foods and beverages such as candy, convenience foods, snack foods, sugar-sweetened beverages and sweetened breakfast cereals that are high in calories and fat and low in fiber and nutrient value. Recent statistics on food advertising to children indicate that annual sales of foods and beverages to young consumers exceeded $27 billion in 2002. The food and beverage advertisers collectively spend $10 to $12 billion annually to reach children and youth. More than $1 billion is spent on media advertising to children (primarily on television), more than $4.5 billion is spent on youth-targeted public relations, and $3 billion is spent on packaging designed for children. In addition, fast food outlets spend $3 billion in television ads targeted to children. At its peak, the main U.S. government nutrition education program ("$5 a Day") was given $3 million for promotion. The food industry spends one hundred times that to advertise fast foods to children.

A recent literature review by Kaiser Family Foundation highlighted a number of studies that suggested that advertising influenced dietary and other food choices in children, which likely contributed to energy imbalance and weight gain. One study found that among children as young as three, the amount of weekly television viewing was significantly related to their caloric intake as well as requests and parental purchases of specific foods they saw advertised on television. Several studies found a relationship between the amount of time children spent watching TV and how often they requested specific products at the grocery store.

The World Health Organization implies that marketing affects food choices and influences dietary habits with subsequent implications for weight gain and obesity. While the American Psychological Association in 2004 made a statement that advertising to children is unfair because of their limited comprehension of the nature and purpose of television advertising and warrants government action to protect young children. Lastly the American Academy of Pediatrics implied that advertising and promotion of en-

ergy dense, nutrient poor food products to children may need to be regulated or curtailed.

REVIEW OF THE EVIDENCE ON ADVERTISING AND OBESITY

A 2003 review of the available literature on advertising and obesity analyzed the relationship between advertising to children and obesity. Following the review of more than 30,000 articles, 120 were determined to be most relevant. Based on these, the study concluded that advertising to children has an adverse effect on food preferences, purchasing behavior, and consumption. However, these findings must be weighed against the fact that the strongest and most cited study in the review does not fully support this notion. This study involves a sample of 262 children selected from white families in Ohio, mainly of high socioeconomic status, and conducted more than 25 years ago. Despite these limitations, much was made of the fact that this study revealed a statistically significant relation between a child's exposure to advertising and the number of snacks eaten. However, even as food-commercial exposure did reduce children's nutrient efficiency, only two percent of the variance was explained by this finding and had no direct effect on calorific intake. The influence of parental behavior was 15 times greater than that of television advertising, and subsequent studies have confirmed that this is the dominant influence on children's eating habits. A second study looked at the influence of advertising relative to other factors and once again the influence of food advertising was small.

Taken together, these and other observations effectively undermine the main conclusions of the literature review. Despite media claims to the contrary, there is no absolute evidence that advertising has a substantial influence on children's food consumption and, consequently, no reason to believe that a complete ban on advertising would have any useful impact on childhood obesity rates.

This conclusion is also supported by evidence from Quebec, where despite food advertising to children being banned since 1980, childhood obesity rates are no different from those in other Canadian provinces. In Sweden, a similar advertising ban has existed for over a decade, but again this has not translated into reduced obesity rates. A study in the United Kingdom evaluated 42 elementary-school aged children and found that lean, overweight, and obese children who watched television programs with snack food advertising were more likely to choose high fat savory food options than lower fat sweet options. This group also consumed a greater volume of food than their similar weight peers in a non-advertisement control group. This study also established that weight status modified the ability to recall advertised products among a list of similar products (where children that are more obese displayed greater recall). These authors suggested that these results support the notion that exposure to food advertising on television can affect eating behavior, stimulating energy intake from a range of advertised foods and exaggerating unhealthy choices in foods.

PORTION SIZE

Increasing portion size is one of the many factors that are likely to contribute to the current obesity crisis, particularly the increase in beverage portions. Researchers have concluded that when the calories from a caloric beverage is added to energy from food, total energy from intake at lunch was increased significantly ($p<.05$) compared with non-caloric beverages. The consumption of sugar-sweetened drinks is associated with increased total caloric intake for many individuals, especially children and adolescents and contributes to obesity in children.

The concept of more product for less money is not inherently bad, but in the case of food, this strategy is applied disproportionately to unhealthy choices. Supersizing at fast food restaurants is well known, but the concept is used in other settings as well (e.g., movie theaters urge customers to buy large containers of popcorn and soft drinks). When one buys broccoli, oranges, or whole grains, the unit price does not decrease as one purchases more. A 2001 study examined the pricing and promotion of low fat snacks from vending machines. Low fat snacks in school vending machines were priced at four levels (equal price, 10 percent, 25 percent and 50 percent reduction) as compared to regular snacks. Results of this study showed that price reduction of low fat snacks increased sales significantly and average profit per vending machine did not change. Lowering the prices of low fat vending snacks had a strong effect on sales of low fat snacks from vending machines at diverse worksites and secondary schools.

Consumer research shows that people believe that larger packages are a better value, but also use more of a product when it comes in a larger package. This has been shown with eating behavior (i.e., people eat more when given larger portions) and use of non-food products such as laundry detergent (i.e., people use more detergent per wash when it comes from a larger container).

SUMMARY

Although advertising and the media may contribute to obesity, they are not the only ones at fault. The changing home environment such as single-family households, food insecurities, and lack of supermarkets in low socioeconomic areas are major contributors to obesity. In 1970, about 25 percent of total food spending occurred in restaurants. By 1995, 40 percent of food dollars were spent away from home. Americans' spending on fast food increased from $6 billion to $110 billion over the last 30 years. On average, children ages 11–18 eat at fast food restaurants twice a week. The percentage of food consumed by children in restaurants and fast-food outlets nearly tripled between 1977 (6.5 percent) and 1996 (19.3 percent). A study in New Orleans found a higher proportion of fast-food restaurants in low income and African-American neighborhoods.

The decline in physical activity in children (and adults) has been exacerbated by the failure of the government to provide an environment in which physical activity can be incorporated into everyday life. Parents who have concerned about traffic or safety will probably opt for the car and not the pavement. The role of schools in promoting an active lifestyle needs more emphasis and funding. Many schools have insufficient resources to purchase basic items of sports equipment. Emphasis on competition and sporting performance in schools alienates those children who are less physically gifted and diminishes the importance of regular physical activity in relation to health. The National Curriculum should include a lifestyle module in which children learn about the health benefits of physical activity for life, not simply competitive sport during their school years. Health policy makers and those who control the public purse should also bear in mind that active children are more likely to become physically active adults with lower rates of heart disease, diabetes and cancer. Even those who are obese but physically fit can expect better long-term health outcomes than their sedentary, lean but unfit counterparts.

FUTURE RESEARCH

Obesity, poor diet and inactivity are severe global problems, a consequence of the current environment. To make progress the environment must change. This will require close attention to both food and physical activity environments, and, support of inventiveness at the community level, increased funding, and perseverance despite deeply rooted systemic problems.

For every complex problem, there is a simple solution and it is always wrong. The claim that food advertising is a major contributor to children's food choices and the rising tide of childhood obesity has obvious appeal, but as an argument, it does not stand up to scrutiny. In order to establish a causal relationship between food advertising and childhood obesity, many questions need to be answered using longitudinal studies designed with a sufficient statistical power.

SEE ALSO: Children's Television Programming; Computers and the Media; Fast Food; Food Marketing to Children; Supersizing; Television.

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Africa, North

THERE ARE REFERENCES to gluttony in several surviving papyri from Ancient Egypt, and medical historians who are interested in Egyptology have tried to work out diagnoses for the deaths of the various Pharaohs of the XVIIIth dynasty. This has allowed them to dismiss the idea that the Pharaoh Akhenaton (d. 1334/1336 BCE) suffered from Florisch's syndrome, which is usually accompanied by becoming overweight, although the Queen of Punt (15th century B.C.E.), shown on a relief in the temple of Hatshepsut at Deir el-Bahari, was clearly suffering from obesity. The Egyptian king, Ptolemy VIII "Physcon" (182–116 B.C.E.), became well known for his obesity.

With a much larger wealthy urban elite than the other countries in North Africa, Egypt has had more of a problem with obesity than the rest of the area. In modern times, several important Egyptians have certainly suffered from obesity, perhaps the most well known being the famous Egyptian general Ibrahim Pasha (1789–1848) and the last king of Egypt, Farouk I (1920–65) who was deposed in 1952. In exile, Farouk became steadily more obese and weighed 300 pounds (136 kg) when he died on March 3, 1965, in Rome, Italy, after collapsing while eating an enormous meal.

During the second half of the 20th century, starting with the land reclamation law of 1953, there has been a major change in the lifestyle of most Egyptians, and this has been combined with a drastic alteration in the diet, with much food being imported, and many new foods introduced as agricultural production has not kept up with the increase in the country's population. The consumption of dairy products, red meat, and sugar has increased, along with processed cheese. The resulting higher level of obesity in Egypt has led to a rise in the prevalence of certain types of diabetes and also coronary heart disease. To try to combat the problem,

A mosque reflects traditional Muslim values and lifestyle in Africa. Major lifestyle changes in significant areas of Africa have brought with them equally significant changes in the indigenous peoples. Of these changes, in developed nations, obesity is an increasing problem.

there have been a number of surveys to ascertain the level of obesity in Egypt, the best publicized one being the Egyptian Integrated Household Survey conducted in 1997. This was critical of the Egyptian food subsidy program, which served to reduce the price of nutrient-poor but energy-dense food, which has caused both obesity and also an increasing prevalence of micronutrient-deficient mothers in the country. Because of the problem in Egypt, there has been the growth of obesity surgery. Dr. Khaled Gawdat, professor of general surgery at the Ain Shams School of Medicine, has been involved in morbid obesity surgery since 1996.

In Morocco, there have been problems with obesity in urban areas, especially among the middle-class elite. The researchers Drs. M. Rguibi and R. Belahsen, from the Training and Research Unit on Food Science, Laboratory of Physiology Applied to Nutrition and Feeding, School of Sciences, Chouaib Doukkali University, El Jadida, have conducted a number of studies on obesity in Morocco and found that in 1984/1985 only 4.1 percent of the adult population were obese, but that had risen to 10.3 percent in 1998/1999. A survey in 2000 showed that 13.3 percent of all people aged 20 years or older were obese—the rates being 22 percent among women and 8 percent among men. They were also able to show that obesity was more prevalent in urban areas; the likelihood of being obese increases with age, and is inversely proportional to the level of schooling achieved. A later survey Rguibi and Belahsen in 2006 showed that the causes of obesity included social problems. Their studies of 249 Saharawi women showed 90.4 percent of the women wanting to gain weight, which they did through overeating, or in some cases, by steroid use. A 2005 study of Moroccans living in the Netherlands showed a higher level of obesity in Moroccan children than their native Dutch counterparts.

Many of the same trends from studies in Morocco and Egypt are evident in Algeria, with much higher levels of obesity among the middle-class urban elite. The causes seem largely to be higher energy consumption, combined with a sedentary lifestyle and a lack of exercise. Some researchers have also believed that the Mediterranean diet, which is heavier in cholesterol and saturated fats, has contributed, in a major way, to obesity in Algeria and elsewhere.

In Tunisia, with a larger level of urban population to Morocco and Algeria, studies have been conducted on the level of obesity in the country under the auspices of the National Health and Nutrition Examination Survey (NHANES). They studied boys and girls between the ages of 13 and 17 at two high schools, and found that the highest level of obesity—the overall being 5.1 percent—was in youth aged 13–14. This was more statistically common for boys from wealthy families; the rates from both schools were similar. They also studied parents showing that 51 percent of obese adolescents had obese parents, indicating the importance of family history. They found that the causes were excessive caloric intake in 52 percent of cases and excessive fat intake in 82 percent of cases. Of all the countries in North Africa, Libya has the lowest level of obesity, and consequently better health indicators in other areas, such as the prevalence of diabetes mellitus, as shown by studies conducted at the Diabetes Hospital in Tripoli.

SEE ALSO: Africa, Sub-Saharan; World Patterns.

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Africa, Sub-Saharan

IN PARTS OF sub-Saharan Africa, there has been a tradition of some rulers being overweight or obese, a tradition that has continued through to the modern era. As well as this, the increasing affluence of some areas has led to a rise in the prevalence of obesity among the middle class, particularly westernized families. From the late 1980s, but especially during the 1990s and the 2000s, the impact of human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS) has been devastating, and AIDS is often called "slim" in Africa because of the wasting effect it has on its victims. This has led some people to think that those who are overweight or obese are less likely to be suffering from AIDS, although this, obviously, is not the case.

In former French West Africa, especially the northern region, obesity has not been a major problem until recently. A 1975 study by Dutch researchers R. De Hertogh, I. V. Vanderheyden, and M. de Gasparo of the glucose tolerance in the Toubou Broayas of northeastern Niger found that obesity was unknown in their community because of diet and physical activity. Although obesity remains relatively rare in Mauritania, it has been found that a very significant number of patients involved in treatment for cardiology problems have been suffering from obesity. In a study conducted in 2000 by M. L. Ba of the Centre Hospitalier National, Nouakchott, Mauritania, he found that just over a third of patients involved in cardiology treatment were obese. Of the 227, 187 were women and 40 were men, illustrating a much higher level of obesity among women. There have been a number of studies in Burkina Faso, which aimed to confirm the study by Dr. Ba in Mauritania, linking obesity to cardio-vascular problems. A study in 2001 by D. Ye, Y. J. Drabo, D. Ouedraogo, A. Samandoulougou and A. Sawadogo in Ouagadougou, the capital of Burkina Faso, showed the extremely low levels of obesity. Of the 1,470 students from primary schools and secondary schools, there were only 28 overweight cases, and only 4 of obesity—one of these four students having high blood pressure.

In Senegal, with a much higher urban population, there is an increased prevalence of obesity. These people have been shown to have a much greater likelihood of having problems from heart disease and asthma. In Guinea, a study by N. M. Balde, et al. of the Endocrinology Service, into tuberculosis and diabetes in Conakry, the capital of the country, the condition was closely associated with obesity. The much higher levels of the urban population in the Côte d'Ivoire, and the affluence of the population in Abidjan, the capital, has seen an increase in obesity in recent years among the urban middle class, as has also happened in Lome, the capital of Togo, and Cotonou, the capital of Benin.

In the former British colonies of West Africa, obesity has also been a problem in some areas. In Gambia, where there has been much tourism and increased urbanization, a study in 2006 by M. Siervo, et al. of the MRC Human Nutrition Research, Elsie Widdowson Laboratory, Cambridge, United Kingdom, has shown the Gambian population to be far more accepting of

obesity, perhaps influenced by the increasing levels of obesity in their society, than Americans. A 1998 study in Sierra Leone has shown that the Mende tribe—which makes up nearly a third of the country's population—has an increased prevalence of obesity—the war and widespread food shortages in Sierra Leone resulting in a decline in obesity levels in recent years, even though the recent civil war has ended.

There have been a number of studies of obesity in Ghana; that in 2005 by F. Ofei of the Endocrine and Metabolic Clinic, Department of Medicine and Therapeutics, University of Ghana Medical School, Accra, published in the *Ghana Medical Journal,* is significant. He highlighted obesity as a major and preventable condition. Dr. Ofei outlined a range of strategies largely around increased education of the population, as many people, through greater affluence, went through "an economic and nutrition transition."

Nigeria, traditionally the wealthiest country in West Africa—and the one with the largest and richest urban elite—continues to have a major problem with obesity. As with all countries, it is difficult to survey the general public, so E. N. Obikili and L. O. Nwoye of the Department of Anatomy, College of Medicine University of Nigeria, Enugu Campus, conducted a survey of a cross-sectional study of students at the University of Nigeria Enugu Campus. Rather than collecting data, they were aiming to work out an accurate method of figuring out levels of obesity in the country, and concluded that the body mass index (W/H2) was the most suitable index to employ. However, the level of obesity, however measured, has certainly been particularly dramatic among some women. A study by N. P. Edomwonyi and P. E. Osaigbovo of the Department of Anaesthesia, University of Benin Teaching Hospital, Benin City, Edo State, into women scheduled for cesarean section, showed that 50.7 percent were obese.

In Liberia, owing to the poverty of the country and the incessant civil strife since 1979, the level of food available has been low, and consequently, the country has not faced a major problem of obesity. Similarly, in Guinea-Bissau, Cape Verde Islands, and São Tomé e Principe, all former Portuguese colonies, there has not been a problem of obesity; neither has there been a problem in the former Spanish colony of what is now Equatorial Guinea.

In former French Central Africa, there are areas of recent affluence, and this reveals itself in the increasing problems of obesity in Cameroon, Gabon, and Congo. A study in 2007 in Cameroon, by researchers from the Department of Community Health and Psychiatry, University of the West Indies, Mona Campus, Kingston, Jamaica, has shown that 36.3 percent of urban men are overweight, with 47.1 percent of women from urban areas—compared with 9.5 percent of women and fewer men from rural parts of the country. The seriousness of the problem in the region is shown in a study for Gabon, of the 108 patients suffering from diabetes who sought treatment at the Internal Medicine Department of the Libreville Hospital Center, 64 of them were suffering from being overweight or obese. In the remainder of former French Central Africa, food shortages has resulted in malnutrition in some parts of Chad and to a lesser extent the Central African Republic, with obesity not being a problem. This was also the case in the Sudan, Ethiopia, and Somalia, as well as Djibouti. However, the 19th-century explorer Sir Samuel Baker in *The Albert N'Yanza: Great Basin of the Nile* (1866) refers to a custom by which wives of the King of Kisoona were forced to consume large quantities of milk—up to a gallon daily—thereby becoming obese, or face a whipping. It is a custom that has long fallen out of practice.

In East Africa, in Uganda, there has not been a major problem with obesity, although Idi Amin, who ruled the country from 1971 until he was overthrown in 1979, became noticeably overweight. In Kenya, the prosperity in the country and the much larger middle class has seen a steady increase in obesity and its related problems such as diabetes. The affluence in parts of neighboring Tanzania has resulted in obesity there. As a result, there have been a number of studies, the most detailed probably being that by C. N. Nyaruhucha, et al. of the Department of Food Science and Technology, Sokoine University of Agriculture, Morogoro, into the level of obesity of students of different ages in schools and other educational institutions in the city of Morogoro, in the southern highlands of Tanzania, west of Dar-es-Salaam. It shows that as much as 70 percent of younger children were not aware of the medical problems with obesity, and married adults had an obesity rate of 22.2 percent compared to 4.7 percent for unmarried adults.

The Democratic Republic of the Congo, formerly Zaire, there have been a level of obesity in the govern-

ment circles with Mobutu Sese Seko (1930–97), president from 1965 until 1997 was slim when he came to power but became steadily overweight during his long period of rule, and Laurent Kabila (1939–2001), who seized power from him in 1997 being significantly overweight. Jonas Savimbi (1934–2002), the leader of the rebel UNITA movement in Angola was also overweight in his latter years.

In former British southern Africa, the affluence of much of the region and larger urban areas has led to greater levels of obesity, with the Zimbabwean nationalist leader Joshua Nkomo (1917–99) being the most well-known African leader to suffer from obesity, as did King Sobhuza II of Swaziland who reigned from 1899 until 1982. President Paul Kruger (1825–1904) of the Transvaal was overweight but not obese, although some British cartoons during the Boer War exaggerated his size.

A 2001 study by A. R. Walker, F. Adam, and B. F. Walker of the Human Biochemistry Research Unit, Department of Tropical Diseases, University of the Witwatersrand, South Africa, showed the general level of obesity in rural South Africa, Botswana, Namibia, and Zimbabwe remaining very low—between one and five percent. However, in urban areas, it became much higher, reaching eight percent in Cape Town for men and 34 percent for women. In South Africa, people suffering from obesity had a much greater likelihood of facing problems from arthritis, diabetes, and heart disease. This has led to an increased awareness campaign run by the Southern African Society for the Study of Obesity which has Professor Tessa van der Merwe of Netcare Bariatric Centres of Excellence, Unitas Hospital, Lyttleton, Pretoria, being the national representative on the International Association for the Study of Obesity.

SEE ALSO: Africa, North; World Patterns.

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African Americans

AFRICAN AMERICANS ARE Americans whose origins may be traced to any of the black racial groups of Africa; it, therefore, includes recent émigrés from Africa and the Caribbean as well as African Americans who have lived in this country for generations. According to the U.S. Census, in 2000, there were 36.4 million African Americans in the United States, making up 12.9 percent of the total population. In 2003, the Census Bureau announced that the Hispanic/Latino group had surpassed the African-American group in size, making African Americans the third largest racial group in the United States. New York has the greatest number of African Americans, followed by California and Texas; about half of African Americans live in southern states.

Although the health status of African Americans has improved in recent years, they still experience health disparities, particularly in relation to Caucasian Americans. African Americans suffer disproportionately from many diseases and have among the highest death rates and lowest life expectancies of any U.S. ethnic group. African Americans as a group also score lower on many socioeconomic indicators related to health, including income, education, employment, and access to healthcare.

OVERWEIGHT AND OBESITY

Overweight and obesity have been increasing for some years in the U.S. population as a whole, but rates are higher among most minority groups, with the exception of Asian Americans, and African Americans have experienced some of the highest increases.

This is a matter of both medical and public health concern because overweight and obesity in adults are associated with lower levels of health and higher risk of many diseases, including heart disease, stroke, diabetes, and some types of cancer, and are also associated with increased severity of other diseases including hypertension and arthritis. In children and adolescents, overweight and obesity increase the risk of becoming an overweight adult, and also increase the risk of diabetes, high cholesterol, and liver abnormalities.

For U.S. adults aged 20–74, the age-adjusted rate of overweight (defined as body mass index, BMI ≥25) has increased steadily since 1960. According to data from the National Health Interview Survey (NHIS) and National Health and Nutrition Examination Survey (NHANES), in 1960–62, 44.8 percent of adults were overweight.

This increased in 1971–74 to 47.7 percent, in 1976–80 to 47.4 percent, in 1988–94 to 56.0 percent, and in 2001–04 to 66.0 percent. In the non-Hispanic Caucasian population, men are more likely to be overweight than women; this pattern is reversed in the African-American population, where women are more likely than men to be overweight. In 1976–80, 53.8 percent of Caucasian, non-Hispanic men and 38.7 percent of Caucasian, non-Hispanic women were overweight; while for African Americans, the numbers were 51.3 and 62.6 percent, respectively. In 1988–94, 61.6 percent of Caucasian men and 47.2 percent of Caucasian women were overweight compared to 58.2 percent of African-American men and 68.5 percent of African-American women. In 2001–04, 71.1 percent of Caucasian men and 57.1 percent of Caucasian women were overweight compared to 66.8 percent of African-American men and 79.5 percent of African-American women.

Rates of obesity (defined as BMI ≥30) have also increased steadily in the U.S. population since 1960. Note that in the sources for these statistics, obesity is treated as a subcategory within overweight, so the

two categories should not be added together. In 1960–62, 13.3 percent of the U.S. population was obese; in 1971–74, this increased to 14.6 percent, in 1976–80 to 15.1 percent, in 1988–1994 to 23.3 percent, and in 2001–03 to 32.1 percent. The rate of obesity has therefore increased more rapidly in the United States since 1960 than the rate of overweight. In distinction to overweight, women are more likely to be obese than men in both the Caucasian and African-American racial categories.

In 1976–80, 12.4 percent of Caucasian men and 15.4 percent of Caucasian women were obese, as compared with 16.5 percent of African-American men and 31.0 percent of African-American women. In 1988–94, 20.7 percent of Caucasian men and 23.3 percent of Caucasian women were obese, compared with 21.3 percent of African-American men and 39.1 percent of African-American women, and in 2001–04, 31.2 percent of African-American men and 51.6 percent of African-American women were obese.

DISEASES RELATED TO BEING OVERWEIGHT AND OBESITY

Overweight and obesity are matters of medical and public health concern because they are risk factors for several serious diseases, and are implicated in both years of life lost and years of healthy life lost due to disease and disability. The fact that African Americans are disproportionately overweight and obese is logically related to the fact that they suffer disproportionately from a number of serious diseases for which overweight is a major risk factor, including diabetes, hypertension, myocardial infarction, and stroke.

Type 2 diabetes, which accounts for over 90 percent of all cases of diabetes, is increasing rapidly throughout the world as well as within the United States. Type 2 diabetes used to be thought of as a disease of older adults, but is occurring more often in children and adolescents as well. Obesity and physical inactivity are major risk factors for Type 2 diabetes, and increased levels of both risk factors are implicated in both the overall increase and the increase among children and adolescents. The Behavior Risk Factor Surveillance System (BRFSS), an annual, nationally representative random-digit-dial health survey, found that the prevalence of diabetes (primarily Type 2 diabetes) increased 60 percent between 1990 and 2001, and the greatest increases were among members of minority groups. The relative rate of increase, compared to Caucasian Americans, was 1.5 times as great among African Americans and 2.5 times as great among Hispanic Americans. Several factors have been hypothesized to account for the greater increase among African Americans and other minorities, including genetic factors (including the thrifty gene hypothesis), environmental triggers such as changes in lifestyle, and substandard medical care.

Hypertension is a common disease worldwide as well as in the United States. Among American adults, approximately 29 percent are hypertensive. African Americans have a higher rate of hypertension after puberty and this difference persists into adult life; 33.5 percent of African Americans are hypertensive compared to 28.9 percent of non-Hispanic Caucasians. The racial difference increases with age; in the age group 40–59 years, 50 percent of African Americans are hypertensive compared to 30 percent of non-Hispanic Caucasians. Hypertension is also more severe among African Americans and is a greater risk factor for morbidity, kidney disease, and heart disease. Among African Americans, as compared to the general population, hypertension is associated with an 80 percent higher stroke mortality rate, a 50 percent higher heart disease mortality rate, and a 32 percent higher rate of end-stage renal disease (ESRD). Various explanations have been offered for these differences, including substandard medical care for African Americans, increased sodium sensitivity (possibly related to survival during the passage to America on slave ships), and suppressed anger due to the experience of racial discrimination.

Myocardial infarction (MI), commonly referred to as a "heart attack," is the leading cause of death for both Caucasian and African Americans. However, while the age-adjusted death rate for MI decreased 20 percent in the United States from 1987 to 1995, for African Americans the decrease was only 13 percent. Caucasian Americans have an age-adjusted death rate from MI of 253.6 per 100,000, while for African Americans the rate is 326.5 per 100,000, the highest death rate from heart disease for any racial or ethnic group in an industrialized country.

Stroke is the third leading cause of death as well as a leading cause of disability in the United States. African Americans, both men and women, have a higher age-adjusted prevalence of stroke than do Caucasian Americans, and a higher rate of mortality from stroke in every age category. The types of

stroke most common also varies by race; African Americans have higher rates of cerebral infarction, subarachnoid hemorrhage, and intracerebral hemorrhage, and among ischemic strokes, have disproportionately high rates of lacunar infarcts and large artery intracranial occlusive disease.

FACTORS RELATED TO BEING OVERWEIGHT AND OBESITY

Many hypotheses have been offered for the disproportionate burden of overweight and obesity in the African-American community. One is the differing standards of beauty or desirable body type among African Americans, who have demonstrated in studies to prefer a larger body type relative to non-Hispanic Caucasians and to have a greater satisfaction with their own bodies. Another explanation is the well-demonstrated inverse relationship between socioeconomic status (SES) and overweight: because most African Americans have lower SES than non-Hispanic Caucasian Americans, it is predictable that they also have higher levels of overweight and obesity. This relationship is often attributed to lack of access to healthy foods, lack of nutritional and health-related knowledge, and lack of inclination or opportunity to exercise. As mentioned above, African Americans on average have lower rates of physical activity, and consume fewer servings of fruits and vegetables than non-Hispanic Caucasian Americans; both behaviors are associated with higher rates of obesity.

The Centers for Disease Control and Prevention (CDC) recommends regular physical activity (at least 30 minutes of moderate activity five times per week, or 20 minutes of vigorous activity three times per week) to improve physical functioning, aid in weight control, and reduce risks for many diseases including heart disease, diabetes, colon cancer, hypertension, and osteoporosis. Exercise is also associated with improved mental health and lessening of arthritic symptoms. However, most American adults do not meet these recommendations, and African Americans rank among the lowest among U.S. ethnic groups in regular leisure-time physical activity. In 1998, according to the National Health Interview Survey (NHIS), only 22.6 percent of African Americans reported regular leisure time physical activity compared to 30.7 percent of Caucasian Americans, 31.8 percent of Asian Americans, 21.1 percent of Hispanic Americans, and 31.8 percent of American Indians and Alaska Natives (AI/AN).

In 2004, the results were similar except for reduced leisure activity in the AI/AN population: 23.3 percent of African Americans, 31.3 percent of Caucasian Americans, 21.9 percent of AI/AN Americans, 22.3 percent of Hispanic Americans, and 27.5 percent of Asian Americans reported regular leisure-time physical activity. Lower rates of physical activity were also related to lower levels of income and education, a fact which is confounded with the racial/ethnic results, since the groups with lower activity levels (African Americans and AI/AN Americans), tend to also have higher levels of poverty.

A diet high in fruits and vegetables is associated with decreased risk for many chronic diseases, and is also recommended for weight management. Current goals from Healthy People 2010 include increasing the number of adults to consume at least two servings of fruits and three servings of vegetables daily. However, data from the 2005 BRFSS, disclosed that only 32.6 percent of American adults met the first recommendation, and only 27.2 percent met the second. African Americans were intermediate among U.S. ethnic groups in meeting both recommendations. About 35 percent of African Americans reported eating at least two servings of fruit per day compared to 37.2 percent of Hispanics, 31.2 percent of Caucasians, and 35.5 percent of those classified into other racial categories. Almost 24 percent of African Americans reported eating three or more servings of vegetables per day compared with 20.4 percent of Hispanics, 28.6 percent of non-Hispanic Caucasians, and 29.3 percent of those classified into other racial categories. Looking at all racial/ethnic groups together, higher levels of fruit and vegetable consumption were negatively related with overweight and obesity: 36.0 percent of people with normal body weight met the recommendation for fruit consumption and 28.9 percent met the recommendation for vegetable consumption compared to 32.0 percent for fruits and 26.0 percent for vegetables for overweight Americans; only 28.1 percent of obese Americans met the fruits recommendation and 26.3 percent the vegetables recommendation.

In addition to differences in dietary patterns, biological and genetic differences between African-Americans and non-Hispanic whites may explain some of these variations in prevalence for obesity and obesity-related comorbidities.

CHILDHOOD OBESITY

The increase in childhood obesity in the United States has been so extreme and sudden that some researchers refer to it as an epidemic. According to NHANES III, a nationally representative survey conducted 1988–94, 14 percent of U.S. children are overweight. Rates of overweight are higher in minority ethnic groups, for instance, 21.5 percent in African-American children versus 12.3 percent of Caucasian children. Within ethnic minority groups, girls were more likely to be overweight than boys, and children living in impoverished communities were more likely to be overweight than those living in more prosperous communities. These results are particularly worrying because persistent overweight beginning in childhood is associated with greater morbidity and mortality than adult-onset overweight.

Many explanations have been offered for this increase in childhood obesity, which is not limited to the United States. Genetic factors are one possibility; even though the increase in overweight and obesity has occurred too quickly to be accounted for by genetic change, it is possible that genetic predisposition to overweight, as suggested for instance by the "thrifty genotype" theory of Neel and others, may interact with the modern environment to produce high levels of obesity. Increased television watching, which results both in exposure to advertising of unhealthy foods and to physical inactivity, is another possibility. Studies, including NHANES III, have shown a correlation between hours of television viewed and BMI. Studies have also found that African-American children watch more television than their Caucasian peers, and that prime-time television programs aimed for African-American audiences included more overweight characters and carried more advertising for high-fat products.

Changes in food consumption and exercise patterns are also logical explanations for the increase in weight gain. There has been a trend over the past decade in increasing consumption of fat, sodium, cholesterol, sweet beverages, and grains, and a decline in lean meat, milk, raw fruits, and eggs by children and adolescents. Minority children show greater increases in fat and calorie consumption, and disparities among racial groups are evident as early as 6–9 years of age in girls and 10–13 years in boys. There has been an overall decline in participation of physical activity among children and adolescents, and minority children are also less likely to participate in physical activity than their Caucasian peers. A study by the Centers for Disease Control and Prevention (CDC) found physical activity levels highest (73.0 percent) among Caucasian boys and lowest (41.8 percent) among African-American girls.

SEE ALSO: Africa, Sub-Saharan; Body Image; Cardiovascular Disease in African Americans; Office of Minority Health; Sisters Together; Stress; Stroke; Type 2 Diabetes.

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Agouti and Agouti-Related Protein

THE COMMON TERM *agouti* refers to several rodent species of the genus *asyprocta* that look similar to guinea pigs and inhabit areas of Central America, the West Indies, and northern South America. The gene (called the agouti gene) that is responsible for the

specific hair-color in these rodents (dark hairs that are lighter at the tip) also makes these animals fat. A similar gene is also found in various other mammals including humans. Its human homologue (version) is located on chromosome 20 and encodes for the agouti-related protein, consists of 132-amino-acids and is 81 percent identical to the one found in the agouti. The agouti-related protein (or agouti-related peptide, AgRP) is produced by a collection of neurons called the arcuate nucleus in the hypothalamus region of the brain.

In 1997, two independent sets of researchers discovered AgRP and showed that it stimulates appetite and is a putative cause of obesity. Understanding the role AgRP plays in weight gain holds the promise of enabling researchers to probe the metabolic processes that lead to obesity and discover therapeutics to treat this health disorder.

AgRP is critical to the mechanism by which the brain controls eating behavior. The arcuate nucleus of the brain has two sets of neurons. One set, the AgRP/NPY (neuropeptide-Y) neurons, produces AgRP. The other set, the POMC neurons, produces proopiomelanocortin (POMC), a precursor to the melanocortin (MC) family of hormones that includes adrenocorticotropin (ACTH) and the alpha-, beta-, and gamma-melanocyte-stimulating hormones (MSH-α, β, and γ). Currently, there are five known receptors of melanocortins, MC1R through MC5R, that are agonized (activated) by them and antagonized (deactivated) by AgRP. In humans, MC1R is expressed in melanocytes; MC2R in the adrenal and adipose (fat) tissue; MC3R in the brain, placenta, and pancreas; and MC4R in the brain, muscle, and adipose tissue. MC5R is expressed ubiquitously in most tissues. Together, the MC receptors control lipogenesis (fat storage), lipolysis (fat burning), and food intake, as well as skin pigmentation, thermogenesis, sexual behavior, memory, and antiinflammatory and antipyretic effects. MC3R and MC4R, found in the brain, are known to control energy expenditure.

The presence of endogenous agonists (melanocortins) and antagonist (AgRP) acting on the same receptor system suggests tight control of energy metabolism by the brain. Central to the brain's control of eating behavior is the protein hormone leptin produced in adipose (fat) tissue. When animals overeat or the body accumulates more fat, more leptin is produced. High levels of circulating leptins in the body decrease AgRP release and increase production of ACTH and MSH-α. In turn, this activates the MC receptors (especially MC3R and MC4R), which results in an increase in metabolism and energy utilization, and a decrease in appetite. Conversely, fasting results in loss of adipose tissue and low leptin levels, which inhibits the activity of POMC neurons, thereby reducing energy usage and increasing appetite. The mechanism via deactivating melanocortin receptors is, however, not the only way in which AgRP is believed to prevent breakdown of fatty tissue. Some recent studies have indicated that AgRP can have similar effects through alternate physiological routes (calcium signaling channels in fat cells) as well.

Because AgRP has been shown to promote food intake and weight gain, it is thought to be a causal factor of obesity in the absence of leptin. It is also known as a hormone that prevents self-starvation. However, variants of the agouti-related protein can lead to a "decreased feeding signal" and is sometimes associated with susceptibility to anorexia nervosa.

SEE ALSO: Melanocortins; POMC Proopiomelanocortins.

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Alcohol

SINCE PREHISTORIC TIMES, people around the world have consumed alcoholic beverages for various reasons. Alcoholic beverages have been used for the standard diet, for health related purposes, for religious purposes, for social and cultural purposes, and for the relaxant effects. The effect of alcohol on the body and mind has been the focus of various research groups. Research is continuing to reveal the contribu-

tion of alcohol toward obesity through calories from the beverages and various effects on adipose tissue, also known as fat. In addition, alcohol and obesity have been independently shown to contribute to similar diseases and health risks, such as cancer, diabetes, hypertension, metabolic syndrome, and fatty liver disease.

Alcoholic beverages contain an active chemical compound called ethanol, which is produced by the metabolism of carbohydrates by yeast during a process called fermentation. Fruits, vegetables, and grains are used for fermentation. The fermented products are then separated by a process called distilling, making wine, sake, beer, or mead.

When alcohol is consumed, about 20 percent is absorbed in the stomach walls and 80 percent is absorbed in the small intestine. Food in the stomach, especially carbohydrates and dietary fats, may reduce the absorption of alcohol by reducing the rate of food emptied into the small intestine from the stomach. The alcohol is then directly transported to the bloodstream, which distributes the alcohol to various tissue systems, such as the brain, liver, heart, pancreas, lungs, and kidneys. The alcohol is diluted by water in the blood and tissues. Eventually, enzymes in the liver detoxify the body.

Alcohol and obesity have been shown to cause similar health problems, including some cancers, hypertension, and diabetes.

Some research studies have shown that moderate intake of alcohol was associated with the lowest body mass index (BMI), which is measured by the weight and height. However, greater amounts of drinking have been associated with higher BMI scores. Higher BMI scores have been associated with various comorbidities, such as cardiovascular disease and diabetes. An individual is considered overweight if their BMI is over 25. An individual is categorized as obese if the BMI is over 30. A study involving 37,000 subjects, conducted by the National Institutes of Health, showed that indivuals who consumed about one drink per day had the lowest BMI. While, heavy drinkers were the most overweight. In another study, BMI increased from the light-moderate to heavy drinkers. Heavy drinkers, who reduced their alcohol consumption, lost greater amounts of weight or gained less weight compared to stable heavy drinkers or new heavy drinkers.

Various mechanisms are being investigated to determine how alcohol induces weight gain. When a food is consumed, the caloric density of the food is a measure of how much energy the food will provide for our bodies. Alcohol contains about seven calories per gram, which is compared to nine calories per gram in fat, four calories per gram in protein, and four calories per gram in carbohydrates. When the consumed calories is greater than the calories used by our bodies via energy requiring physiological requirements for life, such as breathing, or exercise, the calories are stored as fat.

Alcohol also seems to stimulate appetite and reduce self-control, which may lead to an increase in caloric intake. Various research has shown evidence for the ability of alcohol to decrease the amount of energy used by our bodies, which may lead to weight gain. Alcohol is also believed to encourage weight gain by altering hormone levels and other biologically active molecules. Alcohol may interrupt the sleep cycle and cause hormones such as leptin, which is a hormone involved in the control of food intake, energy balance, and body weight, to decrease. Ghrelin, an appetite-stimulating hormone, may also be increased

with sleep deprivation. Research has shown that the release of these biologically active molecules from fat tissue is influenced by alcohol.

Alcohol appears to be a risk factor for cancer in the mouth, esophagus, pharynx, larynx, liver, breast, and ovary. There is a debate whether alcohol is a carcinogen or a cocarcinogen. A carcinogen is a substance that promotes cancer. A cocarcinogen promotes the carcinogenic effects of carcinogens such as tobacco. In comparison, obesity has been associated with cancers of the colon, breast, endometrium, kidney, esophagus, gallbladder, ovaries, and pancreas.

Diabetes is a disease that is characterized by the inability of the body to metaboloize glucose.Various hormones are secreted in our bodies in response to the availability of glucose, which is the sugar that is the main source of energy. Heavy consumption of alcohol impairs the balance of hormones, which impairs the use of glucose, by influencing the hormone producing organs. Alcohol inhibits the biological processes that allow glucose sources to become available for use, so alcohol may cause a person to have a low blood sugar level, especially if insulin or other diabetic drugs are used. Insulin and diabetic drugs helps the body to utilize available glucose in the blood. Alcohol may also damage the pancreas and influence the secrection of insulin, which may lead to diabetes. Obesity has been shown to the resistance of insulin, which leads to an increase of blood sugar. Insulin resistance can lead to diabetes if uncontrolled.

Hypertension is a condition that is characterized by increased blood flow to the arteries. Hypertension is diagnosed by measuring the blood pressure of an individual. Atherosclerosis, stroke, heart attack, and kidney failure are a few complications caused by hypertension. Although the specific mechanism is not well known, alcohol consumption has been linked to an increased risk of high blood pressure. In obesity, hypertension is believed to be induced by an increase of blood to the fat tissue, due to an increased requirement of oxygen and nutrients by the fat tissue. The increased blood flow will increase the blood pressure on the arterial walls.

Alcohol and obesity has been shown to increase the risk of metabolic syndrome. The metabolic syndrome consist of various cardiovascular risk factors including high blood pressure, elevated triglycerides, low levels of high density lipoprotein (HDL), impaired fasting glucose, and increased abdominal fat. An individual is believed to have metabolic syndrome if the individual has three or more of the risk factors. In contrast, studies have shown a protective benefit of alcohol against cardiovascular disease in moderate amounts.

Alcohol and obesity has been shown to increase the risk of fatty liver disease. Obesity induced fatty liver disease is also known as nonalcoholic fatty liver disease. Fatty liver disease has three stages, which include steatosis, steatohepatitis, and cirrhosis. Steatosis is the accumulation of fat in the liver. In steatohepatitis, the accumulation of fat leads to inflammation, necrosis, which is the damage of liver cells, and fibrosis, which is the scarring of the liver. Cirrhosis is characterized by irreversible damage of the liver.

SEE ALSO: Addictive Behaviors; American Obesity Association; Fatty Liver; Food and Drug Administration; National Institutes of Health

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American Academy of Pediatrics

THE MISSION OF the American Academy of Pediatrics (AAP) is to "attain optimal physical, mental, and social health and well-being for all infants, children, adolescents, and young adults." The AAP was founded in 1930 as a not-for-profit organization with the primary purpose of promoting the idea within the medical community that children had special developmental and health needs distinct from adults. The AAP was founded by 35 pediatricians: in 2007, it has a membership of over 60,000 pediatricians and employs a staff of over 350. There are 59 AAP chapters in the United States and Canada and the AAP offices are located in Elk Grove Village, Illinois, and Washington, D.C.

A primary activity of the AAP is furthering the professional education of its members; to this end, it sponsors continuing education courses, scientific meetings, seminars, and publications. *Pediatrics*, a monthly scientific journal, is published by the AAP, as is *Pediatrics in Review*, a continuing education journal; *AAP News*, a membership newsletter; and a series of manuals, patient education brochures, and child care books. The AAP executes original research and promotes the funding of research, and advocates for children's health needs at the federal and state levels. Funding for the AAP comes from membership dues, governmental and private grants, and revenues from publications and continuing education courses.

The Committee on Nutrition of the AAP issued a statement in 2003, which was later reconfirmed, that overweight was epidemic among pediatric populations, and issued a number of recommendations concerning this problem. These include a focus on prevention of overweight, research into genetic and environmental factors, routine monitoring of excessive weight gain in pediatric office visits, education of families in correct child nutrition and appropriate physical activity levels, advocacy in the areas of physical activity and food policy for children, and improvement in insurance coverage for obesity care.

The AAP maintains a website dedicated to providing information about childhood overweight and obesity (www.aap.org/obesity/) which includes links to a number of AAP publications and news releases, including the 2003 Committee on Nutrition statement and a 2004 policy statement from the AAP Committee on School Health discussing soft drinks in schools. Other resources available from the site include information about car safety seats for overweight and obese children, curriculum guides for schools, educational materials for physicians, and information about the We Can! Program, a national program for parents of children aged 8–13 years to help their children maintain a healthy body weight and physical activity level.

SEE ALSO: Bariatric Surgery in Children; Behavioral Treatment of Child Obesity; Eating Disorders in School Children; Medical Interventions for Children.

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American College of Sports Medicine

THE AMERICAN College of Sports Medicine (ACSM) is the largest sports medicine and exercise science organization in the world, with over 20,000 members. These members work in a wide range of medical specialties, allied health professions, and scientific disciplines. The ACSM was founded in 1954 as the Federation of Sports Medicine during the annual meeting of the American Association for Health, Physical Education, and Recreation; the name was changed to ACSM in 1955. According to its mission statement, the ACSM "promotes and integrates scientific research, education, and practical applications of sports medicine and exercise science to maintain and enhance physical performance, fitness, health and quality of life."

The National Center of the ACSM has been located in Indianapolis, Indiana, since 1984. The first regional chapter of the ACSM was founded in 1970, and there are currently 12 regional chapters covering the entire United States. Over 10 percent of ACSM members live outside the United States, in over 75 countries, and about 20 percent of attendees at the ACSM annual meeting are non-U.S. citizens.

The ACSM has established standards for health and exercise professionals and offers educational workshops and testing opportunities that allow individuals to qualify for four types of certifications. The ACSM Certified Personal Trainer certification is intended for fitness professionals involved in developing an individualized approach to exercise for individuals with medical clearance to exercise. The ACSM Health/Fitness Instructor certification is designed for people who lead preventive health programs aimed at individuals with controlled diseases such as diabetes and for low-to-moderate-risk individuals. The ACSM Exercise Specialist certification is for professionals who perform exercise testing and train clients with metabolic,

pulmonary and cardiovascular diseases. The ACSM Registered Clinical Exercise Physiologist is for individuals who develop exercise programs for patients with chronic diseases. A searchable directory of ACSM-certified professionals is available from the ACSM website.

Other educational activities of the ACSM include holding an annual scientific meeting and sponsorship of a number of scientific conferences, and continuing education courses, including the Team Physician Course, intended for clinicians who care for members of athletic teams.

The ACSM Foundation, the fund-raising arm of the College, was organized in 1984. The Foundation accepts unrestricted gifts and also maintains a number of targeted funds, including the Carl V. Gisolfi Memorial Research Fund which supports research in thermoregulation, exercise and hydration; the Clinical Sports Medicine Fund; the Education Endowment, the Michael L. Pollock Student Scholarship Fund, which assists students in attending the ACSM annual meeting; the ACSM Minority Scholarship Fund, the Paffenbarger-Blair Endowment for Epidemiological Research on Physical Activity; and the Research Endowment, which annually awards $10,000 to an outstanding research project. The Foundation awards research grants valued at over $100,000 annually to graduate students and young investigators working in sports medicine and exercise science.

SEE ALSO: Exercise; Fitness; Obesity and Sports; Physical Activity and Obesity.

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American Diabetes Association

THE AMERICAN Diabetes Association (ADA), founded in 1940, provides diabetes education, research, and advocacy. The mission of the ADA is "to prevent and cure diabetes and to improve the lives of all people affected by diabetes." The ADA has two types of members: healthcare membership for professionals such as physicians, nurses, and research scientists, and consumer membership for people with diabetes and their families.

The ADA research-funding program complements the diabetes research program of the National Institutes of Health (NIH) by supporting new investigators and new research ideas. In 2005, the ADA provided over $40 million in research funding for peer-reviewed research projects on topics ranging from cell biology and transplantations techniques to education and behavioral issues. Besides the usual professional medical journals, information about ADA-funded research is disseminated through *Forefront*, a journal published twice per year, which contains profiles of researchers and their discoveries, written for a general audience. *Access: Diabetes Research*, also written for a general audience, is published irregularly by the ADA and contains summaries of recent diabetes research from medical journals. Each issue of *Access* focuses on a particular topic, such as blood glucose control or diabetic heart and blood vessel disease. Both *Forefront* and *Access* are accessible through the ADA Web site, as is other information about ADA-funded research and applications for ADA research funding. The ADA Research Database is also accessible through the ADA Web site: the Web site interface allows the user to search for ADA-funded projects by a number of fields, including topic, grant type, researcher, and start and end dates.

The Government Affairs and Advocacy division of the ADA works to improve access to medical care, promote government funding of diabetes education and research programs, and fight discrimination against people with diabetes. ADA Advocacy goals for 2006 included protecting state diabetes insurance programs; fighting the federal Association Health Plan legislation, which would have allowed small businesses to deny health insurance coverage for diabetes supplies; establishing the National Diabetes Prison Coalition to improve healthcare for inmates with diabetes; developing standards of employment for people with diabetes; establishing the Diabetes Legal Advocacy Fund; and protecting/restoring federal funding for diabetes research.

Obesity is a major concern for the ADA, because it is a major risk factors for Type 2 diabetes, a disease which is rapidly increasing in the United States, yet can frequently be prevented, delayed, or controlled through lifestyle changes, including dietary changes and increased physical activity. The ADA website includes information for the general public about risk factors for Type 2 diabetes, information about pre-diabetes (a condition which frequently precedes diagnosis with Type 2 diabetes), and suggestions on how to make changes in dietary and physical activity habits. The ADA also promotes a number of community events to raise funds, increase public awareness of diabetes, and promote activities known to delay and control Type 2 diabetes, including the annual Tour de Cure (a bicycling event) and America's Walk for Diabetes.

SEE ALSO: Nutrition Education; Physical Activity and Obesity; Type 2 Diabetes; Women and Diabetes.

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American Dietetic Association

THE AMERICAN DIETETIC Association (ADA) was founded in 1917 by a group of women, including Lenna F. Cooper and Lulu C. Graves (the first president of ADA), who were dedicated to improving public health and nutrition and aiding the government in conserving food during World War I. Today, the ADA has approximately 65,000 members and is the largest organization of food and nutrition professionals in the United States. About 75 percent of ADA members are registered dietitians; other members include registered dietetic technicians, clinical and community dietetics professionals, educators and researchers, nutritionists, food service managers, and students. The ADA headquarters is located in Chicago, Illinois, and the organization also maintains a Washington, D.C. office. In addition, there are state dietetic associations in all 50 states, the District of Columbia and Puerto Rico, and the American Overseas Dietetic Association.

ADA's mission is to lead the future of dietetics and to help people enjoy healthy lives. They have identified five areas of critical concern: obesity and overweight, particularly in children; healthy aging; ensuring a safe, sustainable, and nutritious food supply; nutrigenetics and nutrigenomics; and integrative medicine, including alternative medicine and nutritional supplements.

The Commission on Accreditation for Dietetics Education (CADE) is the ADA's accrediting agency, which establishes and enforces eligibility requirements and accreditations standards for educational program that train students for careers as registered dietitians (RDs) and dietetics technicians. In order to become an RD, a person must hold a bachelor's degree from an accredited college or university including course work approved by CADE, complete a CADE-accredited supervised practice program which typically lasts 6 to 12 months, and pass a national examination administered by the Commission on Dietetic Registration (CDR). Dietetics technicians, registered (DTRs), must hold at least a two-year associate's degree from an accredited college or university, complete a CADE-approved dietetic technician program including 450 hours of supervised practice, and pass a national examination administered by CADE. Both RDs and CDRs must complete continuing professional education requirements in order to maintain their registration.

The *Journal of the American Dietetic Association* (*JADA*), published monthly by Elsevier, is the official journal of the ADA. *JADA* is a peer-reviewed journal and is the most widely read professional publication in dietetics; its scope includes nutritional science, medical nutrition therapy, public health nutrition, food science and biotechnology, foodservice systems, leadership and management, and dietetics education.

The ADA issued a position paper in 2002, available from the ADA Web site, stating that obesity is epidemic in the United States; that obesity was harmful to health and increasing obesity is a public health concern; that successful weight management requires lifestyle modifications which include healthy eating habits and physical activity; that weight loss interventions should focus on prevention of weight

gain as well as total weight loss; and that weight loss interventions should be evaluated for their success in producing sustained, rather than merely short-term, weight loss.

SEE ALSO: Nutrition and Nutritionists; Nutrition Education; Nutrition Fads; Overall Diet Quality.

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American Heart Association

THE AMERICAN HEART Association (AHA) is a voluntary health agency whose mission is "to reduce disability and death from cardiovascular diseases and stroke." The AHA has its roots in the Association for the Prevention and Relief of Heart Disease in 1915 in New York City, in order to study the progress of heart disease and whether patients could recover and return to work. Heart associations were founded in other American cities, and the need for a national organization became apparent. In 1924, six cardiologists founded the AHA: Lewis Conner, Robert Halsey, Paul White, Joseph Sailer, Robert Preble, and Hugh McCulloch.

The AHA reorganized in 1948 to allow in nonmedical volunteers with skills in education, communication, business management, community organization, and fund-raising. The first national fund-raising campaign was launched in 1949 and raised $2.7 million. In 2005, the AHA's annual revenue was over $697 million, of which about one-third was raised from donations, one-third from fund-raising events, 11 percent from bequests, and the rest from a combination of sources. The AHA's greatest expenditures in 2005 were for public health education (about 40 percent) and research (about 21 percent). The National AHA Center is located in Dallas, Texas; there are also 12 affiliate offices in the United States and Puerto Rico.

Educating the public about heart disease and promoting a healthy lifestyle is a major goal of the AHA. Information about topics including cholesterol, smoking, overweight and obesity, and physical activity and their relationship to heart disease is available from the AHA Web site. The AHA also produces publications for the purpose of promoting a heart-healthy lifestyle, including fact sheets and brochures and cookbooks, and also produces videos and CDs on related topics; many materials are available in both English and Spanish.

The AHA promotes professional education through sponsorship of scientific conferences and continuing education courses for health professionals. Slide sets for use in teaching and professional presentations are available from the AHA Web site. The AHA also funds research related to heart disease in a number of areas, including applied research, basic biomedical research, outcomes research, population health research, and translational research.

The AHA has issued Scientific Position statements on several topics related to obesity, which are available from the AHA Web site. The AHA Scientific Position on *Obesity and Overweight* states that obesity is a major risk factor for coronary heart disease, and explains how obesity is defined using waist circumference measurement and the body mass index (BMI).

Other AHA Scientific Position statements relating to obesity and overweight include statements on *Obesity and Cardiovascular Disease* (2006), *Overweight in Children and Adolescents* (2005), *Clinical Implications of Obesity* (2004), *Cardiovascular Health Promotion in the Schools* (2004), *Clinical Management of Metabolic Syndrome* (2004), and *Obesity, Insulin Resistance, Diabetes and Cardiovascular Risk in Children* (2003).

SEE ALSO: Congestive Heart Failure; Coronary Heart Disease in Women; Metabolic Disorders and Childhood Obesity.

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American Medical Association

THE AMERICAN MEDICAL Association (AMA) is a membership association of physicians, resident physicians, and medical students, limited to those who reside and practice in the United States, Puerto Rico, Guam, and the Virgin Islands (physicians), who are completing an accredited residency or fellowship program in one of those locations (resident physicians), or who are enrolled in a Liaison Committee on Medical Education (LCME) or American Osteopathic Association (AOA) program (students). The AMA was founded in 1847 by Nathan S. Davis, who was also the first editor of the *Journal of the American Medical Association* (*JAMA*), a scholarly journal founded in 1883.

The mission of the AMA is "to promote the art and science of medicine and the betterment of public health." The AMA is guided by two bodies: the 21-member Board of Trustees is charged with keeping the AMA focused on the promotion of medicine and the betterment of public health, while the House of Delegates is the policy-making body of the AMA and conducts most of the business of the Association. The AMA Alliance, founded in 1922 as the Women's Auxiliary to the AMA, is the volunteer arm of the AMA; membership is reserved for physicians and the spouses of physicians and medical students. The AMA Foundation is the philanthropic arm of the AMA; it develops and supports programs in medical education, public health, and research.

A number of special-interest groups exist within the AMA. The Medical Student Section provides medical students with a forum to discuss and have an impact on issues in medicine and health policy. The Resident and Fellow section educates residents about issues in medicine, provides career development resources, and represents and advocates for residents. The Young Physicians Section, for members under age 40 or in their first eight years of practice, provides information, resources and representation for physicians in the early stages of their career. The Organized Medical Staff Section offers education, information, advocacy, and networking opportunities for physicians working in hospitals and other healthcare delivery systems. The Group Practice Physician Section provides information about trends and patterns in group practices. The Women Physicians Congress helps develop women as leaders within medicine and advocates for women's health issues. The Minority Affairs Consortium helps develop minority leadership within the AMA and advocates for issues such as reducing health disparities. The International Medical Graduates section advocates for the concerns of international medical graduates within AMA and the larger medical community. The Senior Physician Group is for AMA members aged 65 or older who are retired or semi-retired. The Section on Medical Schools provides a forum for all medical schools within the United States to discuss and develop policy on medical education and national healthcare issues. The Advisory Committee on Gay, Lesbian, Bisexual, and Transgender (GLBT) Issues provides a mentoring and networking forum for GLBT physicians and students and represents their interests concerning AMA policy, advocacy, and education issues.

The AMA is involved in several advocacy efforts related to issues affecting healthcare and the practice of medicine; these include medical liability reform, Medicare physician payment reform, expanding insurance coverage for the uninsured, improving public health, funding for graduate medical education, and reforming managed care. The Advocacy Resource Center (ARC) was created in 1997 in order to conduct advocacy with state legislatures. The ARC is a partnership between the AMA and state medical associations and is governed by 14 Executive Committee members from the state medical associations.

AMA advocacy activities relating to public health fall into three main categories: healthy lifestyles, health disparities, and disaster preparedness. In the healthy lifestyles area, the AMA conducts research and develops and disseminates clinical resources related to issues such as obesity, alcohol and drug abuse, and violence prevention. The AMA states on its website that obesity "kills more Americans every year than AIDS, all cancers and all accidents combined" and is engaged in several efforts to halt the spread of obesity in children and adults. Among these, the AMA Working Group on Managing Childhood Obesity first met in 2003 to develop strategies to reduce obesity and overweight in children, and to reduce racial and ethnic disparities in childhood obesity. In addition, the AMA produced a road map for adult obesity, which aims to help physicians integrate obesity intervention into routine managed care.

SEE ALSO: Ethnic Disparities in the Prevalence of Childhood Obesity; Fitness; Future of Medical Treatments for Obesity; Medical Interventions for Children; Physical Activity and Obesity.

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American Obesity Association

THE AMERICAN OBESITY Association (AOA), founded in 1995 by the obesity researchers Richard L. Atkinson, MD, and Judith S. Stern, ScD, RD, is a nonprofit corporation dedicated to changing public policy and perceptions about obesity. The mission of the AOA is "to act as an agent change, move society to re-conceptualize obesity as a disease and to fashion appropriate strategies to deal with the epidemic." The AOA is supported by the nonprofit organization the American Obesity Association Research Foundation, which raises money and commissions studies on obesity. Since 1997, AOA headquarters have been located in Washington, D.C.

Membership in the AOA is available in several different categories, including individual, professional provider, obesity-specific clinic, corporation, nonprofit/government/small business, and student. AOA members who provide obesity-related services may be listed in the AOA Provider Directory, an online resource to help consumers find professional assistance. Members providing obesity-related services are expected to adhere to the AOA Patient's Bill of Rights, available on the AOA website. The document spells out patient rights in 15 different areas, including the right to be informed of treatment options and the likelihood of various outcomes, to be informed about the anticipated costs and duration of services, to be informed about realistic weight-loss goals, to be informed that rapid weight loss may cause serious health problems.

The AOA has six programmatic areas of activity: education, research, prevention, treatment, consumer protection, and discrimination and stigma. Specific goals of the AOA include making obesity a public health priority, achieving recognition of obesity as a disease, supporting obesity prevention, getting obesity treatment included in health insurance coverage, advancing new treatments for obesity, expanding obesity research, improving the understanding of obesity, protecting consumers from obesity-related frauds, fighting discrimination against obese person, and supporting the obesity community.

AOA activities in support of these goals include lobbying and advocating before Congress, the White House, and federal agencies and departments for policies to support the AOA mission; interacting with the healthcare industry to develop, disseminate, and expand obesity treatment options; developing educational materials to explain AOA positions; creating coalitions with other organizations and corporations; and communicating AOA positions through news releases, testimony before Congress and federal agencies, appearances on television and at conferences and meetings, and the AOA e-newsletters and website.

In the area of education, AOA efforts are focused on gaining recognition of obesity as a chronic disease, educating practitioners and the general public about the health costs of obesity, and advocacy to make obesity one of ten Leading Health Indicators for Healthy People 2010. The AOA fact sheet *Health Effects of Obesity*, available from the AOA website, details the association of obesity with risk for many diseases and medical conditions, including diabetes, coronary heart disease, high blood pressure, and osteoarthritis.

Research is supported by the AOA primarily through advocating for increases in research dollars award to obesity-related projects, and informing obesity researchers of funding sources from federal agencies and elsewhere. The AOA *Fact Sheet Obesity Research* summarizes the amount of public funding for obesity research, relative to health risks and diseases, and summarizes its arguments for increased funding for obesity research. The AOA has also developed a proposal for the creation of a National Institute for Obesity Research within the National Institutes of Health (NIH).

The AOA supports obesity prevention and treatment by summarizing and disseminating information about weight-gain prevention and weight-loss programs. The

AOA website also includes information about the economic cost of obesity and the effectiveness and cost effectiveness of weight-loss interventions. In the area of consumer protection, the AOA's primary activities are the collection and dissemination of information about consumer fraud in general, and weight loss and exercise products in particular.

Obesity-related discrimination is divided into three categories by the AOA: employment, healthcare, and education. The AOA position on obesity-related discrimination, as stated on its website, is that "Persons with obesity are frequently the victims of discrimination. Obesity is often described as the last 'acceptable' form of discrimination based on physical appearances." In the area of employment, the AOA website summarizes studies from several researchers which found that overweight people were subject to employment discrimination, were stereotyped as having negative personality traits, and earned lower wages than normal-weight persons, and discusses legal protections and remedies available to overweight people. In the area of healthcare, the AOA website presents studies about the attitudes of medical personnel concerning the obese and discrimination against the obese in healthcare. Concerning education, the AOA website summarizes studies that examine the school experience of overweight students, including teasing and bullying, and lower family support for education for overweight students.

SEE ALSO: Body Image; Fat Acceptance; Stereotypes and Obesity.

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American Society for Bariatric Surgery

THE PURPOSE OF the American Society for Bariatric Surgery (ASBS), founded in 1963, is to advanced the art and science of bariatric surgery (surgery on the stomach and/or intestines to aid weight loss in patients with extreme obesity). The ASBS is a founding member of the International Federation for the Surgery of Obesity (IFSO) and all members of ASBS automatically become members of IFSO.

Regular membership in the ASBS is available to surgeons who are certified by the American Board of Surgery or the American Osteopathic Board of Surgery or are fellows of the American College of Surgeons or the Royal College of Surgeons, and have acted as primary surgeon on at least 25 bariatric surgeries in the last two years. Affiliate Surgeon membership is available to surgeons who lack the necessary certification or have not performed sufficient bariatric surgeries. Other membership classes are available for physicians who are not surgeons, medical students, residents, or fellows who are training in bariatric surgery, and allied healthcare professionals.

The ASBS holds an annual meeting in June and sponsors educational courses for surgeons and other healthcare workers; the latter category includes nurses, dietitians, psychologists, psychiatrists, physician assistants, social workers, counselors, and program managers interested in supporting bariatric surgery and research.

Surgery for Obesity and Related Diseases (*SoaRD*), published by Elsevier, is the official journal of the ASBS. *SoaRD* is published six times a year and includes peer-reviewed manuscripts related to the treatment of severe obesity, including documentation of the effects of surgically induced weight loss on physiological, psychiatric, and social comorbidities.

In 2004, the ASBS issued a consensus statement concerning bariatric surgery for morbid obesity, which is available from the ASBS website. Among the positions advocated in this document are that bariatric surgery is the most effective therapy for morbid obesity, that operative procedures are continuously evolving, that both open and laparascopic bariatric operations are effective therapies, that candidates for bariatric surgery should have attempted to lose weight by nonoperative means, that bariatric surgery can be cost effective before the fourth year of follow-up, and that bariatric surgery should be considered for morbidly obese adolescents (BMIs > 40) and for persons with a BMI between 30 and 34.9 who have a comorbid condition (e.g., Type 2 diabetes) which

would be improved by weight loss. The *ASBS Guidelines for Granting Privileges in Bariatric Surgery,* available from the ASBS website, state ASBS recommendations for experience and training required to perform different types of bariatric surgery. *Suggestions for the Pre-Surgical Psychological Assessment of Bariatric Surgery Candidates*, issued by the Allied Health Sciences Section of the Ad Hoc Behavioral Health Committee of the ASBS in 2004 and available from the ASBS website, contain recommendations for the assessment of candidates for bariatric surgery in the areas of behavior (including previous attempts at weight management), cognitive and emotional functioning, developmental history, current life situations, and motivation and expectations.

SEE ALSO: Bariatric Surgery in Children; Bariatric Surgery in Women; Medication Therapy After Bariatric Surgery; Qualifications for Gastric Surgery.

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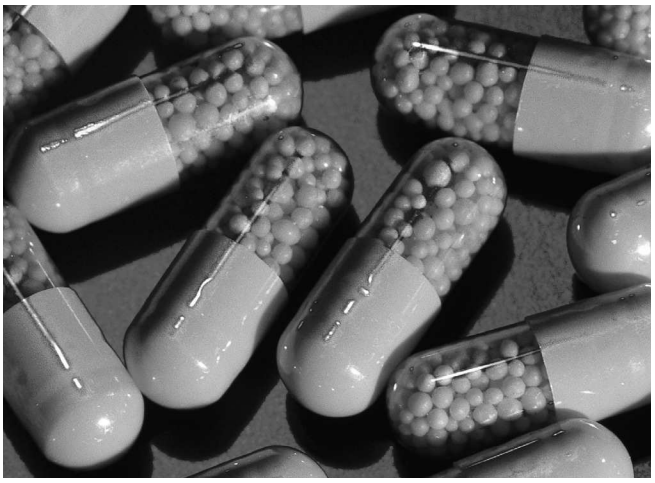

Amphetamines have long been prescribed as an aid in dieting and weight loss, but with a potential cost to the health of users.

Amphetamines

AMPHETAMINES HAVE BEEN commonly prescribed for obesity since the first introduction of amphetamine (Benzedrine) as a dietary aid in 1937 and continuing to the present day. By 1948, 92 percent of physicians surveyed were prescribing amphetamines for weight loss. In 1959, phentermine was approved for weight loss by the U.S. Food and Drug Administration. As shown in the accompanying figure, phentermine differs from amphetamine itself only in the addition of a methyl group. The primary effect of this change is to slow its breakdown and therefore prolong its half-life within the body, so that phentermine can be taken once a day, whereas amphetamine itself is only active for around four hours. Phentermine is also closely related to methamphetamine, which has a methyl group attached to the amine nitrogen. Methamphetamine has a faster onset of action, leading to intense euphoria, whereas phentermine has a much slower onset and is only a mild euphoriant.

Drugs in the amphetamine class are sympathomimetics, because they mimic the action of the sympathetic nervous system. Amphetamines activate sympathetic nerve terminals release norepinephrine in three ways. First, they block the reuptake of norepinephrine and dopamine by the nerve terminal, thereby prolonging the action of norepinephrine within the active zone of the nerve synapse. Second, they directly provoke the release of norepinephrine and dopamine from nerve terminals. Third, the amphetamines slightly inhibit the activity of monoamine oxidase, which breaks down norepinephrine and dopamine. These three actions synergize to stimulate norepinephrine secreting nerve terminals. The nonamphetamine obesity drug sibutramine shares the first action with amphetamine—blocking reuptake—but lacks any effects on release and breakdown. Thus, sibutramine has a milder stimulant action than amphetamines.

Increased norepinephrine and dopamine within the brain—specifically within the appetite centers of the hypothalamus—leads to suppression of appetite and reduced food intake. The effect on food intake tends to fade over time, so that maximum weight loss with amphetamine is achieved within 12 weeks. This is one reason why the use of amphetamines for weight loss is not recommended for more than 12 weeks. The addi-

tional weight loss relative to placebo ranges between 5–20 lb. As would be expected, weight regain occurs rapidly upon discontinuation of amphetamines.

Amphetamines are not only active in the brain, but also throughout the body. Activation of the sympathetic system slows the digestive tract leading to a sensation described as "butterflies in the stomach." Undoubtedly, these systemic effects contribute to weight loss during amphetamine treatment.

The sympathomimetic action of amphetamines can lead to a number of physiological side effects, especially on the cardiovascular system. According to the Hazardous Substances Databank, the primary hazards of phentermine are:

Cardiotoxicity causing tachycardia, arrhythmias, hypertension and cardiovascular collapse; high risk of dependency and abuse; palpitation; chest pain; and hypertension are common. Cardiovascular collapse can occur in severe poisoning. Myocardial ischemia, infarction and ventricular dysfunction are described.

All of these effects are predictable based on the known functions of the sympathetic nervous system. Furthermore, sympathoinhibitory drugs such as beta-blockers are commonly used in cardiology and have been shown to reduce cardiovascular mortality.

Amphetamines are used with relative safety in children for the treatment of attention deficit disorder, but the doses used are much lower and treated children show a paradoxical tranquilizing response to amphetamine that is not common in adult obese patients treated with anorexic doses. Phenylpropanolamine and ephedrine, much milder stimulants used as an over-the-counter obesity treatment, were both removed from the market by the FDA following evidence of increased risk of stroke, a well-known risk of all sympathomimetics.

Side effects of stimulant medications such as amphetamines also include the possibility of addiction and dependence, which is one of the main reasons that treatment is not recommended long-term. The most severe psychiatric side effect has been described as "amphetamine psychosis," which can be indistinguishable from schizophrenia, but usually resolves upon withdrawal of drug therapy.

All of these hazards and side effects are mainly characteristic of amphetamines, including phentermine, and do not apply to sibutramine, a milder sympathomimetic agent. Nonetheless, safety issues regarding elevation in blood pressure and stroke risk have been raised with sibutramine as well.

SEE ALSO: Dopamine; Norepinephrine; Sibutramine (Meridia).

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Animal Models of Obesity

COMPLEX PHYSIOLOGICAL AND behavioral conditions that occur in humans can be investigated in animal species that display, whether spontaneously or as a result of intervention, similar features of critical aspects of the condition being studied; such animals provide what is known as an animal model of the condition. In the case of obesity, the critical aspect is accumulation of excess body fat. Like humans, adequately nourished nonhuman species store body fat in amounts that support growth and reproduction; the percent of body weight that is due to fat at a given age may be quite consistent for the species despite variation in environmental conditions. However, like humans, other animal species can accumulate additional body fat (i.e., show an obese phenotype); this excessive accumulation may be associated with the behavioral and metabolic changes characteristic of human obesity.

Animal models can provide valuable information about human conditions that would be difficult or even impossible to obtain from human subjects. The shorter gestation periods and life spans of rodents, for example, make it possible to examine progression of obesity throughout life, as well as maternal effects on offspring and trends over many generations. Conditions in the animal's life, such as the composition and amount of food eaten, can be controlled and measured

continually. Animal models make it possible, for example, to obtain tissues for study outside of the body, to use radioactivity or substances considered dangerous, to do surgery, or to implant devices in the brain, all of which can provide information crucial to understanding the condition in question. Perhaps the most exciting new use of animal models allows manipulation of the genome (the total deoxyribonucleic acid [DNA] of the organism) to uncover the roles that inherited traits play in controlling body fat accumulation and in mediating its effects.

GENETIC MODELS

Animals living under standard conditions have been observed to spontaneously develop obesity; when this trait can be predictably passed to later generations, a genetic model of obesity may be defined. An example of a single-gene mutation that produces obesity is the ob/ob mouse. The recessive allele ob fails to produce the protein leptin, which the dominant allele Ob codes for; inheritance of both recessive alleles leads to absence of leptin and extreme obesity. Study of this genetic model led to the discovery of the hormone leptin, synthesized in adipose tissue, which inhibits feeding, increases energy expenditure, and alters metabolic pathways such as fat oxidation. Other spontaneous single-gene mutations have been shown to produce obesity in animal models; combinations of several genetic traits (polygenic models) may also be responsible for inherited obesity.

The genome can be manipulated in the animal to prevent the production of the protein coded for by a particular gene; this technique produces a genetic knockout model for the particular trait. The opposite effect, overexpression of a particular gene, can also be produced. These effects are bodywide unless techniques are used to produce tissue-specific effects. For example, nonfunctional membrane receptors for insulin might be produced only in certain tissues but not others; this would assist in the study of insulin resistance (a condition in which insulin is ineffective in producing its effects on cells), one of the metabolic effects of obesity. New traits can be introduced into the genome as well, utilizing viruses to carry the genetic information to targeted cell types. An example is introduction of a functional leptin gene (Ob) into specific tissues of an ob/ob mouse.

Interactions between the genome and the environment can be investigated using animal models of obesity. Epigenetics refers to non-DNA aspects of gene expression, some of which can be passed to the next generation. For example, substances in the diet that promote the addition of methyl (CH3) groups to DNA or its associated proteins can influence the transcription (formation of a strand of ribonucleic acid (RNA) that is complementary to the DNA) of a gene. The epigenetic influence of maternal diet on the predisposition of the offspring to obesity is of current interest.

DIETARY MODELS

Accumulation of excess body fat results from chronically positive energy balance where energy intake from the diet exceeds energy output. Although animals living in the wild or under standard laboratory conditions tend to maintain dietary intakes that match energy outputs (zero balance), humans (and pets) may not. By altering the composition of the diet in ways that override the physiological/behavioral mechanisms that would maintain zero energy balance, animals of various species can be made obese. Diets that are lower in fiber and water content but higher in fat will provide more calories per gram; this higher energy density supports increased energy intake. Diets comprised of a variety of sweet, fatty, aromatic foods can promote greater food intake. Strain of animal or stage of development may affect the susceptibility to dietary effects. Dietary models allow investigation of behavioral and physiological responses to a human-like situation and may offer results with clear application to humans.

DEVELOPMENTAL MODELS

Maternal reproductive function, placental development, and the intrauterine environment may have lasting effects on the offspring's later risk of obesity and obesity-related disease such as hypertension. Changes in these factors through diet, drugs, surgery, hormone administration, and other means allows controlled study of their effects on feeding behavior, body composition, and metabolic function over time, even over multiple generations.

SURGICAL/CHEMICAL MODELS

Alterations in the configuration or function of specific neurological structures, endocrine glands, or

other tissues can produce obesity. Such manipulations can reveal mechanisms that control feeding behavior, physical activity, and fuel storage. For example, destruction of neural pathways that send information from the gastrointestinal tract to the brain has uncovered ways in which the small intestine informs the brain about the nutrients that will be absorbed before these nutrients can reach the brain via the blood supply. Compounds that stimulate or inhibit the release of specific hormones have established the effects of these hormones on hunger and satiety. Surgical or drug treatments in animals may lead to increases in body fat stores, thereby supporting a role for the affected mechanisms in producing or preventing obesity.

SEE ALSO: Db/Db Mouse; Fiber and Obesity; Leptin; Ob/Ob Mouse.

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Animal QTLs (Quantitative Trait Locus)

QUANTITATIVE TRAIT LOCUS, or QTL analysis, is a modern, genetically based way for breeders to tailor their livestock to the needs of consumers. Researchers performing QTL analysis investigate the role of genes, regionally located on chromosomes, in polygenic traits such as body fat, meat tenderness, or milk production.

The genomes, or entire deoxyribonucleic acid (DNA) contents, of many livestock species have been sequenced. These species include cattle, sheep, chickens, and more. Other important model organisms such as mice and rats have also had their genomes sequenced. Researchers can use the vast information available for comparing and contrasting both different species as well as animals within a species. The ultimate goal of QTL analysis is determining the genetic underpinnings of all traits and characteristics, specifically those that are determined by multiple genes. Such traits are termed *polygenic*.

DNA is organized in the cell into chromosomes. Roughly speaking, the chromosomes are organized into noncoding DNA and coding DNA which encodes proteins; the region of a chromosome which encodes a protein is called a gene. The hypothesis that forms the basis of QTL theory is that groups of genes near to each other on a chromosome may have a multigene effect on certain polygenic traits. Using sophisticated mathematics and genetic experiments, investigators can examine these gene regions and their relationships to traits. This is the process for QTL mapping. In other words, scientists look at how often particular genes are expressed together in relation (linkage) to specific traits. Breeders can use this information to generate better, more nutritional meat for consumption. For example, pork backfat and cattle milk production have strong QTL linkages.

QTLs can be used to help study many examples of human obesity as well, and in genome scans from 2002, there were reported to be 68 QTLs for obesity in humans.

To aid researchers in their comparisons, there is a website called the Animal QTL database (AnimalQTLdb). As of 2007, the database includes information on pigs, cattle, and sheep, and will expand to include more species.

SEE ALSO: Genetics; Genomics; Human QTLs; Quantitative Trait Locus Mapping.

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Anorexia Nervosa

EATING DISORDERS ARE a spectrum of illnesses that have serious psychological and medical consequences, both in the short and long term. In the case of anorexia nervosa, the person has an unyielding pursuit of thinness, and may use a variety of behaviors to elicit a rapid and severe degree of weight loss. To understand anorexia, a number of topics must be explored, including the clinical definition, epidemiology, the signs and symptoms, treatment, and consequences.

DEFINITION

Anorexia nervosa, as defined in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV), has four criteria. First, the individual must refuse to maintain a weight within a normal range for height and age, meaning more than 15 percent below ideal body weight. Second, the person experiences a fear of weight gain. Third, there is a severe body image disturbance, and the determination of self-worth is measured based on body image without acknowledgment of the severity of the illness. Last, in women who have begun to menstruate, amenorrhea, or the absence of three or more menstrual cycles, is part of the diagnostic criteria. Within this definition, there are two subtypes: a restricting type and a binge eating/purging type. The restricting subtype maintains a low body weight by restricting caloric intake. The binge/purge subtype will either binge (large intake of calories) or purge (using vomiting and/or laxatives to remove calories) to control their weight. The diagnosis of anorexia is still appropriate with the binge/purge subtype because there is still a body weight at 15 percent less than ideal body weight.

EPIDEMIOLOGY

Most often, it is females who are affected by anorexia, with males representing only 5 percent of all anorexic patients. It is estimated that up to 0.5 percent of young females meet the criteria for anorexia nervosa. Onset is usually four to five years after the onset of menarche. Overall data and trends of eating disorders are difficult to establish because the definition has changed over time and the disease often relies on self-reporting. Many studies demonstrate increasing prevalence of anorexia nervosa over the last 50 years in young women (15–24 years old) in the United States. The incidence of anorexia nervosa and other eating disorders is lower in developing countries. However, some studies suggest that an increasing westernization and modernization of these countries is contributing to a changing self-image and body image among women, and an increased reporting of anorexia.

There is no medical consensus on the etiology of eating disorders including anorexia nervosa. Most models include psychological, biological, family, genetic, environmental, and societal factors. There seems to be an even distribution of anorexia nervosa across social classes. These forces can cause a decreased sense of self-esteem, body image, or self-control. There are several more specific factors that may be influential in the development of anorexia. For example, some literature suggests that an important predictor of eating disorders is dieting during adolescence. In addition, if there is a preoccupation or obsession with thin body image or if one feels a social pressure to be thin, this may be associated with the development of eating disorders. Feminist psychology theory has suggested that societal pressure to be "super" women in the setting of Western society can predispose women to develop eating disorders. Those who are later diagnosed with anorexia are often de-

Anorexia nervosa involves the irrational fear of weight gain as well as significant body image disturbance.

scribed as perfectionists, often excelling in school, athletics, and interpersonal experiences. It is unclear if there is a prodrome of symptoms that precede onset of anorexia. There may be family characteristics that are associated with individuals developing eating disorders. In particular, there is an association with the experience of high parental expectations related to achievement and appearance, and settings with poor communication or marital tension.

In addition, there may be a genetic predisposition to development of eating disorders. If a first-degree relative has an eating disorder, a young woman is six- to tenfold more at risk for developing an eating disorder. Twin studies of identical (monozygotic) twins show a higher concordance rate of eating disorders than between nonidentical (dizygotic) twins. However, these observations do not exclude the role of environment and family influences. There is also a high incidence of psychiatric disorders in individuals with eating disorders. These include affective disorders such as depression, anxiety disorders, obsessive-compulsive disorders, and personality disorders. The psychiatric illness may be present in childhood. There may also be a higher rate of substance abuse.

Altered levels of neurotransmitters, including norepinephrine and serotonin, may be involved in the development of anorexia nervosa. There are decreased levels of norepinephrine in the brains of anorexics experiencing starvation, and this is thought to mediate the slow heart rate (bradycardia) and low blood pressure (hypotension) seen in these individuals. The brain's satiety center is influenced by serotonin, and people with preexisting high level of seratonin in this region of the brain may be predisposed to obsessive behavior and eating disorders. There is also disruption of hormones in those with anorexia nervosa; however, many of them are seen in other states of starvation, and it is therefore unclear if the decreased levels of luteinizing hormone and decreased response to thyroid stimulating hormonal are involved in causation of anorexia or are resultant from starvation.

SYMPTOMS

A universal presenting symptom of anorexia nervosa is a distorted body image. Often, there is a belief that the individual is fat regardless of how much weight is lost. Cognitive reasoning cannot alter the individual's body perception. A person will use both diet and exercise to reduce body weight. The need to control caloric intake often manifests as an intense interest or obsession with food. Individuals will talk at length about food, will prepare elaborate meals, and the process of eating can become very ritualized.

With the same intensity used to monitor caloric intake, individuals pursue exercise such as running, jogging, or bicycling to loose weight. Weight lifting, swimming, or other activities that would increase muscle mass are avoided. Another method of removing calories and losing weight is self-induced vomiting after meals, as seen in the binge/purge subtype of anorexia nervosa. Laxative use to speed bowel transit time is another method of removing food and reducing absorption of calories.

These behaviors may lead to significant weight loss, often apparent to a casual observer. However, if the onset of anorexia nervosa is during puberty, the individual may not technically lose weight, but they will fail to gain weight as their height increases. As anorexia progresses and weight is lost, patients often feel depressed, have trouble concentrating, and experience insomnia. Amenorrhea is common and may manifest as delay in onset of menstruation, or discontinuation of menstruation in a previously menstruating female. The physical response to starvation can be seen in these individuals. There is a low body temperature, decreased heart rate, and a low blood pressure. Skin is dry and scaled, and a fine hair (lanugo) grows to cover the body. In patients with the binge/purge subtype, there may be erosion of teeth secondary to exposure to stomach acid.

There is a low level of red blood cells, platelets, and white blood cells in a patient with anorexia nervosa, increasing the risk of serious infections. Low levels of blood potassium is often seen, either secondary to vomiting or diarrhea or low dietary intake. Liver function tests are often abnormally elevated, and there are deficiencies in nutrients such as magnesium and thiamine. Beta-carotene levels are often elevated, giving skin a yellow coloration. Evaluation of heart electrical conduction with an electrocardiogram (ECG) often shows slowed heart rate and a variety of arrhythmias, including ventricular fibrillation, which may cause sudden cardiac death.

TREATMENT

Treatment of anorexia nervosa is goal-oriented, with a focus on weight restoration and reintegration of the

patient into his/her family and social structure. Medical and behavioral therapy are used toward the goal of weight gain. Initial management involves deciding if an individual needs to be hospitalized or can be followed as an outpatient. However, due to the life-threatening nature of many of the manifestations of anorexia, patients are often hospitalized at the point that they present for medical care.

Initial medical management involves correcting life-threatening alterations in blood potassium, magnesium, and thiamine with supplementation. Forced feeding through nasogastric feeding tubes, or more rarely, through parentaral nutrition administered through the veins, may be used. Subsequently, goal or ideal end weight is established by taking into consideration premorbid weight, the patient's level of comfort, bone structure, and other factors. Establishment of a daily routine, including weighing before and after meals and voiding (urine and bowel movements) is begun with a common goal of gaining at least half a pound per day. Care must be taken to avoid refeeding syndrome in severely underweight individuals. Withholding of rewarding activities is often used, as is the use of isolation with limited contact with family members or medical staff. Meals are presented at regular intervals, and snacks are available as desired.

In addition to intense behavioral modification, there are interventions with the family to establish realistic expectations. Similarly, psychotherapy has an important role in management of anorexia nervosa, on an individual and sometimes on the family level. Patients must develop coping skills both to manage healthy weight gain and to deal with challenges of adolescence. The use of medications in the treatment of anorexia nervosa is limited. Often, use of pharmacotherapy is to treat comorbidities, such as depression or obsessive-compulsive disorders. Medicines that decrease anxiety may be useful before meals.

MORBIDITY AND MORTALITY

There is significant mortality associated with anorexia nervosa. Young women with anorexia have a tenfold increased mortality as compared to unaffected women. A long-term, 10-year follow-up study found that the overall mortality rate was 6.6 percent, although other studies have found relatively lower rates when including women treated as outpatients. The natural course of anorexia demonstrates that without intervention, only 40 percent of patients will experience significant recovery, 35 percent will have some improvement, 20 percent will remain acutely ill and may still meet criteria for anorexia at 12 years after initial diagnosis, and the remaining five percent will die from medical complications related to anorexia or suicide.

There are several serious complications secondary to eating disorders such as anorexia nervosa, including osteopenia or osteoporosis, cardiac impairment, cognitive changes, gastrointestinal problems, hormone changes, and electrolyte abnormalities. Osteopenia or osteoporosis is often seen in anorexic patients, possibly due to estrogen deficiency, or low vitamin D and calcium intake. It is characterized by increased bone resorption and decreased bone formation, and may lead to increased risk of fractures later in life. Several cardiac problems are associated with anorexia nervosa, including mitral valve prolapse, changes in electrical activity, and heart failure. The risk of heart failure is greatest during the first two weeks of refeeding after prolonged starvation. Hormonal changes during starvation can lead to cessation of menstruation, which typically resumes within 6 months of achieving 90 percent of ideal body weight.

SEE ALSO: Bulimia Nervosa; Eating Disorders and Athletes; Eating Disorders and Gender; Eating Disorders and Obesity; Eating Disorders in School Children; Sexual Abuse and Eating Disorders.

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Antidepressants

ANTIDEPRESSANTS ARE AMONG the most commonly prescribed classes of medications. Antidepressants

are effective in the treatment of multiple psychiatric conditions including depression, schizophrenia, bipolar disorder, panic disorder, generalized anxiety disorder, obsessive-compulsive disorder, and other personality disorders. In addition to psychiatric conditions, antidepressants are also effective in treating pain, preventing migraines, and treating eating disorders such as anorexia nervosa and bulimia nervosa. Patients who suffer from obesity are commonly treated for depression or multiple other conditions for which antidepressants are prescribed. Specific considerations of possible weight-related side effects should be made prior to initiating antidepressant therapy in obese patients.

Most medications, including antidepressants, are classified by their chemical structure and how they work in the body. Currently, there are six different classes of medications that are considered to be antidepressants: tricyclic antidepressants (TCAs), nonselective norepinephrine reuptake inhibitors, serotonin/norepinephrine reuptake inhibitors (SNRIs), selective serotonin reuptake inhibitors (SSRIs), aminoketones, and monoamine oxidase inhibitors (MAO-Is). In addition to traditional medications, hyperforin and adhyperforin, substances found in the herb St. John's Wort, has antidepressant effects.

TCAs work in the body by increasing the amount of several natural chemicals in the body; specifically of interest are norepinephrine and serotonin, which directly help to improve nerve function. Improvement of nerve function can help many different medical conditions, including depression and pain relief. TCAs' activity is not limited to one area of the body and it is common for patients to experience side effects including sedation, dry mouth, decreased need to urinate, and decreased blood pressure. TCAs are also associated with moderate weight gain, a side effect that should be considered prior to use in obese patients. Nonselective norepinephrine reuptake inhibitors provide similar benefits and side effects as TCAs.

SNRIs such as venlafaxine (Effexor) and duloxetine (Cymbalta) work similarly to TCAs. Unlike TCAs, SNRIs work specifically by increasing levels of norepinephrine and serotonin and results in greater improvements in depression symptoms with a lower occurrence of side effects in comparison to TCAs. SNRIs are effective in the treatment of chronic pain, nerve pain, and pain occurring as part of depression symptoms. SNRIs are also not associated with weight gain, unlike other antidepressant classes.

SSRIs are the most widely used antidepressants. Examples of these include Prozac, Zoloft, Lexapro, and Celaxa. Unlike the other antidepressant classes, SSRIs specifically work to increase levels of serotonin. The occurrence of side effects from SSRIs is much less than that of TCAs, resulting in better patient satisfaction with therapy. Although TCAs provide greater improvement in symptoms of depression, SSRIs may be beneficial in patients with bipolar depression due to their possibility of triggering manic symptoms. SSRIs may also be used for the management of pain, but are not as effective as other antidepressant classes. SSRIs are not commonly associated with weight gain.

Currently, bupropion is the only available aminoketone available in the United States. Bupropion works to primarily increase levels of dopamine, but may also increase levels of norepinephrine and serotonin at higher doses. The increase in dopamine, a precursor, or norepinephrine, leads to greater improvements in depression symptoms with a lower occurrence of side effects in comparison to other antidepressants. Although rare, bupropion has been associated with the onset of seizures at high doses and should be avoided in patients at risk for or with a history of seizures. Of the available antidepressants, bupropion is the least likely to cause weight gain and sexual dysfunction. Bupropion use should also be avoided in patients with a history of eating disorders.

Monoamine oxidase inhibitors (MAO-Is) prevent the breakdown of norepinephrine, serotonin, and dopamine, hence increasing their levels available in the body. Similar to the other medications that increase levels of these chemicals, MAO-Is improve the symptoms of depression, but may also be used for the management of pain, prevention of headaches, and many other disease states that are affected by these natural chemicals. Due to their ability to affect multiple chemicals, MAO-Is are more likely to cause a greater number of side effects, including insomnia, agitation, changes in blood pressure, and sexual dysfunction. MAO-Is should only be used in patients whose symptoms of depression are not resolved by other antidepressant treatments due to the greater number of drug interactions and dietary restrictions required to prevent negative side effects. Patients receiving MAO-Is should be educated on avoiding tyramine-containing

foods such as wine and aged cheese to prevent drastic, harmful increases in blood pressure.

In addition to the many available prescription medications, an alternative herbal medicine that has shown benefits in relieving the symptoms of depression is St. John's Wort. Hyperforin and adhyperforin are components found in St. John's Wort that appear to provide antidepressant effects by preventing the reuptake of serotonin, dopamine, and norepinephrine. Doses greater than 2–4 grams of St. John's Wort daily may cause negative skin reactions and should be avoided. Similar to prescription and nonprescription medications, alternative herbal medicine interact with other medications. St. John's Wort interacts with a large number of medications; therefore, patients should check with their physician or pharmacist prior to using any herbal medication. Patients should also be aware that the U.S. Food and Drug Administration does not approve alternative herbal medications; hence, the ingredients may vary from product to product and risks or benefits cannot be verified.

Selecting the most appropriate treatment for a patient should be based on the individual patient's needs and goals of therapy while limiting medication side effects.

Anxiety comes in many forms and from many sources, but all feature similar symptoms in patients.

SEE ALSO: Depression; Dopamine; Norepinephrine; Psychiatric Medicine and Obesity.

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Anxiety

FEAR, WORRY AND apprehension are a combination of emotions that are often defined as *anxiety*. Anxiety symptoms are heart palpitations, nausea, chest pain, shortness of breath, or tension headache. The other common symptoms of anxiety are increased blood pressure, heart rate, sweating, and blood flow; inhibited immune and digestive systems; thyroid, respiratory, and gastrointestinal disturbances; arthritis; migraine; and allergic conditions. Pale skin, sweating, trembling, and pupillary dilation are outward signs of anxiety. Obese people have a 25 percent increased risk for developing anxiety disorders.

Severe anxiety leads to generalized anxiety disorders (GAD). Forty million American adults age 18 years and older (about 18 percent) exhibit symptoms of anxiety disorder. Anxiety disorders last at least 6 months and commonly occur along with other mental or physical illnesses, including alcohol or substance abuse, which may mask anxiety symptoms or make them worse. While there are many different symptoms of anxiety disorders, all these symptoms cluster around irrational fear and dread.

TYPES OF ANXIETY

Existential anxiety. This is part of obsessive compulsive disorder, and typically about sex and religion

or death. Eating disorders are commonly seen in adolescent girls with obsessive compulsive disorder.

Test anxiety. Test anxiety is the uneasiness, apprehension, or nervousness felt by students who have a fear of failing an exam. These students may experience any of the following: the association of grades with personal worth, embarrassment by a teacher, taking a class that is beyond their ability, fear of alienation from parents or friends, time pressures, or feeling a loss of control. An association of obesity-binge eating disorders is enhanced by the presence of psychical disorders, like depression, anxiety, or bipolar disorders. Sixty percent of patients with binge eating disorder also present with anxiety. Emotional, cognitive, behavioral, and physical components may present in test anxiety. The common symptoms are sweating, dizziness, headaches, racing heartbeats, nausea, fidgeting, and drumming.

Stranger anxiety. Stranger anxiety is a type of anxiety that occurs when meeting or interacting with people that are unknown, and is a common stage of development in young people. Stranger anxiety in younger people is not a phobia in the classic sense; rather it is a developmentally appropriate fear by young children of those who do not share a "loved-one," caregiver, or parenting role. In adults, an excessive fear of other people is not a developmentally common stage.

Panic disorder. Panic disorder is characterized by sudden attacks of terror, usually accompanied by a pounding heart, sweatiness, weakness, faintness, or dizziness. During these attacks, people with panic disorder may flush or feel chilled; their hands may tingle or feel numb; and they may experience nausea, chest pain, or smothering sensations. Panic attacks usually produce a sense of unreality, a fear of impending doom, or a fear of losing control. Panic disorder affects about six million American adults; this disorder is twice as common in women as in men. Panic disorder is often accompanied by other serious problems, such as depression, drug abuse, or alcoholism.

Obsessive-compulsive disorder (OCD). People with obsessive-compulsive disorder (OCD) have persistent, upsetting thoughts (obsessions) and use rituals (compulsions) to control the anxiety. OCD affects about 2.2 million American adults. OCD may accompanied by eating disorders, other anxiety disorders, or depression. It strikes men and women in roughly equal numbers and usually appears in childhood, adolescence, or early adulthood. One-third of adults with OCD develop symptoms as children, and research indicates that OCD might run in families. OCD patients treated for 2.5 years with clomipramine or selective serotonin reuptake inhibitors observed a non-significant weight gain (34.8 percent) compared with sertraline and fluoxetine.

Post-traumatic stress disorder (PTSD). Post-traumatic stress disorder (PTSD) develops after a terrifying ordeal that involved physical harm or the threat of physical harm. PTSD affects about 7.7 million American adults, but it can occur at any age, including childhood. Women are more likely to develop PTSD than men.

Social phobia (or social anxiety disorder). Social phobia affects about 15 million American adults. People with social phobia have an intense, persistent, and chronic fear of being watched and judged by others and of doing things that will embarrass them. Women and men are equally likely to develop the disorder, which usually begins in childhood or early adolescence. There is some evidence that genetic factors are involved. Social phobia is often accompanied by other anxiety disorders or depression, and substance abuse may develop if people try to self-medicate their anxiety.

Specific phobias. A specific phobia is an intense fear of something that poses little or no actual danger. Some of the more common specific phobias are centered on closed-in places, heights, escalators, tunnels, highway driving, water, flying, dogs, and injuries involving blood.

Generalized anxiety disorder (GAD). GAD is diagnosed when a person worries excessively about a variety of everyday problems for at least six months. Approximately 6.8 million American adults, or about 3.1 percent of people age 18 and over, have GAD. People with GAD can not relax, startle easily, and have difficulty concentrating. Physical symptoms include fatigue, headaches, muscle tension, muscle aches, difficulty swallowing, trembling, twitching,

irritability, sweating, nausea, lightheadedness, having to go to the bathroom frequently, feeling out of breath, and hot flashes.

OBESITY AND ANXIETY DISORDERS

Obesity is associated with an approximately 25 percent increase in odds of mood and anxiety disorders and an approximately 25 percent decrease in odds of substance use disorders. Weight reduction maintained for 2 years was associated with changes in practically all personality traits in proportion to the magnitude of weight loss.

ANXIETY DRUGS AND OBESITY

Azaspirones; benzodiazepines; beta blockers; tricyclics; monoamine oxidase inhibitors and serotonin reuptake inhibitors are antianxiety drugs. Currently, it is known that tricyclics and serotonin reuptake inhibitors may increase weight gain. Betablockers may reduce coenzyme Q10 and decreases HDL-C.

SEE ALSO: Antidepressants.

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Appearance

THOSE WHO ARE overweight or obese suffer from issues regarding and related to their appearance. Western society as a whole values a particular body ideal that does not comport with the outward appearance of those with excess weight. Western society also imbues the ideal with value judgments based on compliance with the accepted body ideal, and those who do not meet the ideal are judged to be lacking and less than acceptable in many aspects.

The prevailing body ideal of the dominant Western culture is predominantly biased against fat and excess weight. Those who carry excess weight are often assumed by the populace to be of lower intelligence, to have less personal discipline, and to be lazy, gluttonous, or slovenly. Overweight and obese individuals are consistently hearing and internalizing messages from society to this effect, and thus either tend to make it a self-fulfilling prophecy, or otherwise let it negatively affect their self-perception and self-esteem.

The overweight and obese internalize much of the negative messaging society and individuals aim at them; this manifests as self-loathing, guilt, and shame. These in turn can lead to anxiety, depression, and further disordered eating patterns. In fact, anxiety and depression rates are three to four times higher in obese individuals than in their leaner counterparts. The frequency of being teased about weight and size while growing up has been proven to be negatively correlated with evaluation of one's appearance, and positively correlated with body dissatisfaction during adulthood. Further, subjects with early-onset obesity reported greater body dissatisfaction than did subjects with adult-onset obesity.

Body dissatisfaction is quite often manifested in a distorted image of one's appearance, attractiveness, and/or self-worth. These manifestations surface in situations such as career choice, clothing and dress, dating, and intimate and sexual circumstances. While overweight individuals tend to choose occupations and careers that are about on par with their thinner counterparts, obese and morbidly obese individuals often choose occupations and careers that do not require them to be out in the public eye, or that require a limited contact with persons outside their comfort zones.

Both groups often have trouble finding clothing that reflects the current fashion trends or that fits properly. They are penalized for their size through increased prices for clothes made in bigger sizes, despite the fact that the manufacturing cost is not proportionate to the increase in retail cost.

Stores catering to larger individuals are fewer and far between, in direct contrast to the fact that 65 percent of the American population is overweight or obese. Clothing is often merely serviceable, or made from lower quality fabric and materials. Larger individuals are rarely encouraged by the media to dress fashionably, tend to their health and fitness in the same ways directed toward thinner persons, or care for their outward personal appearance through hair, makeup, accessorization, or other personal hygiene issues. As a result, larger people tend to feel segregated by society. They feel second rate, which again manifests as shame and embarrassment.

Embarrassment regarding personal appearance crosses over from the internal to the external when larger people seek intimate relationships. Dating is more difficult for heavy people as they field quite a bit of rejection based solely on appearance. Some rejection comes in the form of polite excuses, while others are more pointed and direct about the fact that the individual is not only unattracted to the heavier person, but may actually be repulsed or disgusted by the heavy person's lack of compliance with the social ideal. Some people will even directly express their anger that the heavy individual is not even trying to meet the body ideal. This, of course, is often merely a perception, as most heavy individuals are in a constant state of attempting to achieve the social body ideal and change their appearance.

Even when intimate personal relationships are achieved, personal appearance and the attendant issues insinuate themselves into the sexual relationship. It is extremely difficult for heavy people to literally bare themselves to the examination and potential criticism or ridicule of another human being. It is tremendously difficult to reveal that which one may have spent a lifetime trying to hide or disguise. In fact, it is so difficult that many heavy individuals refrain from visiting medical professionals for fear of physical examination, recrimination, and reprimand. The patient is in much more control in the medical setting than in the sexual setting, as a rule. In an intimate relationship, the bedroom is where one bares both body and soul, and risks hearing again about one's unacceptability because of outward appearance and weight.

Bias, prejudice, and discrimination are prevalent in dating, intimate, and even family relationships. Because of the entrenched societal nature of the body ideal and the attitude toward heavy people, individuals in society are socialized to accept the attitudes and opinions, and perpetuate the same through interactions with each other. As two-thirds of the population is overweight or obese, heavy individuals are constantly running head first into these attitudes and opinions in every interpersonal relationship in their lives. Family, friends, coworkers, medical and other professionals, acquaintances, and sometimes even perfect strangers somehow feel the need or duty to comment on a heavy person's appearance, and rarely in a positive way.

There has been a small turn in the tide of society's negative attitudes about size, but not necessarily about "fat." More larger sized actresses and entertainers are appearing in television, film, and theatre, but they are still in the drastic minority. A major cosmetic company has chosen an attractive, overweight singer as one of its spokespersons, and a major soap and skin care company has launched a campaign "for real beauty" that involves women of varying sizes and shapes, but does not include obviously obese women. In virtually all instances, the media has expressed issues with accepting these women as role models, but the trend continues nonetheless. The women depicted in all of these roles, however, do appear to meet all of the other criteria of the body ideal in that they are attractive, well spoken, fit, young, healthy, confident, and not obese or morbidly obese. Other than being larger than the ideal would dictate, they comport with society's expectations in all other respects.

The appearance of the heavy person to society affects almost all aspects of the interaction of that person with society. Individuals often do not hold doors open for heavy people. Heavy people do not get hired as often when up against a less qualified candidate who is thin. People are less likely to want to sit by a heavy person if they can avoid it. Heavy individuals are discriminated against in a multitude of ways, almost always based solely on their appearance as heavy people, and not on qualifications, attitude, values, or beliefs. This form of discrimination based on weight is not a protected characteristic under law, except in Michigan and a few select major cities. It is, therefore, a well-accepted and well-tolerated form of societal discrimination that permeates all layers of the socioeconomic system in the dominant culture.

Some subcultures and insular groups, however, do express a more tolerant and accepting view of heavy people. As groups, African Americans, Native Americans, and Hispanic Americans are all more culturally accepting of overweight and obese individuals. Not all members of these minority cultures subscribe to the minority view, as many have been socialized to accept the views of the dominant culture through media, education, politics, religious, and familial messaging. Some subcultures may be more accepting of obesity in one gender over another, or in certain circumstances over another, but these same situational exceptions can also apply in small pockets in the dominant Western culture as well.

Overall, the dominant Western culture has maintained the ideal of a thin body type, and has equated overweight and obese individuals with a subpar status. The constant internalization of this message has created a society filled with individuals suffering not only from the disease of obesity and its attendant problems, but also from the ravages of shame, guilt, embarrassment, humiliation, and damaged self-esteem. Heavy persons should not be judged solely by their appearance. Their qualities, attributes, attitudes, value, and worth are, like their thin counterparts, not evident at first glance. We could all do well to remember that unlike beauty, being fat is *not* only skin deep.

SEE ALSO: Anxiety; Body Dysmorphic Disorder; Body Image; Depression; Fat Acceptance; Obesity and the Media; Quality of Life; Self-Esteem and Obesity; Stereotypes and Obesity; Weight Discrimination.

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Appetite Control

APPETITE IS THE drive to ingest food. This drive is regulated by various hormonal and neural stimuli that originate in the adipose tissue and the endocrine, neurological, and gastrointestinal systems. Various functional centers in the brain then integrate these stimuli and regulate the energy homeostasis through energy expenditure and energy intake. Any imbalance in this homeostatic process results in obesity.

A sedentary lifestyle and the availability of food may cause some people to have difficulty in controlling appetite.

Obesity is the most common nutritional problem in the United States, affecting approximately 33 percent of adults. The United States has the highest rates of obesity in the developed world, and the prevalence is steadily increasing. According to a recent U.S. study, 17 percent of children and adolescents are overweight and 32 percent of adults are obese.

The rise in obesity prevalence is attributed to environmental factors rather than individual behaviors. The current environment promotes a sedentary lifestyle with increased calorie consumption. This, coupled with the drive to eat when food is readily available due to evolutionary pressure has led to increasing rates of obesity.

Obese individuals have a greater risk of developing high blood pressure, high cholesterol and other lipid disorders, Type 2 diabetes, heart disease, strokes, and certain cancers. Fortunately, even a small amount of weight loss (as little as 10 percent) will help lower the risk of developing these diseases.

BODY MASS INDEX

Obesity is defined as a body mass index (BMI) of over 30, whereas morbid obesity is defined as BMI over 40. BMI is a measure of body fat based on both height and weight that applies to men and women. BMI is calculated by dividing the weight by the squared height, and is expressed in Kg/m2.

THE APPETITE CONTROL SYSTEM

In most adults, weight is relatively constant despite huge variations in the daily food intake and the energy expended. This is due to the fact that a complex system consisting of various signals and relevant responses balances the energy intake and expenditure. The signals, in the form of various hormones in the blood and neural stimuli, are integrated in the hypothalamus and the brainstem, which influence the energy balance (i.e., energy intake and expenditure) through various neuropeptides (chemicals secreted by neurons in the brain).

Several regions in the brain are important in processing information of food and relating it to body weight. These regions, with their locations and functions, include the following:

(1) The arcuate nucleus (ARC), situated at the base of hypothalamus, plays a pivotal role as it has access to circulating energy levels. There are two populations of neurons in the ARC that regulate the energy balance: one neuronal circuit inhibits food intake and the other neuronal circuit stimulates food intake.
(2) The nucleus of tractus solitarius, present in the hindbrain, integrates the vagal and other neuronal circuits.
(3) The paraventricular nucleus (PVN) and the dorsomedial hypothalamus (DMH) receive neuronal inputs from the ARC and form important part of the response system.
(4) The ventromedial hypothalamus, the destruction of which results in increased food intake and obesity and thus called the satiety center.
(5) The lateral hypothalamic nucleus, which if damaged, may decrease feeding and lower the body weight, thus called the hunger center.

STIMULATORS OF FOOD INTAKE

Neuropeptide Y (NPY) is one of the most potent and abundant neurotransmitters that stimulates food intake. It has been shown that injection of NPY in the paraventricular nucleus not only causes increased appetite and obesity, but it also reduces the energy expenditure, inhibits the thyroid axis, and suppresses the sympathetic nervous system.

Ghrelin is a gut hormone secreted by the stomach and the small intestine and its levels are regulated by the calorie intake and other nutritional signals. Administration of ghrelin increases food intake and decreases fat utilization. The fall in blood ghrelin levels after bariatric surgery for morbid obesity is thought to be partly responsible for the suppression of appetite and weight loss seen after these operations.

Other stimulators of food intake include dynorphin, melanin-stimulating hormone, growth hormone releasing hormone, and norepinephrine.

INHIBITORS OF FOOD INTAKE

Cholecystokinin, predominantly a gut hormone that is synthesized in the brain as well, has been shown to decrease food intake by inducing feelings of satiety.

Leptin is synthesized by the adipose tissues (fat tissues) and its levels correlate with the adipose tissue mass in the body. Systemic administration of leptin reduces food intake and increases energy expenditure. In addition to its effect on appetite, leptin levels

also affect neuroendocrine function of the gonadal, adrenal, and thyroid axes, and the immune function. A mutation in the gene responsible for synthesizing leptin may result in low or absent circulating leptin levels and cause obesity and hypogonadism.

Another gut hormone, peptide YY 3-36, has been shown to reduce food intake in obese as well as normal-weight individuals. PYY 3-36 levels are low in obese individuals.

Insulin also reduces food intake through various neurohormonal mechanisms; nevertheless, hyperinsulinemia and exogenous insulin are associated with weight gain. Other inhibitors include other gut hormones such as obestatin and enterostatin.

ENERGY EXPENDITURE

Energy expenditure is controlled by the sympathetic system, which is an effector system via the thermogenic tissues (tissues that generate heat) and the peripheral nerves that control food intake. The sympathetic activation results in increased heat production and reduced food intake.

Glucocorticoids also form the second key component of the effector system. They act by increasing the food intake in response to the signals from various hormones and neural circuits. If the glucocorticoids are deficient, the deficiency of leptin or the destruction of ventromedial nucleus does not cause obesity.

BALANCING FOOD INTAKE AND ENERGY EXPENDITURE BY LIFESTYLE MODIFICATIONS

Appetite control and adequate exercise is the most effective way to control weight. Approximately 22–25 kcal/kg is required to maintain 1 kg of bodyweight in a normal individual. Intake of fewer than 1,200 kcal/day would result in weight loss for the majority of average weight individuals. More severe calorie restriction is needed to induce weight loss more quickly. One pound consists of 3500 kcal, so an average deficit of more than 500 kcal/day should result in a weight loss of 0.45 kg (1 lb) per week. However, there is a wide variability as weight loss is influenced by a variety of other factors such as gender, age, and genetics. A healthy and a balanced composition of diet consisting of minimal amounts of proteins, essential fatty acids, vitamins, and minerals according to age-specific recommendations is more effective than drastically altered proportions of nutrients for reducing weight. However, low-fat diets may help in short-term weight loss as well as help in maintaining weight in the long term.

The efficacy of weight loss is assessed by the degree of sustained improvement in the associated risk factors such as diabetes and high blood pressure.

DRUG THERAPY TARGETING APPETITE

Drug therapy for weight loss may be offered to those who have failed to achieve adequate weight loss through diet and exercise alone. It is also useful for obese people with sleep apnea and for whom gastrointestinal bypass surgery is being considered. However, there are some concerns regarding the safety and efficacy of these drugs as well as the observation that most people regain weight after the drugs are stopped. Drug therapy does not cure obesity. It has been observed that weight loss ceases after the maximal effect has been achieved and continuation after that results in weight regain.

The medications currently approved for weight loss in the United States fall into two categories: drugs that decrease weight by decreasing appetite or increasing satiety, known as appetite suppressants, and those that decrease the absorption of nutrients. The Food and Drug Administration (FDA) does not currently approve another category of drugs that increases energy expenditure for the treatment of obesity in the United States.

APPETITE SUPPRESSANTS

Appetite suppressant drugs work by increasing the amounts of certain neurotransmitters such as noradrenaline, dopamine, or serotonin in the brain.

Noradrenergic agents are drugs that increase the amounts of noradrenaline in the brain and include agents such as phentermine, diethylpropion, phendimetrazine, and benzphentamine. All these drugs have been approved by the FDA to be used only for a brief period of about 12 weeks or less for the treatment of obesity. Side effects of these medications include insomnia, dry mouth, constipation, palpitations, euphoria, and hypertension.

Serotonergic agents, which increase the levels of serotonin, have been associated with valvular heart disease and pulmonary hypertension and so have been withdrawn from the market.

Sibutramine is a mixed noradrenergic and serotonergic agent, which increases the levels of both these neurotransmitters in the brain and thus induces a feeling of satiety. In various trials conducted, sibutramine has been shown to induce a weight loss of 5 to 8 percent of the body weight when compared with a placebo (group receiving no medication). Sibutramine has also been shown to maintain the reduced body weight compared to other drugs, with which patients tend to regain the lost weight after continuation for long periods. Side effects of sibutramine include mild increases in blood pressure and pulse, insomnia, dry mouth, constipation, and headache. The associated comorbid conditions such as high cholesterol, uric acid, and blood insulin levels improve with weight loss.

EFFECTS OF DRUGS THAT REDUCE NUTRIENT ABSORPTION ON FOOD INTAKE

Of the drugs that work by reducing nutrient absorption, the only drug that has been approved for weight loss in the United States is Orlistat. Although the primary effects of Orlistat are to reduce the absorption of fats, several unpleasant side effects can result, including diarrhea and anal leakage due to excess fat in the stool. These can be prevented, however, by avoiding consumption of high fat foods, and limiting total fat in the diet to less than 30 percent of total calories.

OTHER DRUGS USED FOR WEIGHT CONTROL

Various herbal medications and dietary supplements are available containing ephedra alkaloids (ma huang), chitosan, chromium picolinate, conjugated linoleic acid, and garcinia cambogia which have claimed to bring about considerable weight loss. However, these have not been tested in proper trials, so there is insufficient evidence about the safety and efficacy of these agents promoting weight loss. Herbal preparations containing ephedra alkaloids and caffeine have shown some efficacy in short-term trials. There have been reports of serious cardiovascular and central nervous system events including cardiac arrhythmia, hypertension, stroke, seizure, heart attack, and even sudden death with preparations containing ephedra alkaloids in high doses. Because of these potentially harmful side effects, the FDA banned products containing Ephedra or Ma Huang in 2005.

A variety of drugs used for treating other disorders are being studied for their supplementary effects of decreasing the weight. These include bupropion, an antidepressant; topiramate, an antiepileptic; and metformin, an antidiabetic agent. Other experimental drugs include rimonabant, and various peptides such as leptin, peptide YY3-36, oxyntomodulin, and melonocortin-4 receptor agonists.

EFFECTS OF BARIATRIC SURGERY

For patients with BMI >40 who have failed diet and exercise with or without drug therapy, and for those with BMI >35 with obesity-related conditions (comorbid conditions) such as hypertension, diabetes, or sleep apnea, bariatric surgery is recommended. Bariatric surgery has reduced monthly medication costs and improved the quality of life by improving or resolving the above-mentioned conditions. While diverse in their techniques and performances, these procedures produce a feeling of satiety even after small amounts of food are ingested.

SEE ALSO: Amphetamines; Appetite Signals; Sibutramine (Meridia); Well-Being.

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Appetite Signals

APPETITE, THE URGE to seek food, is controlled by a complex interplay between endocrine, neural, and metabolic signals from multiple body organs and neurons (nerve cell) and neuropeptides (molecules made by nerve cells) in the brain.

Sight, taste, and smell of food are important signals for regulating food intake. There are receptors in the

mouth that recognize the taste, texture, and temperature of ingested food, and taste plays an important role in the selection of foods. There are several peptides (molecules made of short chains of amino acids) that are important for perception of taste, three of which are: substance P, opioids, and cholecystokinin (CCK). Substance P, found in the nerve fibers of taste buds in the tongue, released on ingestion of foods such as chili peppers, reduces intake of water compared to sweet solutions containing sucrose. Opioid peptides seem to stimulate ingestion of sweet-tasting foods. CCK modulates taste preferences through the gastrointestinal tract.

GUT HORMONES

The next set of appetite signals come from various gastrointestinal organs. CCK is released from the duodenum (first part of the small bowel) in response to ingestion of proteins and fats and helps their digestion by stimulating secretion of digestive enzymes and bile. CCK, upon release, inhibits appetite via two mechanisms.

It acts via the vagus nerve to send signals to areas in the brainstem (lower part of the brain, just above the spinal cord). It also acts locally to constrict the last part of the stomach and slow down stomach emptying, thus causing a sense of fullness. It is also possible that CCK may act directly in the brain. However, the effects of CCK are short lived.

Peptide YY (PYY) is released from the cells in the small bowel and colon rapidly after food ingestion. It has been shown to reduce appetite. It acts via neuropeptide Y receptors; it decreases stomach motility, decreases release of secretions from the stomach and pancreas and finally increases absorption of water and electrolytes in the gut. PYY also acts directly in the brain by inhibiting certain neurons in the hypothalamus (specific region in the brain) that stimulate appetite. Unlike CCK, the effect of PYY lasts for several hours after a meal.

Ghrelin is secreted primarily from oxyntic cells in the stomach and, to a lesser extent, from cells of the bowel. Ghrelin is a strong appetite stimulant and it acts via certain neurons in the hypothalamus and brain stem. Blood levels of ghrelin are regulated by endogenous diurnal rhythm and by food intake. Ghrelin levels are high in the morning and low at night. Ghrelin levels increase during fasting (thus increasing appetite) and fall after food intake (decreasing appetite).

However the fall in ghrelin levels after fat intake is transient and returns to normal after 45 minutes, whereas carbohydrate intake causes a longer-lasting fall in ghrelin levels. Interestingly, ghrelin levels do not fall after ingestion of water, suggesting stomach distension has no role in its release. On the other hand, ingestion of small amounts of alcohol increases release of ghrelin and stimulates appetite. Research has found that obese persons do not show the rapid fall in ghrelin levels after meals, which in turn may lead to continued food intake and obesity.

Oxyntomodulin (OXM) and glucagon like peptide-1 (GLP-1) are released from cells in the small bowel following food intake and subsequently act to decrease food intake. OXM release has a diurnal variation (like ghrelin), with peak levels in the evening and nadir in the early morning (unlike ghrelin).

PANCREATIC HORMONES

Insulin is well known for its role in glucose metabolism. However, it also plays an important role in appetite control. Insulin acts as an anorectic (appetite suppressing) signal within the brain by altering expression of genes that regulate food intake. Blood insulin concentration is higher in persons with greater total body fat stores and levels also rise rapidly after a meal. Insulin easily enters the brain, acting on receptors in the hypothalamus to exert its effect on appetite.

Pancreatic polypeptide (PP) is another peptide secreted from the pancreas and its concentration rises based on caloric intake. PP suppresses appetite via multiple mechanisms: It acts on the vagus nerve, regulates hypothalamic neuropeptides, and decreases ghrelin release from the stomach.

ADIPOSE TISSUE HORMONES

Adipose (fat) tissue was originally thought to be inert and solely for storage of energy. Research has shown that it is actually an active endocrine organ and adipocytes (fat cells) release various hormones. Leptin is the most important hormone and it influences multiple systems in the body. Leptin levels in the blood correlate to whole-body adipose tissue mass, and levels are higher in people with increased adipose tissue. Food restriction suppresses leptin

release and food intake increases its release. Leptin acts as an appetite suppressant and over long term leads to loss of fat mass and body weight. Leptin exerts its effect on appetite by acting on specific receptors in the hypothalamus and brain stem. It activates the nerve cells that produce appetite-suppressing signals and inhibits those that produce appetite-stimulating signals.

Adiponectin and resistin are also produced by adipocytes. However, their role in appetite regulation is not fully understood.

NUTRIENT SIGNALS

Certain nutrient molecules upon entry into the blood inhibit appetite. Glucose (from dietary carbohydrates and liver metabolism), fatty acids (from dietary fat), and ketones (produced in the liver) act on the vagus nerve to inhibit signaling to the brain and thus suppress appetite. The hypothalamus is the major site for appetite regulation in the brain.

SEE ALSO: Adiponectin; Appetite Control; Cannabinoid System; Leptin.

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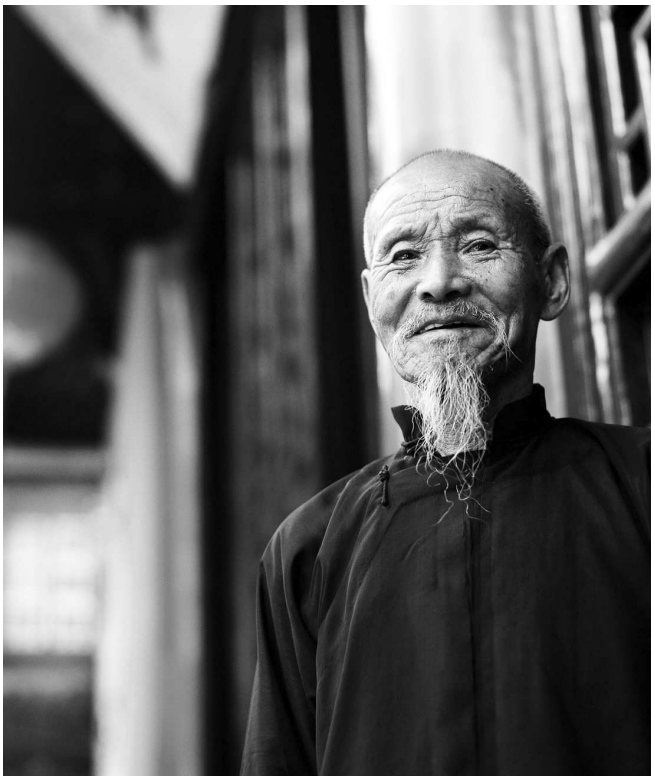
Asia, Central

OBESITY IN CENTRAL Asia has long been a problem affecting small groups of the population. However, in recent decades, it has emerged as a major problem, especially with children, raising serious healthcare issues for the future. This has been particularly true of Iran and also of some of the former Soviet republics in Central Asia.

Although Jalal Talabani, the president of Iraq, has occasionally made jokes about his own obesity, in Iran, the government has conducted many surveys



An old man has a traditional weight of Central Asians, but obesity has become of increasing concern of late.

to try to ascertain the level of the problem, and then implement ways of dealing with it. Some statisticians quote that up to 13 percent of young people in Iran are substantially overweight or obese. To collect the data, a research team from the Department of Pathology at Alzahra University Hospital, Isfahan, studying some of the population of Arak, determined that the waist circumference and the weight-to-height ratio were the easiest statistics to be collected for clinical and epidemiological studies.

Dr. M. Maddah of the Department of Human Nutrition, School of Public Health, Guilan University of Medical Sciences and Health Services, Rasht, has been one of the main researchers in Iran working in the field of obesity. His work shows that contrary to the views of many people in Iran at the time, only 18.2 percent of children who were overweight or obese came from families with low levels of female education, and that there is no extra likelihood of obesity in children of uneducated women. In another study, Dr. Maddah continued to study the level of maternal education against the prevalence of overweight children.

He worked with adolescent girls between the ages 14–17; his study of 1,054 randomly selected schoolgirls in Rasht showed that 21.9 percent of the girls were overweight and 5.3 percent were obese. However, this shows a far higher level of obesity among adolescent girls who have mothers with low educational levels than is the case from mothers who were well educated. This indicates a marked difference between the much higher levels of obesity in adolescent daughters of poorly educated mothers in an urban setting, as opposed to the figures for the entire country. Others doing important work include R. Kelishadi of the Department of Preventive Pediatric Cardiology, Isfahan Cardiovascular Research Center (World Health Organization Collaborating Center) at Isfahan University of Medical Sciences.

To explain these and similar findings in other countries, it has been suggested that children from poor families traditionally ate more fatty food, which was often cheaper, but did not become overweight when they were involved in a more strenuous lifestyle. However, with some of these families moving to towns and cities and adopting a more sedentary lifestyle with little or no change in their diet, overweight became a potential result.

In neighboring Afghanistan, there has been little problem of obesity. Indeed, a study of the population in the country in 2003 showed that as much as 40 percent of the entire population had a high dietary energy deficit, resulting in malnutrition. The lack of access to cars and public transport has often meant people having to travel on foot, which again, has helped with many people not becoming overweight.

In the Soviet Union, much research was devoted to ensuring that the population had good nutrition, and there were health campaigns to try to prevent any increase in obesity. The journal *Voprosy pitaniia* has published much on the field, and since the end of the Soviet Union in 1991, it has continued to help researchers investigating the mounting rates of obesity in Kazakhstan, Kyrgyzstan, Tajikistan, Turkmenistan, and Uzbekistan. However, since 1991, there have been far fewer surveys of health risks, and the health indicators in these countries have fallen.

From the 1920s, there was some malnutrition and occasional food shortages in parts of Soviet Central Asia. Even in the 1970s and the 1980s, the incidence of underweight in Kazakhstan was quite high, as food shortages continued. Leading Kazakh obesity researchers B. A. Salkhanov and R. K. Kadyrova were involved, in 1989, in a study of the etiologic factors of obesity and found that 32.6 percent of those who were obese (above the age of 15) had excessive body mass alimentary disorders; there was low energy expenditure in 26.6 percent; endocrine disorders were noticed in 15.4 percent; and in 6.9 percent there was some acknowledgment of hereditary factors. There were, of course, variations between the urban and the rural population, between males and females, and also between the less mountainous north and west of the country and other areas.

In another survey in the following year, Salkhanov and Kadyrova tried to ascertain the different levels of obesity between people in the north, south, and west of Kazakhstan. Again, restricted to people above the age of 15, they found excessive body mass in 36.1 percent of them, with obesity diagnosed in 23.7 percent of them—the rate increasing with age. Salkhanov and Kadyrova noticed that obesity was also more common among urban women and rural men, with the rate of obesity in the mountainous north being far higher than elsewhere in the country.

Surveys have also been carried out in the neighboring Republic of Kyrgyzstan, which is much poorer than Kazakhstan. In 1988 while Kazakhstan was still a part of the Soviet Union, studies on people living in the capital Frunze showed that the nutritional habits of the indigenous Kirghiz people were very different from Europeans who had settled there. While the former were involved in heavier consumption of proteins and complex carbohydrates, the Russians, Ukrainians, and others included more simple carbohydrates such as sugar, fat, and monounsaturated fatty acids in their diets. However, in spite of these differences, cases of excess body mass were the same for the two population groups, as were cases of coronary heart disease. The study found that much of this could come from equally low rates of physical exercise in both groups of people.

That physical exercise was the main predeterminant for a higher prevalence of obesity was also shown out on a later survey of people in Tien Shan and the Pamirs, which showed clearly that among men aged 40–59, there was a far higher rate of obesity in lowland dwellers, with mountain dwellers being forced by their environment to undertake a more arduous

lifestyle. In 1998, a detailed survey of the population in Kyrgyzstan and Turkmenistan, undertaken by Israeli researchers came up with similar findings.

The most recent survey of obesity in Central Asia was undertaken by V. Mishra, et al. on the epidemiology of obesity in Uzbekistan. Published in 2006, it utilized data from the Uzbekistan Health Examination Survey in 2002 to work out behavioral risk factors. This showed that there was a strong positive correlation between the prevalence of obesity and hypertension in adult men and women in Uzbekistan, although the nature of the relationship differed between that experienced by men and women.

SEE ALSO: Asia, East; Asia, South; Asia, Southeast.

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Asia, East

IN EAST ASIA, there has long been a tradition of obesity among many wealthy officials, even in ancient China. The mythical emperor Shennong (Shen Nung), often known as the Yan Emperor, and who is claimed to have lived in about 3,000 BCE, is said to have asked his people to drink green tea to reduce obesity levels. An Lushan (703–757), the military leader of Turkic-Sogdian origin during the Tang dynasty suffered from obesity which led to diabetes; and Kublai Khan (reigned as Emperor of China 1271–94), was also plagued by obesity and gout during the last 10 years of his reign. This has been attributed to his eating many animal organs. The most well-known overweight emperor was the Wanli Emperor (reigned 1572–1620) near the end of the Ming dynasty. He was ruling when the Jesuit missionary Matteo Ricci visited Beijing, and his reign is seen as representing the decline of the Ming, the grossness of his figure being used by historians to emphasize this.

The Emperor himself had become so large that he apparently was unable to stand without assistance. Some books also credit Yang Guifei (719–756), one of

the "Four Beauties of ancient China" as being obese. However, recent historical research points to the misconception coming from a comparison with the slender Zhao Faiyan (c.32–1 B.C.E.), the wife of Emperor Cheng of Han, possibly suggesting that Yang Guifei was either of normal build or slightly overweight.

The first European artists to visit China often caricatured Chinese officials for being fat, with some of the cartoons associated with the Macartney mission to China in 1793–94 showing clearly obese officials—obesity being used to try to represent slothfulness and corruption. This tradition of caricature continued with Commissioner Lin (Lin Zexu) (1785–1850), the main official who fought opium smuggling, leading to the First Opium War of 1839–42, also being portrayed as obese, with more sympathetic paintings showing him as only a little overweight. The cultural stereotypes continued onto the 20th century, with novelist Ernest Bramah referring to the Chinese gods being old and obese. The New Zealand-born writer Rewi Alley (1897–1987), who lived in China and became a member of the Chinese Communist Party, suffered from obesity during his last years

The main problem facing China in the 21st century has been the massive rise in childhood obesity. One of the main factors of child obesity in China has been that the one-child policy has led to many parents spoiling their children, devoting more money to providing food, often Western-style foods, for them. This has largely come about through overeating, eating fatty food, insufficient exercise, and a sedentary lifestyle, all stemming from a rise in prosperity. A study in 2007 by B. Xie et al. showed that of the 6,863 middle school and high school students measured, 10 percent of the girls and 17 percent of the boys were overweight. The study showed that the higher the level of parental education and the higher the level of family income, the greater the prevalence of the children being either overweight or obese. In another study by H. W. Marsh et al., also carried out in 2007, some 763 Chinese children aged 8–15 also showed high levels of overweight.

For the adult population, the major recent study was conducted by F. Xu, X. M. Yin, and Y. Wang, and investigated areas such as smoking and other lifestyle issues, among people from three urban areas and two rural areas in Nanjing. It found that 36.1 percent of people were overweight, with the prevalence being significantly lower among those still smoking (33 percent), as against nonsmokers (39.9 percent) and ex-smokers (39.2 percent). In fact, the study actually showed that the higher the number of cigarettes smoked daily, the lower the prevalence of obesity. It concluded that there was the possibility of a link between the cessation of smoking and increased body weight.

The major survey of obesity in China has been carried out by the Working Group on Obesity in China, at the Institute of Child and Adolescent Health, Peking Medical University Health Science Center, Beijing. Analyzing figures from 1985 until 2000, they found that the prevalence of overweight and obesity in 2000 for boys aged 7–18 was 11.3 percent and 6.5 percent, respectively, for Beijing, 13.2 percent and 4.9 percent in Shanghai, 9.9 percent and 4.5 percent in large coastal cities, and 5.8 percent and 2.0 percent in smaller coastal cities. The corresponding figures for girls were 8.2 percent and 3.7 percent in Beijing, 7.3 percent and 2.6 percent in Shanghai, 5.9 percent and 2.8 percent in large coastal cities, and 4.9 percent and 1.7 percent in smaller coastal cities—in all cases, lower than boys. The Hong Kong Association for the Study of Obesity is headed by Dr. Gary Tin Choi Ko of the Department of Medicine, AH Nethersole Hospital who is the national representative on the International Association for the Study of Obesity (IASO).

In Taiwan, some 29.3 percent of people aged over 60, in a 1999 study, were found to be overweight, with 6.4 percent being underweight. The Chinese Taipei Association for the Study of Obesity appointed Dr. Hwa-yen Liu, the General Director of the Tianlu Clinic, Hsin-chu City, as the national representative on the IASO. As with many other industrializing countries, there has been a major problem of overweight and obesity in South Korea. The Korean Society for the Study of Obesity was established in 1991, with Professor Hyungjoon Yoo of Seoul as the country's national representative on the IASO. The major study of obesity in Korea, undertaken by D. M. Kim, C. W. Ahn, and S. Y. Nam of the Endocrinology and Metabolism, Yongdong Severance Hospital, Yonsei University, College of Medicine, Seoul, has shown that there has been a dramatic increase in type 2 diabetes in the country, and this has been in spite of the country being geographically isolated, with few migrants and hence a low level of genetic heterogeneity. In North

Korea, where the population is heavily isolated from outside influences, and there have been food shortages, especially of dairy products and meat; thus, there has been no problem with obesity.

Although in Mongolia the diet has traditionally been extremely fatty, the extreme cold climates and the hard physical activity of the Mongolian people has meant that obesity has not been a major problem in the country, although it has had a high rate of prevalence among foreign workers in the country, such as European Russians, many of whom were involved in a more sedentary lifestyle.

In Japan, as with China, traditionally some officials have been obese, and sumo wrestlers are encouraged to be obese, in a sport where size is of great importance. However, Japan has the lowest obesity rate in the industrialized world, with Japanese people having the longest life expectancy rates. The rate of obesity in Japanese women is only 3 percent, with that for men being only slightly higher. This led Naomi Moriyama and her husband William Doyle to write the book *Japanese Women Don't Get Old or Fat* (2005) highlighting the Japanese diet of relatively small helpings and a heavy emphasis on fish. The Japanese Society for the Study of Obesity has Professor Hironobu Yoshimatsu, Department of Anatomy, Biology & Internal Medicine, Faculty of Medicine, Oita University, being the national representative on the IASO.

SEE ALSO: Asia, Central; Asia, South; Asia, Southeast.

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Asia, South

IN HINDU INDIAN mythology, overweight was seen as a sign of inner contentment, with some statues and other representations of several of the gods often showing signs of central obesity. In historical times, early European traders have included accounts of overweight Indian merchants with cartoons often caricaturing obesity. Even allowing for exaggeration, it was clear that there was a level of obesity among some wealthy Indians for several centuries.

Although Bollywood, the Indian film industry, tends to center its films on slim men and women, obesity is increasing in India, as it is around most of the rest of the world. Atal Behari Vajpayee, prime minister of India from 1998 until 2004, has had a problem with being overweight, with medical operations on both

of his knees. Indian prince Kumar Shri Ranjitsinhji (1872–1933) after retiring from cricket, became overweight, and Sri Lankan cricketer turned politician Arjuna Ranatunga (b. 1963) also put on considerable weight after leaving cricket.

However, the modern concern with obesity is not in terms of dealing with a small number of overweight people, but with the massive rise in obesity throughout India, Pakistan, and also Bangladesh, Nepal, and Sri Lanka, especially in the increasing middle class. Much of this has come from the sedentary lifestyle, a lack of exercise and increased access to, and consumption of Western foods. Not only has the increase in obesity created problems of its own, but it has also led to a rise in the levels of diabetes and cardiovascular problems facing many people in South Asia.

As a result, there has been much research on obesity in India, with a website www.obesityindia.com promoting the message "Lose Weight. Gain Health." There is also the All India Association for Advancing Research in Obesity (AIAARO).

As with many other countries in the world, the cause of much of the increasing level of obesity has been the rapid nutritional and lifestyle changes, particularly prevalent in urban areas. As well as obesity, it has also led to a rise in the prevalence of the metabolic syndrome, and this has led to joint studies of obesity and metabolic syndrome, the latter affecting 20 to 25 percent of all urban South Asians.

There is also mounting evidence that obesity in India, as elsewhere in the region, is caused by heredity. Drs. J. Kumar, R. R. Sunkishala, G. Karthikeyan, and S. Sengupta of the Department of Proteomics and Structural Biology, Institute of Genomics and Integrative Biology, New Delhi, carried out a recent study of a genetic variation upstream of insulin-induced gene 2 (INSIG2) (rs7566605) which they believed could be connected with rising levels of obesity. The study was complicated by the lifestyle and diet changes that many Indians have seen during the same period. This led researchers to divide their work between the 1,577 healthy people, and the 610 who were suffering from coronary artery disease. When the two groups were compared, it was found that there was no significant level of association between the polymorphism and body mass index (BMI).

This, as well as other factors, allowed Dr. Kumar and his colleagues to conclude that the variant upstream of INSIG2 could not be shown to be a determinant of BMI in the Indian population. However, there are many other potential genetic links to obesity, and future genome scans may shed insight as to why the overall prevalence of obesity has increased in certain groups.

In Pakistan, the Pakistan Association for the Study of Obesity (PASO) was founded with Dr. Attaur-Rehman being the national representative on the International Association for the Study of Obesity. Recently, in Pakistan, there has been a study by T. H. Jafar, N. Chaturvedi, and G. Pappas analyzing data from 8,972 people aged over the 15 years, working on those with a BMI of 23 kg/m2 or greater as being overweight, and those who have a BMI of 27 kg/m2 or more as being obese. Projected to the entire population, this showed that about a quarter of the people in Pakistan were overweight, with 10.3 percent being obese.

They were also able to show that the factors associated with overweight and obesity were greater age, having a high or very low economic status, and having a high intake of meat. The study revealed that women who were literate and living in an urban area also had an increase prevalence of obesity.

SEE ALSO: Asia Central; Asia East; Asia Southeast.

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Asia, Southeast

UNTIL RECENT TIMES, there has not been a serious problem of obesity in Southeast Asia with the diet generally being relatively good, and also nonsedentary lifestyle ensuring that most people were able to get plenty of exercise. Most early imagery of humans tends to show relatively well-proportioned bodies. However, there are some early representations of human-like figures, such as that of the seated Buddha, where the people are slightly overweight, and certainly not obese, being shown as a sign of contentment.

There is little evidence of obesity in Southeast Asia in medieval times, although it did undoubtedly occur. During the period of French colonial rule in Indochina, the increased prosperity as well as the increase in the sedentary lifestyle for the wealthy did result in a rise in people who were overweight, and even a number who became obese. This led to the tradition that obesity became associated, in the public imagination if nowhere else, with gluttony and also, to some extent, political corruption.

In Cambodia, one of the obese politicians, Trinh Hoanh, for many years a prominent journalist, was one of the men who plotted the overthrow of Prince

Agriculture in Vietnam: The traditional diet and nonsedentary lifestyle of Southeast Asia has prevented obesity from becoming a significant problem. However, with modern trends toward more sedentary jobs and available food, obesity is on the rise in this area.

Norodom Sihanouk in March 1970, was also highlighted as being corrupt. Several other political activists of the 1960s and early 1970s who were also notably overweight included the Communist Poc Doeuskomar, the Royalist secret police chief Kou Roun, prominent member of Parliament Hoeur Lay Inn, Laau Bounpa, Republican Dy Bellon, and Khaou Bun Sun (brother of millionaire developer Khaou Chuly). From the Royal family, the sisters Princess Pinpeang Yukanthor and Princess Pengpas Yukanthor were both overweight, and also two of the wives of Prince Sihanouk, Princess Sisowath Pongsamoni and Princess Mam Monivann, both became overweight during the early 1970s along with Prince Sisowath Monireth, a former prime minister and uncle and adviser to Sihanouk.

With the privations in Cambodia from the mid-1970s, it was not until the tourist boom from the 1990s that resulted in an increase in deep-fried takeout food, an increase in prosperity, and a significant decline in manual work, which led to a significant prevalence of overweight and obese Cambodians not just in the capital Phnom Penh, but throughout increasing parts of the countryside. A survey in 2005 by King et al. of Centre Européen d'Étude du Diabète, Strasbourg, France, showed that there were higher levels of obesity in Kampong Cham compared to Siem Reap, and this also coincided with much higher levels of impaired glucose intolerance and hypertension in Kampong Cham compared to Siem Reap.

In Vietnam, obesity was generally associated with wealthy businessmen and corrupt politicians in southern Vietnam during the French Indochina War (1946–54) and then the subsequent U.S. involvement in Vietnam, with the increasing availability of Western-style food and drink. From the end of the Vietnam War until the tourist boom from the early 1990s, obesity was not a problem, but it has become important with the easy availability of fatty food and the more sedentary lifestyle followed by more and more urban Vietnamese. Much was made of the overweight of former emperor Bao Dai (1913–97) who was slim when he ascended the throne in 1926 and during the 1930s, but put on weight in the 1950s and 1960s.

There has not been a major problem of obesity in Laos, although the introduction of Western-style food and a change in the lifestyle has seen some cases of obesity in the urban populations of Vientiane and Luang Prabang.

With many refugees from Cambodia, Vietnam, and Laos living in the United States, Australia, Canada, and other parts of the Western world, there have been many studies on how these communities have adapted after migration and these have tended to show much higher levels of obesity in the West than in Indochina, causing worrying higher levels of cardiovascular disease and diabetes.

In Thailand and Myanmar (formerly Burma), there are many obese characters in fiction, many being corrupt businessmen such as those in Steven Sills' novel, *Corpus of a Siam Mosquito* (c. 2002); and the portrayal of the fictional slimy businessman Leh Shin as "greasy" and "obese" in Marjorie Douie's *The Pointing Man: A Burmese Mystery* (1920). In Thailand, overweight has recently become noticeable in Bangkok and other cities, among both men and women, as greater access to Western-style food and less exercise has contributed to a higher prevalence of obesity. By contrast in Myanmar, owing to its relative isolation from the Western world and the lack of access to Western food, has had a much lower level of overweight and obesity than its eastern neighbor.

In Malaysia, there has been a rising level of overweight in the population with data studied by the National Health Morbidity Survey showing that 20.7 percent of adults in Malaysia in 2002 were overweight with 5.8 percent being obese. The data also showed that the prevalence of obesity was significantly more for women than men. In addition, an analysis of the data revealed that obesity rates were higher in women from the Indian and Malay communities than those from the Chinese communities, but for men, the highest prevalence of obesity was for Chinese, followed by the Malay and Indian communities. The survey also confirmed earlier studies showing that the energy intake of Indians was often significantly less than those from other ethnic groups, with Malay women having a substantially higher energy intake than other groups.

In comparing urban and nonurban dwellers, urban women consumed less energy than their country counterparts, whereas the data showed the opposite for urban men. Certainly fat intakes were much higher for Chinese and urban people in general. However, with Chinese women ordinarily less active than Chinese men, the cause of obesity in urban Chinese communities seemed to have more to do with intake than lack of exercise. Those working the land had far lower levels of

obesity. There can also be seen to be an increasing level of obesity among the Dayak population in east Malaysia, owing to increased energy intake and a massive reduction in physical exertion, a lot caused by moving the people out of the forests into organized settlements.

The spread of obesity from urban areas to all parts of Malaysia, and the consequent health problems that result, has led the Malaysian government to embark on a program of education to try to reduce the problem, and also sponsoring more research. The Department of Social and Preventive Medicine in the Faculty of Medicine at the University of Malaya in Malaysia has tried to use the promotion of health in the workplace as a major way of reducing obesity by promotion of more physical activity.

In a 2006 study (published in 2007) by Yusof et al. of the Department of Food Science, Faculty of Agrotechnology and Food Science, Kolej Universiti Sains dan Teknologi Malaysia, showed that 10.1 percent of indigenous Orang Asli adults in Lembah Belum, Grik, were either overweight or obese, although 26.7 percent were underweight, possibly suffering from undernutrition. In those who were overweight or obese, the waist circumference and waist-to-hip ratios were high and these figures correlated to high alcohol consumption with smokers tending to have a lower body mass index. Research in Malaysia is now coordinated by the Malaysian Association for the Study of Obesity (MASO) with Dr. Mohd Ismail Noor of the Department of Nutrition & Dietetics, Faculty of Allied Health Sciences, Universiti Kebangsaan Malaysia, Kuala Lumpur, being the national representative on the International Association for the Study of Obesity (IASO).

With the increasing prosperity of Singapore, there has been a sharp rise in the level of obesity with it particularly noticeable among the youth during the 1970s with the introduction of Western-style food and a more sedentary lifestyle. The introduction of compulsory national service for men at around the same time helped initially to create a healthier environment for these young men, and has certainly stemmed some of the rise in obesity. Combined with compulsory sports at schools in Singapore, there have been many measures which have served to delay the onset of obesity with many obese men gaining weight during their 20s rather than as teenagers. Pua, Lim, and Ang of the Physiotherapy Department, Rehabilitative Services, Alexandra Hospital, Singapore, completed an important study of the effects of obesity indices in nonsmoking Singaporean women. Other studies have also shown that there is a higher rate of overweight and obesity in Singaporeans living outside Singapore than in the country itself. Research in Singapore is coordinated by the Singapore Association for the Study of Obesity (SASO) with Dr. Chung Horn Lee of Gleaneagles Medical Centre being the national representative on the IASO. In fiction based in Malaysia and Singapore, obesity is often still connected with corrupt businessmen such as the fictional grossly obese and sleazy businessman Morgan Khoo, addicted to ice cream, who appears in Ian Stewart's novel, *The Seizing of Singapore*.

As with Singapore, the massive prosperity in Brunei from the 1960s has resulted in increased levels of obesity, although for the most part, it has been reflected in an increasingly overweight population, with genuine cases of obesity still extremely rare.

In Indonesia, obesity has become a major problem among the urban Chinese, where prosperity, access to Western-style food and a sedentary lifestyle have all combined to lead to many overweight children, some of whom have become obese adults. In fact, this has led to the establishment of the Indonesian Society for the Study of Obesity (ISSO) with Professor Sidartawan Soegondo of the Division of Endocrinology & Metabolism, Department of Internal Medicine, Faculty of Medicine, University of Indonesia, being the national representative on the IASO.

In the Philippines, there have been higher rates of overweight and obesity in recent years in the cities. In the Philippines, older adults (aged 60 and over) have a prevalence of overweight of 12.2 percent, and a prevalence of underweight of 29.9 percent. Traditionally, novels and short stories associated obesity with business and political corruption. To raise the profile of the problem, the Philippine Association for the Study of Overweight and Obesity (PASOO) has been established with Dr. Rosa Allyn G. Sy of the Medical Plaza, Ortigas Centre, Pasig City, being the national representative on the IASO.

SEE ALSO: Asia, Central; Asia, East; Asia, South.

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Asian Americans

ASIAN AMERICANS ARE defined by the federal government as Americans who trace their ancestry back to the original peoples of the Far East, Southeast Asia, or the Indian subcontinent. According to the U.S. Census Bureau, in 2005, there were about 14.4 million Asian Americans living in the United States, representing about 5 percent of the total population. The largest concentrations of Asian Americans are in California, New York, Hawaii, Texas, and New Jersey. Asian Americans are a diverse group of people with regard to national origin, customs, language, genetic makeup, acculturation, and economic and social status in the United States, and any statistics computed for this group as a whole must be interpreted with caution, particularly in reference to any particular ethnic subgroup. In fact, creation of the ethnic category Asian American is based more on bureaucratic convenience and sociological validity, because it would not be practical to collect and report data on so many small ethnic and tribal groups.

Because Asian Americans constitute a relatively small proportion of the U.S. population, for reporting purposes, data concerning them are often combined with that gathered from another relatively small group, Native Hawaiians and other Pacific Islanders (NH/PI), creating the classification Asian and Pacific Islander (API). Native Hawaiians and other Pacific Islanders are people whose origins lie in the original people of Hawaii, Guam, Samoa, or the other Pacific Islands. According to the U.S. Census Bureau, there were just under 1 million NH/PI people residing in the United States in 2005, representing about 0.1 percent of the U.S. population; about 46 percent were native Hawaiians, with the next largest groups being Samoan and Chamorro/Guamanian. About 280,000 NH/PI people live in Hawaii; other states with substantial NH/PI population include California, Washington, Texas, New York, Florida, and Utah. In 2000, according to the Census Bureau, the six largest ethnic categories within the API group were Chinese, Filipino, Asian Indian, Vietnamese, Korean, and Japanese.

Although the combination of the Asian and NH/PI groups may be a practical necessity and can be defended on some geographical, historical, and cultural grounds, it also creates an even more diverse and heterogenous group of people (including at least 50 identifiable subgroups) for whom summary statistics may be misleading. However, individual API subgroups in the United States usually constitute too small a proportion of any national sample to allow accurate estimation within the subgroups, and even the com-

bined API category often represents so small a percentage of the total that it is not reported separately. This lack of data is a serious problem in studying the health of Asian or API Americans, and scholars must often draw on several sources, including results from national surveys, results from smaller and more narrowly targeted studies, and results from studies collected on Asians living in outside the United States.

Asian Americans are sometimes considered to be a "model minority" who have achieved the highest social and health status of any U.S. ethnic group, exceeding even that of Caucasian Americans. However, this characterization overlooks the diversity of experience within the Asian or API groups. For instance, life expectancy in 2002 was higher among the API group than any other racial/ethnic group in the United States, but showed considerable variability: Female life expectancy among API subgroups ranged from 86.1 for Caucasians and 74.9 for African Americans. To take another example, Asian Americans overall are 20 percent less likely than non-Hispanic Caucasians to die from diabetes, but Native Hawaiians living in Hawaii are over 5.7 times as likely as Caucasians living in Hawaii to die from diabetes.

Data from the 1997 National Health Interview Survey (NHIS), a nationally representative cross-sectional survey of U.S. adults conducted annually, found that 54.3 percent of U.S. adults were overweight (defined as BMI >25, based on self-reported height and weight) and 19.1 percent were obese (BMI >30). However, only 29.8 percent of non-Hispanic API Americans were overweight and only 6 percent were obese, in both cases the smallest percentage among the ethnic groups studied. The same pattern was found in gender-specific breakdowns: 35.2 percent of non-Hispanic API men were overweight and 5.8 percent were obese, compared to national averages of 62.3 percent and 18.8 percent, and 25.2 percent of non-Hispanic API women were overweight and 5.9 percent were obese, compared to national averages of 46.6 percent and 19.3 percent.

Klatsky and Armstrong looked at mean BMI and obesity rates among four categories of Asian Americans (Chinese, Filipino, Japanese, and other Asian) belonging to a particular health maintenance organization (HMO) in northern California. The mean BMI adjusted for age, marital status, education, and alcohol intake was similar among the four groups for both men and women, and was within the normal range (18–25) for all groups, but the percent overweight varied considerably. For women, 12.8 percent of Chinese, 25.5 percent of Filipinos, 18.0 percent Japanese, and 14.6 percent of other Asians were overweight (BMI >25)., while for men, 26.9 percent of Chinese, 41.8 percent of Filipinos, 38.0 percent of Japanese, and 28.9 percent of other Asians were overweight.

Historically, Asian Americans have had the lowest rate of obesity of all American ethnic groups.

Lauderdale and Rathouz combined data from 4 years of the NHIS to have sufficient data to look at obesity rates among adults the largest Asian subgroups (Chinese, Filipino, Asian Indian, Japanese, Korean, and Vietnamese). They found that the percent overweight varied widely among these subgroups, but was in each case less than among the U.S. population as a whole. For instance, they found that the percent overweight among men ranged from 17 percent for Vietnamese to 42 percent for Japanese, versus the 57 percent for the total male population. For women, the percent overweight ranged form 9 percent for Vietnamese and Chinese to 25 percent for Asian Indians, versus a national average of 38 percent. Very few Asian Americans were obese, but the rates were higher for those born in the United States than for the foreign born. Lauderdale and Rathouz also found that among foreign-born Asian Americans, years living in the United States was associated with a higher risk of being overweight, suggesting that the prevalence of overweight may rise among Asian Americans in the future, as more Asian Americans are born in the

United States and as émigrés have lived in the United States for longer periods.

Asian-American adolescents also have lower rates of obesity than other U.S. ethnic groups. The 1998 National Longitudinal Study of Adolescent Health found that 26.5 percent of adolescents in the United States were overweight. Among ethnic groups, Asian Americans had the lowest rate (20.6 percent), versus 30.9 percent for Blacks, 30.4 percent for Latinos, and 24.2 percent for Caucasians. Overweight was much higher for Asian-American males (25.7 percent) than for Asian-American females (15.0 percent). This study also found that similar to Lauderdale and Rathouz's results, overweight among Asian-American adolescents was associated with assimilation to American culture. Asian-American adolescents born abroad were least likely to be overweight (15.6 percent), while among second-generation Asian Americans (born in the United States with at least one foreign-born parent), 30.8 percent were overweight, and among third-generation Asian Americans (born in the United States to parents born in the United States), 34.6 percent were overweight.

This relationship between assimilation and acculturation and decreased health is often seen among immigrants groups. In this case, it is hypothesized that as Asians live longer in the United States, they adopt American eating habits, leading to obesity. These changes include greater consumption of meat and dairy foods, greater fat intake, lower consumption of fruits and vegetables, increased consumption of sweets, greater consumption of convenience foods rather than home-cooked traditional foods, and greater overall caloric intake which is not matched by an increase in physical activity.

SEE ALSO: Asia, Central; Asia, East; Asia, South; Asia, Southeast; Australia and Pacific; Body Image; Ethnic Variations in Obesity-Related Health Risks; Immigration and Obesity.

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Assessment of Obesity and Health Risks

OBESITY IS THE accumulation of excess adipose tissue within the body. The degree of adiposity and the distribution of body fat have been consistently correlated with relative risk of adverse health. Originating in the early 20th century with the Metropolitan Insurance Company weight and height-for-weight tables, assessment techniques now include a variety of methodologies that involve varying levels of accuracy in estimation and measurement of adiposity and body composition. Choosing how to measure adiposity and associated health risk depends on the setting and purpose of the assessment.

MEASUREMENT METHODS

Sophisticated adiposity assessment methodologies are generally used in research rather than large-scale screening. Technologies such as dual energy X-ray absorptiometry (DEXA), computerized tomography (CT) scanning, and air displacement-plethysmography (Bod Pod) all offer a high level of accuracy but are expensive and time consuming. Others methods, including underwater weighting and stable isotope dilution measurement, although safe, are invasive and inconvenient in clinical settings. Bioelectrical impedance analysis and the measurement of body dimensions, such as arm circumference and subscapular skin fold thickness, estimate body fatness and are easy to perform, but lack reliability and are inaccurate with obese persons making them less than optimal for measuring obesity across populations.

BODY MASS INDEX (BMI)

Determined by the indirect calculation of weight in kilograms to the square of height in meters (kg/m2), BMI is a simple and convenient proxy measure for excess adiposity in clinical settings. Many health-related indices, such as mortality risk, heart disease, high blood pressure, and diabetes, have a graded and continuous correlation with BMI. BMI is highly correlated with the precise DEXA method of fat assessment and is thus the universal acceptance as the standard index for the definition of overweight and obesity. Three methods are used to determine BMI: the calculation of the individual's weight divided by the height in meters squared, the use of several Web-based computation sites, or comparison with published reference tables that plot BMI as height and weight intersect. In adults, regardless of sex or age, one set of BMI values is used to assess adiposity and health risk. Based on classification by the National Heart Lung and Blood Institute of the National Institutes of Health (NIH), a BMI under 25 is normal, 25–29.9 is overweight, and a BMI of 30 and above is obese.

BMI is particularly useful for large population surveys and for screening purposes; however, it may not always categorize individual risk well. Although BMI represents the degree of body fat, it does not distinguish between excess weight due to fat mass and nonfat mass such as muscle, edema, or bone. For example body builders have a low percentage of body fat, but their BMI may be in the overweight range because of their large lean muscle mass. Individuals with higher fat mass may be classified as normal BMI despite having a low bone density or muscle mass. Another limitation of BMI is that the relationship to body fatness for those of different gender, age, and ethnicity has not been firmly established. In a family study of 665 African American and Caucasian men and women over the age of 17, the relationship of BMI and fat mass was dependent on gender and age, particularly at lower BMI levels. For women, race was also a factor.

BMI USE IN CHILDREN

For children and adolescents, there is controversy regarding the definition of normal BMI values due to concerns about possible interference with normal growth, self-esteem, and the desire to promote the development of healthy food behaviors and habits. During childhood, BMI changes with growth and development. There are several periods in which sex, growth, and maturation patterns affect muscular gains and account for BMI variation, rather than adiposity. Thus, several authorities have developed child BMI levels including the International Obesity Task Force, the British Child Growth Foundation, and the U.S. Centers for Disease Control and Prevention (CDC). In the United States, the CDC charts are based primarily on data collected during national health examination surveys conducted by the National Center for Health Statistics between 1963 and 1994, and as such, are not skewed by recent increases in the prevalence of overweight children. A BMI equal or greater than CDC's 95th percentile for age and gender has a sensitivity of 49 percent and specificity of 90 percent in identifying children with three or more risk factors for cardiovascular disease.

Excess adiposity in children is known to produce a number of comorbidities in childhood as well as increasing the risk for obesity in adulthood. Thus, the American Academy of Pediatrics and U. S. Preventive Services Task Force (USPSTF) recommend annual screening with the BMI growth charts for children between 24 months and 19 years of age. As in adults, the criterion-referenced BMI scale for children does not quantify body composition variations that occur with race and/or ethnicity.

HEALTH RISKS AND BMI MEASUREMENT

The use of BMI is an accepted method of classifying a patient's risk of the mortality and morbidity due to numerous chronic diseases associated with obesity, including hypertension, hypercholesterolemia, and Type 2 diabetes mellitus (T2DM). These conditions are known to predispose individuals to cardiovascular disease and stroke and may further increase the risk of subsequent mortality. Numerous health risks that are not usually life threatening are also known to be linked to obesity, including sleep apnea, osteoarthritis, gall bladder disease, gastroesophageal reflux disease, respiratory problems, and depression. In women, obesity is associated with higher levels of pregnancy complications, menstrual irregularities, stress incontinence, and hirsutism (hair growth in places where it is usually minimal or nonexistent). Breast, endometrial, prostate, and colon cancer have also been found to occur more frequently in obese individuals. For all complications, the risk is graded beginning at a BMI level of 20 and rises more steeply as BMI increases.

ABDOMINAL FAT

The distribution of body fat has significant health implications. Abdominal, particularly visceral, fat as compared to subcutaneous or retroperitoneal abdominal fat is associated with higher risk of metabolic syndrome and T2DM. Abdominal obesity, as measured by waist circumference (WC), is known to be a better predictor of health risk among those of normal weight, overweight, and obese categories than percent fat measured by DEXA or BMI. Waist circumferences >102 centimeters or >40 inches in men and >88 centimeters or >35 inches in women are associated with increased risks for T2DM, dyslipidemia, hypertension, and cardiovascular disease. CT and MRI can accurately measure the amount of visceral fat, but these methods are too expensive for routine use.

Waist circumference coupled with BMI is a better predictor of health risk than BMI alone, except for individuals with BMIs ≥35. In these individuals, the WC provides no additional predictive power as WC is likely to measure above the recommended cutoff. In those with a BMI between 25–34.9, WC is important for assessing obesity disease risk. Monitoring for changes over time provides a reference to risk increase or improvement. In older persons, who are likely to have more fat in relationship to muscle mass, measurement of WC should be considered even for those of normal weight. In addition to the gender and age differences, ethnic differences are seen in abdominal fat and WC associations with disease risk; in particular, Asian Americans or those of Asian descent living outside Asia have increased risk at levels below the recommended cutoffs.

In children and adolescents, there are no published WC parameters for assessment of health risk. Assessing WC has potential for identifying health risk in this population and standards are likely to be developed. In 9- to 11.5-year-old boys and girls, significant correlations with WC and fasting insulin, high-density lipoprotein cholesterol (HDL-C), total triglyceride (TG), and C-reactive protein (CRP) have been found.

ASSESSING FOR HEALTH COMPLICATIONS OF OBESITY

The routine screening guidelines recommendations for obese persons include annual fasting lipid panel, which includes a total, low-density lipoprotein (LDL), HDL-C, and triglyceride levels along with a blood glucose (fasting or random), and blood pressure measurement. Measurement of blood pressure in obese persons presents a special challenge because of the need for adult large (15 centimeters) or thigh (18 centimeters) sized cuffs. Should a cuff too small be used, the patient may be inappropriately diagnosed with hypertension. The correctly sized bladder cuff will cover 40 to 50 percent of upper arm and fit one to one-and-a-half times around the circumference of the arm. If there is doubt as to the appropriate size of cuff, arm circumference should be determined. Typically, an arm circumference of 23 to 33 centimeters warrants an adult cuff; 33.1 to 40 centimeters warrants an adult large cuff; and 40.1 to 50 centimeters warrants a thigh cuff.

Health conditions associated with obesity typically rise with increasing levels of obesity. Obese persons are at higher risk for numerous conditions such as asthma, nonalcoholic fatty liver disease, gout, hernias, varicose veins, thrombophlebitis, lymphedema, infertility, depression, social stigmatization, and low self-esteem. The extensive list of potential problems suggests that routine laboratory or diagnostic test assessment might not reveal the associated health complications. Instead, a thorough health history and physical exam should be employed so as to identify areas needing further diagnostic testing and treatment.

SEE ALSO: Body Mass Index; DEXA (Dual Energy X-ray Absorptiometry); Doubly Labeled Water; Elevated Cholesterol; Ethnic Variations in Body Fat Storage; Metabolic Disorders and Childhood Obesity; Type 2 Diabetes; Waist Circumference; Waist-to-Hip Ratio.

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Asthma

THE PREVALENCE OF obesity in the United States is rising. This rise has led to an increased awareness of medical conditions associated with obesity. One such association is that of obesity and asthma. It is intriguing to note that the incidence of both disorders has increased simultaneously, doubling in the past 20 years.

Previously, it was accepted that asthma leads to obesity due to the sedentary lifestyles of the patients, but now, there is evidence to the contrary. Studies are now suggesting that obesity leads to asthma or asthma-like symptoms, which is worsened with weight gain.

The fact that there is a relationship between the two is becoming clear. However, what is not understood is the nature of the association: if it is directly causal or if there is a common factor, such as airway inflammation, linking the two. Also, research is being performed to determine if there is a common genetic link between the two disorders. Other comorbidities of obesity, such as gastroesophageal reflux disease (acid reflux) and obstructive sleep apnea (breathing problems during sleep) can also impact the course and severity of asthma.

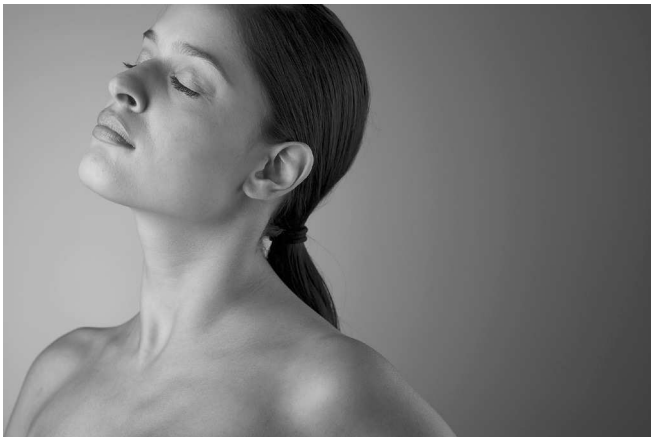
RISK ASSOCIATION

It has been noted that there is a stronger association between obesity and asthma in females than in males, especially in the adolescent population. The risk is increased if there is a family history of allergies and eczema. Obese females have more nonallergic asthma, compared to obese males with asthma.

The risk of both conditions is increased in the inner-city population, affecting minorities, especially the African-American population, and is associated with environmental exposures such as cigarette smoke.

AIRWAY FUNCTION

The hallmark of asthma is reversible airway obstruction. There is obstruction to the airflow, temporarily relieved by medications that dilate the airways, called hyperresponsiveness. In obese patients, a likely explanation for development of asthma is that airway obstruction is due to mechanical compression. These patients have excess weight, which causes them to breathe shallow, resulting in decreased lung volumes on lung function tests. This decreased lung volume results in airway narrowing, causing altered muscle function in the airway, leading to increased response with bronchodilator administration.



The hallmark of asthma is reversible airway obstruction, and studies have shown an improvement in the lung functions with weight loss.

Studies have shown an improvement in the lung functions with weight loss. However, if left unrecognized and untreated, the airway tends to go through a permanent, irreversible remodeling that results in a lack of response to medication administration.

AIRWAY INFLAMMATION

A growing number of animal and human studies are reporting airway inflammation as the common thread between obesity and asthma. It has been found that factors such as leptin and adiponectin, produced by fat cells or adipocytes along with mediators produced elsewhere, contribute to airway inflammation in response to environmental triggers. Other promoters of inflammation are cells such as leukocytes, and mediators such as cytokines and TNF alpha.

SUMMARY

Although longitudinal studies are needed to confirm the link, obesity has been suggested to be a strong but modifiable risk factor for asthma. A focus on prevention of obesity would be a great investment from the perspective of public health. Also, because weight loss has been related to an improvement in the symptoms and lung functions, it is evident that efforts need to be made for early diagnosis and treatment of obesity.

SEE ALSO: National Heart, Lung, and Blood Institute.

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Atherosclerosis

ATHEROSCLEROSIS IS A term used to describe the accumulation of fats and lipids (molecules consisting mainly of cholesterol) that ultimately results in the formation of plaque within arteries in the body. This process can eventually result in the disruption or complete obstruction of blood flow through these arteries, causing a heart attack or stoke.

Atherosclerosis is a well-recognized process that results directly in coronary and cerebrovascular disease and, thus, cardiovascular death. It remains the major cause of death and disability in developed nations. It is estimated that one-half of all cardiovascular deaths each year are secondary to coronary artery disease and roughly 20 percent more are due to stroke. Additionally, atherosclerotic changes in blood vessels begin to occur in the first two decades of life.

As the human life span has continued to increase, so too has the prevalence of atherosclerotic disease and the comorbidities and mortality with which it is associated. Certain risk factors have been identified that put people at a higher risk for developing such changes. These include smoking (specifically tobacco products), high blood pressure, high cholesterol, diabetes, a family history of cardiac disease, and the person's age. In addition, obesity is also a risk factor for atheroclerosis and coronary artery disease. Recent research studies have suggested that obesity may accelerate the progression of coronary atherocleroris. Often atherosclerotic change due to these risks is not discovered until an acute event occurs.

Atherosclerosis typically begins with the formation of small lesions, referred to as fatty streaks, or atheromas, in the internal wall of arteries (blood vessels in the body that deliver oxygenated blood from the heart to the body). Accumulation of such fatty streaks results in a loss of mobility in the arterial wall. Further accumulation of fatty streaks results in the migration of inflammatory mediators to these sites. Invasion of the deeper layers of the arterial wall then occurs with eventual formation of atherosclerotic plaques. These plaques will eventually calcify over time and contribute to coronary artery remodeling. Such changes result in a decreased intraluminal (inner space of artery) size within arteries and a decrease in oxygenated blood flow through these blood vessels. Such changes can ultimately result in heart attack and stroke.

Atherosclerosis has not, in the past, been monitored via imaging studies such as X-ray, computerized tomography (CT) scan, or magnetic resonance imaging (MRI). Testing has usually been limited to blood samples that are used to measure a person's lipid panel. Such a panel measures the total cholesterol that is circulating, low-density lipoprotein (also known as "bad" cholesterol), high-density lipoprotein (also known as "good cholesterol"), and triglycerides (cells that store and transport fat molecules). While measures of cholesterol are useful in monitoring values on an annual basis, they have never provided an adequate internal view of plaque accumulation. With the advent of stress testing in the 1960s, physicians gained a tool for monitoring physical and electrocardiographic changes during exercise that may indicate significant atherosclerotic disease in the coronary arteries. During this same period, cardiac catheterization was perfected as a technique for gaining visual access to the anatomy of the coronary arteries.

Today, cardiac catheterization is performed on a routine basis to visualize coronary and peripheral arteries and to diagnose atherosclerotic plaques and calcifications that may, or are, posing a threat to the patient. Cardiac catheterization has also become a mode of therapy for atherosclerotic disease, as interventional cardiologists are able to insert balloons and stents to correct defects in the coronary and peripheral arteries caused by atherosclerotic plaques.

More recent progress includes the development of radiologic testing, including CT scan and MRI. These allow physicians to visualize atherosclerotic disease in coronary arteries without having to enter the patient's body. Such modalities are aimed to increase patient

safety and allow greater convenience in diagnosis of such a common disease process.

In treating atherosclerosis, there has been a major shift in paradigm over the last 20 years. Specifically, an effort has been made to treat the effects of high cholesterol levels by utilizing methods other than medications. Coronary artery and carotid artery stenting have aided in restoring blood flow in arteries that have significant atherosclerotic change. However, these act as measures to correct changes that have already occurred. Measures are now being taken to prevent these changes from initially occurring, thus, preventing future events from taking place. In addition, physicians are now targeting changes in the lifestyle of their patients. As stated before, most of the major risk factors for atherosclerosis and cardiovascular disease are modifiable. Of these factors, smoking is clearly the most important. Smoking accounts for 400,000 deaths annually, with 35 to 40 percent of all smoking-related deaths due to ischemic heart disease. More importantly, those who smoke 20 or more cigarettes per day have a two- to threefold increase in the incidence of heart disease.

In addition to reduction of risk factors, treatment of atherosclerosis is aimed mainly at reducing the causative agent of this disease. Cholesterol is known to aid in production of atherosclerotic plaque. Additionally, studies have shown that higher cholesterol levels correlate with increased amounts of atherosclerotic and cardiovascular disease. Reduction of cholesterol levels decreases the amount of atherosclerotic plaque and cardiovascular disease based on these same studies. With these findings in mind, multiple classes of medications have been created that aid in the reduction of serum cholesterol levels. Statins (HMG-CoA reductase inhibitors), fibrates, nicotinic acid, bile acid sequestrants, and others have been identified as having the ability to decrease cholesterol levels and, in turn, decrease the risk of coronary artery and cardiovascular disease. Of these, the statins have shown to be the most beneficial in providing substantial reductions in low-density lipoprotein (LDL) (bad cholesterol) levels. While statins and other medications aid in improving high-density lipoprotein (HDL) levels, exercise remains an integral tool in raising intrinsic HDL levels. Concern over HDL levels stems from the knowledge that increased HDL levels act to offset atherosclerotic change.

The future for treatment of atherosclerosis remains making prevention a primary concern and, doing so at earlier ages. This consists of advocating physical activity, proper diet, as well as medication when necessary to prevent formation of atherosclerotic plaques.

SEE ALSO: Atherosclerosis in Children; Computerized Tomography; High Density Lipoproteins; Low Density Lipoproteins.

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Atherosclerosis in Children

ATHEROSCLEROTIC CARDIOVASCULAR DISEASE is the leading cause of death in the adult population of Western societies. It is a process of progressive thickening and hardening of the walls of the arteries as a result of fat deposits on their inner lining. However, the pathological processes and risk factors associated with its development begin during childhood. Environmental and genetic factors such as diet, obesity, exercise, and certain inherited dyslipidemias influence the progression of such lesions.

Atherosclerosis risk factors are present in children and adolescents. Traditional risk factors include obesity, hypertension, and diabetes mellitus. However, endothelial dysfunction is the key event in atherosclerosis with a significant number of young people having advanced coronary artery plaques. As the number of cardiovascular risk factors increases, so does the severity of asymptomatic coronary and aortic atherosclerosis in young people. Also, the degree of atherosclerotic changes in children and young adults can be correlated with the presence of the same risk factors seen in adults. This trend is associated with increasing blood pressure and the occurrence of Type 2 diabetes mellitus in young individuals. Together, these trends

may result in increased cardiovascular morbidity and mortality as these overweight pediatric patients become obese adults.

However, only the first stage of atherosclerosis, the highly reversible fatty streak, occurs in childhood. The more harmful second stage of atherosclerosis, the atheromatous plaque, does not appear until after puberty in boys and much later in girls. The association of lipoprotein risk factors with intermediate type atherosclerotic lesions becomes evident in subjects in their late teens, whereas associations with raised lesions become evident in subjects greater than 25 years of age, which is consistent with a transitional role of intermediate lesion in the formation of advanced plaques. Early atherosclerosis is accelerated by lipoprotein risk factors. Thus, long-range prevention of atherosclerosis should begin in childhood and should include measures to control hyperlipidemia.

Systemic inflammation is present in children and adults with obesity. Inflammation associated with obesity appears to be central to the development of insulin resistance and atherosclerosis and may be important in the pathogenesis of other comorbid conditions. However, inflammation associated with obesity declines after weight loss and with exercise. High levels of blood homocysteine may be an important factor of the obesity-induced early arterial atherosclerosis during childhood. High levels of blood homocysteine may be an important factor of obesity-induced early arterial atherosclerosis during childhood. Other atherosclerosis risk factors include (Lp(a), Apolipoproteins A1 and B, and certain markers of fibrinolysis, the process where fibrin clots are broken down.

Rates of overweight and obesity in both adults and children have risen sharply during the past 20 years. Although the reasons for this escalation in obesity are not fully determined, sedentary lifestyle and dietary changes in combination with genetic predisposition are probably involved. Obesity beginning in childhood often precedes the hyperinsulinemic state. Metabolic syndrome is a constellation of disorders that produces a high risk of atherosclerosis. Children of patients with metabolic syndrome have higher values of the serum markers of inflammation, which may be associated with increased risk for development of cardiovascular disease. Current knowledge suggests that better control of blood glucose is likely to lead to improved long-term microvascular and macrovascular outcomes. Thus, the best approach to prevention of future cardiovascular disease in these young patients is early recognition and aggressive therapy.

There are currently a variety of noninvasive tests to assess the structural and functional properties suggestive of "early atherosclerosis." It should be noted that many of the major risk factors can be modified through diet, body mass control, exercise, and pharmacological intervention, if necessary. Also, it is reasonable to suggest that lifestyle modification and weight control in childhood could reduce the risk of developing the insulin-resistance syndrome, Type 2 diabetes mellitus, and cardiovascular disease.

SEE ALSO: Atherosclerosis.

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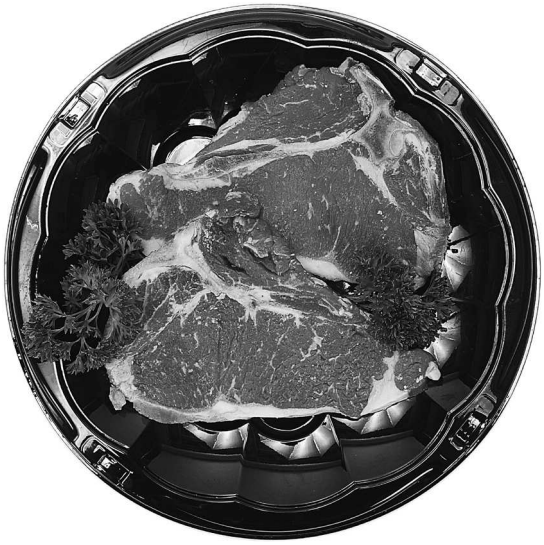
Atkins Diet



Seeming to go against conventional thought, the Atkins Diet stresses protein and fat and avoidance of carbohydrates.

THE ATKINS DIET, also known as the Atkins Nutritional Approach, is a high-fat, high-protein, low-carbohydrate weight-loss diet developed by Dr. Robert Atkins in the 1960s. Atkins published a series of books explaining this type of diet, beginning with *Dr. Atkin's Diet Revolution* in 1972, and in 1989 founded the company Atkins Nutritionals, which produces food products and other merchandise intended to support people following the Atkins Diet. The Atkins Diet has waxed and waned in popularity over the years and has spawned a number of imitators. Although severely criticized by many nutritionists since its inception, few clinical studies were conducted until recently, and most have shown that the diet does have at least short-term efficacy in promoting weight loss and improvements in health without endangering the health of normally healthy individuals. Exercise is encouraged within the Atkins program (for instance, on the Atkins Nutritional Web site) although it is not strictly speaking a requirement for following the diet.

The Atkins Diet runs counter to most mainstream nutritional advice, including that of the Food Pyramid popularized by the U.S. Department of Agriculture, which recommends that most of a person's diet should be based on complex carbohydrates and fruits and vegetables. Instead, the Atkins Diet is based on the belief that consumption of carbohydrates, particularly refined carbohydrates, is the primary cause of obesity in the Western world, and also that saturated fat is less harmful to health than is commonly supposed. The early stages of the Atkins Diet are intended to put a person into ketosis, a state in which their body is sufficiently deprived of carbohydrates that it will break down fatty acids into ketone bodies that are burned for energy (rather than glucose, which is the typical energy source for humans). Prolonged states of ketosis can damage the liver and kidneys, and therefore, standard medical advice for persons following strict low-carbohydrate diets intended to produce ketosis are recommended to do so for only short periods of time (such as 14 days).

The Atkins Diet program is divided into four phases. The first phase, induction, lasts two weeks and is intended to put the individual into a state of ketosis. This is achieved by restricting carbohydrate consumption to 20 grams per day, while allowing consumption of large quantities of meat, eggs, cheese, butter, and oils, as well as low-carbohydrate vegetables. Rapid weight loss is typical during this period (1–2 pounds per day), although some of this loss may be attributed to dehydration. The second phase, ongoing weight loss, allows an increase of 5 grams of carbohydrates per week while still keeping the level low enough that weight loss continues. Ongoing weight loss continues until a weight close to the optimal (i.e., within 10 pounds) is achieved. Pre-maintenance is the third phase; in this period, carbohydrate consumption is further increased and may raise the body out of ketosis. Lifetime maintenance is the final phase in which individuals follow a healthy diet and monitor their weight, so they may return to one of the earlier phases if they start to regain previously lost weight.

Although the Atkins Diet has been around since the 1960s, scientific research into its safety and efficacy stems mostly from 2000 and later. The more recent research may have been prompted by the

extreme popularity of the diet in 2003 and 2004, at which point, some estimate that as many as 9 percent of adults in North America were following it or a similar diet. The influence of the low-carbohydrate diets during this time was blamed for sharply declining sales of carbohydrate foods such as potatoes and pasta, and spurred the creation of low-carbohydrate products and the advertising of naturally low-carbohydrate products (such as salad dressing) as such. The Atkins Diet suffered a drop in popularity after Robert Atkins died in 2003. Although his death was caused by a head injury suffered in a fall, the release of medical records revealing that he had a history of congestive heart failure tended to discredit the diet.

The Atkins Diet has been criticized on many fronts. One is that it is expensive: *Forbes* magazine calculated in 2005 that following the Atkins Diet would cost about 80 percent more than the typical food expenses for an American. Many medical professionals also point out that the rapid early weight loss is typical of many diets that begin with very low caloric intake, and is due largely to dehydration rather than actual loss of fat. Many health concerns have been raised about the Atkins Diet, including the fact that it may cause diarrhea, osteoporosis, bad breath (due to ketosis), fatigue, and increased risk of heart disease. Others critics believe that the basic logic behind the diet is flawed, because the daily diet of people in much of the world consists largely of carbohydrates (e.g., rice or noodles) and yet obesity is not endemic throughout East Asia, for instance. Finally, many nutritionists believed that individuals following the Atkins Diet would suffer increases in blood cholesterol and triglycerides, both associated with heart disease.

Because scientific examination of the Atkins Diet and similar high-protein, high-fat, low-carbohydrate diets is relatively recent, little can be said about its long-term effects on health or how well weight loss is maintained over a longer period of time. However, numerous studies have shown the Atkins Diet to be effective for weight loss and health improvement in the short term. For instance, Foster and colleagues conducted a randomized controlled trial for 1 year testing a high-protein, high-fat, high-carbohydrate diet against a conventional high-carbohydrate, low-fat diet. They found that individuals on the low-carbohydrate diet lost significantly more weight at 6 months (averaging about 7 percent versus 3 percent of body weight), but that the differences were not significant at 1 year (4.4 percent versus 2.5 percent). Both groups showed improved health, in terms of significantly reduced glucose response and decreased diastolic blood pressure, and the low-carbohydrate groups showed greater increase in high-density lipoprotein ("good") cholesterol and decrease in triglyceride concentrations. Samaha and colleagues found that severely obese individuals (mean body mass index 43) lost more weight over 6 months on a low-carbohydrate versus a low-fat diet, and also had greater decreases in triglyceride levels and improvement in insulin sensitivity.

SEE ALSO: Fat Intake; Food Guide Pyramid; High-Protein Diets.

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Australia and Pacific

IN AUSTRALIA, PRIOR to European settlement in 1788, because of their diet and lifestyle, there appears to have been no prevalence of obesity among the native Aboriginal population. The diet of the Aboriginals involved relatively low consumption of meat, the vast majority of it being from lean animals. It was not until

the second half of the 20th century that the increasingly sedentary lifestyle, fatty foods, and the dairy products resulted in a significant level of obesity in the Aboriginal community, leading to problems with diabetes and other medical conditions.

The arrival of European convicts and settlers from 1788 resulted in the raising of beef and dairy cattle and pigs. A number of the early settlers were able to consume fatty meat and dairy products, as well as drink beer, at much higher rates than they had done in Europe. Although contemporary photographs do not show him as such, the early governor of New South Wales Philip Gidley King (1758–1808) was overweight. By the 19th century, obesity was starting to become a problem, mainly for some of the squatters and other large landowners, publicans, and politicians. The most well-known case of obesity was that of the "Tichborne claimant"—the butcher from Wagga Wagga, New South Wales, who, in 1866 went to England to claim that he was the missing Sir Roger Tichborne who was believed to have been lost in a shipwreck off the coast of Chile 12 years earlier. Although overweight before the subsequent court cases began, he quickly gained 27 stones (378 pounds), and became popular with caricaturists. Politician Alfred Thomas Clarke (1845–88) came under attack in 1874 after his paper, the *Williamstown Advertiser*, claimed that Queen Victoria was "an obese, not overburdened with brain, old woman."

Caricaturists also had a field day in the land boom of the late 1880s and early 1890s in Melbourne, with some of the famous "land boomers" and politicians associated with them, such as Tommy Bent (1838–1909), being overweight. Other famous people of this period who suffered from obesity included the boxer Albert Griffiths (1871–1927); the professional cyclist Robert Adam Spears (1893–1950); sporting commentator Cyril Joseph Angles (1906–62); journalist Warren Edwin Denning (1906–75); newspaper editor and journalist Clarence Sydney McNulty (1903–64); farmer and soldier George James Rankin (1887–57); and the prominent surgeon Sir William D. C. Williams (1856–1919). Roman Catholic archbishop Thomas Joseph Carr (1839–1917) was also well known for his obesity, and politician Sir George Reid (1845–1918), who was premier of New South Wales from 1894 until 1899, and prime minister from 1904 until 1905, became obese toward the end of his political career. When army officer and banker Murray John Moten (1899–1953) applied to serve in World War II, he was rejected due to his being overweight.

In recent years, numbers of prominent politicians have been known for being overweight, often becoming subject to attacks by cartoonists and satirists. These include Queensland minister of main roads and racing, Russ Hinze (1919–91); former federal minister of defense Robert Ray (b. 1947); former minister for immigration Amanda Vanstone (b. 1952); backbench Tasmanian politician Dick Adams (b. 1951); and minister for employment and workplace relations, Joe Hockey (b. 1965).

Although there have been many prominent well-known obese and overweight people, the major worry for healthcare professionals is a massive rise in the general level of obesity throughout Australia. As a result, there has been increased attention given to the problem by the Australasian Society for the Study of Obesity, and especially by Professor Louise Baur of the Children's Hospital at Westmead, New South Wales, who is the national representative on the International Association for the Study of Obesity. The 10th International Congress on Obesity was held on September 3–8, 2006, in Sydney.

With the rising level of obesity, in March 2007, Professor Mike Daube of Health Policy, Curtin University, Western Australia, proposed a 15 percent tax on "junk food," whereby taxes are placed on goods which are high in fat, sugar, and salt. This would be a way of reducing obesity by reducing the amount of people eating fatty food, and at the same time provide money to fund a health campaign to alert people to the dangers of obesity. Gavin Mooney of Health Economics, also of Curtin University, urged that the taxation system should hit the "junk food" operators harder through higher taxation, but his idea, and that of Mike Daub were both rejected by the Australian Federal Health Minister Tony Abbott. Another recent study, published in May 2007 in the *Medical Journal of Australia* by Dr. Lisa Gibson of the Telethon Institute for Child Health Research in Perth, investigated the higher prevalence of overweight among single mothers. It concluded that this could have come about from bad dietary habits as a result possibly eating larger quantities of energy-rich and also fatty food. Having considerably more restricted time, some single mothers have had to resort to poor diets and have had less opportunity for regular recreational exercise. There have also been suggestions

that obesity should be raised to a disease status, with a section of the health budget diverted to deal with prevention of obesity which has resulted in the deaths, each year, of between 12,000 to 15,000 people from obesity-related complaints.

In New Zealand, nutritionist and medical researcher Muriel Emma Bell (1898–1974) was very interested in the connection between diets and heart disease. In the 1950s, she had tried unsuccessfully to persuade some insurance companies to collect data on obesity. Although they did not feel it was necessary to collect the data, Bell persisted with her research and studied Maori and Pacific Islander diets. Although the Maori and Pacific Islander communities in New Zealand have a high prevalence of obesity, owing to a change in their diet and lifestyle, many other New Zealanders of European ancestry have also suffered from obesity. David Lange (1942–2005), from a German family, was a leading trade unionist and prime minister of New Zealand from 1984 until 1989. He suffered from obesity for much of his life, and even attributed his caustic wit as having to come from the need to defend himself as a teenager. In 1982, he weighed 165 kilograms and embarked on surgery to staple his stomach to force him to lose weight. This succeeded to some extent, but by 2002, he was suffering from a variety of medical problems, including complications from diabetes, dying from renal failure and blood disease in 2005. In New Zealand, the national representative of the Australasian Society for the Study of Obesity on the International Association for the Study of Obesity is Elaine Rush of the Faculty of Health and Environmental Science, Auckland University of Technology.

In the Pacific Islands, there has been a major problem of obesity owing to inappropriate diet and the change in the lifestyle of the Pacific Islanders. One of the worst examples of obesity is the Kingdom of Tonga where some 92 percent of the total population is officially overweight, with a massive rise in recent years in heart disease, diabetes, and complications resulting from high blood pressure. The rates of diabetes run at 20 percent, seven times that in the United Kingdom, yet there are only five diabetes specialists in the entire country. Until the 1960s, the government had a policy of allocating 8 acres to each person, and half of the country's population worked on the land until the mid-1980s. With popular foods including lamb flaps imported from New Zealand, and a high consumption of spam and corned beef, this has exacerbated the problem.

In Fiji there has also been a period of rapid social change, resulting in many cases of obesity among the native Fijian population, and also among some of the Indian community. Traditional Fijian diet involved the consumption of starchy food such as breadfruit and taro, as well as fish and coconut milk, the latter of which has 25 percent saturated fat. A 1998 survey found that 84 percent of the ethnic Fijian women were overweight, of whom a significant number were obese, compared to only 60 percent in 1989. The situation during that period was even more marked in Nauru, which went through a period of immense prosperity in the 1980s. Similar problems of obesity have also affected Samoa, Guam, and the other Pacific islands.

SEE ALSO: Asia, Central; Asia, East; Asia, Southeast.

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Autonomic Nervous System

THE AUTONOMIC NERVOUS system (ANS) is a complex system of nerves and ganglia primarily involved in the control of involuntary activity. It consists of two arms: the sympathetic nervous system (SNS) and the parasympathetic nervous system. Although it has been suggested that alterations in ANS tone may play a role in the development of obesity, it remains unclear whether defects in ANS tone are a primary etiological factor in the development of obesity or, rather, whether ANS imbalance is a consequence of the obese state.

MEASUREMENT OF ANS FUNCTION

Traditionally, whole-body sympathetic activity was most commonly assessed by measuring plasma, platelet, or 24-hour urinary levels of catecholamines and their metabolites. More recent techniques include isotope dilution-derived measurements of noradrenaline release to plasma, and microneurography, to measure muscle sympathetic nerve activity (MSNA). ANS function can also be assessed by spectral analysis of heart rate variability (HRV), which provides information regarding both sympathetic and parasympathetic (vagal) nerve activity.

IS ANS ACTIVITY ALTERED IN OBESITY?

Although it has been controversial regarding whether "obesity" (i.e., excess adipose tissue deposition) is associated with altered sympathetic neural activity, more recent studies demonstrate that a state of basal/resting sympathetic overdrive characterizes obese individuals. Some of this discrepancy between studies is likely to be due to differences in the methods used to assess ANS function in the literature. Furthermore, failure to measure and account for the demonstrated effects of central adiposity, insulin resistance, elevated fatty acid levels, and glucose intolerance on ANS tone may also explain some of this inconsistency.

In spite of increased basal sympathetic neural activity, recent data suggest that obese individuals may exhibit reduced "responsiveness" to sympathetic stimuli. For example, it has been shown that although the basal (unstimulated) LF/HF ratio (an HRV measure of sympathovagal balance) is higher in obese versus lean individuals, insulin (delivered during a hyperinsulinemic-euglycemic clamp) fails to increase the LF/HF ratio in obese subjects. Furthermore, a recent study examined the effect of physiological (meal-induced) hyperinsulinemia on the LF/HF ratio in lean and obese subjects using high- and low-carbohydrate meals. The main findings were: (1) SNS tone increased postprandially; (2) the postprandial increase in SNS tone was significantly greater following the high-carbohydrate meal compared to the low-carbohydrate meal; and (3) the carbohydrate (insulin)-induced increase in SNS tone following the meal was blunted in obese subjects.

However, it cannot be concluded from these reports that reduced sympathetic responsiveness contributes to the development of obesity, as altered sympathetic tone may be a consequence of the obese state and, perhaps, its associated comorbidities, particularly insulin resistance. To answer this question, it is imperative that ANS activity is examined prior to the onset of these disorders in pre-obese individuals. Although reports in Pima Indians provide some evidence that reduced sympathetic activity may predict weight gain, it has been argued that the unique genetic makeup of the Pima Indians makes it difficult to extrapolate these findings to other populations.

POTENTIAL MECHANISMS LINKING OBESITY WITH ALTERATIONS IN ANS FUNCTION

If confirmed, is it biologically plausible that alterations in autonomic neural activity (i.e., reduced sympathetic responsiveness) could contribute to the accumulation of body fat in insulin resistance? One possible mechanism is that hyperinsulinemia is an initiating factor leading to activation of basal SNS tone in insulin-resistant individuals. As there is some evidence to suggest that persistently elevated SNS activity leads to adrenoreceptor downregulation, this may, over time, lead to a blunted sympathetic response to stimuli such as insulin, which may have important implications in the postprandial state. Indeed, the SNS plays an important role in the regulation of energy balance, particularly in relation to suppressing appetite following a meal. There is also evidence from both animal and human studies that the SNS is a key regulator of adaptive thermogenesis. Furthermore, studies using pharmacological blockade of the SNS suggest that oxidation of fat in the postprandial state is particularly dependent on SNS activation.

Alternatively, activation of SNS tone may be a consequence of the obese state, rather than a primary

etiological factor. Indeed, the accumulation of body fat is known to be associated with a proportional release of leptin from adipose tissue. Furthermore, there is some evidence to support the notion that leptin activates central sympathetic outflow within the hypothalamus. Therefore, coupled with hyperinsulinemia, leptin may be a potential mechanism by which the SNS is activated in obese individuals.

SEE ALSO: Central Nervous System; Peripheral Nervous System; Pima Indians.

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Axokine

AMONG THE SECOND-GENERATION biologic factors being investigated as antiobesity agents, ciliary neurotrophic factor (CNTF) appeared among the more promising. As its name suggests, CNTF was originally identified as a hormone-like factor responsible for growth and differentiation of neuronal tissue. Unlike hormones such as leptin, CNTF is not secreted peripherally and is only found naturally inside developing neuronal cells. Despite this, specific receptors for CNTF are present on multiple nonneuronal cells, including skeletal muscle tissue.

Activation of the CNTF receptor on skeletal muscle can prevent age-related degeneration. This property was investigated when CNTF was given to patients with amyotrophic lateral sclerosis (ALS), a chronic degenerative muscle condition. Serendipitously, patients treated with CNTF experienced significant involuntary weight loss, which led to its investigation as a possible antiobesity agent.

The mechanism by which CNTF might promote weight loss involves its effects on both the central nervous system and peripheral tissues. In the brain, CNTF receptors are found in the hypothalamus, the region responsible for appetite control. Stimulation of those receptors in leptin-resistant mice leads to weight loss and decreased food intake. On a molecular level, this may be due to the inhibition of an intracellular signaling enzyme (AMP-kinase) similarly inhibited by activation of central leptin receptors.

CNTF may also act peripherally to prevent obesity. It has been demonstrated that CNTF administration leads to decreased lipid accumulation in the skeletal muscle of animals fed a high-fat diet. Increased deposition of lipid in skeletal muscle may contribute to peripheral insulin resistance, which in turn can cause weight gain. CNTF receptor activation may also interact with the PPARγ pathway, in turn promoting increased insulin sensitivity.

In humans, CNTF (brand name Axokine) has been tested as a potential antiobesity agent. After 1 year of treatment, individuals given CNTF experienced an average weight loss of 6 pounds compared to 2 pounds in patients given a placebo. Unfortunately, 70 percent of patients treated with CNTF developed blocking antibodies that limited its continued effectiveness.

Current research strategies to address the shortcomings of CNTF as an antiobesity agent are focusing on the peripheral effects of CNTF. By designing novel molecules that specifically target the peripheral CNTF receptors, CNTF may yet prove to be an effective new tool in the fight against obesity.

SEE ALSO: Leptin; Leptin Supplements; PPAR (Peroxisome Proliferator-Activated Receptor).

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Back Pain

THE BACK IS used in nearly every activity people do in the normal course of a day. On top of its extensive use, the human back has a characteristic letter "S" shape that creates some concavities, notably near the neck and lower back. Due to the involved nature of the back and its S shape, humans are very prone to developing back pain.

The human back has many bones and muscles that work in unison. Pain can occur if something disrupts this function. In many such cases, identifiable causes can be found using modern medical diagnostic methods. Instances of herniated discs, cancer, sciatica, spinal infection, and direct trauma are typically identifiable and treatable. In these cases, established medical procedures that can treat the pain exist.

Despite the medical advances available to identifiable causes of back pain, modern medical science cannot concretely identify the mechanisms responsible for all back pain. Nearly 80 percent of Americans will experience lower back pain and 30 percent will have chronic lower back pain. The interconnectedness of the muscles of the back, the physical motions made capable by the human skeleton, and the broad surface area that the back muscles occupy all contribute to a very dynamic system that can be easily disturbed. The human back tends to be very much affected by the daily course of human activity and the overall well-being of the person. Many areas of human concern are incorporated into the healthy function of the back. Paying attention to the natural biomechanics of the human skeleton can greatly reduce the frequency of a person's back pain. Additionally, stress tends to accumulate in the back and can cause a distortion in muscular tension. Because of the back's rather broad surface area, a tightly contracted muscle, also known as a knot, can have a greater effect than in more compact muscles.

Being overweight is at the top of the list of causes of back pain. Being even a few pounds overweight can place undue stress on the back, particularly the lower back. The back supports the weight of the upper torso and can be subtly shifted out of alignment by the forward pull of excess belly fat. Over time, such a condition can create muscular imbalances, strains, and uncomfortable spinal adjustments. Being overweight does not only include weight due to excess body fat; too much muscle mass can also have ill effects. Due to the S shape, the vertebral columns are not held together in a completely vertical column. Having extra weight, fat, or muscle, will place strain on the angled sections and can cause pain.

Back pain can be caused by many different factors. While many are readily identifiable, some are not. For those cases that do not seem to have a clearly identifiable mechanism of pain, the lifestyle of the person,

Though anyone can have back pain, obesity can cause severe back pain due to the strain of the additional weight on the body.

including body weight, is generally suggested as a potential source of the discomfort.

SEE ALSO: Body Mass Index; Central Obesity.

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Bardet-Biedl Syndrome

BARDET-BIEDL SYNDROME (BBS) is a pleiotropic autosomal recessive disorder. This clinically complex genetically heterogeneous disorder is associated with mostly monogenic causes. BBS is a rare condition and is said to occur in 1 in every 150,000 persons born in the population.

The syndrome is defined by a range of primary and secondary features including retinitis pigmentosa, retinopathy, obesity (early-onset morbid obesity is associated with abnormalities chromosome 15), hypogonadism, renal dysfunction, postaxial polydactyly (said to be linked to abnormalities in chromosome 3), and mental retardation. Other manifestations include diabetes mellitus, heart disease, hepatic fibrosis, neurological abnormalities, reproductive abnormalities, endocrine disturbances, short stature, myopia, strabismus, and cataracts. Individuals may also have partial or complete anosmia due to loss of function of the BBS protein which affects the olfactory epithelium.

Patients often experience a late onset of symptoms and, thus, the diagnosis of BBS is usually made during childhood. Obesity appears around age 2–3 years in the patient; however, as this is not diagnostic of the disorder, the actual disorder is diagnosed at approximately 9 years of age when visual problems first appear.

BBS may be occur due to mutations in certain genes (BBS1, BBS2, BBS3 (ARL6), BBS4, BBS5, BBS6 (MKKS), BBS7, BBS8 (TTC8), BBS9, BBS10, BBS11, BBS12). All these gene codes for proteins are associated with the centrioles part of the cytoskeletal system and play a role in cilia function. BBS is possibly caused by a defect of the basal body of ciliated cells. BBS1 is caused by mutation in a gene that maps to chromosome 11q13. The BBS1 is associated with the greatest occurrence of the disorder followed by BBS2. BBS2 is caused by mutation in a gene that maps to chromosome 16q21. BBS3 is caused by mutation in the ADP-ribosylation factor (ARF)-like-6 gene (ARL6) on chromosome 3p13-p12. BBS4 is caused by mutation in a gene that maps to chromosome 15q22.3-q23. BBS5 is caused by mutation in a gene that maps to chromosome 2q31. BBS6 is caused by mutation in MKKS, located on 20p12. BBS7 is caused by mutation in a gene that maps to chromosome 4q27. BBS8 is caused by a mutation in a tetratricopeptide repeat protein, TTC8. The BBS8 gene mutations encodes a protein

with a prokaryotic domain, pilF; this is involved in pilus formation and twitching mobility. BBS9 is caused by mutation in the parathyroid hormone-responsive gene B1 (PTHB1). BBS10 is caused by mutation in the C12ORF58 gene. Mutation in the tripartite motif-containing protein-32 gene (TRIM32) BBS11. Mutation in the C4ORF24 gene causes BBS12. Reports suggest that the BBS genes may predispose male heterozygote patients to obesity.

There has been long-standing uncertainty as to the relationship between the Laurence-Moon syndrome and BBS. Patients suffering from these disorders report similar symptoms and, thus, it was previously difficult to distinguish between the two disorders. However, it is generally reported that the patients of Laurence and Moon have a distinct disorder with paraplegia and without polydactyly and obesity.

The prognosis for BBS varies according to the severity of symptoms, visual prognosis is poor and complication of obesity and renal impairment may increase morbidity and mortality rates.

SEE ALSO: Genetic Mapping of Obesity-Related Genes.

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Bariatric Surgery in Children

BARIATRIC SURGERY IS a treatment option for carefully selected severely obese youth for whom all other medical, dietary, and behavioral approaches have been unsuccessful. The history, indications, risks, and benefits of bariatric surgery in children are reviewed briefly, ending with controversies and future directions. The terms *children*, *adolescents*, and *youth* are used interchangeably.

The history of surgical treatment of extreme obesity in children and adolescents dates back to the 1970s, with initial reports appearing in the literature on small series of youth treated with surgery for weight loss, including some with Prader-Willi Syndrome. In 1991, a National Institutes of Health (NIH) consensus development conference recommended that gastric restrictive or bypass procedures could be considered for well-informed and motivated patients with acceptable operative risks, but indicated that children and adolescents had not been sufficiently studied to allow a recommendation for surgery. Since 1991, Roux-en-Y gastric bypass has become the most commonly performed procedure in the United States. However, the published literature on bariatric surgery in children remains scant, and no definitive, evidence-based guidelines exist for youth.

Patient selection begins with a detailed, multidisciplinary assessment, including medical, surgical, nutritional, and psychiatric expertise, ideally conducted in a specialized and experienced program setting. Due to the permanent impact of gastric bypass on gastrointestinal anatomy and lifestyle, presurgical evaluation and education in children is extensive. Eligible patients have attained or nearly attained physiologic maturity, failed more conservative approaches to treatment, and must meet additional eligibility criteria including weight greater than 100 pounds overweight. The family environment plays an important role in postoperative support and compliance, and both parents and child are involved in the process of obtaining informed consent.

The risks and benefits of gastric bypass in children appear to include the same outcomes seen in adults. Rarely seen, but early complications include bowel obstruction, bleeding, blood clots, and persistent nausea. Late problems can include gallstones, hernias, vitamin and iron deficiencies, inadequate weight loss, or weight regain. Potential benefits include improvements in long-term weight control, reduced medical comorbidities such as diabetes, and enhanced psychosocial functioning. Unfortunately, no reliable predictors of outcome have been identified. Lifelong medical and nutritional surveillance is mandatory.

The role of bariatric surgery in the treatment of severe obesity in children remains controversial. Some express concerns over the potential adverse long-term impact on growth and development, and

question a child's ability to consent to a procedure that has lifelong effects; others focus on the serious health and psychosocial consequences of childhood obesity that may persist into adulthood in the absence of surgical intervention. Future directions indicate the need for long-term prospective outcome analyses of various bariatric surgery procedures in younger patients, including the role of behavioral weight control as an adjunct for effective longitudinal management of this patient population.

SEE ALSO: American Society for Bariatric Surgery; Behavioral Treatment of Child Obesity; Child Obesity Programs; Medical Interventions for Children; Morbid Obesity in Children.

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Bariatric Surgery in Women

THE AMERICAN SOCIETY for Bariatric Surgery estimates that more than 200,000 bariatric, or weight-loss surgery, procedures were performed in the United States in 2007. The typical bariatric surgery patient is a woman, age 25–45, overweight since childhood, having faced a lifetime of discrimination, with multiple attempts at weight loss via diet and exercise. They average 2 years of research into surgery before making the decision that surgery is the choice for them.

Why would a woman turn to surgery for weight loss? It works when all other therapies fail. Once someone has reached the morbidly obese level, diet and exercise alone is only successful in less than 5 percent of the population. Bariatric surgery has a 50 percent success rate at 16 years after surgery. A meta-analysis presented in 2004 that represented 136 studies and over 22,000 patients demonstrated that after bariatric surgery, 86 percent of diabetes was resolved or improved, 70 percent of hyperlipidemia was improved, 78 percent of hypertension was resolved or improved, 83 percent of obstructive sleep apnea was resolved or improved, and perhaps most impressively, there was a 400-percent reduction in the incidence of cancer. The weight loss achieved and maintained after bariatric surgery results in decreased healthcare costs, decreased rates of disability, and a decreased risk of death.

In 1991, the National Institute of Health released a consensus statement on the state of bariatric surgery. The indications for surgery from the NIH are as follows: BMI >40 or BMI 35–39 with comorbid condition; documented ineffective weight-loss attempts; and treatment with a multidisciplinary team approach. At the time of the statement, gastric restrictive (vertical banded gastroplasty), or bypass procedures, were deemed appropriate for motivated patients. They went on to state that surgery is the only approach providing consistent, permanent weight loss for morbidly obese patients. The NIH is expected to release an updated consensus statement in 2008 which will include laparoscopic adjustable gastric banding.

In 2006, the Centers for Medicare and Medicaid Services released a National Coverage Determination for bariatric surgery. The criteria for surgery listed are as follows: BMI >35 with comorbid condition; documented ineffective weight-loss attempts; treatment at a Center of Excellence (American College of Surgeons level 1A–1B and/or SRC full status designation). Specific procedures covered include: Roux-en-Y gastric bypass, Laproscopic Gastric Bypass, and Laparoscopic Bilopancreatic Diversion w/wo duodenal switch. This excludes vertical banded gastroplasty (VBG). Surgery is for the treatment of comorbidities and medical complications related to obesity, and is not considered cosmetic surgery.

Surgical options include restrictive procedures that limit the size of the stomach without changing the basic anatomy (primarily, this is laparoscopic adjustable gastric banding); malabsorptive procedures that change the basic stomach anatomy and bypass a portion of the small intestines (an example is the

biliopancreatic diversion with or without duodenal switch; the most frequently performed procedure in the United States and considered to be the gold-standard procedure is a combination of restrictive and malabsorptive procedures, the gastric bypass (Roux-en-Y) open or laparoscopic.

Women should be advised that with malabsorptive procedures, the absorption, and therefore the effectiveness of oral contraceptives, will be diminished. They will need to either change to another form of contraception or utilize a second form in conjunction with oral contraceptives. As weight loss occurs and the amount of hormones secreted by the peripheral adipose tissue decreases, women who were previously thought to be infertile may now find themselves ovulating regularly and now fertile. It is recommended that pregnancy be avoided for the first 12 to 18 months after any bariatric surgery procedure to allow for optimal weight loss prior to conception and to allow the woman to reach a stage where she can take in a more normal amount of nutrition.

Bariatric surgery is frequently referred to as a tool. Just like any tool, it must be used correctly to optimize the results. A multidisciplinary team at a Center of Excellence will have the mechanisms in place to educate patients properly prior to surgery.

SEE ALSO: Body Mass Index; Lap Band; Morbid Obesity in Women.

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Barker Hypothesis

THE BARKER HYPOTHESIS is named for David Barker, M.D., Ph.D., currently Professor of Clinical Epidemiology at the University of Southampton in the United Kingdom and Professor in the Department of Medicine at Oregon Health and Science University. Also known as the fetal origins hypothesis, the Barker hypothesis posits that common chronic diseases such as coronary heart disease, hypertension, and type 2 diabetes mellitus (T2DM) occur at greater rates among adults who were born at lower birth weight and remained thin during the first year or two of life. While the pathogenesis of these chronic diseases is a complex combination between genetic and environmental factors throughout the life span, Barker points to the fetal nutritional environment as a source of fetal "programming" for a greater predisposition to these conditions. He and his colleagues first discovered the link between low birth weight and higher rates of coronary heart disease in 1989, and the *British Medical Journal* had coined the term "Barker hypothesis" by 1992.

Much of Barker's own work has come from a handful of longitudinal studies; his findings have been corroborated by others. He has collaborated extensively with Dr. Johan Erikkson at the National Public Health Institute in Finland, which has followed 20,000 men and women born in Helsinki between 1924 and 1944, all of whom have detailed information on birth weight and childhood growth. He has also followed a cohort born between 1920 and 1930 in Hertfordshire, England, whose birth weights and weights at one year of age were recorded. With Dr. Dan Lackland at the Medical University of South Carolina, he has used a cohort of Medicaid beneficiaries with birth weight records dating from 1950. Additionally, his collaborative efforts in India, Holland, and China have supported his previous findings.

Barker's initial work reported an association between birth weight and coronary heart disease (CHD). In a study of 8,760 children from the Helsinki study, Barker and colleagues found that those who developed CHD as adults were small at birth and during the first 2 years of life but gained weight more rapidly than the other children between ages two and 11. Compared to children with a birth weight of >3,500 grams and a body mass index (BMI) of 17

at two years old, those born at <3,000 grams who achieved a BMI of only <16 by age two had a hazard ratio for adult CHD of 1.9 (1.3–2.9), adjusted for sex, adult occupational status, and household income. This study also showed that systolic blood pressure, fasting plasma glucose and insulin, and fasting serum triglycerides decreased with increasing birth weight and BMI at 2 years old. Correlation between CHD and low birth weight and/or low weight in infancy has been replicated in other studies in Europe, India, and the United States. In some of these studies, this effect has proven to be independent of potential confounding variables such as smoking, physical activity, and occupational status.

Increased blood pressure is a known risk factor for CHD and may be one of the mechanisms through which low birth weight increases the risk of CHD. In a systematic review of eight studies, Huxley and colleagues showed a modest negative association between birth weight and adult systolic blood pressure (SBP), which increased 2 mmHg for every 1-kg decrease in birth weight. The mechanism of this association likely involves the in utero development of the kidneys. Autopsy studies have shown that adults with hypertension have fewer glomeruli in their kidneys, the microscopic units of blood filtration. Animal studies show that individuals born at lower birth weights have fewer glomeruli, the result of poorer nutrient delivery to the fetal kidneys. As a result, the kidneys have a smaller filtration surface area and thus face increased hydrostatic pressure, leading to renal scarring and hypertension. Barker's South Carolina study showed that the blood pressure of those individuals with low birth weights was more difficult to lower despite the use of multiple medications. The Helsinki study showed, however, that the effect of birth weight on blood pressure was confined only to those participants being treated for hypertension, not in normotensive individuals. In people without hypertension, neuroendocrine mechanisms are able to keep blood pressure within a normal range of values. Barker has proposed that the effect of birth weight on blood pressure is hidden by these counter-regulatory mechanisms in normotensive individuals and that it is evident only when these mechanisms cannot overcome the underlying deficit in glomeruli.

T2DM is also a significant risk factor for CHD and may be another mechanism through which low birth weight is associated with CHD. Several studies have confirmed that the risk of T2DM and glucose intolerance, a prediabetic condition, increases with decreasing birth weight. Among 468 English men aged 64, the BMI-adjusted odds ratio of glucose intolerance increased with each 1-pound decrease in birth weight, such that a baby born at ≤5.5 pounds had an odds ratio of 6.6 for glucose intolerance by age 64 compared to a baby born at >9.5 pounds. These results have been repeated in other studies and in women, and a similar trend has been seen between weight at one year of age and the risk of glucose intolerance in adulthood. These associations are independent of social class, smoking, BMI, and alcohol consumption. In fact, Henrik Sørensen showed in a Danish cohort that BMI in young adulthood actually increases with birth weight, suggesting that factors other than absolute adiposity influence the risk of T2DM. In addition to absolute values of weight at birth and 1 year of age, the tempo of growth in childhood is correlated with the risk of T2DM in adulthood. The adiposity rebound is the normal decrease in BMI from birth until early childhood, followed by increase starting between ages 3 and 8 years. The Helsinki study showed that earlier adiposity rebound is associated with low birth weight and thinness at one year of age and that the earlier this rebound occurs, the greater the risk for T2DM in adulthood.

The link between low birth weight and altered glucose metabolism may be due to the developmental plasticity of the human fetus, an ability to adapt to environmental conditions during fetal development. Poorer maternal nutrient delivery to the fetus in utero may indicate an outside environment in which food is scarce, and the fetus adapts to this "forecast" by remaining smaller and by switching on genes that allow it to use energy more efficiently, resulting in the so-called "thrifty phenotype." Lower birth weight may be a crude indicator that such an adaptation has occurred, but the adaptation likely occurs at the molecular level. This "thrifty phenotype" uses mechanisms such as insulin resistance to make the most of scarce food energy and ensure sufficient glucose delivery to the brain. However, when food supplies are adequate in childhood or adulthood, these adaptations predispose these individuals to glucose intolerance and T2DM. Less nutrient delivery in utero also results in the development of less muscle mass and

consequently a greater body fat percentage in adulthood that contributes to insulin resistance and glucose intolerance.

On the macroscopic environmental level, there may also be an interaction between the effects of birth weight and adult socioeconomic status. Lower socioeconomic status is a well-known risk factor for CHD, but in the Helsinki study, the negative association between household income and CHD was evident only in those men who were thin at birth. Citing studies that show persistently elevated cortisol levels in adults born at low birth weights, Barker has suggested that those who are small at birth are less well equipped to handle the stress of low socioeconomic status and are therefore more susceptible to its deleterious health effects. Such interaction is probably just one of many phenotype-environment interactions affecting the risk of chronic disease.

The largest criticism of the Barker hypothesis is the possibility that low birth weight is associated with other environmental risk factors for CHD, hypertension, and T2DM and is therefore not by itself a true independent risk factor for these conditions. Such potential confounders may include risk factors such as smoking, lack of exercise, high caloric intake, high fat consumption, and low socioeconomic status. Most of the studies supporting the Barker hypothesis adjusted their analyses to control for some of these environmental risk factors, but the possibility exists that residual confounding remains or that important risk factors were not measured and accounted for. Still, a sizable body of epidemiologic evidence and a growing body of basic science research supports the Barker hypothesis, which has considerable public health import. If Barker's ideas hold true, the prevention of the chronic diseases that kill millions of people yearly may require intervention before birth.

SEE ALSO: Infant Growth Rate; Low Birth Rate; Thrifty Gene Hypothesis..

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Behavioral Treatment of Childhood Obesity

BEHAVIORAL TREATMENT PROGRAMS for overweight youth uniformly incorporate three main elements: diet, exercise, and behavior therapy. The behavioral component of treatment emphasizes positive behavioral changes, as well as the application of operant conditioning and social learning strategies for making lifestyle changes. The body of work by Leonard Epstein and colleagues, as well as others has demonstrated the success and importance of including behavior therapy in the treatment of childhood obesity. In addition, parental involvement in this therapy has been shown to improve treatment effects and outcomes. Such therapy assumes that helping parents and children develop a set of skills that will help them correct maladaptive eating and exercise habits will allow them to lose excess weight and to maintain healthy weight. Behavioral treatment for obesity also promotes cognitive approaches and assumes that cognitive change will lead to behavior change, although the efficacy of cognitive components for child overweight is limited.

LEARNING THEORY

Learning theory about eating postulates that eating is prompted by environmental cues and generally reinforced by positive or a lack of negative responses (either internal or external) to such behavior. Behavioral treatment aims to identify cues that trigger inappropriate eating and sedentary behaviors and helps individuals modify such cues (i.e., environment

control) or to have different responses to eating behaviors (e.g., parents praising healthy food choices or lower consumption). Behavioral treatment is goal-oriented, with goals often specified in precise and measurable terms. In addition to clear goals, the treatment also provides children and parents means by which to achieve such goals via the development of various skills. Last, success is often attained in small, approximated steps toward the optimal behavior, based on operant conditioning learning strategy of shaping. Rewards for achieving the smaller steps thus provide multiple successful experiences along the pathway to major change. Given the role of family and particularly parents in determining the environment in which children live and in providing reinforcement for performed behaviors, family involvement is seen as essential in the behavioral treatment of childhood obesity, particularly for elementary school-aged children.

BEHAVIORAL SKILLS

Behavioral treatment provides children and parents with a set of skills to help modify dietary and physical activity habits. These skills include self-monitoring, goal setting, stimulus control, and behavioral substitution.

Self-monitoring is the tracking of specified behaviors, usually through written or electronic documentation (e.g., writing down everything eaten in a day). Behavior therapy often first educates children and parents on how to use measurement tools, food labels, and calorie-counting guides to help them implement self-monitoring of dietary intake appropriately. Such monitoring has been shown to be important in impacting weight loss among adults and children. The ideal form of self-monitoring and what (e.g., calories, food groups) and how often to self-monitor for optimal success is not yet known, but many programs encourage daily monitoring of food and beverage consumption. Self-monitoring can also monitor physical activity performance and sedentary behaviors.

As mentioned above, behavioral treatment encourages participants to set specific and clear goals. Goals should be measurable so that participants can easily determine success and/or failure. Vague goals such as "trying harder" or "improving" are discouraged and replaced by specific goals such as "I will exercise 10 minutes on Mondays, Wednesdays, and Fridays" or "I will eat at home on Tuesdays and Fridays." Goals are also required to be time limited with a usual time frame of a week to accomplish determined goals. Given the specificity of goals, goals should be realistic and small in scope to encourage the likelihood of success and thus reinforcement. Within behavioral treatment for childhood overweight, goal attainment is often accompanied by rewards, preferably those that encourage healthy eating and more physical activity. Parents are often asked to aid in the goal setting and achievement process via creating home environments that encourage the desired behaviors (i.e., via modeling and/or providing positive cues and rewards for desired behaviors).

Based on operant conditioning principles that reinforcing stimuli or cues will increase behavior performance, identification of stimuli that prompt maladaptive eating and inactivity behaviors is an important aspect of behavioral treatment of obesity. Once such cues or stimuli are identified, avoidance or reprogramming of such cues can be performed. In addition, cues that reinforce healthy behaviors should be identified and increased in the environment so as to promote desired behaviors. For example, eating stimuli can be restricted or controlled by having patients avoid exposure to high calorie, problem foods (i.e., by avoiding fast-food, all-you-can-eat venues, convenience stores, etc.). This often involves parents making substantial changes in the home food environment such as no processed food or carbonated beverages, thus positively impacting the eating behavior of the entire family. Physical activity stimuli can be promoted by encouraging children to go to recreational sites.

BEHAVIORAL SUBSTITUTION

In cases where the stimulus associated with a maladaptive behavior is internal (emotional), behavioral substitution may be a more feasible option to modify an individual's lifestyle. In behavioral substitution, the prior maladaptive behavior is substituted by another behavior in response to a given cue. For example, one might substitute walking the dog for eating in an attempt to reprogram the common stress-eating (cue-behavior) cycle.

COGNITIVE APPROACHES

In addition to the standard behavioral techniques described above, cognitive approaches to behavioral

change are also used to implement change. Cognitive approaches assume that cognitions precede and influence behavior, that cognitions can be modified, and that cognitive change can produce behavioral change. Such approaches, in the case of children, obviously must take into account the cognitive development of the client. The cognitive skills most commonly taught to achieve behavioral change are problem solving and cognitive restructuring. The efficacy of adding cognitive skills training to the core behavioral treatment package has not been shown for childhood overweight.

PROBLEM SOLVING

The problem-solving process involves identification of the problem, identifying the predecessors of the problem, generation of potential solutions, and selection of the best or most feasible solution via a pro and con or cost–benefit analysis. After a solution is selected, the solution is then put into practice and its outcomes evaluated. If the outcome is not the desired effect, then the process begins again.

COGNITIVE RESTRUCTURING

In cognitive restructuring, negative thoughts that may hinder achievement of behavioral goals are identified and reprogrammed. Usually such negative thoughts are distortions of reality, and cognitive restructuring aims to replace the distorted thoughts with more realistic or rational cognitions.

Ultimately, the cognitive and behavioral skills taught to patients in behavioral treatment plans for childhood obesity are meant to not only induce weight loss and maintenance but also to prevent weight regain and maladaptive behavior relapse. While short-term success is often attainable by behavioral treatment programs for obesity, long-term success remains difficult to achieve, although some evidence suggests that children have better long-term outcomes than adults in such programs. Research in adults suggests that long-term treatment is required for long-term success but retention rates of such programs are often poor. Among children, evidence to date suggests that family involvement is essential to weight management success, at least among younger children. In fact, some evidence suggests children under 7 may not even need to attend treatment, with efficacy derived from their parents' participation in a child-targeted program. Further research is required regarding whether nonpersonal contacts such as via Internet, mail, or phone might provide additional means or methods for providing effective long-term weight management.

SEE ALSO: Childhood Onset Eating Disorders; Child Obesity Programs; Children and Diets.

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Beverage Choices in Children

BEVERAGE CHOICE, particularly over-consumption of sugar-sweetened beverages such as sodas and juices, might be associated with obesity in children. An increase in the amount of high-calorie drinks consumed by children at home and school is strikingly high. Many parents and adults are not informed about the importance of encouraging healthy beverage choices in children. Proper nutritional uptake is dependent greatly on the beverages a child is given or chooses to drink. Further, the media negatively impacts children who are attempting to make healthy choices about the beverages they drink.

High-calorie beverage choices for children include soft drinks and several juices. An increase in the consumption rates in the past few decades is evident through statistics that show that children (ages 6–11) are now drinking double the amount of soft drinks and nearly triple the amount of high-calorie juices compared to children in 1978.

Further, both children and teens drink about 20.6 gallons of soft drinks per year. In less than two decades, that amount nearly tripled to approximately 64.5 gallons of high-calorie drinks. During around the same time, reports show that childhood and adolescent obesity significantly increased. This strongly suggests that the beverages impact the overall increasing obesity rate.

CANS OF SODA

Calories in popular beverages for children are very high. For example, anywhere from 124 to 189 calories can be found in a 12-ounce serving of soda. On average, a male adolescent consumes three 12-ounce cans, and females drink approximately two 12-ounce cans a day. Energy booster drinks are just as unhealthy as soda. On average, energy drinks have about 160 calories and are packed with sugar. Artificially flavored as well as drinks with added sugars are full in calorie value but empty in nutritional value. On the other hand, 2-percent low-fat milk is approximately 183 calories, but provides vitamin A, folate, vitamin B12, calcium, magnesium, and other important nutrients for the body.

With respect to obesity, the drinks consumed by children are just as important as the foods they eat. Although children choose which beverages they prefer, parents and guardians can play an important role in helping them decide. About 21 percent of American children (aged 12–19) are obese. This number is rapidly increasing. Not only parents, but also pediatricians, caregivers, and nutritionists are making an effort to teach children to make healthy beverage choices.

To break the high-calorie fruit drink and soft drink habit, Cornell University's Nutrition Information Center has set forth recommendations for adults that can potentially address the ongoing obesity crisis in children. For example, healthy beverages can be made more flavorful and accessible to kids. By adding natural flavoring, the beverage remains healthy and delicious. Caregivers can also begin substituting healthier drinks for nutritionally deprived liquids during children's meals. By offering flavored 2-percent low-fat milk, children can gradually become introduced to healthier options.

BAD DRINKING HABITS

Many health issues are characterized by improper beverage consumption. Because of undersupplied calcium-intake levels, osteoporosis and tooth decay can result from bad drinking habits. The lack of water consumption in children has become an increasingly important problem. With insufficient drinking water outlets in shopping malls, entertainment locations, and other public areas, the problem of dehydration is growing.

During intense physical exercise, unhealthy fruit drinks and soft drinks are often served when water is the best option. Healthy drinking habits are formed at a young age; therefore, caregivers are recommended to introduce water as the primary thirst quencher during physical activity.

A high priority should be set on cultivating an environment that allows children to make healthy decisions. Advertisement of unhealthy food and beverages handicaps children from making the right choices. Big beverage industries such as Coca-Cola, Pepsi, and other carbonated drink companies are constantly placing new advertisements make their drinks more appealing. However, the marketing of unhealthy drink options serves as a direct threat to the health of American children.

Overall, the rate of high-calorie/low-nutritional beverage consumption has increased, which has aided the obesity epidemic. This problem is multifaceted, but can be helped in various ways, ranging from lowering the calorie counts in drinks, to educating parents about healthy diet habits, to disregarding media influences, or just by making sure children make healthier choices.

SEE ALSO: Changing Children's Food Habits; Childhood Onset Eating Disorders; Children and Diets.

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Binge Eating

APPROXIMATELY ONE IN three individuals seeking weight-loss treatment reports some form of binge eating and one in five meets criteria for binge eating disorder (BED). With about 65 percent of Americans overweight or obese, binge eating has also increased in prevalence and is receiving more attention. Binge eating is characterized by eating, in a discrete period of time, an amount of food that is definitely larger than most individuals would eat under similar circumstances, accompanied by a sense of lack of control. BED is defined as recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviors characteristic of bulimia nervosa, such as vomiting or using laxatives. This entry presents a brief overview of the diagnosis, prevalence, potential causes, consequences, and treatment options for binge eating and BED.

DIAGNOSIS

BED is currently listed in the appendix of the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* of the American Psychiatric Association (DSM-IV; used to diagnose psychological disorders). BED currently falls into the eating disorder not otherwise specified (EDNOS) category; however, it is likely to be included as a recognized eating disorder (ED) in the next revision of the DSM (DSM-V). Although the majority of research in this area has focused on individuals meeting the diagnostic criteria for bulimia nervosa (BN), individuals with BN make up only a small fraction of those who regularly binge eat.

The larger "subclinical" group includes both men and women who meet some, but not all, of the diagnostic criteria for BN, as well as others who meet most or all the research criteria for BED. A summary of the diagnostic criteria for BED, according to the DSM-IV, is as follows:

To qualify as binge eating, a person must eat more than normal and exhibit a lack of control over food intake.

A. Recurrent episodes of binge eating characterized by both:
   1. eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat under similar circumstance in a similar period of time
   2. a sense of lack of control over eating during the episode (i.e., feeling that one cannot stop eating or control how much or what one is eating)

B. Binge eating episodes associated with 3+ of the following:
   1. eating much more rapidly than normal
   2. eating until uncomfortably full
   3. eating large amounts of food when not hungry
   4. eating alone because of being embarrassed by how much one is eating
   5. feeling disgusted with oneself, depressed, or very guilty after overeating

C. Marked distress regarding binge eating.

D. The binge eating occurs 2+ days per week (on average) for 6 months.

E. The binge eating is not associated with the regular use of inappropriate compensatory behaviors (e.g., purging, excessive exercise) and does not occur exclusively during the course of Anorexia Nervosa (AN) or BN.

The current gold standard for the diagnosis of BED is the structured clinical interview, the Eating Disorders Examination (EDE). However, the EDE can take up to an hour to complete and requires training to ensure proper administration and scoring. As such, several brief paper and pencil measures of binge eating have been developed, although none are considered as valid as the EDE.

PREVALENCE

According to the DSM-IV, the prevalence of BED in community samples was estimated to be about 3.5 percent in 1994. However, numerous studies have found that rates of subclinical binge eating are more common. An interview of 1,500 college women in 1995 found that 35 percent reported a history of binge eating at some point in their lives. The prevalence of BED is also higher within the obese population, estimated at 8 percent and at about 30 percent in obese individuals seeking weight loss treatment. Unlike BN, BED is common in minorities and in men, with a female-to-male ratio of 3:2. Finally, individuals suffering from BED typically remain symptomatic for about 15 years, which is nearly three times as long as with AN and BN.

ETIOLOGY

As with most psychological disorders, genetic and environmental influences combine in the development of BED. Studies indicate a heritability of 40 to 50 percent for BED, with environmental factors accounting for 50 to 60 percent and no differences between sexes. Children of parents with BED are more than twice as likely to develop BED and 2.5 times as likely to develop severe obesity (body mass index [BMI] >40). Most scientists ascribe to a "diathesis-stress" model of eating disorders, where an individual with eating disordered parents or close relatives has an increased likelihood of developing an ED. According to this model, this predisposition may or may not result in an ED depending upon environmental stress. It is also possible for someone to develop an ED without this predisposition. Variables such as parental and peer modeling, social influence, media messages, culture, and body image ultimately determine who develops an ED.

The most commonly accepted explanation for the perpetuation of binge-eating behaviors is based on the influence of negative affect; binge eaters may learn to regulate negative emotions inappropriately by overeating. Studies have revealed that binge episodes are often precipitated by negative emotional states. Enduring negative affect, as reflected in depression, is also associated with binge eating. Binge eaters often experience a heightened concern with their body shape and weight, and typically attempt to restrict their eating for fear of gaining weight. They are often successful in restricting intake until they encounter some form of "disinhibiting stimulus," typically an environmental stressor or ingestion of a "forbidden food." Frequently, a binge episode follows in response to the negative affect experienced because of these stressors. The binge episode temporarily improves mood and serves to reinforce the binge eating behavior. Within a few moments after a binge meal, however, the individual's mood typically reverts to a depressed state, often compounded by feelings of shame and guilt associated with the binge behavior.

CONSEQUENCES

Binge eating has been implicated in the development of obesity and often precedes excess weight gain and initial dieting attempts. Binge eating has also been shown to be a major risk factor for weight regain following a weight loss diet. By definition, BED also causes marked psychological distress. Significant relationships have been found between binge eating and the lifetime prevalence of major depression, panic disorder, borderline personality disorder, and avoidant personality disorder.

Note that although several features associated with binge eating are categorized as causes or consequences, some features may both precede and result from binge-eating behavior. Although helping to perpetuate the behavior, dietary restraint often does not precede the first binge episode. Dieting precedes the onset of binge eating only about 50 percent of the time, and it is likely that binge eating may itself contribute to attempts to further restrain eating. Similarly, negative affect and body image disturbance can contribute to the development of, as well as result from, binge eating.

BINGE EATING IN CHILDREN

Binge eating is not only found in adults. Habits of secretive and overeating, characteristic of BED, can be found in very young children. A study in 1999 of

over 200 children from birth to age 5 revealed that 18 percent of the children hid their favorite foods, and 34 percent appeared to have difficulty stopping when they ate their favorite foods. A survey in 1998 of over 80,000 students in the 9th and 12th grades revealed that 25 percent of females and 13 percent of males reported binge eating in the past year. Similar to adults, binge eating appears more prevalent in overweight and obese children, and loss of control over eating is associated with distress and other morbidity. Unlike adults, however, binge eating in children appears less related to attempts at dietary restriction and eating large amounts of food is not typically associated with distress. In diagnosing BED in children, loss of control of eating remains a hallmark feature; however, more emphasis is placed on eating in the absence of hunger rather than eating an objectively large amount of food. Children who have a personal or family history of obesity may be at risk for the development of binge-eating problems when adverse childhood experiences and risk factors for psychiatric disorder, particularly depression, also are present.

TREATMENT

Treatment for binge eating and BED includes psychological, behavioral, and pharmacological approaches. Psychological treatments consist mainly of cognitive-behavioral therapy (CBT), considered the gold standard, and interpersonal therapy (IPT). CBT combines cognitive and behavioral techniques to break the cycle of bingeing in response to negative affect. The cognitive component attempts to identify and correct "core cognitive disturbances" such as doubts about self-worth, which encourage negative self-evaluation in terms of body shape and weight, as well as the acceptance of a larger-than-average body size. The behavioral component focuses on moderation of food intake that is neither over- nor under-restrictive. CBT has been shown to result in rates of binge-eating abstinence of over 50 percent at 1 year following treatment. CBT has also been shown to decrease dietary restraint, improve distorted attitudes about shape and weight, and decrease associated psychiatric symptoms. IPT focuses on the interpersonal relationships that contribute to the development and maintenance of the eating problem. Specifically, IPT encourages patients to examine the impact of their body shape and weight on relationships and address perceived interpersonal deficits. IPT has been shown to reduce binge eating at rates similar to those after CBT treatment, both at treatment end and at 1-year follow-up. Weight reduction for obese binge eaters following CBT and IPT has been shown to be quite modest. Individuals who remain abstinent from binge eating tend to lose some weight, while individuals who remain even partially symptomatic tend to gain weight.

Behavioral weight loss treatments alone may also reduce binge eating. These treatments seek to reduce binge eating and body weight simultaneously through traditional "lifestyle change" dieting techniques, such as reducing the caloric density and portion sizes of meals and increasing physical activity. Although effective in the short term, most weight lost through dieting is typically regained within a few years and, as body weight returns, binge eating behaviors tend to return as well.

Pharmacological treatments may include tricyclic antidepressants, selective serotonin reuptake inhibitor (SSRI) antidepressants, or appetite suppressants. Tricyclic and SSRI antidepressants have shown the highest efficacy in reducing binge frequency; however, discontinuation of medication generally results in a rapid return of binge episodes. Results from studies combining pharmacological and psychological treatment for BED generally show short-term reduction in binge eating and body weight. However, long-term gains usually dissipate once the medications are stopped. In head-to-head comparisons, CBT was more efficacious than SSRI antidepressant medication.

Self-help books for the treatment of BED are also available that can be used independently by the individual (unguided) or in conjunction with a professional (guided). Self-help techniques may be more cost effective and more readily available than other interventions. Studies show that self-help techniques are effective in the short term for reducing binge eating but not body weight, and the long-term efficacy is yet to be determined.

SEE ALSO: Bulimia Nervosa; Dietary Restraint; Disinhibited Eating; Disordered Eating; DSM-IV.

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Bioelectrical Impedance Analysis

BIOELECTRICAL IMPEDANCE ANALYSIS (BIA) is currently one of the most exact and easiest ways to determine body fat composition. BIA is conducted by attaching electrodes to various parts of the body while a small electrical signal is passed through. BIA is conducted using the weight, height, age, gender, ethnicity, body type, and physical activity level in combination with the measured resistance the body has to the electrical signal. A simpler version of BIA can be found in commercial products similar to the operation of a bathroom scale with the electrodes placed in special foot sensors.

The measured resistance of the electrical signal varies based on the water that is found in muscle and fat. Resistance or impedance is low in lean tissue and high in fat tissue. The measure of resistance is dependent on total body water of an individual. The more muscle an individual has, the greater the amount of water the body contains. The greater the total body water, the easier the current can pass through the body. Although an electrical signal is passed through the body, BIA is considered safe and does not cause pain. The electrical signal used is so small that neither adults nor children have reported feeling it.

SEE ALSO: Assessment of Obesity and Health Risks; Body Fat Distribution in African Americans; Body Fat Distribution in Asian Americans; Body Fat Distribution in Hispanic Americans; Body Mass Index; Ethnic Variations in Body Fat Storage.

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Blood Lipids

BLOOD LIPIDS ARE fats transported in the blood by lipoproteins. Obesity is a risk factor for dyslipidemia (lipid abnormalities). Dyslipidemia is a risk factor for atherosclerosis, which is the main cause of cardiovascular disease and stroke.

Free fatty acids and cholesterol are absorbed by the gut and packaged into chylomicrons as triglycerides and cholesterol esters, respectively. Chylomicrons are acted upon by lipoprotein lipase in the blood to become lipoproteins.

Low-density lipoprotein (LDL) transports cholesterol from the liver to tissue. Cholesterol is used to synthesize cell membranes, steroid hormones, and bile. When there are high levels of LDL, the artery wall takes up more LDL, forming atheromas on the wall. Excess LDL is also taken up by white blood cells, forming foam cells. Foam cells and atheromas cause wall damage, leading to plaques and thrombi, which can produce low blood flow and high blood pressure. If thrombi break off as emboli, they can cause stroke or death. LDL taken up by the liver is used in synthesizing bile. Levels of LDL can increase due to saturated fat intake or hereditary conditions.

High-density lipoprotein (HDL) returns excess cholesterol to the liver, reducing the incidence of atheromas and resultant cardiovascular disease.

Triglycerides transfer energy to cells. While it is unclear that increased triglycerides independently increase the risk of cardiovascular disease, high triglyceride levels decrease HDL levels.

Screening blood lipids is recommended for men older than 35 years and women older than 45 years. If other risk factors such as diabetes, high blood pressure, smoking, or a family history of premature cardiovascular disease, exist, screening should begin after 20 years of age.

Fasting measurement of total cholesterol, triglycerides, and HDL allows LDL and very-low-density lipoprotein (VLDL) calculation. Optimally, total cholesterol should be under 200 mg/dL, LDL should be under 100 mg/dL, HDL should be over 41 mg/dL, triglycerides should be under 150 mg/dL, and the total cholesterol to HDL ratio should be under 4. The Framingham score considers total cholesterol, HDL, smoking history, blood pressure, and age to predict the risk of developing coronary heart disease.

Treatment of dyslipidemia involves lifestyle modifications including diet and exercise. Several classes of medication, including statins, can be used if lifestyle modifications are unsuccessful.

SEE ALSO: Atherosclerosis; Elevated Cholesterol; Fat Intake; High Density Lipoproteins; Lipoprotein Lipase; Low Density Lipoproteins; Stroke.

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Bod Pod and Pea Pod

DEVELOPED BY LIFE Measurements Instruments, Concord, CA, the Bod Pod is a complete system for measuring fat and lean body mass using patented air displacement technology. Bod Pod was created with the help of a National Institutes of Health (NIH) grant. The Bod Pod was introduced at the University of California at Davis and the Plourde Institute of Chicago in 1994, and since then, it has received much attention.

The Bod Pod is based on the same whole-body measurement principle as the hydrostatic weighing tank. The volume of air a person displaces is measured instead of how much water the body displaces when dunked in a tank. The Bod Pod can give results in about 5 minutes and can accommodate children, the elderly, and the disabled. It is safe and noninvasive. Also, no special technician license is required to use the Bod Pod, so it is easy to use for both subject and operator.

The Bod Pod has about a 3-percent error, making it very reliable. Advantages of a Bod Pod include its ease of use, fast measurement, and accommodation of multiple populations. Disadvantages include its price, which can range from $30,000 to $40,000.

The Bod Pod works by measuring the volume of air a person's body displaces while sitting inside. First, the Bod Pod measures the subject's mass and volume and then whole-body density is determined. Using the person's displaced air volume and whole-body density, the Bod Pod can then calculate fat and lean mass.

The Bod Pod's structure consists of a dual chambered, fiberglass plethysmograph. The front of the Bod Pod is the main test chamber, which contains a seat. The door is secured by a series of magnets. The front of the Bod Pod has a large acrylic window, which provides the subject with a field of view.

From the original Bod Pod, the Bod Pod S/T has also been developed to use specifically in health clubs. The main difference is that an individual can either use an operator or do the test him-/herself. The Bod Pod S/T has an easy-to-use audio and kiosk screen that prompts the user/operator through the entire process.

Similar to the Bod Pod, the Pea Pod is a device also developed by Life Measurements Instruments. The NIH awarded Life Measurements Instruments with additional funding and an infant version of the Bod Pod was introduced in 2004. It measures an infant's body composition. The Pea Pod uses body composition instead of length and weight measurements and gives researchers and clinicians a better understanding of an infant's growth and nutritional status. The Pea Pod is a reliable body composition device that helps provide vital information necessary to assess

the efficacy of diet and medical interventions, especially in low birth-weight infants. The Pea Pod uses the same technology as the Bod Pod of measuring air displacement. It is also fast with results in about 5 minutes. It is safe and noninvasive. Also, this device can accommodate most infant behaviors, such as constant wiggling and stretching.

SEE ALSO: Body Mass Index.

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Body Dysmorphic Disorder

BODY DYSMORPHIC DISORDER (BDD) is characterized by an exaggerated preoccupation with an imagined or negligible deformity in one's appearance. The perceived defect is very real to the person with BDD, causing cognitive and behavioral ramifications. Individuals with BDD may have impediments in social activities and occupational functioning, which may result in self-confinement. BDD may be a significant cause of loneliness, depression, social isolation, shame, and guilt. These difficulties may lead to suicidal tendencies.

In 1997, BDD was formally recognized as a somatoform disorder in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (DSM-IV). A somatoform disorder is a group of presenting physical symptoms that cannot be medically explained. Obsessive-compulsive disorder (OCD) may accompany BDD. If the rituals of OCD are confined to appearance-related behaviors, OCD is unlikely.

BDD is not related to body shape, size, or eating habits. Those with BDD may share traits of an eating disorder, but BDD is instead focused on the skin, hair, teeth, nose, lips, or size and shape of the genitals. Those with BDD do not focus on weight-related body sites.

BDD is not gender specific. It is estimated that 1 to 2 percent of the general population is affected. As there is little conclusive research, it is hypothesized that BDD may begin during adolescence. The disorder may be precipitated by an event such as a disparaging or teasing comment about a specific body part, usually on the face. Sexual abuse or harassment may also be a trigger. BDD is often uncovered with other symptoms.

Those suffering with BDD spend an inordinate amount of time focusing on their perceived flaw by constantly looking in the mirror or picking at their skin. They may wear excessive clothing or makeup to cover their perceived flaw as well as practice extreme and excessive grooming rituals which may last for several hours. An individual with BDD may seek surgical or cosmetic intervention, which usually results in their unhappiness from the procedures. They may then begin a quest for a different doctor or put new focus on another area. Ultimately, this may again lead to avoidance of social activities for fear of the perceived flaw being seen. Loved ones may be unaware of the severity and extremes the person with BDD practices.

BDD is a hidden disorder because the individual is unwilling to admit to his or her peculiar behaviors and the effort he or she puts into trying to camouflage his or her actions.

Diagnosing BDD involves frank discussion with a psychologist or psychiatrist and the administration of the Multidimensional Body-Self Relations Questionnaire and the Body Dysmorphic Disorder Examination Self-Report. Treatment for the condition may involve prescribing serotonin-specific reuptake inhibitors in addition to cognitive and behavioral therapies.

SEE ALSO: Appearance; Body Image Disorders; Cognitive Behavioral Therapy; Depression; Obsessive Compulsive Disorder.

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Body Fat Distribution in African Americans

THE PREVALENCE OF obesity is much higher among African-American adults and children compared to Caucasian Americans. African Americans have significantly lower levels of visceral adiposity but higher levels of truncal subcutaneous adiposity compared to Caucasian Americans. Excess visceral adiposity is associated with more adverse outcomes, such as cardiovascular disease (CVD) and Type 2 diabetes mellitus (T2DM). Although African Americans have lower visceral adiposity, they have higher insulin resistance and hyperinsulinemia, and higher risk of T2DM and CVD associated with obesity than Caucasian Americans.

Body fat distribution refers to total adiposity as well as adipose tissue patterning. Body mass is partitioned into fat mass and fat-free mass which vary with age, sex, and ethnicity. Fat or adipose tissue is located in the subcutaneous tissue (SAT) and in the visceral tissue (VAT). There is no consensus as to the best methods for assessing adipose tissue distribution, which may account for some of the variability seen in the different studies of body fat distribution.

African Americans have higher levels of SAT deposited mainly in the truncal region. They have lower waist-to-hip ratio (WHR) compared to Caucasian Americans and Hispanic Americans. Caucasian Americans, on the other hand, have a more peripheral distribution of subcutaneous adiposity. African-American men and women have lower VAT compared to their Caucasian-American and Hispanic-American counterparts.

The difference for VAT mass is greater for men than women. Obese African-American women with similar measures of total body fat and waist circumference to white women had 23 percent less VAT before, during, and after weight loss, indicating that at similar levels of obesity, African-American women have lower VAT compared to Caucasian-American women.

Although the total body fat percentage is lower in African-American men compared to African-American women, African-American men have higher VAT than African-American women. Therefore, as in Caucasian Americans and Asian Indians, African-American men have more VAT than African-American women.

Compared to their age-matched counterparts, African-American children have greater weight, height, body mass index, and subscapular and suprailiac skinfold thickness. African-American children have less total subcutaneous abdominal and VAT compared to their white counterparts, suggesting early ethnic difference in fat deposition.

Higher subcutaneous fat accumulation and lower VAT mass have also been reported in populations of African decent residing in Africa, Europe, United States, the Caribbean, and South America.

Although African Americans have less VAT mass than Caucasian Americans, the risk of obesity-related comorbidities is higher in African Americans compared to Caucasian Americans, indicating perhaps racial differences in adipose tissue metabolism, or that the higher risk of comorbidities may be due to factors independent of VAT. Obesity-unrelated predisposition factors to hypertension, for example, may be more important in the development of CVD in African Americans. Ethnic differences in lipid markers for CVD have been shown to be established at an early age and track into adulthood.

The exact mechanisms by which body fat preferentially distributes in certain regions are not completely understood. Ethnicity may influence basal metabolism, and dietary and other lifestyle habits such as physical activity. Other proposed determinants of regional fat distribution include sex, age, total body fat, hormonal factors, dietary factors, cigarette smoking, and alcohol consumption.

SEE ALSO: African Americans; Assessment of Obesity and Health Risks; Cardiovascular Disease in African Americans; Ethnic Variations in Body Fat Storage; Ethnic Variations in Obesity-Related Health Risks; Hypertension in African Americans; Magnetic Resonance Imaging Scans; Waist-to-Hip Ratio.

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Body Fat Distribution in Asian Americans

THE DISTRIBUTION OF body fat has been shown to vary among ethnic groups. Body fat distribution may have a gynoid fat pattern, which is characterized with a significantly greater percentage of fat on the extremities. This is in contrast to the android group, or female group, characterized with proportionately more fat on the trunk. For a given body mass index (BMI), Asian adults were found to have a higher body fat percentage and more upper-body subcutaneous fat, than Caucasians. Consequently, weight gain is especially detrimental for Asians. Also, the regional differences in the storage of adipose tissue, or fat tissue, vary in terms of function in the body. Upper body fat has notably been associated with increased risk of chronic diseases. Fat that is centrally located is also referred to as central adipose tissue, visceral adipose tissue, intra-abdominal fat, trunk fat, etc. There is both intra-abdominal (visceral) and subcutaneous abdominal fat. The differences in the accumulation may have distinct impacts on glucose and lipoprotein metabolism. Several studies have shown that the effect of visceral fat on glucose tolerance is independent from total adiposity and subcutaneous fat depots.

Visceral fat is associated with a variety of health concerns. Specifically, even in the absence of other risk factors, central adiposity is associated with cardiovascular risk. Also, previous studies have demonstrated a strong relationship between BMI and cardiovascular risk factors in Asian populations. However, the range of BMI is at the lower end of the distribution for typical western populations. For example, greater and regional adiposity, and family history of diabetes in Asian Americans. The observed ethnic difference in diabetes risk is probably due to an interaction between diet and visceral adiposity has been shown to increase the risk for hypertension in Asian Americans. Compared with Europeans, Chinese and South Asian participants have demonstrated a relatively greater amount of abdominal adipose tissue. Moreover, this difference was more pronounced with visceral adipose tissue.

Diabetes is an additional health concern. Greater visceral adiposity precedes the development of Type 2 diabetes and is considered to play a key role in the metabolic syndrome. The effect is independent of fasting insulin, insulin secretion, glycemia, total lifestyle and increased genetic susceptibility among minority groups. In addition, the pattern of fat on the trunk is also related to high levels of LDL and VLDL cholesterol and low levels of HDL cholesterol, independent of general body fatness.

Ethnic differences in fat distribution are partially explained by differences in skeletal dimensions. Asians are shorter, weigh less, and have a higher percentage of body fat, particularly abdominal visceral fat, compared with African Americans and whites for the same BMI. In order to identify Asians at a higher risk of cardiovascular disease and diabetes, cutoff BMI values for classifying overweight and obesity status are needed. Other preventive measures include diet and endurance exercise.

SEE ALSO: Cardiovascular Disease in Asian Americans.

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Body Fat Distribution in Hispanic Americans

OBESITY AND COMPLICATIONS of obesity are more prevalent among Hispanic Americans than Caucasian Americans. There is evidence to suggest that there is ethnic-race variation in the distribution and patterning of body fat. Hispanic Americans have a larger per-

centage of total body fat that is more centrally or viscerally distributed than their Caucasian counterparts at similar body mass indices (BMIs). Central adiposity has been associated with adverse health outcomes.

Body fat distribution refers to a description of total adiposity as well as adipose tissue patterning. Body mass is partitioned into fat mass and fat-free mass. These vary with age, sex, and ethnicity. Fat or adipose tissue is located in the subcutaneous tissue (SAT) and in the visceral tissue (VAT). There is no consensus as to the best methods for assessing adipose tissue distribution, which may account for some of the variability seen in the different studies of body fat distribution. It has been suggested that VAT versus SAT distribution is associated with more adverse metabolic risks, including hypertension, type 2 diabetes mellitus (T2DM), dyslipidemia, and cardiovascular disease (CVD).

Studies have shown that Hispanic-American women have higher total adiposity and greater levels of centralized obesity measures (higher subscapular to triceps skinfold ratios and waist-to-hip circumferences) than their age-matched Caucasian counterparts. From childhood to adulthood, Hispanic-American women have consistently higher truncal-to-extremity skinfold thickness ratios compared to Caucasian women. Additionally, Hispanic-American women have higher VAT than African-American women of similar BMI.

Some studies have shown that Hispanic-American men have slightly higher total body fat and greater truncal to peripheral adiposity compared to Caucasians, whereas other studies have shown no significant differences in total body fat estimates or fat distribution among men in the different race-ethnic groups.

In children 4–19 years old, Hispanic-American men and women have been shown to have higher body fat than their Caucasian counterparts. Waist-to-hip ratio (WHR) was slightly higher in Hispanic-American boys than Caucasians and much higher than African-American boys. Among girls, Hispanic Americans had slightly higher WHR than Caucasian or African-American girls.

The higher prevalence of T2DM and CVD risk profile seen in Hispanic Americans could be a reflection of the higher VAT depot. The exact mechanism for preferential visceral adiposity is not known. Possible factors for differential body fat distribution include genetics, hormones, lifestyle or behavioral factors (smoking, drinking, physical activity) and dietary factors, and socioeconomic factors.

Comparison body composition studies between Hispanic Americans and other race-ethnic groups are limited. The majority of studies that included Hispanic-American samples invariably surveyed only Mexican Americans. As Hispanic Americans is not a homogenous group, caution needs to be applied when using the term *Hispanic American* or generalizing the study findings. The Hispanic-American ethnic group comprises members with various admixtures of genetic and cultural background with combinations of Amerindian, European, and African ancestry.

SEE ALSO: Assessment of Obesity and Health Risks; Cardiovascular Disease in Hispanic Americans; Ethnic Variations in Body Fat Storage; Ethnic Variations in Obesity-Related Health Risk; Hispanic Americans; Hypertension in Hispanic Americans; Magnetic Resonance Imaging Scans; Mexican Americans; Waist-to-Hip Ratio.

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Body Image

CHILDHOOD OBESITY HAS been demonstrated to not only impact the physical health but also the mental health of affected children and adolescents. The psychological effects of childhood obesity have been well studied over the past several decades. One par-

ticular focus of research has been the area of body image (or how children view their own bodies in the context of feedback received from their social community and/or environment) and how body image relates to overall health among children with overweight. Such research demonstrates that body image is related to current weight status and to other psychological measures. In addition, more recent studies demonstrate that body image can affect the adoption of healthy weight-related behaviors by children. Treatment of overweight among children therefore must incorporate interventions that address body image and satisfaction in order to promote and maintain healthy weight among youth over the long term.

BODY IMAGE IN CHILDREN

Body image has been widely researched in children, particularly in girls, owing to prior concerns about weight related obsessions and dieting behaviors in this population. Most studies have sampled from the general population, with a majority of evaluations limited to girls; however, more recently, studies have included boys as well, perhaps reflecting the uniform increase in childhood obesity among both boys and girls worldwide. Tools utilized to assess body image have included standardized scales as well as pictorial instruments depicting various body sizes. Regardless of the year of publication, the demographics of the community population sampled, and the methods used, greater body dissatisfaction has been documented among heavier children and adolescents as compared to their normal weight counterparts.

Potential moderators and mediators of the relationship in children between weight status and body image that have been identified in prior research include: age, gender, ethnic/racial background, psychosocial and psychological functioning. Several studies (both cross-sectional and longitudinal) suggest that older youth are more likely than younger youth to demonstrate strong relationships between lower body image and higher weight status.

This finding taken together with the development of body awareness and image during middle childhood highlights a vulnerable period in childhood where future body image concepts can be affected and modified by social experiences. Similarly, several studies have shown that girls are more likely than boys to exhibit body dissatisfaction. However, more data is needed to confirm these findings given that fewer studies have sampled from boys, and research instruments may not reflect the distinct gender-specific ideal body types promoted by society or take into account body image development variability between boys and girls. The published data addressing ethnic or racial effects on the relationship between body image and weight status indicate significant differences in body satisfaction among various racial groups, which may reflect different cultural body ideals. In addition, body image has been found in cross-sectional and longitudinal evaluations of adolescents to be associated with self-esteem, and other measures of psychological (depressive mood) and psychosocial functioning (peer interactions). These relationships are most evident among adolescent girls, as compared to adolescent boys for whom the nature of the relationship appears to be less clear.

Body image has also been shown to affect how children manage their weight. In a longitudinal cohort of 2,516 adolescents from the EAT (Eating Among Teens) project, adolescents who demonstrated lower body satisfaction were likely to engage in unhealthy weight control behaviors, such as fasting, skipping meals, smoking, and using diet pills or laxatives. Thus, while negative body perception may increase weight loss attempts, these weight loss attempts are likely associated with weight-related behaviors that may actually increase weight and reduce overall health. In contrast, programs to date targeting healthy nutrition and exercise behaviors that also promote a healthy or positive body image as part of their intervention appear to have a positive or at least no negative impact on body image over time regardless of the weight status of enrolled children.

POOR BODY IMAGE

The contribution of the environment in regards to the development of body dissatisfaction has been more recently evaluated. Weight-related teasing has been shown to predict current and future poor body image, particularly among overweight youth and independent of gender. In addition, media modalities with a focus on appearance have been shown to be related to and predictive of body satisfaction in children, where increased exposure to these media modalities increases body dissatisfaction, especially among girls. Similarly, peer and parent opinions regarding weight

appear to influence the body concept of children, although the relative contribution of each depends on the demographics of the population studied.

The strong relationship of body image and childhood weight status is an important issue that must be addressed in the setting of childhood obesity. Interventions addressing weight maintenance and weight loss in this vulnerable population should promote body satisfaction not only to avoid negative physical and psychological consequences but also to increase adoption of healthy weight-related behaviors. Data would also suggest that such interventions may need to tailor their addressing of body image issues according to individual demographics, including gender, age, and prior social weight-related interaction history.

SEE ALSO: Peer Influences on Obesity in Children: Self-Esteem and Children's Weight; Self-Esteem and Obesity.

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Body Image Disorders

BODY IMAGE DISTURBANCES are generally defined as negative thoughts and feelings about one's body, but the concept of body image is multidimensional in nature. Body image disturbances generally fall into two categories: body size distortion in which the individual has an erroneous perception of his/her body size, and body dissatisfaction, which relates to the cognitive and attitudinal aspects of the individual's body image. Body image disturbance receives attention in current research as it has been implicated as a risk factor for eating disorders such as anorexia nervosa and bulimia nervosa and in obesity. However, research yields conflicting results as to which of these two distinct categories is a stronger predictor of disordered eating.

BODY IMAGE AND GENDER

Adolescent girls and women consistently report dissatisfaction with their overall body shape and size. One study found that nearly half of all American women are preoccupied with their weight, while another study reported up to one-third of the same population are actively trying to lose weight. In addition to overall complaints of body shape and size, women report dissatisfaction with specific areas of their bodies such as their stomachs, hips, and thighs.

Although women tend to emphasize appearance more than men, men are not immune to forming negative body images. In general, both men and women report dissatisfaction with their bodies, but negative body image among men is of a complex nature. Unlike women, men do not simply want to become thinner; they desire a muscular physique that is lean as well. Men are also influenced by some of the same factors that contribute to negative body image in women (i.e., media and print images).

BODY DYSMORPHIC DISORDER

First recognized in 1997 in the *Diagnostic and Statistical Manual of Mental Disorders, 4th ed.* (*DSM-IV*), body dysmorphic disorder is characterized by a consuming preoccupation with an imagined defect in physical appearance. This preoccupation can be highly disruptive and can cause significant emotional distress and impairment, both socially and professionally. Body dysmorphic disorder is distinct from body image dissatisfaction and is frequently undiagnosed in the general population. Individuals with body dysmorphic disorder often have low self-esteem and may engage in ritualistic behaviors related to their perceived defect that may include excessive grooming and constant observation of themselves in a mirror.

BODY IMAGE AND IMPLICATIONS FOR EATING DISORDERS

Body dissatisfaction has emerged as a major risk factor for both development and maintenance of disordered eating behaviors. There are several theories accounting for this relationship, and several sources of sociocultural pressures have been implicated in the risk for body dissatisfaction including the media, peers, family members, and intimate partners. One theory suggests that the dogmatic pursuit of an unattainable ideal inevitably increases body dissatisfac-

Sufferers of anorexia nervosa and bulimia nervosa also suffer from body image disorders; patients have a distorted, unrealistic view of their own body, believing themselves overweight or obese contrary to evidence.

tion. Another theory posits that body dissatisfaction increases with increasing weight (i.e., farther away from the "thin ideal"). With regard to the development of specific eating pathologies, two mechanisms linking body dissatisfaction to eating disorders have been identified. The more credible of the two reasons is that body dissatisfaction increases dieting activities, which then renders the dieter vulnerable to disordered eating behaviors seen in anorexia nervosa and bulimia nervosa. The second theory describes the relationship between body dissatisfaction and a "negative affect" which may promote binge-eating behaviors, ultimately resulting in an eating disorder.

BODY IMAGE AND OBESITY

In Western societies where the "thin body ideal" is pervasive, individuals who are obese, women in particular, are subject to poor body image. The problem worsens; research has found that as the rates of societal pressure to be thin and the prejudice against obese individuals increase so does the rate of obesity itself. Obese individuals tend to suffer a more negative body image than individuals of normal weight and, as a result, may be so preoccupied with their appearance that they avoid social situations because of their weight.

Body image has been correlated with psychological distress and depressive disorders. Research with obese individuals suggests that obese men and women experience higher levels of major depression and other mood and anxiety disorders than individuals of normal weight. Lower levels of self-esteem are also associated with obesity. However, it is not yet clear whether obese individuals in treatment for weight loss would significantly benefit from specifically addressing body image as a therapeutic component.

SEE ALSO: Anorexia Nervosa; Body Dysmorphic Disorder; Bulimia Nervosa; Eating Disorders and Obesity; Self-Esteem and Obesity.

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Body Mass Index

BODY MASS INDEX (BMI), also known as the Quetelet index, is a measure combining the weight and height of an individual. BMI can be calculated by dividing the weight (kg) by the square of the height (m). Although invented in the mid-1800s by Belgian Adolphe Quetelet, BMI is currently used as a simple clinical tool for healthcare professionals to determine a patient's potential risk for disease and call for intervention. However, BMI has recently and incorrectly been seen as an "absolute" tool for medical diagnosis and care, rather than a suggestive indicator of risk.

BMI is used to classify individuals into categories. The most common definitions used clinically in the United States for BMI categories include underweight (BMI <18.5), ideal (BMI 18.5–25), overweight (BMI 25–30), obese (BMI 30–40), and morbidly obese (BMI >40). According to the U.S. National Health and Nutrition Examination Survey in 1994, 59 percent of American men and 49 percent of women have BMIs over 25 and, therefore, categorized as either overweight or obese.

BMI categories have important correlations with disease risk. Higher BMI values are associated with cardiovascular disease and higher morbidity and mortality rates for almost any disease. According to some studies, a BMI of 30 and greater translates to an increase risk of death from any cause by 50 to 150 percent. Extremely low BMI values, below the ideal category, may indicate malnutrition or an eating disorder. Although a correlation may exist, researchers are also quick to acknowledge that BMI is not entirely accurate in predicting the risk of cardiovascular disease and death because it does not distinguish body muscle and fat. However, when used appropriately, categorization of an individual using the BMI can indicate to a healthcare provider that some intervention may be needed.

While the strength of BMI is its ability to be calculated quickly and without the use of expensive equipment, the tool is not without limitations. The largest limitation of BMI, which explains why its use should be carefully considered, is the fact that BMI is only calculated using weight and height measures. Consequently, BMI does not consider important factors such as frame size, fitness level, muscle mass, bone structure and mass, gender, and ethnicity. For example, a healthy athletic patient who has an ethnic background of short stature may have a BMI that would categorize the patient as obese, when in fact, the patient should really be considered normal with low disease risks. The combination of muscle mass and short stature skews the simple BMI calculation. Other individual types that may not be well suited for the BMI scale include athletes, children, and the elderly. However, despite these limitations, the ease of use has led to the World Health Organization to adopt BMI to calculate obesity statistics since the 1980s.

The use of BMI for children is different than for adults. Currently, BMI-for-age is a special BMI index used clinically for children. BMI-for-age takes into account the differences of BMI normally expected for children based on whether they are a boy or girl and their current age. Age is important for BMI to account for because young children who may initially have a high body fat will get leaner as they get older. For individuals between the ages of 2 and 20, healthcare providers can use growth charts to follow the devel-

opment of height, weight, and age. These variables are then compared to other children of the same age and gender to help determine the risk the child has for being overweight in the future or his or her risk for developing certain diseases associated with excessive weight and body fat. Importantly, the normal BMI category increases for girls as they grow to their teenage years. This is to account for the fact that girls have more body fat than boys. Additionally, clinicians best use BMI data by tracking its change over time because of the impact of growth spurts in children.

Alternatives to BMI calculation methods include measuring actual body fat percentage and using the individual's waist–hip ratio. Measuring the body fat percentage has an advantage over BMI because of its ability to differentiate between muscle and fat mass; however, accurately measuring body fat is difficult and expensive. The use of waist–hip ratio has been shown to accurately predict disease risk because of its ability to account for abdominal obesity.

SEE ALSO: Assessment of Obesity and Health Risks; Body Image; Children and Diet; Exercise; Ethnic Variations in Body Fat Storage; National Weight Loss Efforts for Children.

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Bombesin

THE PEPTIDE HORMONE, bombesin, was first isolated in 1970. This hormone is composed of 14 amino acids. Its amino acid sequence is written as follows: H-pGlu-Gln-Arg-Leu-Gly-Asn-Gln-Trp-Ala-Val-Gly-His-Leu-Met-NH. Bombesin's unique name was derived from the organism in which it was separated from, the frog Bombina bombina. The significance of this hormone is partly attributed to its role as a mitogen. In other words, bombesin takes part in inducing mitosis. It is for this reason that high levels of it are found in thyroid and pulmonary tumors. Bombesin is also found in brain tissue as well as in the gastrointestinal tract. Furthermore, it has been discovered that there is a meal-elicited rise of bombesin-related peptides in humans.

Bombesin is classified as a neurohormone that acts as a releaser of two important hormones of the gastrointestinal system: cholecystokinin and gastrin. Gastrin stimulates the production of hydrochloric acid from parietal cells, while cholecystokinin stimulates protein and fat digestion. As the releaser of these peptide hormones, bombesin is acknowledged as an inhibitor of food intake. The bombesin-related peptides that are found in humans are neuromedin B and gastrin-releasing peptide. As the name implies, gastrin-releasing peptide is responsible for stimulating the release of gastrin. On the other hand, neuromedin B takes role in regulating body temperature, cell growth, glucose levels, blood pressure, and endocrine as well as exocrine secretions. Bombesin as well as bombesin-related peptides evidently act in a negative-feedback fashion. An increased amount of food intake results in an increased amount of bombesin released. In an article from the *Journal of Neuroscience*, it was proposed that even the administration of bombesin exogenously causes the suppression of food intake. In view of the fact that it acts in such a manner, it is understood that suppressing the release of bombesin or bombesin-related peptides will instigate just the opposite, feeding. Thus, with continued research on this hormone, much can be learned about its response to appetitive stimuli as well as its association with obesity and various eating disorders.

SEE ALSO: Hormones, Hypertension.

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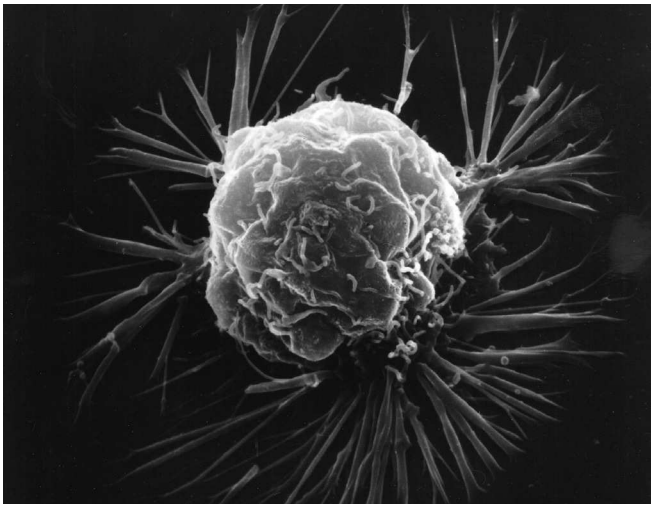
Breast Cancer

CARCINOMA OF THE breast is the most common nonskin malignancy in women, exceeded only by lung cancer as the greatest cause of cancer deaths in women. The major known risk factors are hormonal and genetic (family history). Chronological age is the strongest predictor of nongenetic risk; a woman who lives to age 90 has a one in eight chance of developing the disease. Hormonally based risk factors include age of menarche, age of first live birth, and first-degree relatives with breast cancer. Of breast cancer with a genetic basis, about 25 percent can be attributed to two autosomal-dominant genes, BRCA1 and BRCA 2; however, inherited mutations at these loci account for only about 3 percent of all breast cancers. Thus, sporadic breast cancer is by far more common than familial, and risk increases with exposure to excess estrogen stimulation, especially in cells that express the estrogen receptor (ER).



Magnified breast cancer: While breast cancer is often caused by genetic factors, doctors have discovered a link to obesity as well.

Transformation from normal to malignant phenotype requires that the deoxyribonucleic acid (DNA) become damaged and unstable (initiation), and that damaged cells be exposed to growth stimulants in a permissive environment that allows unrestrained proliferation (promotion and progression). Estrogen plays at least two roles in this process: (1) metabolites of estrogen can cause DNA mutations and DNA damage, and (2) estrogens act as growth promoters, driving premalignant proliferation. A subset of breast carcinomas do not express the estrogen receptor (ER negative); however, it is not certain whether these cells were initially estrogen independent or became neoplastic under the influence of estrogens and converted to an ER-independent phenotype in a later phase.

OBESITY, CIRCULATING HORMONES, AND POSTMENOPAUSAL BREAST CANCER

Three major hypothesis have been advanced to explain the observed association between obesity and postmenopausal, but not premenopausal breast cancer. The first is that elevated concentrations of circulating estrogen metabolites found in obese women and men expose breast tissue to excess hormone. While ovarian estrogen synthesis is the major source of estrogen in the premenopausal women, estrogen metabolites are also formed by peripheral conversion of adrenal androgens in adipose tissue in the breast, abdomen, thighs, and buttocks. As indicated above, chronic estrogen exposure has potential both to initiate and promote breast cancer. Alternatively, metabolic syndrome, associated with obesity, elevated circulating concentrations of insulin, insulin-like growth factors, glucose, and triglyceride-rich lipoproteins can provide an anabolic environment conducive to neoplastic growth. Cross-talk potentiation between insulin and the estrogen receptor pathway has been postulated; increased signaling through this pathway can directly activate gene transcription and secondary signaling pathways within the cell. Because hyperinsulinemia has been associated with risk for recurrence and mortality from breast cancer regardless of its ER status, other pathways must also be involved.

ADIPOCYTE AS ENDOCRINE TUMOR

Adipocyte metabolism has also been linked to the development of breast cancer. Adipocyte is an endocrine organ that secretes factors that both act locally in paracrine fashion, and enter the circulation. Mature adipocytes secrete leptin, known to regulate food intake and energy balance. Breast tissue does not normally express leptin receptors, but the presence of leptin receptors on a breast tumor is associated with lower survival and distant metastases. Inflammatory cytokines including tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6) are also synthesized and secreted by adipocytes and adipose tissue-associated

macrophages. These cytokines have diverse activities, including cell signaling and control of dendritic cell growth, which have potential to regulate connective tissue integrity and control the degree of tumor invasion. Undifferentiated preadipocytes secrete estrogen metabolites through the action of aromatase, an enzyme that converts adrenal androgens to estrogens. Control of this enzyme is poorly understood, but it is known to respond to signals from mature adipocytes, including TNF-α and IL-6. Aromatase activity can elevate the local estrogen concentration manyfold, increasing exposure of breast tissue to this potential carcinogen and growth promoter.

SEE ALSO: Obesity and Cancer.

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Among the benefits of breastfeeding are increased protection against infection and reduced risk of obesity.

Breastfeeding

SCIENTIFIC KNOWLEDGE HAS demonstrated that breastfeeding is the preferred method of feeding and nurturing infants and is the primary means of achieving optimal infant health, growth, and development. Breast milk provides almost all of the necessary nutrients, growth factors, and immunological components a healthy term infant needs. The American Academy of Pediatrics recommends breastfeeding for at least the first year of life, and as long thereafter as is mutually desired by mother and child. However, breastfeeding rates differ substantially by race, socioeconomic level, and other demographic factors. In regard to health, growth, and development, the breast-fed infant remains the reference model against which all alternative feeding methods are measured.

Beneficial long-term effects of breastfeeding include the protection against infectious and immune-related diseases, enhancement of cognitive development, visual function, and a decreased risk of metabolic syndrome and of obesity. Other advantages of breastfeeding include reduction of incidences and severity of infections and the prevention of allergies. The most important short-term immunological benefit of breastfeeding is the protection against infectious diseases. Also, breastfeeding influences the development of the infant's own immune system. For example, there is some evidence of lower prevalence of inflammatory bowel diseases, childhood cancers, and Type 1 diabetes in breast-fed infants.

There seems to be a small protective effect against later overweight and obesity. Breastfeeding reduces the odds ratio for obesity at school age by about 20 percent, relative to formula feeding. It has been reported that the early feeding mode affects growth and body composition and it could be considered a critical factor for metabolic development.

The protective effect of breastfeeding may be partially explained by lower rates of infant weight gain,

which may be related to differences in substrate intakes with breast milk and standard infant formulas. Protein intake per kilogram body weight is approximately 55 to 80 percent higher in formula-fed than in breast-fed infants. According to the "early protein hypothesis," the early high-protein intakes in excess of metabolic requirements may enhance weight gain in infancy and later obesity risk.

Human milk is species-specific and offers a superior method of feeding. Milk formulas are designed to mimic human milk as much as possible, but there are important compositional differences between human milk and formulas. Human milk has a dynamic nature and varies with time postpartum. The variations of its composition match the changing needs of the growing infant. The properties of breast milk promote the growth of beneficial bacterial flora such as bifidobacteria and suppress the growth of coliform and other potentially pathogenic organisms. In addition, human milk is a source of different nutrients and bioactive factors, especially hormones and growth factors including leptin, ghrelin, insulin, and insulin-like growth factor (IGF-I). Each plays a role in food intake regulation, metabolism, and body composition. In particular, breast milk leptin may provide a physiological explanation for a number of advantages seen in reaching proper growth and energy balance in breast-fed infants compared with formula-fed ones. Other nutritional advantages of breast milk include a low sodium-to-potassium ratio, an appropriate fat content, optimal absorption rates for each compositional factor, and high taurine levels, which may promote nerve cell growth.

Health benefits for breastfeeding mothers include lactation amenorrhea, early involution of the uterus, enhanced bonding between the mother and the infant, and reduction in incidence of ovarian and breast cancer. Breastfeeding is also associated with increased parasympathetic nervous system modulation, greater vascular stress response, lower perceived stress levels, and fewer depressive symptoms. In contrast, bottle-feeding is associated with increased sympathetic and decreased parasympathetic cardiac control, whereas breastfeeding is associated with decreased neuroendocrine response to stressors and decreased negative mood in the mother.

For children, not breastfeeding is associated with increased health risks including otitis media, respiratory tract infections, diarrhea, and necrotizing enterocolitis. In addition, blood pressure and blood cholesterol seem to be slightly lower in breast-fed infants although a link has not been demonstrated in regard to cardiovascular disease later in life. Constipation is also lower in breast-fed infants. From the economic perspective, breastfeeding is less expensive than formula feeding. In most cases, maternal ingestion of medications and maternal infections are not contraindications to breastfeeding. However, there are rare situations and medical reasons when human milk is not recommended, when alternative options should be considered, or when breastfeeding must be closely monitored. For example, breastfeeding is contraindicated in infants with galactosemia. Also, some diseases may be transmitted from the mother to the infant by breastfeeding.

Breastfeeding is associated with enhanced physical and mental health compared with non-breastfeeding mothers as well as benefits for the breast-fed infant. However, contamination of human milk is possible due to decades of inadequately controlled pollution by toxicants, persistent pesticides, or chemical solvents. The enormous health benefits of breastfeeding should be emphasized as well as the need to reduce environmental pollution of breast milk.

SEE ALSO: Infant Growth Rate; Infant Weight Gain and Childhood Overweight.

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Built Environments

THE BUILT ENVIRONMENT has been commonly defined, within the health and behavior literature, as the physical structures of living space that either promote or obstruct healthy living. As it relates to obesity in industrialized countries, the built environment has been examined as a possible place to intervene on obesity-promoting behaviors. Many components of the physical landscape make access to a walking-friendly environment important to engaging in a physically active lifestyle. People who live in neighborhoods with easy access to clean and safe sidewalks and diverse shopping and recreation areas are more likely to walk to these locations and be more physically active compared to people who live in neighborhoods without these amenities. Similarly, the proximity and access of food markets contributes to the dietary choices that people make.

Neighborhoods with more fast-food restaurants and fewer food markets with healthy food options, for instance, tend to have a higher prevalence of obesity. Several studies have found that people living out of urban city centers in more suburban and rural areas are more likely to be obese than city dwellers, possibly because of a greater dependence on cars and fewer places that can be accessed by walking.

Understanding exactly how the built environment affects obesity requires knowledge of both the environment and the people living in it. By considering numerous aspects of the physical environment simultaneously, reasonable conclusions about how a person's weight is affected by the environment may be drawn. Therefore, studies that examine the effect of the built environment on obesity will often incorporate numerous variables from many sources. These variables characterize the physical environment, such as the presence or absence of sidewalks, as well those that characterize the individuals living in the physical environment, such as a person's body weight or body mass index.

The built environment may be defined in many ways in an effort to show how it might influence behaviors that promote weight gain. For example, features of the physical landscape that might affect a person's likelihood of walking in his or her neighborhood include sidewalk quality, whether grocery or other stores are located within walking distance, and levels of violent crime in the community. The access to certain neighborhood amenities such as parks, playgrounds, and fitness centers are also important built environment characteristics when attempting to assess an individual's opportunity to be physically active. Furthermore, not only are the numbers of food establishments important in influencing health behaviors, but when determining dietary choices, the types of restaurants and grocery stores are also of issue. For this reason, the number of fast-food restaurants and the access to grocery stores with healthy food options are also considered when defining the built environment.

The field of study on the built environment and obesity risk refers not only to the characteristics and location of the "brick and mortar" buildings of the area landscape, but also includes how people interact within the boundaries of these physical structures. All of the variables considered thus far should be interpreted within the context of the people living in these environments, whose interaction with their surroundings may be shaped by race/ethnicity, culture, economics, educational background, marital status, immigration, perception, and health constraints, among other things.

SEE ALSO: Accessibility of Foods; Eating Out in the United States; Income Level; Physical Activity and Obesity.

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Bulimia Nervosa

EATING DISORDERS ARE a spectrum of illnesses that have serious psychological and medical consequences, both in the short and long term. In the case of bulimia nervosa, the general trend is to binge, or eat profound quantities of food, and then use compensatory measures to remove calories and prevent weight gain. To understand bulimia, a number of topics must be explored, including the clinical definition, epidemiology, the signs and symptoms, treatment, and consequences.

DEFINITION

The *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (DSM-IV) defines bulimia nervosa with the following four criteria. There are episodes of binge eating with a sense of loss of control. The binge eating is followed by compensatory purging behavior, such as self-induced vomiting, laxative, or diuretic abuse. The binges and compensatory behavior must occur a minimum of two times per week for at least three months. The individual must also have dissatisfaction with his or her body shape and weight.

Bingeing is described as eating, in a defined and self-limited amount of time, an amount of food that is larger than most people would eat during a similar time period and under similar circumstances. During this time, the person feels out of control with his or her eating, as though he or she cannot stop. Purging can take many forms and is an attempt to compensate for the extreme food intake during a binge. Purging can be achieved by self-induced vomiting, using laxatives, diuretics, or with excessive exercise. Bulimia is frequently divided into two subtypes: purging and nonpurging. Purging involves those who induce vomiting, or use laxatives, diuretics or enemas to remove calories, whereas those who use excessive exercise or periods of fasting to compensate for calorie intake fall into the nonpurging type. Nonpurging is more rare and accounts for only 6 to 8 percent of bulimic patients.

EPIDEMIOLOGY

There is great difficulty in accurately evaluating the epidemiology of eating disorders due to changes in diagnostic criteria over time, the reliance on self-reporting, and the tendency for those affected to deny the disorder. Bulimia nervosa was first described in 1977, at the Royal Free Hospital in London and was recognized as an eating disorder by the American Psychiatric Association in 1980. It is estimated that between one and two million women in the United States would meet the criteria for bulimia nervosa. Over 90 percent of bulimic patients are female. There are two peaks in incidence of bulimia, at ages 14 and then at 18, although presentation can be into adulthood.

There is no medical consensus on the etiology of eating disorders including bulimia nervosa. Most models include psychological, biological, family, genetic, environmental, and societal factors. These forces can cause a decreased sense of self-esteem, body image, or self-control. There are several more specific factors that may be influential in the development of bulimia and other eating disorders. For example, some literature suggests that an important predictor of eating disorders is dieting during adolescence. In addition, if there is a preoccupation or obsession with thin body image or if one feels a social pressure to be thin, this may be associated with the development of eating disorders. Activities that emphasize body shape, such as gymnastics, dance, and cheerleading may also influence the development of eating disorders.

Bulimia nervosa also has a familial component, as the incidence is increased if a first-degree relative also has bulimia. In addition, twin studies of eating disorders in the United States and Europe have

shown between 28 and 83 percent heritability for bulimia nervosa and binge-eating disorders. These studies avoid the bias of specific household environmental factors, but do not eliminate overarching societal pressures or influences. Feminist psychology theory has suggested that societal pressure to be "superwomen" in the setting of Western society can predispose women to develop eating disorders. There have been conflicting results regarding the association of eating disorders and sexual abuse. The rates of sexual abuse among patients with bulimia were found to be higher than healthy controls, but these increased rates of sexual abuse were similar to that seen among other psychiatric populations. Basic science research also suggests that there is a disturbance in neurotransmitter balance in patients with bulimia, particularly with serotonin. There may also be differences in the role of appetite and satiety in these patients. The release of cholecystokinin, a hormone that influences the sensation of fullness, is low at baseline in bulimic patients, and does not rise to as high levels after a meal.

Patients with eating disorders such as bulimia have higher rates of comorbid psychiatric disorders, such as obsessive-compulsive personality traits when they are children. In addition, there is a higher rate of substance abuse in those with eating disorders. In particular, alcohol problems have a higher prevalence in those with bulimia nervosa.

SYMPTOMS

Bingeing episodes may be either gradual or acute. Gradual episodes are preceded by plans to binge, including purchasing or preparing the food, whereas acute binging episodes are urgent and are immediately fulfilled by any food that is available. These binging episodes are most often done in secrecy. Once a binge has been initiated, the patient experiences a feeling of loss of control, food is consumed rapidly, and the appetite seems insatiable. These episodes may last for up to an hour and are ended when the patient is unable to physically eat any more, and may experience nausea, bloating or pain, as well as feelings of guilt and shame. In contrast to those with anorexia, patients with bulimia can acknowledge that their behavior is abnormal but conceal the disease. These binges may occur several times a day, but must occur at least two times per week in a three-month period to meet the definition.

Removal of calories to prevent weight gain is most commonly achieved by induction of vomiting by irritating the back of the throat with a finger. In severe cases, those with bulimia may not need to induce vomiting and it becomes almost reflexive and food regurgitates automatically. Bulimics also employ laxatives or diuretics to stimulate rapid bowel movements or loss of fluid weight via urine. In addition to physically removing the binged food from the body, bulimics may also exercise to eliminate calories.

A bulimic patient usually has a normal body weight, or may be slightly overweight, which is a marked difference between bulimia and anorexia. The physical consequences of bulimia include acute gastric dilation during a binge episode, with acute and severe abdominal pain, which may lead to gastric rupture if untreated. Frequent vomiting may lead to erosion of the teeth and dental caries, tooth discoloration and increased temperature sensitivity, the patient's knuckles and fingers may have lesions or scratches from inducing vomiting. Vomiting may also cause irritation of the esophagus, causing esophagitis or esophageal tears. The parotid glands, or salivary glands located in front of the ears, may swell secondary to constant irritation from vomiting. Electrolyte imbalances such as low potassium may also result from the constant vomiting, laxative, and diuretic use. Laxative abuse may also lead to irritation of the intestinal lining and rapid transit of stool through the intestines, leading to pain, the development of hemorrhoids, rectal bleeding, or rectal prolapse. Dehydration may lead to dizziness, fainting, and thirst.

Women who are affected by bulimia nervosa have amenorrhea, or lack of menstrual cycles and subsequent infertility. If they do conceive, they are at higher risk of having a miscarriage.

TREATMENT

As with all eating disorders, treatment for bulimia nervosa involves an interdisciplinary care approach. This includes medical personnel, dieticians with experience with bulimia, and mental health professionals. For bulimia in particular, a combination of antidepressant medication and psychotherapy in combination with cognitive behavioral therapy has been shown to be the most efficacious. In contrast to anorexia nervosa, most treatment for bulimia can be managed on an outpatient basis. The medical staff

can manage acute electrolyte abnormalities and dehydration secondary to frequent vomiting. They can also monitor weekly weigh-ins to assess weight gain and monitor vital signs. The dietitian can provide education about a healthy diet and can work with the patient to develop behavior change around eating habits. They can establish caloric requirements and can help to determine goal weight. The mental health professional works with both the patient and his or her family to address underlying issues that may have precipitated the eating disorder. The mental health provider can address comorbid affective disorders, such as depression, which often accompany eating disorders, and can screen for self-injurious behavior, which is common in those with eating disorders. Medical and mental health professionals must also address comorbidities, such as alcohol abuse.

Cognitive behavioral therapy emphasizes the relationship between thoughts and feelings to behavior, and helps the patient to recognize cues that can trigger binge eating. This can assist in managing anxiety, and can lead to the development of coping strategies. One facet of the disorder that should be addressed is the feeling of loss of control that accompanies the bingeing sessions, as well as the self-deprecating thoughts and distorted body image. The second stage of treatment involves understanding balanced and healthy eating habits. The final goal of cognitive behavioral therapy involves the maintenance of new, learned eating behavior. Therapy for bulimia has been conducted successfully on an individual basis and in group therapy, and may be used via outpatient treatment or in daytime therapy sessions.

In combination with the cognitive behavioral therapy and psychotherapy, medication has been shown to be more successful in the treatment of bulimia nervosa than other eating disorders. Pharmacotherapy often consists of antidepressants, such as tricyclic antidepressants and selective serotonin reuptake inhibitors. Antipsychotic medicines are also used, though in lower doses than that used to treat schizophrenia. In addition, the antiepileptic medicine topiramate and the selective serotonin antagonist ondansetron may also be helpful in reducing the urge to binge eat.

The hospitalization of patients with bulimia is considered in cases of severe malnutrition, dehydration, or electrolyte abnormalities; instability in vital signs such as heart rate or rhythm; arrest of normal growth, puberty, or development; uncontrollable bingeing or purging; and medical complications such as seizures or psychiatric emergencies such as suicidal ideation.

MORBIDITY AND MORTALITY

Little data exist on the long-term recovery and survival of a person with bulimia nervosa. It has been suggested that as the follow-up treatment time extends, there is a decline in the proportion of women that meet criteria for bulimia. Nearly half of all bulimic patients will remain symptomatic at 6 years after diagnosis, and in studies with 10 years of follow-up, 30 percent of women continue to binge and purge. More recent data also suggest that for bulimia nervosa, a significant factor in long-term mortality is the incidence of suicide.

SEE ALSO: Anorexia Nervosa; Eating Disorders and Athletes; Eating Disorders and Gender; Eating Disorders and Obesity; Eating Disorders in School Children; Sexual Abuse and Eating Disorders.

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Caffeine

CAFFEINE IS ONE of the oldest drugs. It is one of the most commonly used psychoactive drugs in the world, with approximately 80 percent of adult individuals consuming caffeine-containing foods and beverages. The most popular forms of caffeine are coffee, tea, cocoa, and cola drinks. Currently, caffeine consumption may have benefits for performance and safety at work.

Coffee is a complex mixture of chemicals that provides significant amounts of chlorogenic acid and caffeine. Coffee is a powerful stimulant. For adults consuming moderate amounts of coffee (3–4 cups/d providing 300–400 mg/d of caffeine), there is little evidence of health risks and some evidence of health benefits. Unfiltered coffee is a significant source of cafestol and kahweol, which are diterpenes that have been implicated in the cholesterol-raising effects of coffee. In addition, coffee consumption may help prevent several chronic diseases, including Type 2 diabetes mellitus, Parkinson's disease, and liver disease (cirrhosis and hepatocellular carcinoma).

Caffeine may cause a wide range of side effects, either due to abuse or due to abrupt discontinuation of its use. Some groups, including people with hypertension, children, adolescents, and the elderly may be more vulnerable to the adverse effects of caffeine. Pregnant women may need to limit coffee consumption to three cups per day providing no more than 300 mg/d of caffeine. This is to exclude any increased probability of spontaneous abortion or impaired fetal growth. Also, caffeine and caffeinated coffee have been shown to acutely increase blood pressure and thereby pose a health threat to persons with cardiovascular disease risk.

The global prevalence of obesity has increased considerably in the last decade. Tools for obesity management, including caffeine, ephedrine, and green tea have been proposed as strategies for weight loss and weight maintenance, because they may increase energy expenditure and have been proposed to counteract the decrease in metabolic rate present during weight loss.

Caffeine consumption is associated with a substantial reduction in insulin-mediated glucose uptake independent of obesity, Type 2 diabetes mellitus, and chronic exercise.

Increases in caffeine intake may lead to a small reduction in long-term weight gain. For example, high caffeine intake was associated with weight loss through thermogenesis and fat oxidation and with suppressed leptin in women. Improved weight maintenance was also seen in habitual low caffeine consumers, using a green tea–caffeine mixture. This was achieved partly through thermogenesis and fat oxidation.

Caffeine may cause a wide range of side effects, either due to abuse or due to abrupt discontinuation of its use.

A combination of caffeine and ephedrine has shown to be effective in long-term weight management, likely due to different mechanisms that may operate synergistically. However, adverse effects of ephedrine prevent the feasibility of this approach. Positive effects on body-weight management have been shown using green tea mixtures. Green tea, by containing both tea catechins and caffeine, may act through inhibition of catechol O-methyl-transferase, and inhibition of phosphodiesterase. The mechanisms may operate synergistically. In addition, tea catechins have antiangiogenic properties that may prevent development of overweight and obesity.

SEE ALSO: Addictive Behaviors; Amphetamines.

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Calcium and Dairy Products

WEIGHT-CONSCIOUS PEOPLE have often shied away from dairy products for fear of weight gain. Many, however, have changed their opinion in recent years because dairy products are high in calcium, and recent evidence suggests that calcium intake might have benefits for weight regulation. A current area of interest, and controversy, among dieters and researchers alike is the potential association between increased intake of dietary calcium and decreased body weight and improvements in body composition (fat vs. lean). In numerous studies, calcium intake has been negatively associated with body weight, body mass index (BMI; weight corrected for height), and body fat in both adults and children.

Additional studies have found a negative relationship between calcium intake and the odds of being obese, waist circumference, and abdominal adipose tissue. Recent evidence also suggests that calcium may aid in weight loss maintenance. Interestingly, dietary calcium from food sources such as dairy prod-

ucts has been found to be more effective for weight control than supplemental calcium.

CALCIUM CONTROVERSY

Despite research revealing the calcium might have beneficial effects for weight regulation, many scientists are not convinced. Several studies supporting calcium claims have been funded by the National Dairy Council and resulted in patents related to the use of calcium for weight loss. These potential conflicts of interest do not invalidate results from this body of research; however, attempts to replicate many of the original findings have met with mixed results. Several recent studies of calcium, in both adults and children, have revealed no effects of dietary or supplemental calcium on body weight, BMI, or body composition. The debate currently continues with staunch advocates on both sides.

CALCIUM IN RELATION TO CALORIC INTAKE

As caloric intake increases, people tend to ingest a greater variety of foods, many containing dietary calcium. With the same amount of energy expenditure, increasing caloric intake increases body weight. However, although not unequivocal, much of the calcium literature suggests that increasing dietary calcium intake may oppose the effects of greater caloric intake. This means that dietary calcium and caloric intake may be positively correlated with one another, but might have opposing effects on how they influence body weight. Further research is necessary before definitive conclusions can be drawn.

DAIRY AND DIETING

Regardless of whether they contribute to weight loss, dairy products such as milk, cheese, and yo-

The benefits of calcium from milk: In a variety of studies, calcium has been shown to be associated with lower body weight, body fat, and body mass index, and calcium intake may assist in maintaining weight loss.

Cutting out calcium-rich foods, such as these fine cheeses, above, may cause more harm than benefit in pursuing a diet.

gurt contain essential nutrients, protein, zinc, B-vitamins, and calcium, which has well known benefits for the prevention of osteoporosis. Cutting out these foods due to fat content may be more harmful than beneficial.

In addition, no- and low-fat dairy options are readily available providing the same, or more, nutrients than their full-fat counterparts. Given the nutrient content, possible anti-obesity effects, and relative safety of low-fat dairy products, there is no convincing scientific evidence to exclude these foods while dieting.

SEE ALSO: Body Mass Index; Osteoporosis; Supplements and Obesity.

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Caloric Restriction

CALORIC RESTRICTION IS a dietary method that has been suggested to promote longevity. Followers of caloric restriction consume all their essential micronutrients (i.e., vitamins and minerals) but reduce their consumption of calories by 30 to 40 percent. The first study to suggest that caloric restriction could extend life was from 1935.

In this study, rats that followed a caloric restriction diet lived nearly twice as long as the rats that were allowed to consume as much food as they wanted. Since this study, there have been numerous animal studies that have examined how caloric restriction may promote longevity. Caloric restriction is associated with reduced incidence of heart disease, cancers, diabetes mellitus, neurodegenerative disorders (e.g., Alzheimer's disease), and autoimmune disorders (e.g., Parkinson's disease).The benefits of caloric restriction appear to be mediated through three different aspects of metabolism: changes in hormones, physiological factors, and biochemical factors. Animals that follow caloric restriction had a decrease in their insulin levels, thyroid hormone, and sex hormones (i.e., testosterone). Some of the physiological changes that are associated with caloric restriction include a decrease in body temperature and blood pressure. Additionally, the amount of fat and muscle both decrease. Some of the biochemical changes to the body include a decrease in blood glucose (sugar) and lipids. Studies have also demonstrated that there is a decrease in oxidative damage to the body because there is less oxidation of foods occurring. Finally, there is a decrease in the expression of genes that are related to aging and inflammation in the body.

While there have been many caloric restriction studies in animals (e.g., rats, mice, flies, earthworms, and monkeys), caloric restriction studies

in humans are incredibly scarce. These studies are very laborious and take a long time to achieve an outcome. However, there have been some opportunities to study longevity in humans. One example is from Okinawan population, the southernmost island region in Japan. Resident from this region have the largest portion of their population over 100 years of age and have significantly lower rates of heart disease and cancer compared to the rest of Japan as well as the United States. Okinawans consume a diet that has all of the essential vitamins and minerals but is still nearly 40 percent fewer calories than an American diet. Another example was gained during the Biosphere 2 study. During this experiment, the eight scientists had decreased crop production and subsequently consumed 30 percent fewer calories than they thought they would. After the experiment was completed, these scientists had a metabolic profile similar to animals that had undergone caloric restriction (i.e., lower insulin, lower blood pressure, etc.).

There are several side effects that have been documented from caloric restriction. These have been observed in the above-mentioned studies as well as in some starvation experiments from the 1950s. Some of the negative effects of caloric restriction include very low blood pressure, irregular menstrual cycles, infertility, osteoporosis, extreme coldness, decreased energy, extended time for injuries to heal, depression, and irritability.

Given the difficulty of achieving true caloric restriction in humans, some alternatives have been suggested. Some studies have shown that following caloric restriction of just 8 percent had some of the longevity benefits with decreased amounts of side effects. Also, there has been interest by the

In addition to helping control weight, caloric restriction can also reduce incidence of cancer, Alzheimer's disease, and diabetes. While there have been many caloric restriction studies in animals, studies in humans are scarce.

pharmaceutical industry to develop caloric restriction mimetics.

These would be drugs that can act on the body as if the body had reduced food intake. One of the first compounds tested in animals was 2-deoxyglucose. This compound is a glucose analogue (similar to the sugar molecule), but does not get metabolized in the same way. Rats that were given 2-deoxyglucose for 6 months had a decrease in their insulin levels and body temperature despite no change in the amount of food they ate. Antioxidants such as resveratrol are also being studied to determine if they can produce some of the favorable metabolic effects associated with caloric restriction.

SEE ALSO: Dieting: Good or Bad?; Food Intake Patterns.

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Cannabinoid System

CANNABINOIDS ARE A group of related compounds found in the plant *Cannabis sativa*. The major psychologically active component in marijuana is Δ9-tetrahydrocannabinol (THC). THC acts on mammalian cannabinoid receptors to elicit various responses. Mammals also have endogenous molecules (termed *endocannabinoids*) that activate the same cannabinoid receptors as THC. The endogenous cannabinoid system in mammals thus includes the cannabinoid receptors, endocannabinoids, and enzymes involved in synthesizing and degrading endocannabinoids.

There are 70 known cannabinoids in *Cannabis sativa*. Marijuana refers to the dried leaves and flowering tops of the female hemp plant. Marijuana smoking can elicit a variety of psychological and physiological effects. The physiological effect is best exemplified by increased heart rate and increased appetite. The psychological effects are highly subjective experience, and the experience varies enormously depending on many variables such as dose, environmental setting, and prior experience. Common effects include euphoria, diminished psychomotor performance (coordination of cognitive process and motor activity), and impairment of short-term memory. Some people have reported dysphoria and a change in time perception. The most psychologically active component in marijuana is THC. Other cannabinoids may have effects different from those of THC.

The mechanism of action of THC was clarified when a brain receptor was found to be activated by THC in 1990. This brain receptor was termed *cannabinoid-1 receptor* (CB1R), and is present in the brain of humans, mammals, and vertebrates. A second receptor, cannabinoid-2 receptor (CB2R), was found most predominantly in immune cells. In 1992 and 1995, two molecules with cannabinoid-like activity were identified in mammalian brain and gastrointestinal tract. These endogenous molecules were named anandamide and 2-arachidonyl glycerol, and both activate CB1R and CB2R in a manner analogous to THC.

In the nervous system, endocannabinoids are made from membrane precursors when specific neurons are activated. Anandamide and 2-arachidonyl glycerol are synthesized at the cell body of neurons through two separate biosynthetic pathways, and are degraded by different enzymes, respectively. When endocannabinoids are synthesized, they serve as retrograde neuromodulators, diffuse across the synapse and act on CB1R to inhibit neurotransmission. Thus, endocannabinoids are feedback molecules that dampen both excitatory and inhibitory transmissions if CB1R is expressed in those nerve terminals.

The involvement of the endocannabinoid system in energy balance and feeding control is well supported by the appetite-enhancing effect of marijuana smoking. In addition, endocannabinoid system interacts with other brain mechanisms to exert influence on energy balance. The synthesis of endocannabinoids is inhibited by leptin, indicating that endocannabinoids serve as one of the downstream effectors of leptin action. CB1R is co-expressed with several neuropep-

tides, such as melanin-concentrating hormone and orexin, which are known to affect food intake and energy expenditure. Furthermore, the endocannabinoid system can also exert its effect in parallel with other systems, leading to an additive or synergistic influence on energy balance.

Several experimental medications are in clinical development. These include inverse agonists such as rimonabant, taranabant, and SLV319 for the treatment of obesity.

SEE ALSO: Leptin; Rimonabant.

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Carbohydrate "Addictions"

WHILE NOT LISTED in the official diagnostic manual of the American Psychiatric Association (*Diagnostic and Statistical Manual,* 4th edition [*DSM-IV*]), addiction to carbohydrates has found its way into the vocabulary and psyche of the American consumer. This is partly fueled by popular diet plans that recommend limiting or avoiding carbohydrates as a way of promoting weight loss through reduced food intake. There are many mechanistic problems, however, with the concept and existence of an addiction to carbohydrates, although many people experience an intense desire for carbohydrate consumption similar to that for abused drugs.

The first problem with defining a desire for carbohydrate as an addiction comes from the fact that all living creatures require food, and many available foods found in nature contain carbohydrates. Therefore, you could say everyone is addicted to carbohydrates in some respect.

In order to ensure sufficient food intake for survival, the body has special systems that detect the levels of needed nutrients and signal the brain that it is time to eat and replenish the body's nutrient supply. These "time to eat" systems are countered by other systems that signal to the brain that it is "time to stop" eating. Aberrations in either the "eat" or "stop eating" systems can cause overconsumption of food with respect to the amount needed to maintain a healthy weight. This overconsumption of food can form the basis for the experience of "loss of control" over eating.

The second problem with defining a desire for carbohydrate as an addiction lies in the composition of carbohydrate foods people usually say they are addicted to, namely, ice cream, cookies, cakes, pies, chips, potatoes, pasta, and pizza.

All of these foods contain fat and a small amount of protein as well as carbohydrate. Animal studies have shown that carbohydrate/fat containing foods, but not solely carbohydrate foods, cause an increase in the body's natural opioids, suggesting that the "addiction," if it exists, may be to the fat, not the carbohydrate. If the carbohydrate in "high carbohydrate" foods is not

While not an addiction in the strictest sense, many people experience intense cravings for carbohydrate-rich foods.

the "addictive" agent, can it still contribute to the feeling of "loss of control" experienced when eating these foods? The answer is yes.

The sweetness of some carbohydrate foods helps promote release of dopamine, a neurotransmitter of the brain reward circuitry. This is perceived as a "time to eat" signal. Another source of "loss of control" with respect to carbohydrate in obese individuals lies in changes in the body's post-ingestive responses to carbohydrate.

STOP-EATING SIGNALS

There is increased stomach capacity in the obese, which requires a greater amount of food to fill the stomach and activate one of the "stop eating" signals. Although many carbohydrate foods contain fat, consuming fat in an obese individual does not result in sufficient release of hormones that signal fullness (another "stop eating" signal). In addition, many obese individuals are resistant to insulin and leptin (other satiety hormones) signaling not only in the periphery but also in the brain. Peripheral insulin resistance creates a pseudo-hypoglycemic condition in which blood glucose (from carbohydrates) is high, but intracellular glucose is low.

This can be perceived by the brain as a "time to eat" signal, and can contribute to a vicious cycle of continuous eating but no feeling of satiety (satisfaction/fullness). An additional source of "loss of control" can occur in individuals who are experiencing stress or other causes of depressed mood; this carbohydrate-induced dopamine release can cause a transient and temporary relief of the stress or depression. The temporary relief of stress promotes conditioned learning that underscores the development of a craving for the carbohydrate during some future period of stress.

SEE ALSO: Addictive Behaviors; "Food Addictions"; Food and Mood.

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Carbohydrate and Protein Intake

IN THE PAST, hypocaloric diets to promote weight loss in the obese patient focused mainly on reducing total fat or saturated fat intake. More recently, interest has shifted to the role that dietary carbohydrates and protein play in the weight loss diet. Emerging research is suggesting that a higher protein intake, as well as a shift from simple to complex carbohydrates, with an increased intake of dietary fiber, may be a more practical approach for weight loss and weight management.

Carbohydrates are the preferred source of energy in the human diet, yielding about four calories per gram. They are usually classified into two categories, which include simple and complex forms. Simple carbohydrates include sugars, while complex carbohydrates include starches and fiber. Some evidence suggests that eating pattern changes may be one of the causes for the increased prevalence of obesity. These eating pattern changes are characterized especially by the increase in the dietary intake of simple sugars, such as sucrose and high-fructose corn syrup

Some regimens like the Atkins Diet shun carbohydrates, but many "carbs" may be beneficial for weight loss and maintenance.

and refined grain products, along with a decreased intake of fiber.

Increasing the intake of fiber-rich carbohydrates, such as whole grains, beans, fruits, and vegetables, may be beneficial in the treatment of obesity. Fiber differs from simple sugars and starches because our body cannot digest it. Because dietary fiber does not provide any calories to the diet, a meal that is high in fiber is usually less energy dense. This may bring on a feeling of satiety due to the added bulk and slower absorption from the indigestible plant materials. Many fiber-containing foods are also rich in micronutrients such as vitamins, minerals, and phytochemicals, which may be of benefit as well. Additionally, fiber has been shown to lower blood cholesterol levels and insulin secretion, and to prevent and manage constipation and diverticulosis, which are complications often associated with obesity.

Similar to carbohydrates, protein yields about four calories per gram. Protein is an essential nutrient needed for growth and development, and is a component of virtually every living cell in the human body. Proteins are composed of amino acids, of which nine are essential, meaning that they cannot be synthesized in the body, and have to come from food sources. Proteins in food that supply all of the essential amino acids are called complete proteins. Most complete proteins come from animal sources, with soy being the only plant-based complete protein.

Recent research suggests that an increase in dietary protein intake may be beneficial in weight loss and weight management. Higher protein diets increase satiety and may increase the obese patient's adherence to a hypocaloric diet. Higher protein diets may also lead to a higher percentage of fat loss, spare muscle tissue during weight loss. In dietary practice, a shift to replace some simple and refined carbohydrates in the diet with high-quality, complete sources of protein that are low in saturated fat may be beneficial.

As part of a healthy lifestyle, a diet for weight management should focus on a low intake of simple sugars and refined carbohydrates, and a high intake of high fiber complex carbohydrates such as whole-grain products, vegetables, and beans. Currently, most American adults are consuming less than half of the recommended dietary fiber intake of 20 to 35 grams per day. Americans are receiving about 15 percent of their dietary energy each day as protein, which is considered adequate. Most studies that focus on moderately high protein intake for weight loss and maintenance suggest about 30 percent of the daily caloric intake should come from protein. The role of higher protein diets to promote weight loss is generally well accepted; however, their longer-term role in weight management is not clear because studies investigating their safety and efficacy have yet to be reported.

SEE ALSO: Fat Intake; Overall Diet Quality.

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Cardiovascular Disease in African Americans

CARDIOVASCULAR DISEASE (CVD) is a result of numerous risk factors, many of which are behavioral in nature such as smoking, diet, and lack of regular exercise. Others include preexisting medical conditions such as metabolic syndrome (a group of metabolic risk factors including hypertension, abdominal obesity, poor regulation of blood lipids, and insulin resistance). The relationship among these various factors is complex; however, it is apparent that African Americans are disproportionately affected by each, placing them at greater risk for CVD.

In 2004, cardiovascular disease was responsible for 27 percent of all deaths in the United States. However, the CVD death rate (per 100,000) among white males was significantly less than the rate reported among black males (335.7 and 448.9, respectively). Differences were also evident between white and black females (239.3 versus 331.6, respectively). CVD accounted for 32.7 percent of deaths among African-American males and 40 percent of all deaths among African-American

The prevalence of cardiovascular disease among African Americans is due to environmental and psychosocial elements that influence risk-taking behaviors, belief systems, and healthcare utilization.

females. Disparate estimates place African Americans at greater risk of dying from cardiovascular disease compared to other racial/ethic groups.

Racial disparities with regard to CVD are due to a unique constellation of environmental and psychosocial elements that influence risk-taking behaviors, belief systems, and healthcare utilization. These elements, coupled with targeted media campaigns designed to promote smoking and intake of high-energy foods, predispose African Americans to a number of comorbidities that lead to CVD. For example, research indicates that blacks in the United States are at greater risk of developing diabetes compared with non-Hispanic whites. Subsequently, cardiovascular disease is reported as a leading cause of death among those with diabetes. The threats of CVD and diabetes intensify given that both conditions are more common among those who are overweight or obese, with minorities representing a larger percentage of individuals with weight-related health conditions. Among Americans 20 years of age or over, 67 percent of non-Hispanic blacks are considered overweight or obese, while 75 percent of Mexican Americans are so classified. Minority females tend to be at greater risk of CVD with 73 percent of Mexican Americans and 80 percent of African-American females with body mass index estimates greater than 25. The threat of CVD is exacerbated given the increasing trend toward higher percentages of black children who become overweight or obese. Additional risk factors specific to CVD include hypertension, smoking, and lack of physical activity, each of which tend to be more prominent among African Americans.

To curtail the threat of CVD among blacks, a number of approaches have been introduced, each primarily guided by objectives outlined in Healthy People 2010. Education is a strong preventative with regard to CVD as well as associated risk factors such as metabolic syndrome.

SEE ALSO: Body Mass Index; Centers for Disease Control and Prevention; Prevention.

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Cardiovascular Disease in Asian Americans

AMONG ASIAN-AMERICAN men, cardiovascular disease is the number one cause of death, followed by cancer. Among Asian-American women, cancer is the number one cause of death followed by heart disease and stroke. And within the Asian-American group, stroke is the leading cause of death among Chinese, Filipino, and Japanese. Although stroke is the leading cause of death for many Asian-American subgroups, there is a lack of information and data about risk factors in these subgroups and additional research is needed. Moreover, even though cardiovascular disease represents one of the major causes of death for Asian Americans, there are disparities that persist for Asian Americans, especially among limited English speakers who may have less access to information on the importance of preventative screenings and also lack of information on the medical advancements made in this area. Because of the lack of access to information, many Asian Americans are unaware of their risk for cardiovascular disease.

The main risk factors for cardiovascular disease are high blood pressure, high blood cholesterol, cigarette smoking, obesity, lack of physical activity, and diabetes. Because most Asian Americans are not aware that they are hypertensive, they are less likely to seek care, compared to non-Asian Americans. In addition, for Asian Americans, blood pressure levels tend to vary by age and by ethnic sub-group. Filipino Americans, as a group, have significantly higher levels of high blood pressure. This has been attributed to their high-sodium diet.

High cholesterol increases an individual's risk for coronary heart disease. Studies on serum cholesterol levels of Asian Americans are limited. Moreover, only a limited number of studies have investigated cholesterol levels of Asian Americans. The limited studies that do exist show that Asian Americans do not regularly get blood cholesterol exams. Southeast Asians are the least likely to get their blood cholesterol checked. At the same time, the cholesterol levels of Asians living in the United States are significantly higher than those living in Asia.

In addition to the above-mentioned risk factors, cigarette smoking is also a known risk factor for cardiovascular disease. Asian-American men aged 22–44 tend to have the highest prevalence smoking rates compared with other races, gender, and age groups. Among Asian ethnic groups, Chinese adults have the lowest smoking rates. In California, Korean men have extremely high smoking rates. According to various local surveys and studies, there is widespread cigarette advertising in Asian-American communities. A lack of physical activity also contributes to the development of cardiovascular disease.

While obesity is less pronounced in Asian Americans, cardiovascular disease and stroke are serious problems.

MIGRATION PATTERNS

Obesity has also been linked to cardiovascular disease. For Asian Americans, obesity is directly linked to migration patterns with the individual's length of stay in the United States increasing their likelihood to be overweight or obese. The longer an individual has resided in the United States, the greater his or her chances of eating unhealthy foods high in fat. For

immigrants from Asian countries, many adapt to the new Western lifestyle by consuming great amounts of high-fat and high-calorie foods such as fatty meats, dairy products, and processed snacks and desserts. All of these take the place of traditional meals of rice, fish, and vegetables.

SEE ALSO: Asian Americans; Body Fat Distribution in Asian Americans; Hypertension in Asian Americans;

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Cardiovascular Disease in Hispanic Americans

CARDIOVASCULAR DISEASE (CVD) is the leading cause of death and major cause of disability across racial and ethnic minority groups in the United States. Close to a third of deaths are attributed to CVD. Data compiled by the National Center of Health Statistics revealed that in 2004, among adult Hispanic Americans (18 years of age or older), 9.2 percent have heart disease, 6 percent have coronary heart disease, 19.6 percent have hypertension, and 2.8 percent have stroke. Among Mexican Americans age 20 and older who make up the largest share of the U.S. Hispanic population, 32 percent of men and 34 percent of women have CVD.

There is evidence suggesting that Hispanics are less likely to die from CVD than non-Hispanic whites (Caucasians). According to the Office of Minority Health of the Department of Health and Human Services, in 2004, Hispanic men were 30 percent less likely to die from heart disease relative to Caucasian men, and in 2005, Hispanics were 10 percent less likely to have heart disease, compared to non-Hispanic Caucasians.

The attributed lower risk of CVD among Hispanics occurs in despite that they are more likely to have lower socioeconomic status than Caucasians. Socioeconomic indicators such as poverty and low educational attainment are risk factors that influence CVD patterns. However, there are some protective factors that may compensate for the socioeconomic disadvantage suffered by Hispanics. Those related to their culture and ethnicity, including their strong informal support networks and familism, are associated with better CVD outcomes among this ethnic group.

However these better CVD outcomes among Hispanics seems to be changing, getting closer to those observed among whites, partially explained by the demographic changes experienced by Hispanics, particularly their growing in numbers, their assimilation into the American culture and the fact that this group, as the rest of the population, is living longer than before.

Moreover, there are health-related risk factors associated with CVD that affect more Hispanics than Caucasians. Rates of overweight and obesity, two of the leading factors for CVD, are higher among Hispanics relative to Caucasians. Mexican-American women, for example, are 1.2 times more likely to be obese than Caucasian women. Leisure-time physical inactivity is also more prevalent among Hispanics than Caucasians. Among Hispanic adults age 18 and older, 32 percent of men and 27 percent of women report no leisure-time physical activity.

Hispanics are the largest single minority ethnic group in the United States, constituting 14 percent of the total population. According to 2005 estimates from the Census Bureau, there were 43 million Hispanics, excluding 4 million in Puerto Rico. And the increase of the Hispanic population will continue at a fast rate. By 2050, they will constitute 24 percent of the total population, with a projection set at about 103 million people. This is a population group with specific socioeconomic and cultural characteristics, which need to be taken into consideration when addressing their increasing risk for CVD. Health pro-

fessionals need to understand and control the patterns associated with ethnic disparities observed in CVD among Hispanics when compared to the general U.S. population.

SEE ALSO: Congestive Heart Failure; Coronary Heart Disease in Women; Dominican Americans; Ethnic Disparities among Obesity in Women; Ethnic Variations in Obesity-Related Health Risks; Hispanic Americans; Hypertension in Hispanic Americans; Mexican Americans; Puerto Rican Americans.

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CART Peptides

CART PEPTIDES ARE named for early observations that they arise from a cocaine- and amphetamine-regulated transcript. CART peptides are neuropeptides (proteins that signal in the brain) expressed in the brain and other organs. In the past 10 years, CART has been studied in several contexts, including energy balance and body weight regulation.

CART peptides are expressed in many brain areas and neuroendocrine tissues. This includes the hypothalamus, pituitary gland, gastrointestinal tract, adrenal gland, and pancreas. CART is synthesized as a larger, inactive precursor (pro-CART) that is cleaved to yield smaller, active forms. As with other neuropeptides, CART functions depend on the neurons in which they are expressed and how those neurons communicate with other specific tissues or neural circuits. CART peptides may serve as neuromodulators (regulatory proteins) in multiple systems.

CART peptides can decrease short-term food intake when administered directly into the brain in animal models. Chronic brain infusions of CART produce lasting weight-loss beyond the short-lived hypophagia (undereating). Brain sites governing these effects are being investigated. For example, CART expression in the arcuate nucleus, a key hypothalamic site for processing body signals of energy status, is upregulated by leptin, a hormone made and secreted by fat cells. These CART-containing neurons can release the neuropeptide at second-order relays in hypothalamus and elsewhere that carry out CART's catabolic effects.

Some of CART's effects on energy balance and body weight may be secondary to effects on systems whose primary functions are not specifically to regulate appetite. For example, CART peptides injected into some brain areas involved in dopamine activation of motivated behaviors (which include drug-seeking and sexual behaviors, as well as food and water intake) can alter food intake, suggesting that CART can modulate the rewarding qualities of food. CART also appears to be involved in brain-to-adrenal and brain-to-thyroid axes that regulate physiologic responses to stress, body temperature, and metabolic rate, with much potential for associated effects on body weight and adiposity.

Little is known about putative CART receptors. They have not been identified or cloned, and therefore, there is currently a lack of pharmacological tools for CART research. It is anticipated that when these obstacles are surmounted, the potential for CART-based antiobesity and other therapies may emerge.

SEE ALSO: Addictive Behaviors; Amphetamines; Anxiety; Appetite Control; Appetite Signals; Central Nervous System; Cortisol; Dopamine; Food Reward; Leptin; Neuropeptides; Stress; Sympathetic Nervous System.

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Caucasians

OVERWEIGHT AND OBESITY have been rapidly increasing in both children and adults in the United States and worldwide over the last 40 years. This increase is of major medical and public health concern because obesity is associated with premature mortality and many types of morbidity, including heart disease, stroke, diabetes, some types of cancer, and arthritis. Many explanations have been offered for the increase, which some have termed an epidemic: modern life which has removed most physical activity from people's lives, the increasing consumption of fast food high in calories and fat, greater acceptance of large body types, and even increased television viewing. It is notable that obesity has been increasing across all racial and ethnic groups, but not evenly, in the United States; Caucasian Americans generally have lower rates of overweight and obesity than do members of minority groups such as African Americans and Hispanics. Many explanations have been offered for this phenomenon as well, ranging from the socioeconomic (people with greater social capital have more opportunities to buy healthy foods and exercise, and more knowledge of the importance of both activities) to the genetic.

DEFINITIONS

Caucasian, or white Americans are defined by the U.S. Census Bureau as people whose origins may be traced to any of the original peoples of Europe, the Middle East, or North Africa. According to Census 2000, on April 1, 2000, there were 216.9 million Caucasians in the United States, representing 77.1 percent of the total population. Of these, 211.5 million reported only one race, while 5.5 million reported Caucasian along with one or more other races. Not surprisingly, because white Americans are the majority racial/ethnic group, the states with the largest populations (California, New York, and Texas) also have the largest number of white Americans. However, the states with the highest percentage of white Americans are smaller, such as Vermont (98.6 percent), Maine (98.4 percent), New Hampshire (98.0 percent), and Iowa (96.6 percent).

The Census and other federal surveys also collect information on ethnicity, using two categories: "Hispanic or Latino" and "Not Hispanic or Latino." A person who is Hispanic or Latino is defined as being a person of Cuban, Mexican, Puerto Rican, South or Central American, or other Spanish culture or origin. Race and ethnicity are considered distinct categorizations by the federal system, so a Hispanic or Latino person may be of any race, and decisions about when to include, for instance, White Hispanics in the white total for any particular survey or study is a decision left to the researcher. According to Census 2000, 92 percent of the white population was non-Hispanic, and the white non-Hispanic population represented 70 percent of the total U.S. population.

Because white Americans constitute the majority of the U.S. population, studies are seldom done specifically examining their health or health behaviors. Instead, research studies or surveys of the population as a whole are generally assumed to apply to white Americans, and targeted studies are more likely to look at minority groups such as African Americans or Asian Americans. There has also been relatively little interest in studying different ethnic groups within the white classification (e.g., Italians versus Swedes), as there has been, for instance, with Hispanic, Asian, or American Indian subgroups based on national origin or tribal identification.

This creates something of a paradox because although white Americans are certainly the most studied group of people in the United States, seldom are studies identified as applying specifically to white people or to specific white subgroups (as would typically be done with, for instance, studies based on

samples of African Americans or Korean Americans). In addition, review articles are seldom written summarizing what is known about the health of the white population, risk factors specific to white people, or to specific ethnic or cultural groups classified as white. Therefore, while it is possible to speak on a national basis about, for instance, obesity rates among white Americans, more detailed breakdowns by national origin may be difficult to locate.

OBESITY RATES

According to the 2005 National Health Interview Survey (NHIS), 35.3 percent of American adults (age 18 and older) are overweight (body mass index [BMI] ≥25), and 24.7 percent are obese (BMI ≥30). More men than women are overweight, while approximately equal numbers are obese: 42.6 percent of men and 27.9 percent of women are overweight, while 24.9 percent of men and 24.3 percent of women are obese. Overweight and obesity are not evenly distributed across racial/ethnic groups, with differences in obesity rates particularly striking. As with many health-related conditions, Asian Americans have the lowest rates of both overweight (27.9 percent) and obesity (8.5 percent), while white Americans generally score equal or better in health factors compared to the other racial groups. The overweight rate for white Americans is 35.5 percent overweight and 24.1 percent obese, compared to American Indians and Alaska Natives (AI/ANs) with 38.6 percent overweight and 37.6 percent obese, African Americans with 35.4 percent overweight and 32.4 percent obese, and Native Hawaiian and Pacific Islanders (NH/PIs) with 34.0 percent overweight and 26.9 percent obese.

Considering Hispanic/Latino as a separate category produces a slightly different breakdown. Hispanics who are overweight constitute 39.6 percent, compared to 34.8 percent of non-Hispanic whites and 35.1 percent of non-Hispanic blacks; 27.5 percent of Hispanics are obese, compared to 23.5 percent of non-Hispanic whites and 32.5 percent of non-Hispanic blacks.

Overweight and obesity also differ among men and women: 42.6 percent of men are overweight, as compared to 27.9 percent of women, while 24.9 percent of men and 24.3 percent of women are obese. When the population is broken down by racial and ethnic groups, however, this pattern is not entirely consistent. For non-Hispanic white Americans, the pattern is similar to that found in the general population: 42.9 percent of men and 26.9 percent of women are overweight, and 25.3 percent of men and 21.7 percent of women are obese. Among non-Hispanic blacks and Hispanics, however, women are more likely to be obese: 47.3 percent of Hispanic men and 31.7 percent of Hispanic women are overweight, and 23.9 percent of Hispanic men and 31.0 percent of Hispanic women are obese. Among non-Hispanic blacks, 38.0 percent of men and 32.7 percent of women are overweight, while 28.3 percent of men and 35.9 percent of women are obese.

Consideration of the racial or ethnic influence on overweight and obesity cannot be separated from the fact that racial groups differ, on average, on measures of socioeconomic status (SES) which are also associated with many health behaviors and health states. For instance, the 2005 NHIS found, as many previous surveys have also found, an inverse relation of poverty with obesity rates. Slightly over 29 percent of persons classified poor (below the Census Bureau's poverty threshold for the previous year) were most likely to be obese compared to 28.3 percent of the near-poor (those whose income was 100 to 200 percent of the poverty threshold) and 23.6 percent of the nonpoor. Similarly, obesity was higher among those insured by Medicaid (34.1 percent) than among those with private insurance (23.8 percent). Looking at educational levels, people without a high school diploma were most likely to be obese (30.7 percent), while those with a bachelor's degree or higher were least likely to be obese (17.5 percent).

Overweight has been increasing in the American population as a whole for a number of years; this trend is evident among white Americans, although at a lower rate than among African Americans and Hispanics. According to the NHIS (1960–62 data) and National Health and Nutrition Examination Survey (NHANES; later data), in 1960–62, 44.8 percent of adults (aged 20–74) were overweight. This increased in 1971–74 to 47.7 percent, in 1976–80 to 47.4 percent, in 1988–94 to 56.0 percent, and in 2001–2004 to 66.0 percent. In the non-Hispanic white population, men are more likely to be overweight than women; this pattern is reversed in the African-American population, where women are more likely than men to be overweight. In 1976–80, 53.8 percent of white, non-Hispanic men

and 38.7 percent of white, non-Hispanic women were overweight, while for African Americans, the numbers were 51.3 percent and 62.6 percent, respectively. In 1988–94, 61.6 percent of white men and 47.2 percent of white women were overweight, compared to 58.2 percent of African-American men and 68.5 percent of African-American women. In 2001–04, 71.1 percent of white men and 57.1 percent of white women were overweight, compared to 66.8 percent of African-American men and 79.5 percent of African-American women.

Rates of obesity have increased even more rapidly in the U.S. population since 1960, and have been observed across all racial and ethnic groups. In 1960–62, 13.3 percent of the U.S. population were obese; in 1971–74, this increased to 14.6 percent, in 1976–80 to 15.1 percent, in 1988–94 to 23.3 percent, and in 2001–03 to 32.1 percent. In distinction to overweight, women are more likely to be obese than men in both the white and African-American racial categories. In 1976–80, 12.4 percent of white men and 15.4 percent of white women were obese, compared to 16.5 percent of African men and 31.0 percent of African-American women. In 1988–94, 20.7 percent of white men and 23.3 percent of white women were obese, compared to 21.3 percent of African-American men and 39.1 percent of African-American women, and in 2001–04, 31.2 percent of African-American men and 51.6 percent of African-American women were obese.

Overweight and obesity have also been increasing among American children and adolescents. Rates of obesity are lower among white children and adolescents than among members of minority groups, but are still worrying because persistent overweight beginning in childhood is associated with greater morbidity and mortality than adult-onset mortality, overweight or obese children and adolescents are more likely than normal-weight children and adolescents to become obese adults. According to NHANES III, a nationally representative survey conducted 1988–94, 13.2 percent of Caucasian boys (aged 6–11 years) and 11.9 percent of Caucasian girls were overweight, as compared to 14.7 percent of non-Hispanic black boys and 17.9 percent of non-Hispanic black girls, and 18.8 percent of Mexican-American boys and 15.8 percent of Mexican-American girls. Among adolescents (aged 12–17 years), 11.6 percent of non-Hispanic white boys and 9.6 percent of girls were overweight, compared to 12.5 percent of non-Hispanic black boys and 16.3 percent of non-Hispanic black girls, and 15.0 percent of Mexican-American boys and 14.0 percent of Mexican-American girls.

RISK FACTORS

Many studies have identified risk factors for overweight and obesity; among the most salient are lack of sufficient physical activity, and regular consumption of a diet that includes too much fat and processed food and too few whole grains, fruits, and vegetables. This makes intuitive sense because for most people, weight gain is a product of more calories consumed than calories expended over a period of time; lack of physical activity lowers the number of calories expended, while intake of dense, high-fat foods tends to increase the number of calories consumed. Not surprisingly, the distribution of these risk factors parallels the observed rates of overweight and obesity.

According to data from the 2000 NHIS, white adults were the among the least likely to report leading inactive lifestyles and the most likely to report a high rate of physical activity. Only 8.7 percent of white Americans said they were never active, compared to 15.9 percent of African Americans and 13.0 percent of AI/ANs. Nearly 20 percent of whites and 16.3 percent of Asians reported that they were very active, compared to 14.3 of African Americans and 16.0 percent of AI/ANs.

When Hispanics are considered as a separate ethnic group, only 8.5 percent of non-Hispanic whites report inactive lifestyles and 20.4 percent report highly active lifestyles, compared to 11.2 percent of Hispanics who said they were never active while 15.2 percent said they were highly active, and non-Hispanic African Americans, 16.0 percent of whom reported inactive lifestyles and 14.2 percent highly active lifestyles. As has been found in previous studies, the 2000 NHIS found high levels of activity associated with SES factors which are disproportionately distributed among non-white racial and ethnic groups and therefore confounded with genetic and cultural factors; for instance, poverty and lack of education are both negatively associated with a highly active lifestyle.

Consuming a diet high in fruits and vegetables is associated with lowered risks for many chronic dis-

eases and conditions, including heart disease and certain cancers. For this reason, the Centers for Disease Control and Prevention (CDC) recommends consuming at least five servings of fruits and vegetables daily. No studies have specifically examined the relationship between consuming a diet high in fruits and vegetables and effective weight loss or weight control, but a few studies have demonstrated that consuming foods low in energy density and high in fiber (both of which apply to most fruits and vegetables) are related to lower food consumption. In addition, several studies that increased fruit and vegetable consumption for other reasons (such as to prevent intestinal polyps) found that weight maintenance or loss was often a by-product. Data from the 2005 Behavioral Risk Factor Surveillance System (BRFSS) demonstrated that the number of American adults meeting the CDC recommendations was surprisingly low, but that white non-Hispanic Americans had among the highest rates of compliance. In this survey, 17.4 percent of white non-Hispanic Americans reported consuming five or more servings of fruits and vegetables daily, compared to 19.6 percent of Asian Americans, 12.6 percent of non-Hispanic black Americans, and 14.8 percent of Hispanic Americans.

SEE ALSO: Childhood Obesity as a Risk for Adult Overweight; Ethnic Disparities in the Prevalence of Childhood Obesity; Exercise; Fast Food; Fruits and Vegetables.

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CD36 and FAT (Fatty Acid Transporters)

CD36 (KNOWN AS SCARB3, GP88, gpIV, and gpIIIb) is a membrane glycoprotein with different roles, which is found on the surface of different cell types. The functions of CD36 which is called scavenger receptor B include facilitation of fatty acid uptake, acting as a signaling molecule and a receptor for several of ligands (apoptotic cells, modified forms of low density lipoprotein, thrombospondins, fibrillar beta-amyloid, components of Gram-positive bacterial walls, and malaria infected erythrocytes).

This multifunctional receptor is expressed in different cell types such as macrophages, dendritic and endothelial cells, platelets, erythrocytes, monocytes, differentiated adipocytes, mammary epithelial cells, spleen cells, and some skin microdermal endothelial cells and in different tissues such as muscle, heart, and fat.

Circulating low-density lipoprotein (LDL), which is not taken up by the LDL receptors, can enter macrophages via unregulated scavenger such as CD36 (scavenger receptor B). This process requires LDL to be modified by enzymatic, nonoxidative change and oxidation. This will facilitate the accumulation of cholesterol, glycosylation, or glycooxidation. It is confirmed that vitamin E can diminish the uptake of oxidized LDL by decreasing the expression of the CD36 receptor.

Fatty acid translocase (FAT)/CD36 is an 88 kDa fatty acid (FA) transporter protein responsible of regulation of the uptake of fatty acids via the plasma membrane in the heart and skeletal muscle. It is believed that a mutation in FAT/CD36 decreases uptake rate of fatty acids and its overexpression leads to increased fatty acids uptake.

It is evident that defective FAT/CD36 translocating machinery is related to an elevated surface abundance

of FAT/CD36 and increased fatty acid influx into the heart and muscle cells in Type 2 diabetes mellitus.

SEE ALSO: Atherosclerosis; Blood Lipids; Fatty Acid Transport Proteins; LDL Receptors.

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The National Center for Maternal and Child Health promotes a range of initiatives to ensure infant and child wellness.

Center for Maternal and Child Health

THE NATIONAL CENTER for Education in Maternal and Child Health (NCEMCH) is a research entity within the Public Policy Institute of Georgetown University, located in Washington, DC. NCEMCH was founded in 1982 to provide leadership to, and serve as a national resource for the maternal and child health community. The Center, in collaboration with federal agencies, professional organizations, academic institutions, and corporate and philanthropic partners, is involved in three key activities: launching national health initiatives, developing and disseminating culturally competent child health and development materials, and providing a virtual library of maternal and child health information.

Bright Futures, an ongoing national health promotion initiative of the NCEMCH, is a typical NCEMCH project. The purpose of Bright Futures is to improve child health and well-being by focusing on four goals: fostering partnerships between families, health professionals and communities; promoting desired outcomes for children; increasing family participation in health promotion and prevention activities; and fostering culturally and developmentally appropriate healthcare for children. Other initiatives and centers founded by NCEMCH include the Healthy Start National Resource Center, the National Resource Center for Health and Safety in Child Care, the National Maternal and Child Oral Health Center, and the Children's Safety Network.

NCEMCH has produced a number of publications, many of which can be downloaded from the NCEMCH website, concerning child and adolescent health. Topics covered by NCEMCH publications include alcohol, tobacco, and drug use; child care; health insurance;, infant mortality; maternal and child health policy; oral health; and racial and ethnic disparities. The NCEMCH website also provides links to relevant materials from other sources, including a materials in languages other than English.

The Maternal and Child Health Library at Georgetown University provides access, through the NCEMCH Web site, to a variety of information. *MCH Alert*, the Library's free weekly newsletter, is distributed by e-mail and on the website and carries information about new research, policy developments, programs, and publications relevant to maternal and child health. The Library website also provides access to five databases relevant to maternal and child health, and provides access to NCEMCH Resource Guides (annotated lists of organizations) and Knowledge Paths (annotated bibliographies of websites, publi-

cations, databases, and electronic resources). Both Resource Guides and Knowledge Paths are organized around specific topics; those most relevant to childhood obesity include Child and Adolescent Nutrition, Physical Activity and Children and Adolescents, and Overweight in Children and Adolescents.

SEE ALSO: Changing Children's Food Habits; Fitness; Physical Activity in Children.

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Center for Nutrition Policy and Promotion

THE MISSION OF the Center for Nutrition Policy and Promotion (CNPP) is to improve the health of Americans by providing food and nutrition guidance, assessing diet quality, and advancing consumer, nutrition, and food economic knowledge. The CNPP was created in 1994 within the U.S. Department of Agriculture (USDA), and reports to the Office of the Under Secretary for Food, Nutrition, and Consumer Services. The CNPP staff includes nutritionists, nutrition scientists, dietitians, economists, and policy experts who help to define and coordinate nutritional education policy within the USDA and translate nutrition research into information and materials for consumers, policy makers, educators, health professionals, industry, and the media.

The CNPP's best-known project is probably the food pyramid, which provides recommendations about the types and amounts of food to include in a healthy diet. The current version, the MyPyramid Food Guidance System (and the Spanish version, *MiPirámide*), includes an online menu interface allowing users to enter their age, sex, and activity level, and calculates the servings of grains, vegetables, fruits, milk, meats, and beans they should be consuming each day.

The *Dietary Guidelines for All Americans* is produced by the Departments of Agriculture and Health and Human Services and is the cornerstone of federal nutrition policy and nutrition education activities. The *Dietary Guidelines* present the government's science-based advice on nutrition and physical activity to promote health and reduce chronic disease and are updated every five years; the most recent version (2005) is available from the CNPP website. The 2005 edition, which contains 41 key recommendations, emphasizes the importance of maintaining a body weight within reasonable guidelines, and discusses the need for many individuals to reduce calorie consumption and increase physical activity in order to maintain or achieve a healthy body weight.

The *Healthy Eating Index* (HEI), first produced by the USDA in 1995, was revised in 2005 to conform to the recommendations in that year's *Dietary Guidelines*. The HEI is a measure of diet quality that assigns points for the percent of calories represented by different types of foods in a person's diet, and can be used in nutrition monitoring, nutrition interventions, consumer education, and research. HEI data for 2001–02 are available for download from the CNPP website.

SEE ALSO: Department of Agriculture; Food Guide Pyramid; Government Agencies.

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Center for Science in the Public Interest

THE CENTER FOR Science in the Public Interest (CSPI) was founded in 1971 with the purpose of evaluating the effects of science and technology on society and promoting national policies responsive to the interests of consumers. Most CSPI funding is supplied by individual donors and the approximately 900,000 subscribers to the *Nutrition Action Healthletter*, and

about 5 to 10 percent comes from private foundation grants; CSPI accepts no government grants or corporate support, and the *Nutrition Action Healthletter* accepts no advertising. The CSPI office is located in Washington, DC.

The *Nutrition Action Healthletter,* which first appeared in 1974, is currently published 10 times a year by CSPI and has the largest circulation of any health newsletter in North America. The *Healthletter* is a popular journal that includes articles about health and nutrition, deceptive advertising and faulty science related to food products, editorials, and feature stories. Many articles are available through the *Healthletter* website.

The three main goals of CSPI, according to its mission statement, are to conduct research and provide useful and objective information to the public and policy makers concerning food, alcohol, health, the environment, and other science and technology issues; to represent the interests of citizens on food, alcohol, health, the environment, and other issues before regulatory, judicial, and legislative bodies; and to encourage scientists to engage in public-interest activities and ensure that science and technology are used for the public good.

Many of CSPI's efforts have involved promoting legislation banning dangerous food additives or requiring informative labels on food products. One of CSPI's early campaigns was launched in 1973 to bar the use of sodium nitrite in bacon and other cured meats; this campaign ultimately resulted in lower levels of nitrites in many foods. Sodium labeling of all foods and fat-content labeling of processed meats was the result of a CSPI petition to the Food and Drug Administration (FDA) in 1978; CSPI efforts resulted in a new FDA rule requiring such labeling in 1982.

In 1982, CSPI began an effort to ban sulfite preservatives in most fresh foods; this effort succeeded in 1987. In 1988, CSPI succeeded in championing a federal law requiring a health warning label on alcohol beverage containers. In 1994, CSPH campaigned for labeling of transfats in foods, an effort that succeeded in 2003. In 2000, CSPI was successful in promoting legislation providing funding for more food safety inspections and expanded bacteria testing for meat. In 2004, CSPI successfully convinced Congress to require disclosure on food labels of common allergens such as peanuts, wheat, and eggs.

In 1993, CSPI began the first of a series of investigative reports into the nutritional value of restaurant foods. The first focus was Chinese restaurants, because Chinese food was often believed to be a particularly healthy cuisine. However, the CSPI report revealed that many popular Chinese restaurant foods contained unhealthy amounts of fat, cholesterol, calories, and salt and recommended that portions be divided among several diners and that popular dishes like Moo Shu Pork and Kung Pao Chicken should be consumed alongside equal parts of rice and in conjunction with dishes based on steamed vegetables, which are much lower in fat and calories.

SEE ALSO: Eating Out in America; Food Labeling; Nutrition and Nutritionists.

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Centers for Disease Control and Prevention

THE CENTERS FOR Disease Control and Prevention (CDC) is one of the major operating components of the U.S. Department of Health and Human Services and is the principal agency responsible for improving public health in the United States. The CDC has two primary purposes: to respond to health emergencies and to improve people's health in their daily lives. In support of those goals, the CDC conducts research and public health interventions across the United States and globally.

The CDC originally was concerned primarily with infectious diseases, but its purview has expanded

to include all diseases and conditions that impact human health, including chronic diseases and risk factors for them such as obesity, lack of physical activity, tobacco use, and exposure to environmental toxins. The name Center for Disease Control was adopted in 1970; in 1981, this became Centers for Disease Control, and in 1992, Centers for Disease Control and Prevention; however, the acronym CDC is still used for the organization.

The CDC has a workforce of over 8,000 employees in various locations throughout the world; CDC headquarters are located in Atlanta, GA. As the name suggests, the CDC consists of a number of Centers which focus on particular aspects of public health: these include the National Center for Environmental Health/Agency for Toxic Substances and Disease Registry (NCEH/ATSDR), the National Center for Injury Prevention and Control (NCIPC), the National Center for Health Statistics (NCHS), the National Immunization Program (NIS), and the National Institute for Occupational Safety and Health (NIOSH).

The CDC Division of Nutrition and Physical Activity takes a public health approach toward nutrition and physical activity and their roles in preventing chronic disease and promoting good health. The Division maintains a website devoted to the topics of overweight and obesity, which states unequivocally that overweight and obesity are increasing sharply among American children and adults, and that these conditions increase the risk of many chronic diseases including coronary heart disease, stroke, hypertension, and osteoarthritis. The website also includes information about nutrition, physical activity and the health risks of being overweight or obese, links to CDC press releases on those topics, information about relevant CDC programs and campaigns, and links to relevant CDC publications.

Several major CDC campaigns are relevant to obesity prevention and weight control. The 5-a-Day for Better Health program is a national effort to increase per-capita fruit and vegetable consumption to five per day. Program materials are also available in Spanish: the Spanish-language program is called *5 al Día*.

The Active Community Environments (ACES) Initiative promotes environmental and policy interventions that will increase levels of physical activity, including the development of pedestrian- and bicycle-friendly environments, the promotion of active forms of transportation such as walking and bicycling, and the development of accessible recreational facilities. The Kids-Walk-to-School program is a community-based program that encourages children to walk to and from school in groups accompanied by adults, and communities to create a safe environment to facilitate walking and other regular physical activity. VERB is a social marketing campaign to encourage children aged 9–13 years (also known as tweens) to be physically active every day. The campaign is national and multicultural (materials are available in English and Spanish) and combines advertising, marketing, and partnership efforts to reach audiences of tweens and adults who influence their behavior.

Morbidity and Mortality Weekly Report (MMWR) is a weekly online and print journal published by the CDC which contains primarily current reports of disease occurrence and risk factors. Each week includes a section of Notifiable Diseases and the remainder of the articles is on various topics, including reports of disease outbreaks and focused analyses of publicly available data sets such as the National Health Interview Survey (NHIS). Electronic copies of *MMWR* dating back to 1982 are available through the CDC website or by e-mail subscription without charge, and paper copies may be purchased through the same website.

SEE ALSO: Department of Health and Human Services; Fitness; Government Agencies; National Center for Health Statistics; Nutrition and Nutritionists; Physical Activity and Obesity; Prevalence of Childhood Obesity in the United States; Prevalence of Obesity in U.S. Women; Social Marketing and Obesity.

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Central America and Caribbean

IN CENTRAL AMERICA and the Caribbean before the arrival of Christopher Columbus and the subsequent European colonization, there is some evidence that obesity existed in the Aztecs and other cultures seen through surviving statues. However, it has been suggested that the shape of these figures might be as much to do with exaggerated style and possibly to help their balance than to do with accurate representation of the size of people during this period.

The early European settlers in the Caribbean and Central America did not suffer much from obesity, and neither did the slaves brought over to work the plantations. However, with many of the plantations growing sugar, a primary cause of obesity, it seems to be likely that members of some of the European plantation families might have become obese. By contrast, given their hard working schedules, few slaves would have become obese except perhaps a handful of household slaves, especially those who were involved in work in the preparation of food or connected with the refining of sugar. Certainly, sugar seems to have been an early cause of obesity with recent studies surveying some of the population of Port-au-Prince, the capital of Haiti, have shown that there is an inherited glucose intolerance which in some people has developed into diabetes, and has shown itself in abdominal obesity.

There have been a number of recent studies of obesity among the former slave populations of the Caribbean and although these people share a common genetic heritage, their environment varies considerably. Curiously, an important study by T. Forrester and colleagues of the Tropical Metabolism Research Unit, University of the West Indies, Mona, Kingston, Jamaica, surveying people in Barbados, Jamaica, and St. Lucia has shown that obesity levels are significantly higher in areas where the gross national product is higher, showing a clear link between wealth and obesity.

Today, the glucose intolerance along with a much larger diet and less exercise has contributed to a large rise in obesity rates throughout the Caribbean, with obesity having doubled in the Caribbean in the last 10 years. This has led to increases in diabetes mellitus, hypertension, and various types of heart diseases. Indeed, according to an October 2004 report by Fitzroy J. Henry, director of the Caribbean Food and Nutrition Institute in Kingston, Jamaica, to the Caribbean Commission for Health and Development, obesity is now the underlying cause of most deaths in the English-speaking Caribbean. This is even more marked with women, and the body mass index shows the greatest occurrence of obesity in people aged 45–54 years living in Belize. The main cause has generally been the change from nonsedentary jobs to sedentary jobs, which certainly helps explain some of the higher obesity rates in Jamaica and Barbados. However, this alone does not explain the rise in obesity in young children, with the increasing consumption of unhealthy food clearly being important. This has led to a study of childhood obesity that has shown massively increased levels in Antigua, Dominica, and St. Kitts and Nevis during the 1990s, with that of Nevis reaching a rate of 10 percent in the years 1990–99. Curiously, the rise of obesity in St. Vincent during the same period was much less marked.

In Cuba, where there is not a major problem of obesity, research is coordinated by the Cuban Obesity Association with Dr. Mirtha Prieto of Havana as the national representative of the International Association for the Study of Obesity (IASO). The Asociación Salvadoreña de Obesidad (ASOBE) runs out of San Salvador, with Dr. Ana Margarita Nuila de Villalobos of Colonia Escalon, San Salvador, being the national representative of the IASO. The Panamanian Association for the Prevention and Treatment of Obesity (APPTO) deals with matters concerning obesity in Panama, with Dr. Itzel Velasquez being the national representative of the IASO.

There have also been studies in Central America of the increasing levels of obesity, with a focus on the changing nature of work and in diet. With rising rates of obesity in Central America, especially in children, there have been marked increases in other health problems. This rise in obesity has been in spite of periods of great food shortages in Central America, with Guatemala and Panama both having

experienced general rise in the rate of malnutrition in the early 1990s. Some countries have had higher rates of obesity than other countries, with the highest rates in the Americas for 20- to 29-year-olds being recorded in Chile, and the rates in Costa Rica and Cuba closely following. The same survey found the prevalence of obesity among children aged under 6 was the lowest in Nicaragua (about 2.2 percent).

The rate of malnutrition in children under the age of 4 in the Americas is at its highest in Guatemala (38.5 percent), Guatemala also recording the lowest height for age in the same age group (57.9 percent). A Japanese study by T. Sekiya of the Faculty of Education, Hiroshima University, in Honduras in 1994 showed that although the physique of Honduran children was less than that of Japanese, their chest size and body weight was the same size, with girls being heavier and more thickset, something he put down to the low levels of protein in the diet and the high levels of fat, leading to increasing obesity in girls. From the 1990s, there were other studies of obesity in children. One by H. P. Nunez-Rivas and colleagues of the Costa Rica Ministry of Health and the Costa Rican Institute for Research and Education on Nutrition and Health, carried out between July 2000 and April 2001, showed a distinct higher prevalence of overweight in children from higher socioeconomic backgrounds—often exceeding a third of the boys under 12 whom they studied.

This association of obesity with greater wealth has been borne out by another study into obesity in adult women in Guatemala and Haiti, showing that being obese is often associated with better-than-average educational attainments, although a similar study in Bolivia, as a part of a control exercise, showed the opposite. Another study by A. D. Stein, A. M. Thomson, and A. Waters of the Department of Global Health, Rollins School of Public Health, Emory University, found that some forms of late-childhood obesity in Guatemala and also in other countries, were associated with a heightened risk of elevated blood pressure, glucose, and serum lipids in adulthood. As in other countries, Guatemala has been involved in a campaign to reduce obesity, and as a result, prevent diabetes.

With the general rise in obesity throughout Central America and the Caribbean, there has been an attempt to introduce public policies such as encouraging exercise rather than relying on treatment by expensive drugs. This has seen many countries in the British Caribbean introducing healthier diets in schools, and encouraging children to take part in sports as a way of preventing childhood obesity. This has sometimes been followed by campaigns to improve the food provided in workplace canteens, with more money spent on leisure activity facilities. Shortages of government money in many Central American countries mean that many of those countries not being able to undertake such options.

SEE ALSO: North America; South America.

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Central Nervous System

EVERY ACTION, EMOTION, response, and human characteristic involves the central nervous system (CNS). The CNS is comprised of the brain and spinal cord and is involved with physiological actions throughout the entire human body. The CNS is one of the two major divisions of the nervous system. The associated system that coordinates with the CNS is the peripheral nervous system (PNS), which is outside the brain and spinal cord in the extremities and trunk. The CNS is involved with hunger, taste, and overall food intake regulation. The CNS is also involved with overall metabolism. The past decade has witnessed an explosion of information regarding the role of the CNS in the development of obesity and the influence of peripheral hormonal signals that regulate nervous system function.

The PNS connects the CNS to sensory organs (such as the eye and ear), other organs of the body, muscles, blood vessels, and glands. The peripheral nerves include the 12 cranial nerves, the spinal nerves and roots, and what are called the autonomic nerves. These are concerned specifically with the regulation of the heart muscle, the muscles in blood vessel walls, and glands.

These glands such as the pituitary, parotid, and thyroid release hormones throughout the blood, and when they dysfunction they can be linked to many issues and newly understood pathological mechanisms involving obesity. For example, an overproduction of human growth hormone (hGH) from the pituitary gland in the brain can lead to uncontrollable weight gain and obesity. The capacity to adjust food intake in response to changing energy requirements and environment is essential for survival. Any change in these biochemical processes can lead to obesity and long-term health concerns.

One of the new questions much research is aiming to answer and better understand is why not all people become obese when in an obesogenic environment. *Obesogenic* is a newer medical term meaning factors tending to make individuals obese. Much of this new research is looking at genetic dispositions and failures in hormonal levels that lead to obesity. Metabolism is the sum of all chemical processes, assimilation, incorporation, detoxification and excretion of food in the body. Inborn and acquired abnormalities with metabolism can lead to obesity and, in some instances, excessive weight loss.

The CNS controls and oversees homeostasis. Homeostasis is the inherent tendency in the human body toward maintenance of physiological stability. The physiological importance of this homeostatic control system is highlighted by the severe obesity that results from dysfunction of any of the several key components. Basically, when the CNS or another component fails, homeostasis is no longer maintained and one such outcome can be excessive weight gain and eventual morbid obesity. New research is looking at the detailed reasons and causes behind such pathologies and putting things into context of the global obesity epidemic. Last, some new research relating to the CNS and obesity aims at identifying potential avenues for therapeutic intervention and reversal of this obesity epidemic.

For example, some such studies include animals and look at the genetic propensity to develop diet-induced obesity (DIO) by observing a reduced responsiveness to signals such as leptin and insulin. Obesity and type 2 diabetes are on the rise in the United States and are major health concerns. The two hormones thought to play a critical role in both energy homeostasis and glucose metabolism are the adiposity hormones insulin and leptin. Both of these hormones circulate in proportion to body fat stores and interact with their respective receptors expressed in key brain areas such as the hypothalamic arcuate nucleus (ARC) that regulate food intake and glucose metabolism.

The arcuate nucleus is a collection of neurons present in the hypothalamus, the center of and main CNS operator with masses of neurons that can send messages to other areas of the brain and body. When activated, these masses of neurons can produce ravenous eating desires and may be regulated by glucose, insulin, and leptin. Thus, arcuate neurons are responding to information on whether the body has sufficient calories and nutrients and acting on needs of the body. It is possible that leptin signaling in the arcuate nucleus is an important determinant of both energy homeostasis

and glucose metabolism. Any malfunctions with the above homeostatic mechanism can lead to obesity.

Leptin is a protein hormone that plays a key role in regulating energy intake and energy expenditure, including the regulation of appetite and metabolism. Leptin is secreted by fat tissues but regulated by the hypothalamus. The hypothalamus is located in the center of the brain and regulates certain metabolic processes and autonomic activities. Research of hypothalamic leptin regulation of energy homeostasis and glucose metabolism can lead to a better understanding of obesity as it relates to the CNS.

Insulin is a polypeptide hormone that regulates carbohydrate metabolism, most notably with glucose. These hormones normally inhibit the development of obesity when dietary fat and calorie contents are increased in the diet. The assumption is that obesity is very difficult to reverse once it develops in such individuals and prevention is the best treatment with the above-mentioned emerging obesity epidemic. The CNS is the keystone to understanding obesity and its root causes for human beings. With obesity in America reaching epidemic proportions, there is no better time to explore this final frontier of the brain.

SEE ALSO: Autonomic Nervous System; CNS/Hypothalmic Energy Sensing; Dopamine; Hypothalamus; Insulin; Leptin; Obesity and the Brain; Sympathetic Nervous System.

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Central Obesity

IN 1956, JEAN Vague, an endocrinologist, first described masculine (central) and feminine (peripheral) fat distribution and suggested that central fat distribution is associated with an increased risk of diabetes and cardiovascular disease. In recent years, epidemiological studies have clearly established the relationship between obesity and cardiovascular diseases, type 2 diabetes, and many other morbidities in both cross-sectional and longitudinal studies. However, not all obese people are at risk of above-mentioned diseases. Although in debate as to whether a cause or a result, the positive relationship between central obesity and obesity-related health risks independent of total adiposity has been well established in the last two decades, especially after the introduction and wide acceptance of metabolic syndrome. Central obesity is characterized as fat accumulation at the trunk region, namely abdominal region. In contrast to central obesity, peripheral obesity is defined as fat deposit at limbs and hip, especially lower body region. Other prevailing terms that has similar meanings in describing fat distribution pattern as central versus peripheral includes android versus gynoid, masculine versus feminine, "apple" shape versus "pear" shape, and upper body versus lower body.

Central fat or abdominal fat depot includes subcutaneous abdominal adipose tissue and visceral adipose tissue. The latter is fat residing within the abdominal wall and surrounding the organs, although a small amount has been found to have a strong relationship with morbidity and mortality. Visceral adipose tissue's relationship with obesity-related health risks has been supported in most studies and some studies even suggested a causal relationship between visceral adipose tissue and health risks in humans and in animal studies. On the other hand, the role of abdominal subcutaneous adipose tissue in obesity-related health risks has not been as well established independent of visceral adipose tissue.

Accordingly, to refine the understanding of different central fat depot with health risks, methods that can quantify visceral and abdominal adipose tissue separately are desired. Only with the relatively recent application of imaging technology including computerized tomography (CT) and magnetic resonance imaging (MRI), it has been made possible to quantify subcutaneous and visceral adipose tissue separately. With lower cost and wider availability than CT and MRI, dual energy X-ray absorptiometry (DEXA) measured trunk fat is also used as an indicator of central obesity. Simple and low-cost anthropometric measures of central obesity include waist circumference, waist-to-hip

ratio, saggital diameter, skin folds of the abdomen and subscapular area, and so on. While DEXA and anthropometric measures cannot differentiate visceral from subcutaneous adipose tissue, these measurements are practical for large-scale research, especially epidemiological studies and are therefore widely used.

Central obesity has been found to be related to health risks including insulin resistance and metabolic syndrome, morbidity such as diabetes and cardiovascular disease, and mortality in different racial and ethnic groups. Investigated cardiovascular disease risk factors include high blood pressure, insulin resistance, glucose intolerance, high blood lipids, and so forth. Cardiovascular morbidities include myocardial infarction, angina pectoris, stroke, and so forth. It has also been found that in diet, exercise, or weight loss drug intervention, visceral adipose tissue and abdominal subcutaneous adipose tissue tend to be lost more quickly than limb subcutaneous adipose tissue.

The relationship between health risks and central obesity can be related to its subdepots. Potential explanations includes portal vein theory, which means that free fatty acids from visceral adipose tissue delivered into the portal system have potent and direct effects on the liver. For subcutaneous adipose tissue, it has been shown that in vivo lipyolysis increases more from the abdominal adipose tissue than from the gluteal adipose tissue during exercise, especially in women. This sex difference may also explain that women tend to accumulate subcutaneous fat more readily in the hips than in the abdomen. Subcutaneous adipose tissue can be further divided into superficial and deep subcutaneous adipose tissue by a facial plane. Some studies also found that the deep depot is more closely related to health risks than superficial subcutaneous adipose tissue. This may be explained by that deep subcutaneous adipose tissue is more metabolically active or simply have a larger mass than superficial subcutaneous adipose tissue.

Although both men and women accumulate more fat with aging, women tend to accumulate fat with a peripheral and gynoid distribution pattern, while men tend to put on fat with a central or android fat distribution pattern. This may partially explain why women have a lower risk for disease than do men. Sex hormones such as testosterone and estrogen may play major roles in the fat distribution pattern differences in men and women, but other factors may also contribute to this difference. Women tend to deposit fat in abdomen region after menopause and the risk for disease also increase in postmenopausal women.

There are also racial differences in adipose tissue distribution pattern. Asians gain a greater proportion of fat in the trunk region, especially visceral fat, than other races. On the other hand, African Americans tend to accumulate fat in subcutaneous region than visceral region. This may explain the high risk of diabetes in Asians and low risk of diabetes in African Americans at a given level of obesity.

SEE ALSO: Visceral Adipose Tissue; Waist Circumference.

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Changing Children's Food Habits

APPROXIMATELY 9 MILLION U.S. children over the age of 6 are now considered overweight, and the numbers are on the rise. The U.S. Centers for Disease Control and Prevention (CDC) state that the percentage of overweight young people in the United States has roughly tripled since 1980 to about 18 percent. Children are less active, eat more processed food, and have poorer eating habits than ever before, but this does not have to be a permanent condition. Parents can start by examining and understanding what, where, when, why, and with whom their children are eating, and how many extra calories a day they are consuming.

Kids today are suffering from an energy gap, meaning they take in more calories than they burn through growth and daily living. The average child takes in

Education remains the best way to ensure that children make wise choices when it comes to food.

up to 165 extra calories per day, which is about the amount in a can of soda. The heaviest children and teens are taking in as many as 1,000 calories more per day than needed, which is almost as much as two Big Macs. If government, community, schools, families, and food and beverage industries do not start working together, we will see the first generation of children to have a shorter life span than their parents.

Presently, overweight children suffer from a range of psychosocial and physical consequences, including poor self-esteem, Type 2 diabetes, and the beginning stages of cardiovascular disease. Moreover, consumption of poor, high sugar and fat diets may affect learning potential as well. Children who consume large amounts of sugar-sweetened beverages, for example, may find themselves having quick bursts of energy followed by nadirs of energy depletion. Also, these foods tend to be "empty calories" sources that lack substantial vitamins and minerals, but contain palatable and easily accessible sources of energy. Overconsumption of these empty calorie food sources can put children at risk for under-consumption of many vital vitamins and minerals.

Education is the best way to help children understand their food intake. One way is to educate children so they understand their unique bodies. Parents can help children gain more self-assurance about their own bodies while at the same time, teaching them to appreciate the need to achieve optimal nutrition. In addition, parents must role-model proper nutrition and eating habits for their children by providing a variety of healthful, ready-to-eat foods, and "practicing what you preach." It is unlikely a child will grow up to like vegetables if his or her parents do not like to eat those foods.

Children should also be active and exercise several times each week. The CDC reports that 25 percent of U.S. children engage in no free-time physical activity. The average U.S. child spends 20 hours per week watching television, with up to 20 percent of this time accounted for by meals eaten while watching television. Children need to separate meals times from entertainment and relaxation events. Parents need to help children refocus relaxation and entertainment events more on physical activities rather than sedentary ones. Far too many children today are sedentary, which when coupled with poor nutrition habits, creates a recipe for weight issues.

This recipe contains a number of very prominent ingredients, the first of which is high fructose corn syrup (HFCS). HFCS is found in a startling number of processed foods as well as foods specifically aimed at children. It is prominent in many brands of ketchup as well, a staple of most children's diets. But the most significant source of HFCS is in soda pop and sweetened fruit juices, elevated intake levels of which are associated with higher body mass index (BMI) in children.

Trans-, saturated, and hydrogenated fats are also very prominent in the prepackaged, processed, convenience foods aimed at children and their busy parents. Often appearing with those fats are very high quantities of sodium and chemical preservatives. Children do not need these ingredients in their food, but they are not equipped to determine the contents of a food, or whether the contents are appropriate or inappropriate for them. They simply eat what is available, familiar, tastes good, or what they are told to eat.

Because of convenience packaging, school lunches, and maladaptive lifestyles relating to food on the part of parents and other family members, children are taking in too few dairy foods, whole fruits and vegetables, and fiber. They are consuming high quantities of fatty foods, too much refined sugar, too much caffeine, and too many processed and preserved foods. Children also have little control over portion sizes, as foods are often preportioned for them by schools, restaurants, convenience packaging, and parents. Parents need to exercise more than

a modicum of control and restraint on behalf of their children's nutritional health. It should be stated, however, that too much parental restraint or restriction has been found in some studies to be associated with children being more likely to binge on restricted foods when parents are not around. Thus, individual families need to respond to their own child's food, dietary needs, and personality to strike the most effective balance with respect to dietary restrictions.

SEE ALSO: Child Obesity Programs; Childhood Obesity as a Risk Factor for Adult Overweight; Children and Diets.

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Childhood Obesity as a Risk Factor for Adult Overweight

CHILDHOOD OBESITY HAS been shown to predict adult obesity, indicating that early life factors may be important risk factors for adult obesity. A number of studies have shown that overweight children become overweight adults, with a five- to ninefold increase in adult obesity for individuals obese at age 9–13 years. About one-third of obese preschoolers, half of obese school-age children, and 70 percent of obese adolescents were obese as adults. Recent surveys report a higher predictive rate of 65 to 84 percent, which may be reflective of recent trends of increasing obesity, or a redefinition of body mass index (BMI) cutoff points used to classify obesity. Although a higher proportion of obese children become obese adults, only a small proportion of obese adults were obese as children.

Early childhood factors that may affect the propensity to develop obesity in adulthood include birth weight, early feeding patterns, adiposity rebound, early maturation, genetic predisposition, parental obesity, and socioeconomic status. Adiposity rebound is the point at which the BMI inflection occurs in the BMI-for-age curve, usually at about 5–7 years of age. Adults who had adiposity rebound after age 8 had 40 percent lower incidence of obesity compared to those who had adiposity rebound before 5 years of age.

The risk of an overweight child becoming an obese adult rises with age. Among preschoolers, the risk of becoming an obese adult is twice as likely if the preschooler was obese, while among adolescents, the

Overweight and obese children are 18 times more likely to become obese adults than children who are average weight.

risk is 18 times more likely for an obese adolescent becoming an obese adult. The higher the BMI is in childhood, the greater the probability of persistence to adulthood. Tracking of obesity is greatest when both parents are obese. Obesity runs in families, a reflection of shared genes, environment, and culture. However, the relative contribution of genes and inherited lifestyle factors contributing to the parent–child obesity association is unknown.

The velocity of BMI has been shown to be an important predictor of adult obesity. Rapid weight gain in infancy and childhood is associated with development of obesity in childhood and adulthood. Adults with BMI in the upper quartile by age seven had threefold increase in obesity compared to their counterparts with a BMI in the lower quartile.

An association between birth weight and adult obesity has been documented. The relationship is not linear, but a U-shaped association between birth weight and adult obesity. It has been suggested that the effect of birth weight is mainly accounted for by maternal weight. Heavier mothers having heavier babies, who tend to become heavier adults. There is some evidence to suggest that breast-feeding may protect against later development of obesity. Breastfed infants have a slower growth rate. Parental socioeconomic status is inversely associated with adult obesity. Upwardly mobile men and women are less obese than their peers who remained in the same social class.

Lifestyle habits such as diet and physical activity may track from childhood to adulthood. Adult obesity has been associated with low levels of leisure-time physical activities and increased TV viewing in childhood. TV viewing may increase obesity risk by displacing physical activity or through selection of unhealthy food choices influenced by food advertising.

Some, but not all, studies have shown that childhood obesity is associated with more adverse adult health outcomes than obesity developing in adulthood. Overweight in adolescence is associated with insulin resistance and dyslipidemia (high total cholesterol as well as low-density lipoprotein cholesterol and low high-density lipoprotein cholesterol), which tracks from childhood to adulthood. Accelerated weight gain or BMI in childhood is associated with increased risk of hypertension, Type 2 diabetes, coronary heart disease, and a doubling of the risk of strokes in adulthood. Additionally, greater BMI in childhood is associated with increased mortality from cardiovascular disease in adults.

SEE ALSO: Assessment of Obesity and Health Risks; Atherosclerosis in Children; Elevated Cholesterol; Ethnic Variations in Obesity-Related Health Risks; Infant Weight Gain and Childhood Overweight; Maternal Influences on Child Feeding.

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Childhood Obesity Treatment Centers in the United States

CHILDHOOD OVERWEIGHT IS an increasingly prevalent condition in the United States. According to data from the National Health and Nutrition Examination Survey (NHANES), between 1976–80 and 2003–04, overweight among children aged 2–5 years almost tripled, from 5.0 percent to 13.9 percent. Even greater increases were seen in older age groups: for those aged 6–11 years, the prevalence of overweight increased from 6.5 percent

to 18.8 percent, and the prevalence of overweight among those aged 12–19 years increased from 5.0 percent to 17.4 percent. Although previously it was believed that children would simply "grow out" of their weight problems, this is no longer the case as overweight in childhood and adolescence has been found to be a strong predictor of obesity in adulthood. In addition, overweight children suffer from higher rates of many serious health conditions, including Type 2 diabetes, high cholesterol, high blood pressure, and sleep apnea. Overweight children also suffer psychologically from the experience of prejudice and discrimination and have poorer body images than normal-weight children.

DEFINITIONS

Overweight for children and teenagers is usually calculated using the body mass index (BMI), a formula which combines information about height and weight. It is not a direct measure of body fat (an extremely muscular adolescent, for instance, might have low body fat but a high BMI) but correlates well for most people with direct measures of body fat such as underwater weighing. A child's BMI is compared to a gender-specific BMI age-for-growth chart (available, for instance, from the U.S. Center for Disease Control and Prevention) to obtain a percentile ranking, which shows the relative position of the child's BMI among others of the same gender and age. A BMI percentile at the 95th percentile or higher is classified as overweight, and from the 85th to 95th percentile as at risk for overweight. Recently, an alternative classification system has been developed using growth curves created using data from several different countries, which identify BMI scores for children age 2–18 years which project to a BMI of 25 (overweight) or 30 (obese) at 18 years of age.

COMPREHENSIVE PROGRAMS

Because childhood overweight is a complicated phenomenon, deciding which children require therapeutic intervention requires expert evaluation, possibly by a team of specialists, and devising an effective treatment plan may also require input from a team of specialists. Expert panel guidelines outlined in Barlow and Dietz (1998) describe a two-level screening process for children who are overweight or at risk. The first level involves calculating the child's BMI; those who are overweight (95th percentile or above) are referred for an in-depth medical assessment, while those at risk for overweight (85th–95th percentile) are referred to a second level of screening. This screening focuses on five elements: (1) family history of cardiovascular disease or diabetes, and parental hypercholesterolemia or obesity; (2) high blood pressure; (3) elevated cholesterol; (4) large recent increase in BMI; and (5) psychological or emotional problems related to weight, or child concerns about weight. Children with any of these criteria are referred for an in-depth medical examination.

Most successful childhood obesity treatment programs focus on a lifestyle approach and require cooperation and participation from the child's parents or entire family. As with adults, losing weight and keeping it off requires changes in fundamental aspects of daily life (e.g., food consumption, leisure activities), and in the case of a child, this inevitably requires changes from other members of the family as well. For these reasons, comprehensive, family-based behavioral treatment programs have produced the best results over both the short and long term. This is not surprising because most children learn their attitudes and beliefs about food, as well as their habitual behaviors regarding eating, at home. Most successful programs also involve increasing physical activity and/or reducing time spent in sedentary activities such as watching television or playing video games; both types of changes require the cooperation and monitoring from parents and possibly from other family members as well.

Because of the multifaceted approach required for successful treatment of childhood obesity, a number of comprehensive treatment programs have been devised. In addition, some children's hospitals have developed specialized centers to treat weight problems in childhood. The types of services offered may ranged from basic medical and nutritional advice and behavioral counseling or therapy for children, parents and families through evaluation for and performance of bariatric surgery. Services are typically provided through a multidisciplinary team that may include pediatricians, other specialized physicians, dieticians or nutritionists, exercise specialists, mental health professionals, nurses and social workers. A list of "University and Hospital-Based Weight Control Programs and Services for Children and Adolescents" is maintained

by the Weight-control Information Network (WIN), an information service of the National Institute of Diabetes and Digestive and Kidney Diseases, which is one of the National Institutes of Health located within the U.S. Department of Health and Human Services. There are 36 programs listed as of May 2007.

Because the daily lifestyles of children and their families are inherently intertwined, the expert panel recommends that both the child and family be evaluated for readiness to change before an obesity treatment program is initiated. While parents may be able to modify the diet and activity levels of a younger child, such manipulation of an older child may induce resentment and loss of self-esteem, in addition to being less likely to succeed. Parents who have unhealthy attitudes to food (e.g., if they have an untreated eating disorder themselves) or who are not willing to participate in the child's program also lessen the probability of success. Because changes implemented for the overweight child's sake (such as prohibiting certain types of food from the home) may affect all family members, it is important to educate other siblings, grandparents, and so forth, about the health importance of the changes. In addition, a diet history and physical activity history of the overweight child should be taken before the program being. A diet history, which may be taken by the primary care provider or by a clinical dietician, identifies the child's typical pattern of food consumption, including foods eaten outside the home (at day care facilities or in school, at the grandparent's house, etc.). A physical activity history includes both vigorous activities such as physical education or organized sports, and daily activities such as walking to school or doing household chores. Information about time spent in sedentary behaviors such as watching television should also be collected, as should deterrents to activity (including unsafe neighborhoods, medical complications such as asthma, etc.).

GOALS OF CHILDHOOD OBESITY TREATMENT

Expert committee recommendations emphasize that the primary goal for most children should be to establish healthy eating and activity habits rather than achieving a specified body weight. Therefore, childhood obesity programs should be based on the acquisition and maintenance of skills, by the family as well as the child, such as becoming aware of current eating habits, identifying problem behaviors such as overconsumption of particular high-calorie foods, gradual modification of eating and activity habits, and continued monitoring of these habits. Medical goals may also be established with regard to the improvement or resolution of secondary conditions such as high blood pressure or abnormal lipid profile. The initial weight goal should be maintenance of current weight, which will automatically result in a reduction of BMI as the child grows taller. For children with no secondary conditions and whose BMIs are in the 85th–95th percentile, this should be sufficient to return them to a healthy weight. For children with secondary complications or with BMI in the 95th percentile or higher, after an initial period of weight maintenance, additional changes in eating and activity level may be recommended which would lead to the loss of around 1 lb per month. A BMI below the 85th percentile is an appropriate goal, but the focus should be kept on healthy eating and activity.

AN EXAMPLE OF A COMPREHENSIVE CHILDHOOD OBESITY TREATMENT CENTER

The Center for Healthy Weight (CHW) at Lucile Packard Children's Hospital (LPCH) may serve as an example of a comprehensive obesity treatment center for children and adolescents. LPCH is an academic medical center on the campus of Stanford University in Palo Alto, California, and many LPCH physicians are also professors in the Stanford Medical School. CHS is an interdisciplinary, cross-departmental program headed in 2007 by Thomas Robinson, MD, MPH. The goals of CHW are to prevent and treat overweight and obesity in children and adolescents. Efforts toward these goals are organized into three main areas: patient care, community programs, and research, including basic and translational science, prevention science, clinical trials, and studies of health policy and health inequalities.

Activities related to patient care at CHW take place in three primary programs: the Pediatric Weight Clinic, the Packard Pediatric Weight Control Program, and the Adolescent Bariatric Surgery program. The Pediatric Weight Clinic is an outpatient program providing comprehensive medical and psychological evaluation of overweight children and adolescents, and then helps to establish or expand their treatment program as necessary. All patients in the Pediatric Weight Clinic receive a medical evaluation from a pediatrician

experienced in weight management, a meeting with a dietitian to develop diet and activity goals, diagnostic studies as necessary, and referral to other specialists if needed for comorbidities related to overweight. The Packard Pediatric Weight Control Program (PPWCP) was founded in 1996 as a family-based educational and behavioral program to promote healthy eating and exercise habits for children and their families. The PPWCP has two age-specific programs, for children aged 8–12 and 13–15, and a program for Spanish-speaking parents of English-speaking children aged 8–12. The basic PPWCP program consists of 24 group meetings over six months; each group consists of 9 to 12 families and each meeting lasts approximately 1.5 hours. Individual meetings focus on topics such as developing better exercise habits, identifying and avoiding low-nutrient, high-calorie foods, and maintaining a healthy diet in difficult situations such as holiday parties or family gatherings. To join the PPCWP, a child must have a BMI ≥95 or a BMI ≤85 plus an obesity-related comorbidity, and must have at least one parent or legal guardian willing and able to attend each session. Adolescent bariatric surgery is a treatment option available only for severely obese adolescents suffering from severe comorbidities and who have been unsuccessful with other methods of weight loss. LPCH was the first California Pediatric Hospital to perform bariatric surgery, in November 2004. Potential bariatric surgery is evaluated according to strict eligibility criteria and they and their families must commit to lifelong lifestyle changes, including adherence to a low-calorie diet and following a regular exercise program.

Community programs of the CHW fall into three main areas: Community Partnerships, the Provider Education Institute, and Advocacy and Government Relations. LPCH is involved in several community partnerships whose goal is to create healthier environments for children. LPCH is one of seven founding and funding organizations of the Healthy Santa Clara County Collaborative, whose goal is to foster improved nutrition and increased physical activity. Get Fit EPA (East Palo Alto) was formed in 2004 to focus community effort on reducing overweight among school-age children, through nutritional education, access to physical fitness activities, and coordination of education efforts among existing community organizations. The Center for Provider Education of CHS develops and implements programs to help community agencies and providers deliver family-based weight-control programs. These efforts including training, supporting and certifying community agencies so they are able to deliver the PPWCP to their local community; providing Internet-based tools and materials to facilitate wider dissemination and high-quality implement of this program; offering immersion training programs for providers from out of state; and providing ongoing standards-based certification of providers who are able to deliver the PPWCP. Its purpose is to advocate for programs and policies to prevent and treat child and adolescent obesity, including provision of medical student and physician advocacy training; development of school-based advocacy curricula and projects; advocacy training for parents, teachers, and child care professionals; institutional advocacy through Stanford University and LPCH; and provision of professional representation and expertise to community coalitions.

SEE ALSO: Bariatric Surgery in Children; Behavioral Treatment of Childhood Obesity; Child Obesity Programs; Children and Diets; Family Therapy in the Treatment of Overweight Children; Parental and Home Environments; Physical Activity in Children.

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Childhood Onset Eating Disorders

THE MEAN AGE of onset for anorexia nervosa is 17 and the prevalence among females in late adolescence and early adulthood is approximately 0.5 to 1.0 percent. Bulimia nervosa occurs in between 1 to 3 percent in young adolescent and adult females and typically begins in late adolescence or early adulthood. Early onset of eating disorders typically refers to children aged 8–14. Little is known about eating disorders in children; it is rare that children younger than 8 experience anorexia nervosa or bulimia nervosa because of the cognitive features associated with the disorders.

Evaluations of children should rule out other forms of disordered eating and reasons for food refusal including mood disorders, general food refusal unrelated to body concerns, and selective eating. Early onset anorexia nervosa involves weight-loss attempts or food restriction to avoid weight gain and/or purging behaviors. Dieting for weight control and binge eating and purging are indeed a source of concern given that they are associated with the development of eating disorders.

Although the clinical presentation of childhood onset eating disorders is similar to adult eating disorders, the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) diagnostic criteria were developed for use in adults. The Great Ormond Street Diagnostic Checklist was developed to diagnose AN in children and includes criteria for food avoidance, weight loss or failure to gain weight, and two of the following: preoccupation with weight, preoccupation with energy intake, distorted body image, fear of fatness, self-induced vomiting, extensive exercising, and purging.

In children, the prevalence of binge eating disorder (BED) or binge eating symptoms ranges from 0 to 37 percent (depending on the measure or interview used). Given that the DSM criteria for BED were developed for adults and considering the subjective definition of a binge episode, provisional BED criteria for children have been proposed, including binge eating (food seeking in the absence of hunger and lack of control over eating) and at least one of the following: eating in response to negative affect, eating as a reward, sneaking or hiding food).

Finally, for diagnosis, symptoms persist for at least 3 months and are not associated with compensatory behavior. We have compared these criteria via a brief checklist in children aged 5–13 versus diagnosis via the Structured Clinical Interview for the Diagnosis of DSM-IV Disorders (SCID-IV) and found that 44 percent reported wanting to eat when not hungry, 52 percent reported that they lose control over their eating, 63 percent acknowledged eating as a result of negative emotions, 48 percent reported that they use food as a reward, and 28 percent stated that they sneak or hide food. Approximately 30 percent of the children met criteria for either syndromal or subsyndromal BED.

Several risk factors are related to the development of eating disorder including genetics, biological vulnerability (e.g., appetite dysregulation), psychological factors (e.g., perfectionism, low self-esteem, obsessionality), and sociocultural factors (e.g., internalized thin ideal). Factors posited to relate to resiliency include positive self-esteem and sense of life control, positive peer and adult role models, values, and behaviors factoring abstinence from health risk behaviors. Early detection and intervention improve the odds of recovery. In terms of BED, prevention and early interventions are needed to avoid the associated consequences including adult BED, obesity, and other medical and psychiatric comorbidities.

SEE ALSO: Children and Diets; Eating Disorders and School Children.

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Child Obesity Programs

WITH THE INCREASING prevalence of pediatric obesity over the past several decades and the mandate to physicians to manage this important health issue, clinical obesity programs have been developed that coordinate the services of physicians and other specialists from the relevant disciplines to manage and distribute care to young, overweight patients. This model of multidisciplinary care is based on the chronic care model and recognizes not only the need for overweight children to receive care for pediatric obesity over a prolonged period of time, but also the requirement for health and community agencies involved in the care of overweight children to communicate effectively with the patient, his or her family, and each other. Similarly, other commercial options are now becoming more available to children, particularly adolescents, for the management of weight. Although most interventions incorporate the essential elements of counseling to improve diet and physical activity behaviors, and psychosocial support, and many produce short-term success in regards to weight loss, successful weight maintenance in the long-term often remains elusive.

The essential issues that must be addressed in child obesity programs include nutrition, physical activity, and social support. While these components can be delivered from a single source, client/patient interaction with and reliance on family and community resources should be encouraged for long-term weight maintenance and to reduce weight gain and relapse once programs have been completed.

NUTRITION

Poor dietary behaviors are a known risk factor for the development of obesity. In particular, energy intake has increased from the 1970s to 2000; according to the National Health and Nutrition Examination Survey (1999–2000) children aged 6–11 were consuming 133 kcal/day more in 2000 than they were just a decade earlier. Although national surveys indicate a decline in the average proportion of calories from total and saturated fat over the past several decades, the Centers for Disease Control and Prevention (CDC) estimated in 2000 that only 38 percent of individuals 2 years and older met the recommendation for total fat intake and 41 percent of these individuals met the recommendation for saturated-fat intake. In addition, data from CDC's 2003 Youth Risk Behavior Survey indicate that in general, only one in five youth ate five or more servings of fruits/vegetables the week before the survey.

Caloric or energy restriction is an essential and required element for weight loss. However, simple dietary restriction has not been associated with successful weight control and may even result in a nutritionally inadequate diet. Research supports that a diet rich in fruits and vegetables and low in fat is important in preventing obesity and other chronic diseases, and is recommended by the U.S. Department of Agriculture, U.S. Department of Health and Human Services, the Surgeon General, National Research Council, and American Heart Association. Thus, current recommendations promote a diet that is nutrient dense: high in vegetables, fruits, grains, and other fiber-rich plant foods, yet low in fat, at a given level of energy intake. However, a recent review of dietary intervention studies for weight loss among children does not support energy restricted low-fat diets as the most effective method to reduce weight. Rather, there is evidence that alternative diets are just as effective.

Alternative diets that have been used to reduce weight include reduced-carbohydrate and low glycemic index diets. While such diets can be restricted in calories, not all such regimens promote energy restriction and allow ad libitum consumption, or eating until the individual is satiated. In clinical populations, among youth with severe obesity, reduced-carbohydrate, protein sparing modified fasting (PSMF) has been used with short-term success. The goal of the PSMF is to maximize weight loss while achieving positive nitrogen balance (i.e., conserving lean muscle mass) and preserving mineral balance. Generally lasting only 4 to 12 weeks, the PSMF prescribes carbohydrate restriction, a daily nutrient content of 600–900 kcal and 1.5–2.5 g of protein/kg ideal body weight. Children undergoing this dietary regimen require medical supervision for electrolyte abnormalities, and multivitamin and mineral supplementation is often performed. The low glycemic index diet promotes foods with a relatively low carbohydrate content based on the premise that such foods produce a relatively lower rise in blood glucose and thus blood insulin levels

compared to foods high in carbohydrates with benefits on hunger and metabolic fuel availability. Both the PSMF and the low glycemic index diet (ad libitum) have been shown to be as effective as energy-restricted diets in children.

Dietary programs that have been applied to youth populations with reported success have included: the Stoplight Diet, developed by Leonard Epstein and colleagues, and Committed to Kids, developed at the Pennington Biomedical Research Center. In the Stoplight Diet, foods are assigned colors that reflect calorie and fat content and are associated with eating recommendations. In particular, red foods are high-fat, high-calorie foods, and youth are discouraged from eating these foods; accordingly, yellow foods are moderate-fat, moderate-calorie foods, and youth are instructed to eat these foods in moderation; and green foods are low-fat, low-calorie foods, and youth are allowed to eat these foods freely. In the Committed to Kids clinical weight-loss program, children are assigned various dietary regimens based on weight status and prior weight-loss success. These regimens include not only the usual reduced-fat, energy-restricted diet, but also the PSMF. Overall, eating healthy in moderation and healthy eating habits are encouraged.

PHYSICAL ACTIVITY

There is strong evidence of the health benefits of physical activity including improvements in longevity, cardiovascular diseases (CVDs), CVD risk factors, diabetes, obesity, osteoporosis, immune functioning, certain types of cancer, and mental health among adults. There is also some evidence that regular physical activity can improve body composition, insulin sensitivity, hyperlipidemia, and blood pressure among obese children who engage in regular moderate-to-high intensity physical activity. The American Medical Association's Guidelines for Adolescent Preventive Services recommend that all adolescents annually receive guidance about the benefits of exercise and be encouraged to engage in safe exercise on a regular basis. More recent guidelines from the United Kingdom Health Education Authority recommend 60 minutes of daily physical activity for youth. Objective measures suggest less than 40 percent of teens are meeting the 60-minute guideline.

Females, older adolescents, minorities, and disadvantaged youth are even less likely to be meeting this recommendation. In addition, the latest report from the U.S. Department of Health and Human Services (2006) demonstrates that adolescents are not achieving current moderate and vigorous physical activity goals, and evidence suggests that adolescents are reducing their participation in physical activity.Sedentary behaviors, particularly television viewing, are the most consistent behavioral predictor of obesity in childhood. Intervention studies show that decreasing television time is an effective method of increasing physical activity. Parents can influence physical activity through modeling, being active with the child, or transporting the child to an activity location. Substitution of physical activity for sedentary behaviors and increases in activity level predict 6- and 12-month reductions in body mass index (BMI). In addition, targeting either decreased sedentary behaviors or increased physical activity is associated with significant decreases in percent overweight and body fat and improved aerobic fitness.

Owing to the benefits enumerated above, child obesity programs uniformly promote increased physical activity and reductions in sedentary behaviors. Physical activity recommendations to overweight and obese youth must take into account the health symptoms and complications that morbidly obese youth may exhibit such as orthopedic problems, asthma, lack of physical fitness, and early fatigue. Thus, initial goals recommendations should be small and realistic to allow for gradual increases to meet the recommendations.

PARENTAL INVOLVEMENT

Obesity programs must not only improve the dietary and physical activity behaviors of their enrollees, but they must also promote family and social environments that will foster adoption of healthy habits by their clientele. In the case of obesity programs for youth, particularly children in preadolescence or early adolescence, parental engagement and involvement in the weight-loss and weight-management program are often essential for success. Parental engagement may be affected by personal characteristics, parenting style, and family dynamics.

Parents have a strong influence on children's dietary intake and level of activity, both through modeling and reinforcement of eating and exercise behaviors, and through active determination of food options and

opportunities for activity. Research to date demonstrates strong relationships between parental weight status and weight-related behaviors and child weight status. Similarly, parental and particularly maternal feeding behaviors have been shown to affect development of obesity in their children. Differences in parenting methods may also affect which weight-related behaviors children adopt; in particular, more controlling parenting styles are associated with adoption of unhealthy eating and physical activity behaviors. The effect of culture has yet to be fully elucidated because studies to date have not fully studied families from multiethnic/racial backgrounds.

Compared to medical, educational, and school-based treatments, family-based treatment programs have reliably produced the best short- and long-term treatment effects on weight. Moreover, family-based treatment of childhood obesity is associated with significant health benefits including decreases in systolic and diastolic blood pressure, better physical fitness, and improved lipid profiles. Although not all studies have found that parental participation improves outcomes, interventions documenting the largest and longest-term decreases in percent overweight include parental participation as an integral component. Specifically, Epstein suggests that the direct involvement of at least one parent as an active participant in the weight-loss process improves short- and long-term weight regulation. Also, parent BMI change has been shown to be an independent predictor of child BMI change in family-based behavioral treatment of obesity. Additionally, parent activity improved the prediction of obese children's activity levels and the number of bouts of moderate-to-vigorous physical activity. Goldfield and Epstein reason that the more the parent is involved in treatment, the greater the opportunity to change the environment and provide support. Based on expert consensus recommendations regarding the importance of parental involvement and combined family-hild approaches including family-based behavioral weight-loss programs, family-based strategies are crucial when treating childhood obesity.

CLINICAL OBESITY PROGRAMS AND THE CHRONIC CARE MODEL

Given the complexity of the issue, it is becoming increasingly clear that successful clinical programs for overweight and obese children must utilize all of the components enumerated above. Depending on the circumstances of the child and the family, expertise from several medical and nonmedical disciplines is often required. Additional options available to patients in clinical settings that are not generally available to nonclinical programs include medications and bariatric surgery treatments. Such medical therapies include agents that suppress appetite or inhibit energy absorption. Bariatric surgery has only more recently been considered a viable option for children, and is generally only offered at centers with experience performing the procedures in adults. However, little is known about the long-term outcomes of medical and surgical therapies among youth; therefore, formal recommendations about this treatment option should be guarded.

The chronic care model is an appropriate model for clinical care programs dedicated to the treatment of pediatric obesity. Interdisciplinary team input and case management has been demonstrated to be a successful method for treating diseases such as asthma, hypertension, diabetes, and heart failure, all conditions that have been associated with morbid obesity. Such programs emphasize the Chronic Care Model (CCM). Designed to coordinate healthcare delivery, the CCM requires innovations in and revision of healthcare delivery with the intent of providing higher quality chronic illness management within primary care. This model highlights self-management support; practice teams to achieve clinical and behavioral management; disease decision support; and a well-designed clinical information system. Self-management support involves training patients in problem solving and goal setting.

The CCM separates the management of chronic illness from acute care and case management of high-risk patients. In the CCM, routine chronic care tasks promoting healthy behaviors can be performed by nonphysician members of a practice team (nurses or health educators) rather than physicians, sparing physicians' time to deal with more acute issues. Disease decision support consists of making evidence-based knowledge available to all clinic staff. Finally, clinical information systems involve reminders to providers to comply with care guidelines along with physician feedback about chronic disease management performance. Planned visits with patients with

chronic conditions and case management of high-risk patients, and reminder systems for clinicians improve doctors' performance and patient outcomes. CCM interventions can also reduce healthcare costs and/or lower use of healthcare services. Given the notable prevalence of obesity and recognition that it is a chronic disease, the CCM is a relevant and appropriate framework for healthcare delivery for obese adolescents.

SUMMER CAMP OBESITY PROGRAMS

Children's residential weight-loss programs have also shown some promise in producing short-term weight loss for overweight children. Such programs generally include daily physical activity sessions, moderate dietary restriction, and group-based educational sessions. A recent evaluation of attendees at a residential summer weight-loss camp demonstrated improvement across a range of outcomes including: BMI and fat mass reduction, blood pressure reduction, aerobic fitness, physical skill, and self-esteem improvements; a 10-month follow-up of camp attendees also demonstrated continued reductions in BMI over time compared to baseline measurements. The success of a controlled environment in promoting adoption of healthy weight-management behaviors highlights the importance and role of the social setting in supporting both initial and long-term behavior change for children.

COMMERCIAL PROGRAMS

Given the notable prevalence of pediatric obesity and the increased demand for prompt intervention, commercial programs have developed programs tailored for older youth. In particular, Weight Watchers International, the largest provider of commercial weight-loss services in the world with operations in 30 countries worldwide, allows adolescents to enter its programs. The Weight Watchers program, similar to other obesity programs, provides the essential components of obesity interventions via multiple modalities including group in-person meetings, Internet-based consultation resources, and pre-prepared food options. However, in March 2003, Weight Watchers limited access to its programs to children 10 years and older owing to a reported lack of evidence of long-term success in the younger age group.

CHILD OBESITY PROGRAM SUCCESS

Long-term weight maintenance success depends on the adoption of healthy lifestyle changes by children and their parents. Despite featuring components that would logically promote and support such lifestyle modification, child obesity programs have a mixed history of success. While short-term weight loss has been demonstrated by a variety of approaches, long-term success has been difficult to attain. Research is therefore needed to optimize currently available approaches and/or develop new methodologies to improve adherence to prescribed messages of obesity programs by affected children and their families.

SEE ALSO: Bariatric Surgery in Children; Behavioral Treatment of Child Obesity; Children and Diets; Family Therapy in the Treatment of Overweight Children; Pharmacological Therapy for Childhood Obesity; Physical Activity and Obesity; Physical Activity in Children; Physician Assisted Weight Loss; Weight Watchers.

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Children and Diets

OBESITY-ASSOCIATED ANNUAL HOSPITAL costs for children and youth more than tripled over two decades, rising from $35 million in 1979–81 to $127 million in 1997–99. The Centers for Disease Control and Prevention's (CDC's) 1999–2000 National Health and Nutrition Survey (NHANES) estimated that 10.4

As obesity increases in children, the questions of proper diet and education become more important.

percent of children aged 2–5, 15.3 percent of children aged 6–12, and 15.5 percent of children aged 12–19 are overweight, compared to rates of pediatric obesity of 7.2 percent (aged 2–5), 11.3 percent (aged 6–12), and 10.5 percent (aged 12–19) during the 1988–94 surveys. Overweight is more common among ethnic groups. Prevalence rates for African Americans are 8.4 percent (aged 2–5), 19.5 percent (aged 6–11), and 23.4 percent (aged 12–19). Mexican Americans have experienced more dramatic increases in the prevalence of obesity during the lasts 20 years compared to Caucasian Americans. Impoverished children are more often overweight than their economically advantaged peers.

Pediatric obesity is a difficult problem to treat because the clinician is treating the patient and the family. Childhood obesity involves significant risks to physical and emotional health. In 2000, it was estimated that 30 percent of boys and 40 percent of girls born in the United States are at risk for being diagnosed with type 2 diabetes at some point in their lives. Young people are also at risk of developing serious psychosocial burdens due to societal stigmatization associated with obesity.

CRITICAL PERIODS FOR WEIGHT GAIN IN CHILDREN

Early childhood is a critical period in the development of obesity due to adipose rebound. This is the time between ages 4 and 6, when a child's adiposity begins to increase after a low point in early years of life. Researchers found that the younger and heavier a child is at the time of adipose tissue rebound, the more likely he or she is to be overweight as an adult. Genetic as well as environmental and cultural factors play into obesity, but they do not account for the significant increase that has occurred over the last 10 years.

FACTORS INFLUENCING DIET AND OBESITY

Children become overweight for a variety of reasons. The most common causes are genetic factors, lack of physical activity, unhealthy eating patterns, or a combination of these factors. In rare cases, a medical problem, such as an endocrine disorder, may cause a child to become overweight. Children whose parents or siblings are overweight may be at an increased risk of becoming overweight themselves. Although weight problems run in families, not all children with a family history of obesity will be overweight. Shared family behaviors such as eating and activity habits also influence body weight.

A child's total diet and his or her activity level both play an important role in determining a child's weight. The increasing popularity of television and computer and video games contributes to children's inactive lifestyles. The average American child spends approximately 24 hours each week watching television—time that could be spent participating in some sort of physical activity.

Traditional nuclear families eating meals together are uncommon. Dual-income households, single-parent families, divorced parents who share custody, and parents who work nontraditional hours lead to unstructured meal times with little food preparation. As a result, there are more fast-food meals, no portion control, and no modeling by parents. There is evidence that meals eaten together as a family improve the nutritional intake of parents and children. According to a recent government report, pediatric obesity is one of the top threats to children's health. Excess weight in children and adolescents is due primarily to poor eating habits and inactivity.

DIETARY RECOMMENDATIONS

Children are good learners and they learn best by example. Setting a good example for your kids by eating a variety of foods and being physically active will

teach your children healthy lifestyle habits. In 2000, families ate an average of 4.2 commercially prepared meals a week, up from 3.7 in 1981.

Controlling portion size helps limit calorie intake particularly when eating high-calorie foods. Short-term studies showed that the bigger the portion size, the more participants ate. Fisher et al. in a study with preschoolers found similar results; they doubled the recommended portion size which resulted in an increase in energy intake.

The U.S. Department of Agriculture (USDA) recommends that children eat at least five servings of fruits and vegetables each day, engage in moderate physical activity for at least 60 minutes on most days, and limit their TV viewing and computer use to no more than 2 hours a day.

OVERWEIGHT CHILDREN

Reduce the rate of body weight gain while allowing growth and development. Consult a healthcare provider before placing a child on a weight-reduction diet. However, caloric intake is only one side of the energy balance equation. Caloric expenditure needs to be in balance with caloric intake to maintain body weight and must exceed caloric intake to achieve weight loss. To reverse the trend toward obesity, most Americans need to eat fewer calories, be more active, and make wiser food choices (Table 1, following page).

Prevention of weight gain is critical because while the behaviors required are the same, the extent of the behaviors required to lose weight makes weight loss more challenging than prevention of weight gain. The understanding of serving size and portion size is important in following either the Dietary Approaches to Stop Hypertension Eating Plan or the USDA Food Guide. When using packaged foods with nutrient labels, people should pay attention to the units for serving sizes and how they compare to the serving sizes in the USDA Food Guide and the DASH Eating Plan.

Parents and clinicians should strive first to maintain a child's baseline weight. Weight loss of no more than 1 lb per month is recommended in children aged 2–7 who have a secondary weight-related complication such as high blood pressure. Weight loss should be considered for children aged 7 and older if their BMI for age is 95 percent or greater, or they are at risk for becoming overweight (body mass index, or BMI, for age of 85 to 95 percent) and they have secondary complications. Children should never be placed on a restrictive diet to lose weight unless a doctor supervises one for medical reasons. Limiting what children eat may be harmful to their health and interfere with their growth and development.

To promote proper growth and development and prevent overweight, parents should offer the whole family a wide variety of food. Most of the foods in a child's diet should come from the grain products group (6–11 servings), the vegetable group (3–5 servings), and the fruit group (2–4 servings) (see chart for suggested serving sizes). A child's diet should include moderate amounts of foods from the milk group (2–3 servings) and the meat and beans group (2–3 servings). Foods that provide few nutrients and are high in fat and sugars should be used sparingly. Fat should not be restricted in the diets of children younger than 2 years of age.

Encouraging families to reduce fat is a good way to cut calories without depriving children of nutrients. Simple ways to cut the fat in the family diet include eating low-fat or nonfat dairy products, poultry without skin and lean meats, and low-fat or fat-free breads and cereals. Making small changes to the amount of fat in a family's diet is a good way to prevent excess weight gain in children; however, major efforts to change a child's diet should be supervised by a health professional. In addition, fat should not be restricted in the diets of children younger than 2 years of age. After that age, children should gradually adopt a diet that contains no more than 30 percent of calories from fat by the time the child is about 5 years old.

DO'S AND DON'TS OF DIETS IN CHILDREN

Children are good learners, so it isn't often necessary to overly restrict sweets or treats. While it is important to be aware of the fat, salt, and sugar content of the foods, all foods—even those that are high in fat or sugar—have a place in the diet, in moderation. Make available a wide variety of healthful foods in the house. This practice will help children learn how to make healthy food choices. Making mealtimes pleasant with conversation and sharing, and not a time for scolding or arguing can reinforce positive food choices. If mealtimes are unpleasant, children may try to eat faster to leave the table as soon as possible. They then may learn to associate eating with stress.

Table 1. Average Height and Weight Gain

AGE	WEIGHT GAIN	HEIGHT/CM	CAL/KG
0-3 months	1kg	2.5 /month	110-120
3-6 months	.5kg	1.25/month	105-115
6-12 months	.5kg/month	1.25/month	100-105
1-2 years	3kg/year	10-12.5/year	90-100
3-5 years	3kg/year	7.5/year	80
5-10 years	2kg/year	5/year	70
10-14 female*	17kg (double weight)	20% of adult height	40-45
12-16 male*	22kg (double weight)	20% of adult height	45-55

Adolescent growth occurs over 3–4 years related to onset of puberty

These activities offer parents hints about children's food preferences, teach children about nutrition, and provide children with a feeling of accomplishment. In addition, children may be more willing to eat or try foods that they help prepare.

Avoid continuous snacking because it may lead to overeating, but snacks that are planned at specific times during the day can be part of a nutritious diet, without spoiling a child's appetite at mealtimes. Allow children to eat only in designated areas of the home, such as the dining room or kitchen. Eating in front of the television may make it difficult to pay attention to feelings of fullness, and may lead to overeating.

Do not withhold food as a punishment; it may lead children to worry that they will not get enough food. For example, sending children to bed without any dinner may cause them to worry that they will go hungry. As a result, children may try to eat whenever they get a chance. Similarly, when foods, such as sweets, are used as a reward, children may assume that these foods are better or more valuable than other foods.

Caloric expenditure needs to be in balance with caloric intake to maintain body weight and must exceed caloric intake to achieve weight loss. To reverse the trend toward obesity, most Americans need to eat fewer calories, be more active, and make wiser food choices.

SEE ALSO: Behavioral Treatment of Child Obesity; Beverage Choices in Children; Changing Children's Food Habits.

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Children's Television Programming

Inactive behaviors, such as watching television, are considered risk factors for obesity in school-aged children.

NEXT TO SLEEPING, watching television is the most frequent activity engaged in by school-aged children. Included in the broad category of television watching are watching videotapes or DVDs; playing on the computer, including text messaging and computer games; and playing video games on stand-alone game consoles. Television-viewing is the number one leisure-time activity of most school-aged children. Since television was first invented in the 1950s, the number of television sets in the typical home has steadily increased, as have viewing times. With the expansion of programming from local networks to around-the-world broadcasts, with 24-hour-a-day availability, compared to limited evening hours when television was first introduced, and with programming targeted to tots to octogenarians and everyone in between, on every topic from sports to cooking and from space to discovery, television is everywhere. North American children aged 8–18 spend about 45 hours per week on media-related activities and estimates are even higher for preschool-aged children. The average high school graduate will likely spend 15,000 to 18,000 hours in front of a television, but only 12,000 hours in school. The top 25 percent of child viewers watch 4 or more hours of television per day. Almost half of children aged 8–16 watch 3–5 hours of television a day.

Approximately 30 percent of children in this age range are overweight and 15 percent are obese. Inactive behaviors, such as watching television or playing computer games, are considered risk factors for obesity in school-aged children. Kids who watch the most hours of television have the highest incidence of obesity. In fact, as much as 60 percent of the cases of overweight may be related to television viewing.

Obesity in children increases the more hours of television that children watch. As television viewing time rises, time spent exercising declines, especially among girls. Children who watch more than 3 hours of television a day are 50 percent more likely to be obese than kids who watch less than 2 hours. The top 25 percent of children who watch 4 or more hours of television a day have significantly more body fat than those who watch less television. The more time children spend watching television, the greater their weight increase. The situation is similar for preschoolers. In addition, while television viewing predicts weight gain, weight gain itself predicts greater television viewing, as being overweight makes exercising more difficult.

Increased television viewing and subsequent lack of exercise affect children adversely in many areas. For example, early childhood is a time of tremendous physical growth for children and the amount of physical activity positively affects the strength and amount of bone mass; lack of physical activity leads to diminished bone mass. Metabolic rates during television viewing are significantly lower than during resting periods, including sleep, for both obese and normal weight children aged 8–12. Increased health risks are associated with increasing obesity and disorders formerly seen only in adults are appearing more frequently in younger and younger individuals, such as type 2 diabetes and hypertension. So children are not only at risk of developing these diseases as adults, but they are also more likely to develop them as children, with increasing weight. Overweight children tend to grow into overweight adults. Health issues that used to affect only adults are increasingly affecting overweight children with concomitant costs to the individual and to the healthcare system.

Children who watch less television and play fewer video games show a significant reduction in measures of obesity, such as body mass index. As obesity becomes more of a health problem for our children, it is increasingly important to encourage children to

become more active. For example, if children have televisions in their bedrooms, the odds of being overweight jump by one-third for every hour watched. Limiting screen time and removing televisions from bedrooms can be important first steps to encouraging children into a more physically active lifestyle.

In addition to the physical inactivity associated with watching television, there are other reasons why watching television may be linked with childhood obesity. When watching television, children often mindlessly eat high-calorie or high-fat snack foods, which also lead to increased weight gain. So, children are not only avoiding physical activity and consequently burning fewer calories, but they are also increasing their caloric intake by eating "junk" food in front of the television. This eating is generally in excess of the daily caloric requirement and is typically associated with high-fat, high-sugar, and/or high-salt content snacks. Further, while some eating in front of the television consists of the actual consumption of regular meals, children tend to eat more and poorer quality food when eating meals while watching television.

Another factor promoting eating in front of the television is that television in general is associated with commercials for all kinds of food. Even television in schools contains ads for high-calorie, high-sugar, and high-fat foods. Child-oriented television programming in particular contains excessive advertising for child-friendly products, including sugary cereals, soda, candy, and fast food, all of which may encourage more eating of these products. On average, a child watching television sees a commercial from the food industry every 5 minutes or about 40,000 ads a year. Further, children are bombarded with well-designed TV ads from fast-food chains and other distributors of high-fat, high-sugar meals and snacks. The reason children are exposed to so many advertising commercials is that they work. Television advertising is effective or the food manufacturers would not spend as much as they do to advertise their products. Television advertising influences not only children to make direct purchases, but also indirectly as children influence the shopping behavior of family members. Children aged 2–6 who watch television are more likely to choose food products advertised on television than children who do not watch such commercials. Many advertisements are especially seductive in that they combine favorite television characters in the advertising, so that the star of the actual television show promotes the food products or is associated with merchandise such as toys and clothing that are related to the food products. These highly effective advertising campaigns, combined with a physically inactive lifestyle, have produced a generation of children who are at high risk for obesity-associated medical conditions. Not only does watching television increase the likelihood of children successfully influencing their parents to buy sugary cereals and sodas, but it also reduces the likelihood of their eating fruits and vegetables. There is some evidence that watching television leads children to believe that advertised foods such as fast foods are healthier and more nutritious than nonadvertised foods such as home-cooked meals.

Paradoxically, television has also been accused of promoting an unhealthy body image, as the stars of television shows are typically of low body weight. Overall, this can lead to unhealthy diet choices as well. The message to the viewer is contradictory and confusing. On television, it is better to be thin as this is where success and popularity lie, but the advertising suggests that one can be thin and still eat high-calorie, unhealthy food. The food that actors eat on television is also generally of low nutritional quality. Characters are more likely to be seen eating snacks or cereal than meals or fruit and typically drink coffee, soft drinks, and alcohol rather than milk or fruit juice. Overweight characters on television are stereotypically older, unattractive, and unpopular. While some argue that portraying obesity as undesirable is positive, there is concern that the stereotyping might lead to stigmatization.

Obesity has complex and multidimensional causes and treatment needs are no different. It is unlikely that television and related media will cease to be attractive to children, but they can be used more responsibly to help prevent obesity. Children can have limited access to television and parents can model good television viewing habits. Children can be encouraged to engage in physical activities on a daily basis to counter and perhaps replace the sedentary activities associated with watching television and playing computer games. Research suggests that almost one-third of cases of obesity could be eliminated if television viewing by children was severely restricted. While there are currently numerous regulatory guidelines for chil-

dren's advertising, more can be done by manufacturers, companies, and the government. Healthy eating plans can be incorporated into television storylines for even more impact.

SEE ALSO: Computers and the Media; Obesity and the Media; Overweight Children in the Media.

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Cholecystokinin

CHOLECYSTOKININ (CCK) IS a neurotransmitter important in the regulation of satiety and a gastrointestinal hormone responsible for the digestion fats and proteins. There are several molecular forms of CCK, although they are all products of the same gene. As a hormone, CCK is produced in the endocrine cells lining the upper parts of the small intestine. Gastrointestinal CCK receptors are found in the pancreas, gallbladder, stomach, lower esophagus, the end of the small intestine, and the colon. As a neurotransmitter, CCK is most highly concentrated in dopamine-containing neurons in the cerebral cortex, although it can be found in the midbrain as well. CCK is also seen in peripheral nerves innervating the intestines and the vagus nerve. CCK neuroreceptors are found in the brain and gastrointestinal peripheral nerves.

After a meal, especially one that is high in protein or fat, the digested products of fat, protein, and carbohydrates cause CCK release. CCK blood levels peak within 15 minutes and gradually decrease over the next 3 to 5 hours. The half-life of CCK in the blood is 1 to 2 minutes.

When released, CCK's primary role is to stimulate contraction of the gallbladder, facilitating bile secretion into the small intestine to break down fats. CCK stimulates the pancreas to release enzymes into the small intestine that break down proteins. Additionally, CCK also inhibits stomach acid secretion.

CCK regulates bowel motility by delaying emptying of the stomach, increasing satiation and decreasing food intake. Although CCK decreases meal size and duration, it does not alter satiety between meals. As a result, drugs mimicking CCK are not seen as an effective strategy for long-term obesity management because they do not inhibit hunger in a sustainable way. In fact, while CCK infusions reduce food intake, they also reduce the interval between meals such that energy intake returns to baseline within days.

CCK1 is a CCK receptor important in appetite control. When high concentrations of CCK bind to CCK1, appetite is suppressed. However, increased fat intake, while resulting in high concentrations of CCK, also reduces CCK1 sensitivity to CCK. Thus, the satiating effect of CCK is similar in normal weight and obese patients. Selective CCK1 antagonists are used to increase hunger in treatments of anorexia and cachexia.

SEE ALSO: Appetite Control; Disinhibited Eating; Fat Intake; Hunger; Neurotransmitters.

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Chromium Picolinate

OBESITY SIGNIFICANTLY INCREASES the risk for the development of Type 2 diabetes, hypertension, and cardiovascular disease. Chromium supplementation

has variable effects on body weight and body composition in people with and without diabetes. Chromium picolinate is a popular dietary supplement in the United States. The clinical response to chromium supplementation depends on chromium status, diet consumed, type and amount of supplemental chromium, and study duration. Chromium picolinate is a compound made up of a combination of the element chromium and picolinic acid. Chromium is a naturally occurring mineral. Trace amounts of chromium are found in everyday foods such as meat, poultry, fish, and whole-grain breads. When foods are processed, they are stripped of natural chromium, making American diets generally very low in Chromium; studies estimate an average daily chromium consumption of 33 mcg. Recently, studies from Harvard University of Public Health and Johns Hopkins University reported that low Chromium status is associated with higher risk of cardiovascular disease and diabetes. Chromium can be found with other ingredients in many herbal weight-loss products readily available at the local drugstores or on the Internet.

There have been no confirmed negative adverse events or side effects of using chromium picolinate supplementation in any of the controlled clinical trials. Based on the past three decades of research on chromium picolinate supplement, it has potential benefits on decreasing insulin resistance, thereby decreasing diabetes risk, and lowering elevated cholesterol. In a recent study, the addition of chromium picolinate to a regimen consisting of a sulfonylurea in subjects with type 2 diabetes improved glycemic control, increased insulin sensitivity, and significantly attenuated body weight gain as compared to sulfonylurea only. These subjects demonstrated less increase in percent body fat and less accumulation of visceral, subcutaneous, and total abdominal fat in those subjects randomized to chromium picolinate. However, in non-diabetics, chromium picolinate supplementation had equivocal results on glycemic control and weight loss.

In individuals without diabetes, chromium supplementation showed decrease in weight and fat in three larger studies. Most of the studies reporting no change in body weight may be due to specific discrepancies in study design, such as subject selection, sample size, exercise pattern, diet, end-point measures, baseline body fat percentage, variation in diets, population, measuring body fat distribution techniques, urinary chromium, and follow-up for a relatively short duration. Chromium supplementation has at best modest effects on body weight or composition in individuals with diabetes. In the 1990s, there were a few human and animal studies supporting claims that it is a "safe alternative to steroids" for increasing strength and lean muscle mass.

In a recent meta-analysis, it was observed that there was a significant differential effect on body weight found in favor of chromium picolinate (mean difference: −1.1 kg; 95 percent confidence interval (CI): −1.8 to −0.4 kg, n = 489). Sensitivity analysis suggests that this effect is largely dependent on the results of a single trial (mean difference: −0.9 kg; 95 percent CI: −2.0 to 0.2 kg, n = 335). The clinical relevance of the effect is debatable. Austrian scientists reported in a study with 36 obese patients that chromium picolinate is able to increase lean body mass in the maintenance period after a very-low-calorie diet without counteracting the weight loss achieved. Recent reports suggest that chromium picolinate enhances the activity of insulin, thus significantly aiding the body's glucose and fat metabolism, and in managing the breakdown of glucose and fat. Activated mediated protein kinase (AMPK) integrates nutritional and hormonal signals in peripheral tissues and the hypothalamus.

It mediates effects of adipokines (leptin, adiponectin, and possibly resistin) in regulating food intake, body weight, and glucose and lipid homeostasis. A recent animal study showed higher adiponectin levels which could contribute to the indirect effects on AMPK activity. In an 8-week study, supplementation of chromium picolinate reduced waist and hip circumferences ($p < 0.01$) and plasma leptin ($p < 0.001$) levels. Further long-term clinical studies with different doses are encouraged to confirm chromium's effects on AMPK activity and leptin resistance.

SEE ALSO: Supplements and Obesity.

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CNS/Hypothalamic Energy Sensing

ENERGY BALANCE, OR the regulation of energy intake (food) and energy expenditure (activity), is complex due to the high demands of energy throughout the body, and because neuronal activity is tightly coupled with energy utilization. Some cells in the brain, notably in the hypothalamus, are able to interpret and respond to signals from the central nervous system (CNS) and other tissues about the energy demands of the body. Together, the signals indicate the overall metabolic status of the animal. In addition, all neurons monitor and participate in maintaining their own energy status. Recent studies have characterized an important role for an enzyme called AMP-activated protein kinase (AMPK) in the brain's ability to sense energy status, both locally in the brain and on the larger scale of the whole organism. The brain's ability to sense and properly respond to energy state is critical for normal regulation of body weight and adiposity. When brain mechanisms are defective for detecting or responding appropriately to energy imbalances, underweight or overweight/obesity can result.

Hypothalamic neurons receive input about whole-body energy status, integrate these input signals, and coordinate mechanisms that defend energy balance. In a simplified model example, the arcuate nucleus of hypothalamus receives such signals. The arcuate nucleus contains two neuron types with receptors for hormones such as leptin and insulin, which reflect body fat mass and levels of blood glucose, respectively. Some neurons make proopiomelanocortin (POMC), precursor of some neuropeptides that stimulate melanocortin receptors on other neurons in the brain to decrease the organism's food intake and increase its energy use. POMC production is decreased by conditions of food deprivation or underweight (low leptin and insulin) and increased by overeating or overweight (high leptin and insulin).

Other arcuate neurons make neuropeptide Y (NPY) and agouti related protein (AgRP), neuropeptides that increase food intake and decrease energy expenditure. Food deprivation and underweight (conditions of low leptin and insulin) increase NPY and AgRP, and overconsumption and overweight (high leptin and insulin) decrease them, and also affect the general activity of these neurons. NPY/AgRP and POMC neurons project to other hypothalamic nuclei with receptors for these neuropeptides. Projections from these second-order neurons to other brain sites and the pituitary gland mediate the effects on energy balance.

In addition to responding to hormones such as leptin and insulin, and to neuropeptides released in response to leptin- and insulin-receptor activation, some hypothalamic neurons respond to altered local nutrient concentrations. For example, the brain depends upon constant supply of glucose to meet its metabolic demands. Certain hypothalamic neurons alter their activity (increase or decrease) in response to altered local glucose concentration. This is thought to result from altered levels of glucose uptake into cells, and thus effects on the generation of ATP (adenosine triphosphate) from glucose. ATP is a high-energy molecule considered to be "energy currency" in the cell. ATP can bind to certain ion channels in the cell membrane, which then open or close, and affect the neuron's level of excitability. Glucose-sensing neurons may be "metabolic sensors" in general. They can respond to other metabolites such as lactate (product of glucose), ketone bodies (product of fat), and fatty acids (components of fat molecules). Glucose-sensing neurons also express receptors for leptin and insulin, which in glucose-excited neurons

decrease the level of activity by direct action at ATP-influenced ion channels.

Thus, these neurons can integrate a variety of metabolic, hormonal, transmitter, and peptide signals related to metabolic status. Arcuate nucleus NPY neurons are glucose-inhibited neurons, and POMC neurons are glucose-excited, and exemplify such metabolic integrators.

Recently, there has been great interest in how the ratio of ATP level to that of the lower-energy AMP (adenosine monophosphate) may be an important intracellular signal of metabolic status in hypothalamus, via its interaction with the enzyme AMP-activated protein kinase (AMPK). AMPK is a member of a family of protein kinases (enzymes that add phosphate onto other molecules), and has long been recognized as a sensor of cellular energy balance in peripheral tissues such as muscle and liver.

Increased cellular AMP/ATP ratio activates an upstream AMPK-kinase, which adds a phosphate to AMPK (pAMPK). In turn, active pAMPK adds phosphates to other molecules, altering cellular metabolism and gene expression via multiple mechanisms, collectively inhibiting anabolic processes and stimulating catabolic processes. These changes conserve and restore cellular levels of ATP. Recent studies focus on the role of AMPK in neuronal responses to alterations in energy status.

Hypothalamic AMPK activity has recently been shown to control food intake. Brain administrations of anorexigenic agents (food intake reducing) such as insulin, glucose, or a melanocortin receptor stimulator decrease AMPK activity in several hypothalamic nuclei in animal models. Leptin injection into the arcuate nucleus inhibits AMPK activity there. Expression (by use of a virus) of a permanently active mutant form of AMPK in hypothalamus is sufficient to increase food intake and body weight in animal models, and expression of a permanently inactive AMPK form decreases feeding and body weight.

Recent studies suggest that hypothalamic AMPK phosphorylation status is unresponsive to leptin in mice made obese on a high-fat diet. AMPK, and molecules that interact with it, may provide targets for antiobesity therapies. Brain administration of a compound that directly activates AMPK increases food intake in rodents, whereas another compound that inhibits AMPK decreases feeding. Drugs that manipulate the metabolism of fatty acids, altering ATP levels in hypothalamus, can also decrease hypothalamic AMPK activity to reduce food intake and body weight.

SEE ALSO: Agouti and Agouti Related Protein; Appetite Control; Appetite Signals; Central Nervous System; Hormones; Hypothalamus; Insulin; Leptin; Melanocortins; Neuropeptides; NPY (Neuropeptide Y); Obesity and the Brain; POMC (Proopiomelanocortin).

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Cognitive-Behavioral Therapy

THE COGNITIVE-BEHAVIORAL THEORY (CBT) has its roots in the idea that thoughts and feelings precede actions, and that inaccurate thoughts drive unhealthy behaviors. The intention of CBT is to use cognitive techniques to challenge unhelpful or distorted thoughts while using behavioral techniques to reduce problematic behaviors. CBT focuses on current thinking, problematic behavior, precipitating factors, and developmental events. In addition, the approach requires active participation of the patient; it is goal oriented, problem focused, and structured while using many techniques to change distorted thinking, mood, and thus problematic behavior.

Specifically, in terms of disordered eating, patients are asked to maintain daily monitoring logs of foods eaten, eating disordered behaviors, thoughts, feelings, and details about the situation in which these behaviors occurred. Self-monitoring yields objective infor-

mation that can be used with behavioral interventions such as cue recognition, desensitization, or reinforcement. It also reveals patterns of automatic thoughts (e.g., "I am fat," "I can't eat this or I am weak," "I blew it, now I might as well eat more") that reflect broader core beliefs. Cognitive restructuring challenges these thoughts with rational alternatives. CBT is effective when beliefs change, automatic thoughts decrease, and problem behaviors are reduced.

CBT for weight control or obesity consists of core techniques including dietary recommendations, exercise, cognitive techniques, stimulus control, relapse prevention, and social support. Although weight loss is certainly the goal of treatment, weight is deemphasized as it is not a behavior per se, but a result of the behavior changes. Several important techniques are incorporated into this treatment.

NUTRITION EDUCATION

It is important for patients to understand healthy versus unhealthy eating. Unhealthy eating consists of emotional eating (i.e., eating for any other reason than hunger including boredom, anger, frustration, excitement, etc.), eating in response to cravings or urges (which is also often linked to emotional reasons), and unhealthy dieting (either behaviorally via purging or restricting or psychologically in which dieting is conceptualized as a transitional time rather than a healthy permanent lifestyle change). Unhealthy dieting can result in physical problems (e.g., intense hunger, low energy, fatigue, headaches, visual problems, weight gain, electrolyte disturbance, dental problems, gastrointestinal problems), cognitive problems (e.g., focus on food, loss of interest, poor concentration, memory problems, difficulty with comprehension and decision making), and emotional problems (e.g., stress, irritability, and anxiety, depression). Individuals learn about healthy, balanced nutrition, to remain within a certain calorie range necessary for weight control/maintenance, and to consume three meals and approximately two or three planned snacks. Many individuals who overeat may resist eating during the day, which sets them up for overeating as they find their eating behavior out of control later in the day. Establishing a regular eating pattern includes setting meal times, not allowing greater than 3–4 hours between eating times, not skipping meals, and avoiding eating in between planned meal/snack times.

SELF-MONITORING

Self-monitoring of energy intake is the hallmark of weight control interventions as it helps individuals understand their current eating patterns, so that they identify patterns that need to be changed. It also helps them learn about the nutritional value of individual foods, assists in planning meals, supports healthy food choices, and aids in helping individuals to avoid overeating or unhealthy eating given that they are being accountable. It also helps individuals notice their behavior changes and successes and observe the formation of new patterns.

PHYSICAL ACTIVITY

It is recommended that patients gradually increase both lifestyle and structured exercise. Physical activity is essential for weight loss, but it is also highly correlated with long-term weight management.

COGNITIVE RESTRUCTURING

Cognitive techniques are used in weight control for several reasons. First, because many automatic thoughts pertain to weight and factors that influence weight control, cognitive restructuring helps patients accept more rational ideas about weight and weight loss. This is a gradual process in which individuals become aware of the automatic thought, challenge it, question and evaluate the evidence that supports/disconfirms the thought, consider alternative views, determine the effect of the automatic thought on other thoughts, feelings, and behaviors, and identify typical thinking errors. In addition, because negative mood and stress can result in overeating, cognitive techniques are used to reduce emotional distress, ultimately reducing the need to eat for emotional reasons.

BEHAVIORAL CHANGING

Individuals become skilled at understanding both cues and consequences of eating behavior. A cue is anything that may lead to a specific thought, feeling, or behavior. Cues can be internal (a thought; negative thoughts about past, anxieties about future), physiological (a bodily response: stomach growling, fatigue), external (a situation: social isolation, social gathering, interpersonal conflict, walking past a bakery, advertisements of food), or a feeling (depression, anxiety, happiness). On the other hand, a consequence is the result of behavior. Consequences can be of several

different types and either positive or negative. For example, a physiological consequence of eating can be (1) reduce hunger (positive), and (2) high cholesterol (negative). An emotional consequence can be (1) temporary relief of anger or boredom (positive) and (2) depression, guilt, shame (negative). Often, when individuals who are attempting weight loss overeat, they do not think of the consequences in advance. In CBT, individuals practice identifying and resisting cues to eating and thinking about the consequences prior to eating.

BEHAVIORAL STRATEGIES

Aside from challenging thoughts, CBT also incorporates behavioral strategies to help avoid unhealthy eating behavior. CBT helps individuals with stimulus control including following an eating schedule, food shopping from a list, removing serving dishes from the table, buying foods that require preparation while keeping problem foods out of sight. Individuals also practice rearranging cues to unhealthy eating by avoiding high-risk cues, doing one thing at a time (i.e., do not eat and watch television), and strengthen cues for desired behavior (plan an afternoon activity instead of going home alone and eating). Another strategy is to change the response to cues. This includes building in a pause between having thought to eat and the actual act of eating. This pause will allow one to practice thought restructuring, focusing on the consequences, or finding an alternative behavior. The time interval of pauses can gradually increase. Changing the response also includes choosing an alternative behavior to unhealthy eating and "surfing the urge," in which people experience a peak in the urge to eat but then experience the decline. This teaches individuals that the craving will in fact go away on its own. Individuals also practice focusing on consequences. For instance, they may excuse themselves when they face occasional lapses, and administer mental or material rewards to themselves for overcoming unhealthy eating episodes.

RELAPSE PREVENTION

Relapse prevention is paramount to successful weight maintenance. This involves continual practice of the skills learned in CBT. Patients learn the difference between an occasional "slip" or "lapse," in which one engages in unhealthy eating behavior but regains control of one's behavior, versus a "relapse" in which one loses control of one's behavior and then ultimately reverts to one's original eating patterns. An important part of relapse prevention is identifying high-risk situations in advance and learning how to plan and cope with them, so that they do not lead to a relapse. Accordingly, individuals will recognize and anticipate high-risk situations and cope with them by planning both a behavioral response (e.g., avoidance, surf the urge) and an emotional plan should they engage in unhealthy eating behavior (e.g., *"I learned that this is a high-risk episode. I will now plan ahead for the next time I am faced with this situation. I am still doing a good job adhering to my healthy eating"*). A panic card can be developed to help people when they are faced with high-risk situations. The panic card can include positive self-statements, several coping responses, alternative behaviors, and phone numbers.

CBT-based weight control treatments typically consist of 16- to 20-week programs and are usually offered in group formats. While some individuals are successful at losing significant amounts of weight, the majority of patients lose a modest 10 to 15 percent of their initial body weight. This amount of weight loss correlates with improvements in hypertension, hypercholesterolemia, diabetes, and all-cause mortality. Long-term weight-loss maintenance is associated with continued exercise, healthy diet, self-monitoring, and continued patient–provider contact.

CBT is also applied to eating disorders including anorexia nervosa (AN), bulimia nervosa (BN), and binge-eating disorder (BED). For AN, CBT typically focuses on cognitions relating to fear, control, low-self esteem, body image distortion, and perfectionism, with behavioral targets including decreasing caloric restriction, decreasing physical activity, and reducing avoidance. Previous studies do not provide sufficient support to recommend CBT as the primary treatment for AN in the underweight state. However, CBT delivered to weight restored patients is associated with significantly reduced relapse risk and increased likelihood of good outcomes compared with nutritional counseling with education regarding food exchanges.

CBT for BN focuses on common cognitions in women with BN (e.g., loss of control, fear of weight gain, self-esteem) while also addressing behaviors such as binge eating, purging, and maladaptive anxiety reduction strategies. Several studies have reported that CBT is effective in reducing the essential behavioral

(i.e., binge eating and purging) and psychological features of BN. Regardless if delivered in individual or group format, CBT has been associated with decreases in binge frequency, vomiting, laxative use, and psychological features.

Because many individuals with BED are overweight, CBT for BED often targets both decreasing binge eating and managing weight. Group CBT for BED has been shown to be effective at reducing number of days binged, BMI, disinhibition, hunger, depression, and self-esteem. In one study, compared to behavioral weight-loss treatment (BWLT), CBT resulted in faster reduction of binge episodes while BWLT resulted in faster weight loss. However, these differences did not persist at the 12-month follow-up.

Regardless of the type of disordered eating, CBT is particularly helpful in changing thoughts and behaviors. When applied in a group format, individuals are able to learn the successful CBT tools while also receiving social support from other group members. However, individual therapy may also be suggested (particularly for diagnosable eating disorders) to offer individual attention to work on more specific issues.

SEE ALSO: Mood and Food; Non-Diet Approaches; Self-Esteem and Obesity.

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Colon Cancer

COLON CANCER IS unregulated cell replication and accumulation in the colon. Colon cancer is often called colorectal cancer. Colorectal cancer ranks as the fourth most common cancer in the United States among men and women.

Colon cancer is more frequent in obese individuals. In 2002, 41,000 cases of colon cancer were due to obesity in the United States. Studies have shown increase of colon cancer with higher body mass index (BMI) and abdominal obesity. Increased levels of insulin in obese individuals have been suggested to play a role in colon cancer.

Food enters the colon after becoming digested by the stomach and the small intestine. The colon removes water, nutrients, and forms stool from food. When colon cancer spreads to other parts of the body, the cancer is still recognized as colorectal cancer because the abnormal cells are the same. Colorectal cancer spread to the liver is diagnosed and treated as metastatic or distant colorectal cancer. The liver and lymph nodes, which are immunological filters from foreign bodies, are common locations to where colon cancer spreads.

Factors that may increase individual risk for colon cancer include age over 50, colorectal polyps (extra tissue growths in the colon), family history, inflammation of the colon, poor diet, and smoking. The non-polyp-inherited cancer, HNPCC, is more common than the polyp-inherited cancer, FAP. Inherited colon cancers can be detected by genetic testing. Studies have shown with diets high in fat and low in calcium, folate, fiber, fruits, and vegetables to increase the risk of colorectal cancer.

Detecting immunologic proteins in the blood common in cancer patients or a bloody stool test can screen for colon cancer. A lighted tube, such as a sigmoidoscope or colonoscope, is used to examine the colon for polyps. Polyps and tumors can also be detected with X-ray procedures. The detection of polyps may be followed by a tissue sample check for cancer cells. Ultrasounds, X-rays, computerized axial tomography (CAT) scans, and magnetic resonance imaging (MRI) may be used to determine if the colon cancer has spread to other areas. Symptoms of colon cancer include diarrhea, constipation, bloody or narrow stools, bloating, loss of weight, nausea, vomiting, and feeling tired. Colon cancer can be categorized in five stages (0–IV). Higher stages are categorized by the deeper penetration of the cancer in the colon wall and the spread to other parts of the body.

Cancerous polyps are surgically removed, which is the most common treatment. Sections of the colon with cancer cells may also be surgically removed. Radiation and chemotherapy are also used to attack cancer cells. Exercise, fruits, vegetables, and fiber intake are often recommended to prevent colon cancer.

SEE ALSO: National Cancer Institute; Obesity and Cancer.

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Combined Approaches to Treatment

WORLDWIDE PREVALENCE RATES of obesity and overweight are rising, and safe and effective treatment strategies are urgently needed. Obesity has been recognized as a preventable and treatable disease, and there is a growing evidence base showing the efficacy of various clinical strategies for its management. Given the overwhelming research focus on this disease, it is likely that the coming years will bring more treatment options, raising the chance that patients will have meaningful and sustained weight loss.

Obesity is a multifactorial problem. According to the National Institutes of Health (NIH), the main focus of an obesity intervention should be on health improvement instead of body weight reduction. People who are overweight or obese have increased risk for more than 30 other diseases such as heart disease, cancer, breathing problems, and diabetes. For the millions of obese and overweight, the ideal model of obesity treatment is a comprehensive intervention including dietary, activity-related, behavioral, pharmacological, and/or surgical components that is geared toward individual needs.

Not all treatments will work for everyone. When a diet, exercise, or drug plan does not produce expected results, either the individual is blamed for noncompliance, or the treatment is deemed ineffective. Most people can be helped to manage their weight, and healthcare providers must avoid stigmatizing and blaming patients for their obesity. It is important that practitioners understand that patients do not come equipped with appropriate compliance skills, and that the practitioner must assist in teaching the underlying information and attendant skills. The more obese the patient, the more important it is to understand that the weight loss may be outside the control of the patient and the lifestyle modifications without more serious intervention such as bariatric surgery.

For the average weight-loss patient, however, dietary-focused weight-management programs can be successful when combined with appropriate activity, behavioral and lifestyle modification, and education and skill training. These programs are generally utilized for patients with body mass indexes (BMIs) under 30, and with no comorbidities. The key behavior combination for success of these weight-managed programs is reduced energy intake, reduced (but not eliminated) dietary fat and carbohydrate intake, increased physical activity, and nutrition and health education relative to the patient's personal situation.

Activity-related weight-loss programs alone do little to cause short-term weight loss. The ineffectiveness of moderate exercise by itself is not surprising considering 1 lb. of body fat contains about 3,600 calories. Combined with appropriate additional interventions such as dietary, behavioral, and lifestyle modifications, physical activity contributes to avoiding weight gain, maintenance of weight loss, and contributes to the overall health of people at any weight.

Weight-loss medications (pharmacological interventions) are currently recommended for use only as an adjunct to diet, exercise, and behavior modification. Little is known about the benefits of combining behavioral and pharmacological therapies, or about the mechanisms that would make these combined approaches more effective than using either approach alone. Weight-loss medications must be prescribed in combination with, rather than in lieu of, lifestyle modification. Researchers found that best results were obtained when medications were combined with an intensive, group program of lifestyle modification.

Behavioral treatments seem to help obese individuals control the external (i.e., food-related) environ-

ment, whereas pharmacotherapy may control the internal environment by reducing hunger, cravings, or nutrient absorption. Some professionals have voiced concerns that medication may undermine lifestyle modification and inhibit appropriate behaviors that need to be practiced if medication reduces hunger because patients may not be motivated to practice proper eating habits. Patients must take responsibility for managing their obesity because diet drugs will not force anyone to stop eating. An uncommitted patient can "out eat" the weight-loss effects of any drug. Patients must truly understand that the drug is merely one tool in the toolbox and many tools are needed to complete any major reconstruction job.

Drugs are not a quick fix and there are risks in taking any medication. The use of medications may also convey the message that obesity is "biological," and personal efforts to change the disease process are futile. Professionals should inform patients how the medication and the patient's own efforts to modify diet and activity habits potentially complement each other to produce a better outcome. Education of the patient about the disease of obesity and its progressive state is still a key component of the treatment plan. Patients and practitioners must understand the nature of this condition in all its facets.

For the very obese, there are few noninvasive options available that result in long-term weight loss. Bariatric surgery helps reduce energy intake by physically limiting food intake and/or absorption, depending on the procedure. Surgical intervention in combination with behavior, nutritional, and activity-focused programs are the most successful for the obese and very obese. Surgery of any type alone does not work in the long term. A lifelong commitment to each critical aspect of lifestyle change, behavior modification, nutritional reeducation, and activity when choosing this intervention by the individual is essential to long-term success.

The American College of Physicians (ACP) recommends surgeons and surgical centers with high levels of experience in performing these procedures because, typically, they have better outcomes in terms of higher efficacy and lower complication rates. Patients contemplating surgery should seek these providers out. The recommended overall strategy for managing overweight and obese patients should always include appropriate diet and exercise, as well as appropriate education and skill training. Additionally, it is important to emphasize that a positive attitude of support and encouragement from all professionals, nutritionists, exercise physiologist, behaviorists, nurses, psychologists, and physicians is crucial to the continuing success of these patients.

Practices should provide and strongly encourage patients to attend support groups to help them understand the changes they are making, the changes that are occurring in their lives, and to learn the skills needed to be successful in the long term. Patients seeking to make lasting lifestyle changes cannot do so in isolation. They need guidance, information, role modeling, feedback, demonstration, illustration, reassurance, and support. Practitioners should be sure to assist patients in finding support groups that are not therapy based, but rather based on learning the information and skills necessary to solidify the changes being made in the patient's life. Should the patient require therapy for particular issues the patient can be directed to an appropriate professional for that purpose.

SEE ALSO: Bariatric Surgery in Women; Behavioral Treatment in Childhood Obesity; Caloric Restriction; Carbohydrate and Protein Intake; Cognitive Behavior Therapy; Community Programs to Prevent Obesity; Diet Myths; Dietary Restraint; Dieting: Good or Bad?; Disinhibited Eating; Drug Targets that Decrease Food Intake/Appetite; Drugs and Food; Drugs that Block Fat Cell Formation; Exercise; External Controls; Family Behavioral Interventions; Fat Intake; Fenfluramine; Fitness; Future of Medical Treatments for Obesity; High Carbohydrate Diets; Impulsivity; Inaccessability to Exercise; Liquid Diets; Low Calorie Diets; Low Fat Diets; Macrodiets; Medications that Affect Nutrient Partitioning; Medications that Increase Body Weight; New Drug Targets that Prevent Fat Absorption; New Drug Targets to Improve Insulin Sensitivity; New Drug Targets to Increase Metabolic Rate; Night Eating Syndrome; Non-Diet Approaches; Nutrition Education; Orlistat (Xenical); Pharmacological Therapy for Childhood Obesity; Physical Activity and Obesity; Support Groups; Very Low Calorie Diets.

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Community Programs to Prevent Obesity

THE PRIMARY MODIFIABLE causes of obesity are poor nutrition and lack of physical inactivity. Community characteristics can contribute to obesity by raising barriers to eating a healthy diet or promoting the consumption of high calorie/low nutrient density foods, and to having sufficient opportunities for regular physical activity.

For example, individuals living in many urban communities face challenges in terms of access to, and availability of, healthy food choices or availability of safe and appealing community resources for physical activity. Increasing social capital through community participation and local advocacy efforts can help to improve community resources and safe access to enable healthy lifestyles.

In recent years, numerous intervention programs have been developed to address community-level risk factors for obesity. Such programs often take an ecological view of obesity risk factors, which view the community as an environment in which conditions can be modified to lower risk. These programs can be classified in a number of ways, but the following provides a framework for the illustrative overview of research literature that follows. Community intervention programs can be classified as seeking to influence factors such as:

- Physical environment (e.g., availability of fresh fruits and vegetables or low fat milk; recreational opportunities, and point of decision prompts for healthy choices)
- Family and social environment.
- Community outreach and mobilization (e.g., engaging community-based organizations and resources)
- Social capital (to facilitate community outreach, healthy environments, and encouragement of healthy activities of control weight)
- Social marketing and health communication campaigns

A generalized conceptual framework suggests that interventions to enhance social capital, to mobilize communities, and to conduct social marketing can mutually reinforce each other in enhancing the physical environment and the family and social environment to prevent obesity. The impact of interventions is a function of their intensity (e.g., reach, frequency, and duration). The effects of interventions on obesity are mediated by changes in knowledge, attitudes and beliefs, intentions to engage in behaviors, and to maintain behaviors that can prevent obesity.

Examples of these behaviors include increased use of enhanced opportunities for physical activity, or improved dietary practices. There is also mounting evidence that reducing children's TV viewing can help to reduce their risk of obesity. Among infants, increasing breastfeeding incidence and duration appears to reduce the risk for overweight.

OBESITY PREVENTION AND COMMUNITY ENVIRONMENTS

Obesity prevention is an emerging science developing in response to alarming increases in overweight and obesity in children and adults. In a recent quantitative review of pediatric obesity prevention programs, only 10 studies published since 1990 met the author's criteria for long-term (one year or longer)

evaluation. Of these studies, only six targeted both diet and physical activity. All six were school-based and demonstrated null or weak associations with health-related outcomes.

There is now considerable agreement that escalating rates of obesity can be tied to characteristics of the environments in which people live. Accordingly, our consideration of the community obesity prevention is grounded in a socio-ecological framework that acknowledges that the factors that influence pediatric and adult obesity are present at many levels . For instance, a study in London, England found that improved lighting on footpaths increased footpath use by 34 to 101 percent and a study in Toronto, Canada found that the creation of bike lanes increased bicycle use by 23 percent.

One lesson from community studies is that changes in the physical environment should be accompanied by active promotion and point of decision prompts to action behaviors. For instance, signs placed by elevators and escalators can increase the percentage of people who use nearby stairs by more than 50 percent. One recent review adds that point of purchase prompts may have greater impact on food choices in settings, such as worksites, where fewer alternatives were available. Similarly, a systematic review by the Community Preventive Services Task Force (2005) found that increasing access to places for physical activity in worksites (8 studies) or low-income communities, combined with information activities, increased by a median of 48 percent the proportion of adults who exercised three of more times a week.

FAMILY ENVIRONMENT, DIET, PHYSICAL ACTIVITY, AND SEDENTARY MEDIA USE

Families live in physical and social environments, including their local community as well as the larger media and policy environments that surround them, that influence their beliefs and attitudes about health, dietary behaviors, and physical activity. In turn, a number of parental behaviors may moderate children's and larger family obesity-related outcomes. For example, parent-child interactions surrounding food can be instrumental in determining dietary behaviors that persist into adulthood. Three parenting styles—over-control, dietary restriction, and using food as a reward—have received the most attention in terms of their potential impact on weight status. Parents who are excessively controlling with regard to their child's eating may interfere with the child's ability to monitor and regulate caloric intake. The role of meal patterns and family meals has been examined as a potential influence on children's eating habits and risk for obesity. Much of this research, however, involves adolescents rather than children. These studies show that frequency of family meals and the presence of parents at family dinner are associated with better diets including greater intake of fruits and vegetables.

Parents can alter young children's dietary habits and reduce the risk factors associated with pediatric obesity by providing their children with access to healthful foods. A recent study examining the role of shopping behavior found that the likelihood of coming from a household classified as overweight increased 14 percent for every additional 10 grams of fat purchased per person per day. Parents who provide nutrient-rich foods and limit availability of energy-dense and sugar-sweetened foods create an environment that supports good nutritional habits.

Among young children, increasing preference for fruits and vegetables is assumed to lead to greater intake later in life, and parents can play a pivotal role in this process through exposure. For example, consistent exposure to new foods, such as fruits and vegetables, can increase the likeability of these new foods . Increases in intake of fruits can be further enhanced through careful selection processes that identify more appealing choices.

Parents may also directly influence children's weight status by encouraging physically active behavior and discouraging sedentary behavior. The current literature suggests parents are more likely to influence their children by providing social support and encouragement that leads the child to develop a sense of competence related to engaging in physical activity.

Parents can reduce their children's sedentary behaviors by setting limits on such activities. A number of studies over the past two decades have identified a consistent, though moderate, relationship relation between overweight and obesity and the amount of time spent watching television. However, the mechanism for the relationship between TV viewing and obesity—possibly displacement of physical activity; increased snacking while watching TV, or increased consumption of high caloric foods in response to advertising on TV—has not been fully explicated.

Another critical mediator of behavior change, the stages of change construct, holds that intervention success is enhanced when program messages are stage-matched. Accordingly, readiness to change could potentially influence credibility, awareness, and recall of program messages, as well as behavioral outcomes. Studies of dietary and exercise interventions have shown a positive association between readiness to change and social cognitive mediators . These studies identify self-efficacy as a critical factor in motivating progression to higher stages and actualizing behavior change.

MODERATING EFFECTS OF THE PHYSICAL AND SOCIAL ENVIRONMENT

Health promotion campaigns disseminated in community settings need to consider and attend to a host of potential factors in the real-world setting that can influence the magnitude of observed intervention effects. Program effects will be strongest among families who live in communities that share a common interest in healthy lifestyles and provide residents with the materials and resources that support good diet and physical activity. Some of these factors may moderate intervention effects, especially when parents and children perceive them as barriers to behavioral change.

The term "obesigenic environment" has gained traction as a descriptor of the characteristics of the social and physical environment that inhibits these goals. Many factors contribute to this type of environment. For instance, the ability to increase fruit and vegetable consumption may be hampered in neighborhoods that lack convenient access to supermarkets. Access to supermarkets has been associated with fruit and vegetable consumption . Shoppers perceive supermarket produce as higher in quality than produce obtain at small groceries or convenience stores and report greater intake of fruits when they live in close proximity of a supermarket. Access to supermarkets has been shown to vary by neighborhood socioeconomic level and ethnic composition. Supermarkets are three times as prevalent in wealthiest neighborhoods compared to the lowest wealth neighborhoods.

There is speculation that increased frequency of eating meals out of the home has contributed to the prevalence of obesity . Restaurant portions are larger and more energy-dense than foods prepared at home . There is some evidence that the quality of restaurant offerings in lower income areas presents a barrier to eating a healthy diet outside of the home. Studies of the distribution of fast-food restaurants in low-income neighborhoods, however, indicate no association between proximity to fast-food restaurants and obesity in children and, in fact, suggest that significant clustering of fast-food outlets around schools is more prevalent in neighborhoods of higher socioeconomic level.

ENHANCING SOCIAL CAPITAL

Social capital is related to self-reported levels of general health and obesity, and may influence observed health- and obesity-related outcomes. Neighborhoods that have high levels of community participation may increase dissemination of campaign messages via a denser network of social contact and shared interest in the development of community resources. There is also a complex and potentially reciprocal relation among characteristics of the physical environment, social capital, and physical activity. For example, walking-friendly, mixed-use neighborhoods have been associated with increased physical activity in the form of more walking for transportation.

Other studies have examined the role of community-level socioeconomics in the distribution of leisure time physical activity resources. Individuals in moderate to low socioeconomic status communities may have less ability to control their physical activity level in the face of insufficient resources. Even when community resources are available, community members are unlikely to use them when they are perceived as inaccessible due to public safety issues.

SOCIAL MARKETING, HEALTH COMMUNICATION AND OBESITY PREVENTION

Children and their parents are also exposed to a wide array of health information and media messages. All these contribute to the attitudes, beliefs, and knowledge of parents and children and direct their health-related behaviors regarding diet and physical activity.

Mass media and community-based outreach interventions have been employed in a wide array of health promotion activities. Overall, these programs have demonstrated modest statistical effects that, on a population level can translate into large absolute numbers of individuals receiving prevention and health promotion benefits. Approaches that go be-

yond simple mass media and include community organizations offer greater impact.

Social marketing acknowledges that effective behavior change must evolve through a reciprocal process so that the proposed change provides an acceptable and beneficial solution to the audience for the identified problem. In addition, the beneficial aspects must outweigh any perceived costs in order for the target population to "buy in" to the proposed behavior change . In this regard, social marketing shares a participant-oriented perspective with health promotion approaches. Unlike more traditional health promotion approaches, however, social marketing draws effective concepts and techniques from the commercial marketing sector such as audience segmentation, message targeting, and branding. Social marketing approaches incorporate theories of behavior change such as Social Cognitive Theory and Theories of Reasoned Action and Planned Behavior to identify modifiable behavioral determinants that are the targets of persuasive appeals and construed as the mediators of the behavior change; it incorporates communication theories such as the Elaboration Likelihood Model to increase the persuasive impact of its messages and increase the likelihood that those messages will affect specified determinants.

SOCIAL COGNITIVE MEDIATORS AND PHYSICAL ACTIVITY AND DIETARY OUTCOMES

Recent studies on diet and physical activity in children and adults have identified a number of social cognitive constructs that can be expected to mediate the relationship between health promotion messages and program outcomes. There is some evidence that nutrition knowledge may be an important mediating factor. Results of a community-based diabetes education program indicate that increased knowledge led to greater levels of dietary self-efficacy.

The role of self-efficacy in the "5 A Day" programs may be due to the greater behavioral specificity of that program, indicating that efforts to influence self-efficacy require more targeted approaches. Outcome expectancies have also been identified as a potentially important mediator of programs aimed at changing dietary behavior. Effective alteration of outcome expectancies will also require attention to expectations in order that new set of expectancies are well matched to participants' value structure, which may vary along sociodemographic lines. Perceived barriers may limit behavior change when individuals believe that situational factors render change inaccessible or believe they already achieved target goals.

EFFECTS OF HEALTH MESSAGES ON FAMILY DIET AND PHYSICAL ACTIVITY OUTCOMES

A growing number of media-based health promotion campaigns provide evidence of the potential for social marketing campaigns to affect dietary behavior and physical activity levels. The "1% or Less" campaign provides strong evidence that a properly articulated social marketing campaign can produce meaningful changes on food purchasing behavior and dietary behavior. Average intervention effects across four separate implementations of "1% or Less" were very strong. Self-reported purchase of low-fat milk increased 21 to 38 percent in intervention communities relative to control communities; similarly, community-level sales of low-fat milk increased 13 to 125 percent in intervention communities relative to control communities. The "5-A-Day" program was developed to increase consumption of fruits and vegetables and has been implemented in a number of high-risk subpopulations. For instance, "5 A Day" activities targeting women in a low-income, primarily African American population resulted in a relative increased fruit and vegetable consumption of close to one-half serving per day in intervention communities. Other "5 A Day" programs have been less successful, but provide evidence of the mediating factors that influence dietary change.

The research base examining the direct effects of media-based social marketing interventions on obesity-related health behavior is relatively small, but growing. In general, these programs have demonstrated high levels of program awareness and recall. For instance, the "VERB: It's what you do" Campaign, sponsored by the CDC, employed a social marketing approach to increase levels of physical activity and decrease sedentary behaviors among U.S. children. At the end of the first year, the program achieved high levels of awareness and demonstrated increases in physical activity among selected subgroups. High levels of awareness and recall have been reported in other physical activity promotion programs.

Media efforts work most effectively as part of coordinated social marketing campaigns. For example,

consider the Lexington, Kentucky Verb Summer Score Card, in which local businesses teamed with a community coalition to offer free access to opportunities for physical activity to pre-teenagers. The program built on the national VERB media campaign, and developed a community contest in which children earned points by participating in a variety of physical activities in the community. Recently, community-based social marketing campaigns have begun to use strategies such as branding of healthy lifestyles derived from tobacco control, HIV/AIDS, and other successful prevention intervention programs.

DISCUSSION

Several important lessons can be learned from review of the existing community obesity prevention literature that should be considered when developing future public health education campaigns focused on promoting healthy eating and physical activity:

- *Source credibility and message receptivity.* Social marketing and message theory stresses the importance of source credibility and positive cognitive and affective reactions to health messages. Extant literature points to the importance of nutrition messages that take urban lifestyles, health information sources, and healthy eating barriers into account in developing culturally relevant messages and delivery systems.
- *Culturally relevant health information sources.* Many urban communities rely on health information from trusted local sources rather than physicians or mass media sources. Spanish-speaking communities, in particular, obtain such information from their own media and targeted service providers such as local clinics or faith leaders.
- *Multi-pronged outreach strategies.* Given that many urban audiences use local health information sources with relatively low reach (e.g., community newspapers), many community prevention interventions are using multi-pronged outreach and message delivery strategies. By utilizing multiple sources targeting community members in their homes, through businesses or churches, and at public events, these interventions maximize reach using cost-effective strategies.
- *Community partners and "influentials."* Another critical approach to extending reach is through partners, such as community-based organizations and city or other local government resources. Community-based organizations can work with partners to leverage resources and enhance acceptance of behavior change strategies, such as promoting increased use of parks and other physical activity resources in target communities. Community leaders can also serve to reinforce intervention activities.

CONCLUSIONS

There is a substantial research literature on community obesity risk factors, and a growing literature on intervention strategies as illustrated above. However, more research appears to be needed in several areas. The following areas appear ripe for intervention research and program evaluation:

- Develop, sustain, and support evaluation capacity and implementation among obesity prevention and control community-based organizations.
- Increase utilization of evidence-based practices and evaluation as a programmatic and management tool among community-based organizations.
- Family targeted interventions that expand on the limited evidence base on how parent interventions can form part of a comprehensive intervention strategy across settings, including communities.
- Research on effectiveness of social marketing strategies to increase knowledge, promote more positive attitudes and beliefs, and lower lifestyle barriers to improved nutrition and physical activity, particularly in low-income urban communities.

SEE ALSO: Government Agencies; Governmental Policy and Obesity.

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Compulsive Overeating

COMPULSIVE OVEREATING, ALSO known as binge eating, is a disorder accompanied with marked distress and at least three of the following: (1) eating very rapidly, (2) eating until uncomfortably full, (3) eating when not hungry, (4) eating alone, and (5) having feelings of disgust or guilt following overconsumption. This disorder is usually not accompanied by recurrent purging, excessive exercise, or fasting.

Compulsive overeating can also be secondary diagnosis as part of anorexia nervosa or bulimia nervosa. Compensatory behaviors such as purging and excessive exercise help differentiate between anorexia from bulimia.

Men, in general, make up 40 percent of the population of binge eaters. However, obese males who compulsively overeat attempt fewer diets, medications, and weight-loss supplements before seeking weight-loss treatment. These findings suggest less pronounced body dissatisfaction and fewer help-seeking behaviors by males compared to females.

Compulsive overeating is a serious medical condition with short-term and long-term consequences. In the short term, there are numerous reports of esophageal and gastric rupture due to overconsumption. These ruptures are life threatening. The long-term results can include obesity. The effects of obesity include hypertension, hyperlipidemia, coronary artery disease, diabetes, respiratory disease and congestive heart failure, liver disease, edema, gallstones, osteoarthritis, and an increased risk for certain cancers. These cancers include endometrial, breast, kidney, colorectal, pancreatic, and esophageal.

In addition to medical concerns, compulsive overeating is a nutritional issue. Most binges are high in foods with little or no nutritional value, usually consisting of meal high in fat and sugar with no complex carbohydrates or fiber.

Compulsive overeaters also suffer psychologically. Fifty percent of all compulsive eaters suffer from depression. The lower levels of serotonin that are found in binge eaters could mediate this. Drugs that maintain serotonin levels such as sertraline and prozac have been effective in treating compulsive overeating.

Behaviors associated with this disorder include a change in dietary habits, difficulty with social eating, and social and repetitive body checking. The majority of the participants would routinely pinch areas of their body to check for fatness and avoid wearing clothing that increases body awareness. Behaviors such as these point to the social implications of compulsive overeating and may be included with other symptoms of psychiatric disorders.

These behaviors plays a large role in compulsive overeating, allowing healthcare practitioners to use behavioral therapy, such as cognitive behavior therapy (CBT) and group therapy in treating this disorder. Seamore evaluated the effectiveness of group therapy for women with compulsive overeating. He found that with 6 months of group therapy, all participants demonstrated changes in dichotomous thinking, eating behavior awareness, and detachment from food. These dietary changes all led to a reduction in binge eating.

CBT acts to diffuse the fears and anxiety associated with compulsive overeating. By changing thought patterns and creating tools to address self-deprecating thoughts and fears, CBT has been shown to be an effective therapy. Recently, CBT has been combined with selective serotonin reuptake inhibitor (SSRI) treatment and has shown increased effective when compared to either treatment alone.

SEE ALSO: Anorexia Nervosa; Binge Eating; Bulimia Nervosa; Childhood Onset Eating Disorders; Cognitive Behavior Therapy; Eating Disorders and Obesity.

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Computerized Tomography

COMPUTERIZED TOMOGRAPHY (CT) is an imaging technique that utilizes X-rays. This technology is used for both medical and research purposes. During a test that utilizes a CT scanner, a patient lies on his or her back on a bench-like structure and a scanner rotates around the patient taking several pictures using X-rays. These X-rays are then sent to a computer, which converts them into a picture, known as cross-sectional images. From the cross-sectional images, a physician or researcher can determine the amount of fat, muscle, and bone a patient has as well as if certain diseases are present.

CT scans can be used as a diagnostic tool for many medical conditions. It is often used along with other imaging techniques such as traditional X-rays. In the medical field, CT scans have been employed to diagnose problems in the brain, lungs, heart, and gastrointestinal tract (digestive system). Some diseases it may be used to diagnosis include cancers of any of the abovementioned organs, internal bleeding in the brain, emphysema or pulmonary embolism in the lungs, heart disease, pancreatitis, or any bowel obstruction.

CT scans are useful in body composition research, an important component in the study of obesity. CT scans are able to determine how much fat, muscle, and bone a patient has. CT scans can distinguish the types of tissue because each of these tissues has a different density, and subsequently, the X-rays will travel through each of these body tissues at different speeds. CT scans can also provide measurements about organ size such as the liver, kidney, and spleen. Organ sizes are often used as references for body composition measurements. Data from CT scans are presented in Hounsfield units; an image with low Hounsfield values will signify a greater amount of fat present, while an image with high Hounsfield values will signify a greater amount of muscle present.

CT scans can also provide data about how fat is distributed in the body. It is useful for determining the regional distribution of fat (i.e., subcutaneous fat—below the skin; visceral fat—around the organs of the abdomen; intermuscular fat—fat between the muscles). CT scans along with magnetic resonance imaging (MRI) are considered good research tools for the measurement of human body composition. Learning about body composition has been critical in understanding how fat and muscle tissue relate to the development of many diseases such as heart disease, diabetes, and hypertension (high blood pressure).

A major disadvantage of this test is its utilization of X-rays, which exposes a patient to radiation. Therefore, this test is limited in the populations in which it can be used. Children and pregnant women should be limited to the amount of radiation to which they are exposed. MRIs are an alternative option for measuring body composition in these populations because no radiation is used during this test. Additionally, this test is expensive and laborious which limits where it is utilized. Therefore, CT scans tend to be found in most major medical and research centers, but not commonly found in physicians' offices.

SEE ALSO: Magnetic Resonance Imaging Scans.

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Computers and the Media

BOTH MEDIA USE and obesity have increased dramatically during the past decades, causing concern that increased time spent using media products such as computers may be contributing to the growing obesity epidemic in the United States. Although only 8 percent of American households owned a personal computer in 1984, 62 percent owned at least one by 2003, and nearly 60 percent of adults and children aged 3–17 years used the internet, compared to 19 percent of children and 37 percent of adults in 1998. These trends have caused concern that media use may be diverting attention from more active pursuits and increasing exposure to advertisements for nutritionally inferior foods that are high in calories. However, widespread media and computer use may also represent a new channel for communicating health information to reduce obesity through the development of media products that encourage physical activity and healthful eating behaviors. Computer technology can be used to deliver health education, and to provide follow-up, feedback, and reinforcement to participants making lifestyle changes.

Much of the focus on computer use and obesity has been among children and adolescents. About two-thirds of children aged 5–17 years use the computer for schoolwork, and about 90 percent use it to play games. In this population, increasing weight status has been associated with elevated use of computers and computer/video games. Increased time spent in sedentary activities, such as computer and video games, may decrease time spent in more active pursuits. Further, compared to adolescents of normal weight, overweight adolescents are more likely to become isolated from their peers and social networks, and they may consequently spend more time using electronic media. The National Longitudinal Study

Included in the factors causing the epidemic of obesity in America are the roles of computers and the media. As people spend more time in sedentary activities and see advertising for high-fat, unhealthy foods, obesity rates increase.

of Adolescent Health shows that greater participation in sports and school activities and less television viewing are associated with decreased isolation. This might lead one to assume that decreased use of computers, in favor of school activities and sports, might also show the same outcome, a decrease in isolation. However, other factors such as the accuracy of self-reported media use, differentiation between the types of media used (e.g., television vs. computers or sedentary vs. active video games), and the type of activities that media use actually displaces (sedentary vs. active) will be important considerations for future studies to better understand the associations between obesity and overall media use.

Increased media use also elevates exposure to food advertising. Although television is commonly associated with advertisements for foods that are high in saturated fat and/or sugar, such as ice cream, cookies, and soft drinks, the internet is quickly becoming another venue for advertising these items. An estimated 98 percent of children's websites permit advertising, and more than two-thirds of these sites rely on advertising as their primary source of revenue. Food manufacturers have turned to "advergaming," which uses product websites to integrate specific food products and brands with promotions, sweepstakes, clubs, and online clips of television commercials for that brand. Thus, children may view commercials for a food without turning on their television, and the internet may also reinforce the messages seen in television advertisements by repeating these advertisements or linking a specific food with a television personality or cartoon character.

These sites may also include games with brand-related characters and adventure or sports themes or educational activities that integrate lessons on topics such as spelling, history, math and health with brand-related trivia and characters. Games and activities have the additional advantage of providing longer exposure to the product (relative to a 30-second television commercial), and when the specific food item, product package, or logo is incorporated, they can help young consumers remember the brand on subsequent occasions. Many sites also encourage users, particularly children, to inform their friends about the product. In addition, some sites provide nutrition claims and information, particularly for products marketed to teenagers and adults. However, these claims vary by site; although many highlight their product's vitamin and mineral content or make general nutrient claims, few sites give information about cholesterol and sodium, nutrients that are important in preventing obesity-related complications, such as cardiovascular disease. Thus, in addition to causing an increase in sedentary activities, media use can affect eating habits by providing exposure to advertisements for unhealthy foods.

Although computers and media technology are often portrayed as villains in the obesity epidemic, their widespread use gives them tremendous potential as tools for obesity prevention. For example, activity-promoting video games that require players to move their entire body to participate in the game have been reported to more than double energy output compared to traditional video games, in which players remain seated. Active media-related products may therefore represent an alternative method for increasing energy expenditure using an activity that adolescents already value and would want to continue to perform.

Moreover, computers can be used to tailor dietary and other lifestyle interventions to meet the individual needs of participants. For example, a classroom-based nutrition intervention among adolescents showed that a computer-based component was effective in increasing the knowledge about dietary fats and personal confidence to eat more fruits and vegetables and less fat. In addition, the computer-based component was rated as more relevant and personal compared to the classroom-based component. The interaction that computer-based tools provide can engage students, stimulate curiosity and keep them motivated to continue learning. Further, these types of programs are short enough to be effectively completed in a few class periods and can be administered by instructors even if they have not had extensive formal training in nutrition and physical activity.

Computer-based interventions can also help adults adopt dietary and lifestyle behaviors to decrease obesity risk by providing participants with individualized feedback, encouragement about their progress, and tips for sustaining change.

Further, these types of interventions can provide a cost-effective method of reaching many people at once; they are not limited by geographic barriers and can be easily accessed by participants at their own convenience and pace so that they can assimilate

the information. More recently, interest has turned to using personal digital assistants (PDAs) to help patients in self-monitoring their dietary intake and physical activity and to enable those conducting the intervention to provide feedback and reinforcement to the participants. Computer-based nutrition and lifestyle interventions may thus represent a new way to effectively reach a computer-savvy public with health information.

Although computer use is widespread, the frequency with which computers are used and when and how varies widely by demographic characteristics such as race/ethnicity and socioeconomic status, creating what has been termed a *digital divide* in use. According to the United States Census Bureau's 2001 Population Survey, 65 percent of white households and 67 percent of Asian households owned a computer compared to 46 percent of black and 38 percent of Hispanic-American households. Further, among 8- to 18-year-olds, computer use varied with socioeconomic status, as measured by parental education and income.

Young people whose parents had completed college spent more time using a computer than those whose parents had completed only high school (1:12 vs. 0:55 minutes per day, respectively). Youth, whose parents earned more than $50,000 per year spent slightly more time with internet-related activities such as visiting websites and using e-mail and instant messaging. Hence, first determining the computer access and use within the target audience is important for maximizing computer-based programs' potential success.

Throughout history, the introduction of new technology has often been accompanied by the challenge of preventing or reversing the negative health consequences that misuse or overuse brings. Yet, wise and creative use of technological advances can provide a wealth of resources for helping the public develop habits that promote good health across the lifespan.

SEE ALSO: Children's Television Programming; Food Marketing to Children; Television.

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Conditioned Food Preferences

PEOPLE EAT AN enormous variety of plants and animals. Historically, what people have eaten is usually a matter of what is available and even if it is not very acceptable to the palate. A lifetime of eating certain foods conditions people to desire some foods over others. The choices that people make of foods to eat are largely due to patterns of conditioned behavior learned from parents and foods served at schools or in other places.

The availability of food among a culture may historically have been very limited. For example, if the desert-dwelling Bedouin drink camel or goat milk and eat dates, it is very much a matter of availability of a few foods. However, if a person who previously had to live off a poor subsistence diet were to suddenly be presented with the opportunity to eat from the enormous variety of food for sale in contemporary food stores, then it may take some time but the individual could expand his or her diet to include many foods. When people from poor rural areas of the world who have been lean because they were almost malnourished move to modern urban areas, they are confronted with enormous food choices. They may, because of prior conditioning, stay with a simple diet.

The foods that people eat are in large part due to their food consumption training. For example, eating savory foods versus bland food may be due more to eating habits that have conditioned the individual to prefer spicy foods to bland foods (or vice-versa).

Recent physiological studies have begun to explore the reasons why people prefer certain foods. Some foods are chosen because they are "comfort" foods. Comfort foods are those that produce pleasant feeling of comfort. For example, research shows that males

prefer as comfort foods hearty dishes that are warm—dishes such as steaks, casseroles, and soups. Females, in contrast, prefer comfort foods that are more snacklike in quality. These include items such as chocolate or ice cream. Young people prefer snacks.

COMFORT FOODS

Childhood experiences of comfort foods can become set for a lifetime. For example, a family in the 1950s that went to drive-in movies with a thermos of ice water and a paper grocery bag filled with home-cooked popcorn on a regular basis could easily have late-middle-aged adults today who microwave popcorn before watching a movie at home.

Psychological influences that become deep-seated consumption patterns of behavior can affect the dietary habits of children for a lifetime. If they grew up in a family in which almost the only time food was consumed was at the family dinner table where everyone ate the same things, they were conditioned to eat what was served.

However, if children are given a "children's diet" that is different from adults, they will be conditioned to a different dietary habit. Many children become conditioned to eating fattening foods when there are given items such as processed deep-fried chicken or pizza. The item may taste good to the child, who then will demand that this type of diet be continued indefinitely despite its negative health effects.

The profound fact is that parental feeding habits can create conditioned food preferences. If the parental feeding is with healthy foods, then the patterns of healthy consumption will be established. However, if parental behavior allows the consumption of large amounts of sweets, fats, fast food, or junk food, the risk for obesity is greatly increased.

Studies show that children can be neophobic toward trying new foods. It usually takes repeated exposures to new foods to condition children to eat them. This is especially true of foods that may be naturally bitter, such a broccoli. Food acceptance is a learned behavior in children that is a product of parental nurturing.

SEE ALSO: Access to Nutritious Foods; Accessibility of Foods; Flavor Programming and Childhood Food Preferences; Food Preferences; Taste Aversion Learning.

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Congestive Heart Failure

CONGESTIVE HEART FAILURE (CHF) occurs when the heart does not effectively pump blood, and congestion of the pulmonary and venous system results. Obesity is related to most of the risk factors for developing CHF. Additionally, obesity itself can increase the risk of heart failure. However, it is unclear whether weight loss improves survival after the onset of CHF.

Heart failure involves problems with filling chambers with blood or ejecting blood from chambers. Causes of heart failure include normal aging or underlying cardiovascular disease. The ventricle compensates for dysfunction by hypertrophying or dilating. Compensation negatively affects distensibility and contractility, resulting in a backing up of blood within the circulatory system.

Major risk factors for developing CHF include coronary artery disease, high blood pressure, diabetes, and left ventricular hypertrophy (LVH). Risk factors for prematurely developing CHF include high cholesterol, chronic inflammation, smoking, and genetic factors. Obesity additively clusters risk factors and can result in premature manifestation of CHF. Additionally, obesity causes LVH by increasing blood pressure and viscosity. Overall, obesity is associated with an 8 percent population-attributable risk of developing CHF. Prevention of CHF involves prevention of major risk factors.

The incidence of CHF approximately doubles over each successive decade of life, although the incidence rises more steeply in women. After the age of 40, the lifetime risk of developing CHF is 20 percent.

Several factors, including obesity and obesity-related illnesses, increase the chance for congestive heart failure.

Mortality due to CHF is increasing, partly reflecting the avoidance of death due to predisposing conditions. While obesity increases the risk of developing heart failure, being overweight or mildly obese does not worsen mortality due to CHF while involuntary weight loss increases the mortality due to CHF. Cause of death due to CHF is usually sudden arrhythmic death or progressive pump failure.

Major criteria for diagnosing CHF include waking up short of breath, shortness of breath while lying down, increased jugular venous pressure, crackles during lung sounds, a third heart sound, an enlarged heart, and weight loss after treatment with a diuretic. Minor criteria for diagnosing CHF include swelling of the legs, nocturnal cough, shortness of breath on exertion, enlarged liver, and increased heart rate. Patients meeting two major criteria or one major and two minor criteria are considered to have CHF. Imaging measuring heart function confirms CHF diagnosis.

Staging of CHF determines treatment plan. Treatment in all stages involves diet and lifestyle modification, while symptomatic heart failure is treated with medication. Management of CHF involves several classes of heart and vascular drugs as well as drugs for underlying diseases. While weight loss can significantly alleviate underlying risk factors, it is currently unclear if weight loss increases survival rate after onset of CHF. Classification of heart failure is based on function and predicts survival rate.

SEE ALSO: Atherosclerosis; Blood Lipids; Exercise; Hypertension; Mortality and Obesity; Type 2 Diabetes.

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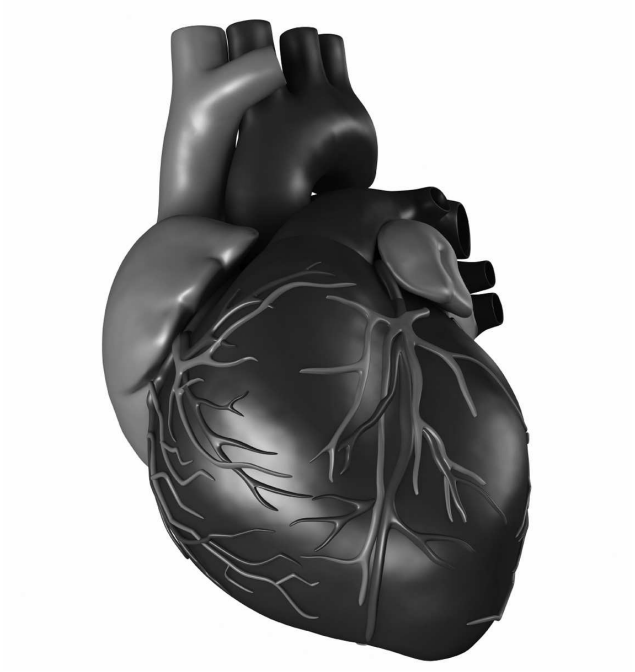
Coronary Heart Disease in Women

CORONARY HEART DISEASE (CHD), also referred to as coronary artery disease (CAD), is the most common type of heart disease. In CHD, small blood vessels that supply blood and oxygen to the heart narrow due to atherosclerotic plaque buildup. Eventually these blood vessels may become blocked and lead to a coronary event (i.e.-heart attack). This condition is the leading cause of death in both men and women in the US, however, age-matched men and post-menopausal women have a significantly higher incidence of CHD than pre-menopausal women. This higher incidence of CHD in post-menopausal women is attributed to the dramatic decline in estrogen after menopause.

Estrogen serves a protective role in pre-menopausal women against risk factors for CHD. These risk factors include other chronic diseases such as obesity and diabetes, as well as physiological factors such as high blood levels of low density lipoprotein (LDL) cholesterol, low levels of high density lipoprotein (HDL) cholesterol, hypertension and central obesity. Additional risk factors include modifiable risk factors such as smoking, physical activity, and diet, as well as unmodifiable risk factors such as age (risk increases with age) and family history/genetics. Menopause leads to a number of metabolic changes which contribute to CHD risk factors. These include perturbations in body fat distribution, reduced glucose tolerance, a worsening atherogenic lipid profile, and a tendency toward hypertension. Body fat distribution changes from a gynoid to an android pattern



Risk factors for coronary heart disease include modifiable activities such as smoking, physical activity, and diet,

during menopause—in essence from a "pear shape," in which most fat is stored in the hips/buttocks/thigh area, to an "apple shape," in which most fat is stored in the abdomen. Research suggests that this abdominal fat may be more atherogenic than fat stored in other areas, contributing to the increased risk of CHD after menopause. This change in body fat distribution can contribute to other risk factors such as reduced glucose tolerance (high blood sugar), hypertension (high blood pressure), and an atherogenic lipid profile (high levels of triglycerides, LDL cholesterol, and total cholesterol, and low levels of HDL cholesterol).

Estrogen therapy is used to help mitigate the effects of estrogen loss in post-menopausal women. However, its use is controversial. The results of recent large clinical trials such as the Women's Health Initiative (WHI) were not supportive of the use of hormone therapy such as estrogens for cardiovascular endpoints. However, there are a number of limitations to that study which render the results and interpretation debatable. For example, the subjects that received hormone therapy were generally an older postmenopausal population, whereas benefits had been previously demonstrated in younger perimenopausal (ie-during menopause) women. Similarly, research demonstrates that hormone therapy may be beneficial if initiated during the early stages of atherogenesis, rather than in individuals with more advanced atherosclerosis.

CHD is a major health concern for older postmenopausal women. As estrogen is purported to be the protective factor in pre-menopausal women, hormone therapy has been utilized to help mitigate the untoward effects of menopause. Further research will determine if estrogen therapy is a reasonable means to reduce the risk of CHD.

SEE ALSO: Atherosclerosis; American Heart Association; Blood Lipids; Central Obesity; Estrogen Levels; Estrogen-Related Receptor; Menopause.

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Corticotropin-Releasing Hormone

HORMONES ARE THE body's chemical messengers that travel through the blood stream to various tissues, working slowly over time to affect a variety of processes. Corticotropin releasing hormone (CRH) is

a 41-amino acid peptide hormone that is made and released by the hypothalamus gland. After its release, CRH travels to the anterior lobe of the pituitary gland where it stimulates the release of corticotropin. Corticotropin increases the production of CRH-related peptides from the adrenal cortex, which interact with CRH1 and CRH2 G-protein coupled receptors throughout the body during the stress response. It is believed that this hormone system may play an important role in energy balance.

Studies have shown that CRH administration prevents weight gain in obese (but not lean) rats. This observation suggests that CRH may be reduced in obesity. CRH also lowers the body weight at which food-deprived animals begin to store food. CRH-related peptides are anorectic agents that decrease food intake. Some of these peptides are also thermogenic, increasing energy expenditure through activity of the sympathetic nervous system. The mechanisms of these anorectic and thermogenic effects are the focus of many studies.

Selectively inhibiting the CRH2 receptor has been shown to block the anorectic effect of CRH, demonstrating that it plays a part in anorexia. The role of the CRH1 receptor is unclear; however, studies suggest that it may also contribute to the anorectic effect of the CRH system. Additionally, the CRH1 receptor may play a role in CRH-related thermogenesis. Other studies have shown that CRH antagonists can block the anorectic effect of leptin, a hormone that promotes the feeling of fullness after a meal. This observation suggests that there may be a relationship between the CRH system and leptin.

Since these hormones may be involved in energy balance, the CRH system is being viewed as a potential target for antiobesity drugs. Nonspecific activation of this system may lead to other undesirable effects; however, recent studies on the CRH1 and CRH2 ligands may allow for the development of drugs that are specific to energy balance in the future.

SEE ALSO: Appetite Control; Cortisol; G-Protein Coupled Receptors; Glucocorticoids; Hormones; Hypothalamus; Pituitary Gland.

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Cortisol

CORTISOL IS A corticosteroid hormone that is produced and secreted by the human adrenal cortex. This hormone is generally responsible for restoring homeostasis in response to stress. General effects of normal levels of cortisol include elevating blood pressure, elevating blood sugar level, suppressing the immune system with a specific effect on T-cell lymphocytes, enhancing catecholamine activity, enhancing short-term memory, and enhancing bone metabolism. However, irregular levels of this hormone may have detrimental effects.

Cortisol in the serum is mostly found bound to proteins. It is a cholesterol-based hormone and its production depends on the levels of pituitary adrenocorticotropic hormone (ACTH). High levels of cortisol, however, inhibit the production of ACTH by inhibiting corticotropin-releasing hormone (CRH), which forms a negative feedback loop that functions to regulate the serum level of cortisol. Acute levels of cortisol undergo diurnal variations, meaning they change throughout the course of the day, with highest levels in the morning and the lowest at night. Disruption of this cycle can be caused by physical factors or psychological factors.

Clinically used as hydrocortisone, also known as compound F, it is injected into arthritic joints to help treat rheumatoid arthritis. Another common pharmacological use is as a topical cream that reduces skin inflammation or itchiness such as Cortisone-10. It is also used to suppress allergic reactions and is orally implemented.

While these treatments using cortisol derivatives are highly useful, improper levels of cortisol in the body may cause serious conditions. Usually, these syndromes are caused by tumors of the adrenal

cortex or the pituitary gland. A highly elevated level of cortisol, hyperadrenocortical syndrome or Cushing syndrome, is usually caused by an adrenal cortex tumor(s) and causes extreme weight gain in the face ("moon face") and in the trunk, hypertension, susceptibility to pulmonary infections, as well as other effects. Another form of hyperadrenocortical syndrome, Cushing disease, also causes increase levels of cortisol; however, it is caused by tumor(s) in pituitary gland, which in turn causes increased levels of ACTH release. On the contrary, hypocortisolism or Addison's disease is characterized by decreased levels of cortisol by either diseased or damaged adrenal glands or pituitary gland. Results of this imbalance may cause excessive weight loss, muscle weakness, and increased blood acidity.

Cortisol levels are typically elevated in obesity, although circulating concentrations usually appear to be normal. Increased cortisol levels in obesity, particularly in abdominal obesity, may be one source of difference in the metabolic efficiency of lean vs. obese individuals, and high cortisol levels may exacerbate certain disease conditions (e.g., Type 2 diabetes). For this reason, several companies have formulated medications such as CortiSlim to lower cortisol levels in obese individuals. These medications have not proven to be effective."

SEE ALSO: Cushing Syndrome; Stress.

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Cost of Medical Obesity Treatments

OBESITY IS FAST becoming a worldwide epidemic and proper actions need to be taken to ensure a future where obesity-related deaths will not be at the top of the list. Obese adults are at an increased risk of type 2 diabetes, hypertension, hypercholesteremia, asthma, arthritis, and overall poor health leading to a poor quality of life. It is estimated that Caucasian men aged 20 years with a BMI of over 45 lose 13 years of life compared with men of comparable age with BMI of 24, making it painfully clear just how hefty of an impact obesity has on the population.

Medical costs associated with overweight and obesity may include both direct and indirect costs. Direct medical costs include preventive, diagnostic, and treatment services, while indirect costs relate to morbidity and mortality rates. Morbidity cost is defined as the value of income lost from decreased productivity, restricted activity, absenteeism, and bed days, while mortality costs are the value of the future income lost by premature death.

DRUG THERAPY

There are several methods that can be utilized to combat the epidemic of obesity. Drug therapy, instillation of an exercise program, and behavioral therapy can all be utilized. Drug therapy with orlistat provided change in weight at 12 months or longer with 360 mg per day. However, as with all medicines, there were adverse side effects to using orlistat: that all the studies with patients using orlistat had gastrointestinal adverse events such as oily stool and fecal incontinence.

However, the drawback with the use of drugs is that often these are given without incorporation of intensive exercise and behavior therapy programs. Although drug therapy with orlistat or sibutramine leads to a significant beneficial effect on high-density lipoprotein (HDL) and triglycerides (TGs), no other risk factors are affected. Specifically, for sibutramine, there was an increase in diastolic pressure observed, so the beneficial effects of sibutramine on weight and risk factors needs to be carefully balanced against the potential increase in blood pressure.

BEHAVIORAL MODIFICATION THERAPY

The inclusion of exercise with diet yielded beneficial results as well and exercise seems to be an important factor in weight reduction when it is used in combination with diet. This combination yields beneficial effects for HDL cholesterol, TGs, and significantly decreasing both systolic and diastolic blood pressure. Adding

exercise to diet and behavior therapy yielded greater results in weight reduction than did just the combination of diet and behavioral therapy. The combination of behavior therapy with diet does not seem to change cardiovascular risk factors.

The exercise regiment in these studies were not necessarily exhausting; the regiment included walking, jogging, and cycling tailored to produce 60 to 80 percent of the maximum heart rate with the minimum of 20 minutes three times a week as the most placid to 90 minutes three times a week as the most intensive.

Behavioral therapy varied among the studies, but usually included self-monitoring, slowing the rate of eating, reducing eating cues, responding to social pressures, preplanning, and relapse prevention techniques. Through all of these studies, the greatest weight loss was associated with the addition of behavior therapy to diet or drug therapy with diet.

SURGICAL THERAPY

With respect to surgical interventions, in 1991, the National Institutes of Health (NIH) published a Consensus Statement citing that most bariatric surgery patients rapidly lost weight and continued to do so for 18–24 months. It seems that bariatric surgery, when patients are carefully selected and have been unable to gain results through less invasive methods, produces beneficial results.

Gastric bypass surgery has demonstrated very impressive results with lower costs and greater long-term weight-loss maintenance when compared to low-calorie diets and behavior modifications. Two types of surgery have been recommended by government consensus panels, both for people with severe cases of obesity (defined as above 100 lb of ideal body weight) who have had ineffective weight loss through other methods.

The first method is gastroplasty, which involves surgically reducing the size of the stomach. The second method is the gastric bypass surgery, which creates a small pouch and connects this pouch to the second portion of the intestine. Although more extensive and difficult than the simpler gastroplasty, it yields more effective weight-loss results.

There are three types of bariatric surgeries: Roux-en-Y gastric bypass, laparoscopic adjustable gastric banding, and biliopancreatic diversion with duodenal switch. Roux-en-Y is the most frequently performed obesity operation, accounting for 85 percent of all bariatric surgery in the United States. It calls for the creation of a pouch which functions as the stomach with the patient's original stomach still functioning to deliver important enzymes for digestion.

The laproscopic adjustable gastric banding is a restrictive weight-loss operation because it works by limiting the actual amount of calories that can be consumed at the same time as making an individual feel "full" despite this reduced intake. Simply, a belt is placed around the upper part of the stomach, separating the stomach into two parts with a port that fills fluid into the stomach, thereby slowing the passage of food from the upper pouch into the lower pouch. Weight loss with an adjustable gastric band is slow and steady and shows great improvements in weight-related medical problems. There is also minimal stress on the body during surgery with a fast recovery time. The uniqueness of the band lies in its adjustability, which makes it possible to adjust the band according to an individual's weight loss goals and needs.

Biliopancreatic diversion with duodenal switch removes the outer margin of the stomach, meaning approximately two-thirds is removed and then the intestines are rearranged so that the area where the food mixes with the digestive juices is short. This procedure requires a much longer recovery period and causes a greater risk for infection.

INSURANCE ISSUES

Despite the publicity of obesity as an epidemic and the many health risks associated with obesity, there is relatively very little support provided by health insurers. Low reimbursement rates preclude the long-term financial feasibility of weight-management programs without other support or a significant proportion of patients who can pay for care out of pocket. It is an increased issue because many insurers will not cover weight-loss treatments unless the patient has an obesity-related condition such as diabetes or hyperlipidemia. According to a study of national costs attributed to overweight individuals (BMI 25–29.9) and obese individuals (BMI >30), medical expenses for this population accounted for 9.1 percent of total U.S. medical expenditures in 1998. Medicaid and Medicare paid approximately half these costs. The difficulty with assessing the cost of obesity is that not only must the cost of obesity itself be accounted for, but also the

added cost of all the various conditions causally related to obesity including cancer, gallbladder disease, type 2 diabetes, and heart disease, among other things. The direct healthcare costs of obesity are estimated to be at $102.2 billion in 1999. According to the American Obesity Association, health insurance expenditures constitute $7.7 billion of the total with 43 percent of all spending by U.S. business on coronary heart disease, hypertension, type 2 diabetes, and so forth. Despite the steep estimation of the impact medical treatment for obesity is causing the United States, it may still be very likely that it is an underestimate as the true burden because it omits intangibles such as pain, suffering, and care provided by nonpaid caregivers.

OBESITY COVERAGE

Still, signs of change are beginning to show as the American Obesity Association is working toward expanding insurance coverage for obesity treatment. State policy makers are slowly beginning to allow Medicaid treatment options for the low-income citizens.

In Iowa, Governor Tom Vilsak signed into law a bill that requires the state's Medicaid program to develop a strategy to provide dietary counseling to child and adult Medicaid enrollees. In Colorado, Governor Bill Owes signed a measure establishing an obesity treatment pilot program for Medicaid patients over 15 years old having a BMI ≥30.

It may behoove each respective state to acknowledge the different prevalence rates of obesity which can then assist state policy makers to determine how best to allocated public health resources and provide information concerning the economic impact of obesity per individual state.

Although people may be given treatment for obesity, their health insurance company may neither cover nor reimburse these services. This is somewhat of a conundrum as they are willing to pay for treatment of conditions that are associated with obesity and cover other conditions which neither have the mortality nor the morbidity of obesity. Furthermore, many insurance companies also charge higher premiums with increasing degrees of overweight. However, in July 2004, Medicare policy changed and now reflects the seriousness of obesity as a medical condition by considering it as a disease and making payments for any services in connection with it.

The difficulty with the lack of health insurance covering obesity is that there is a lack of understanding that obesity should be viewed as a disease, that treatments are very effective, and the heightened role that personal responsibility is given. One of the most significant barriers toward lack of health insurance reimbursements are the beliefs that people have about personal responsibility because many other healthcare conditions involve personal behavior. The NIH and other organizations all agree that obesity should be recognized as a disease and should therefore receive coverage for its treatment. However, this is not the case, and organizations such as the American Obesity Association are taking measures to rectify this misleading belief.

CONCLUSION

The problem with obesity is that it is not a disease which can be treated and cured. It seems to be more of a chronic disease condition; therefore, obesity is very likely to require lifelong treatment. However, the benefits of such lifelong treatment yield a significant difference in both health and economic benefits. Changes need to be made with respect to how we view obesity and the blame we place on those afflicted. Therapeutic intervention is essential for obesity as it will be beneficial for the future health of all individuals and many diseases resulting from obesity can be stemmed. A better quality of life will be assured to all.

SEE ALSO: Exercise; Governmental Policy and Obesity; Health Coverage of Gastric Surgeries; Orlistat (Xenical); Psychiatric Medicine and Obesity; Sibutramine (Meridia).

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Council on Size and Weight Discrimination

FOUNDED IN 1990, the Council on Size and Weight Discrimination is a not-for-profit advocacy group working to end discrimination based on body size, shape, or weight. The Council serves as a consumer advocacy group for large-size people, especially in medical treatment, job discrimination, and media images. It offers information, counseling, and referral for people who have encountered weight discrimination. The Council provides literature and technical assistance to professionals in the fields of eating disorders, body image, and weight issues. It works with regulatory agencies, legislatures, and the media to change prejudicial attitudes and policies regarding weight. The organization provides public education through its website, www.cswd.org.

The Council's Medical Advocacy Project works with the National Institutes of Health (NIH), the Food and Drug Administration (FDA), and the Federal Trade Commission (FTC). In 1995, the Director of the Council's Medical Advocacy Project, Lynn McAfee, testified at an FDA hearing urging the disapproval of Redux (Fen-Phen) on the grounds that the drug had not been thoroughly studied. Two years later, when evidence of serious side effects and deaths had surfaced, the drug was removed from the market. The Council continues to testify for proof of long-term safety and effectiveness of new diet drugs.

The Council advocates evidence-based public health policies, including full disclosure of actual long-term success rates by weight-loss programs. McAfee was appointed a member of the Partnership for Health Weight Management of the FTC. The Partnership put together the first official government publication targeting fraud in the weight-loss industry. This publication asks weight-loss programs to abide by fair consumer practices, warn customers of the risks of diet products, and disclose the fact that most dieters will regain the weight they lose. In 2003, when the FTC announced a crackdown on fraudulent weight-loss programs, McAfee spoke at the press conference.

The Council has had success in convincing regulatory agencies to address medical discrimination and inappropriate care of large patients. In 2003, with the Council's input, the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) published a brochure, aimed at healthcare professionals, promoting respectful treatment for large patients. The Council on Size and Weight Discrimination is listed as a resource.

The Council is part of the Size Acceptance movement as well as of the Health at Every Size community of organizations and professionals. The Council stands for the principles that all people, no matter what their weight, deserve equal treatment in the job market and on the job, and that all people, no matter what their weight, deserve respectful treatment by healthcare professionals.

SEE ALSO: American Obesity Association; National Association to Advance Fat Acceptance; NIDDK.

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C-Reactive Protein

INFLAMMATION IS ASSOCIATED with morphological and clinical progression of atherosclerotic vascular disease. C-reactive protein (CRP) is a plasma protein, an acute phase protein produced by the liver. It is a member of the pentraxin family of proteins. CRP is involved in inflammatory processes occurring in the body. CRP is a predictive marker of inflammation.

The levels of hs-CRP of <1, 1 to <3, and ≥3 mg/L have been suggested to define low-, moderate-, and high-risk groups in cardiovascular disease (CVD). Recent studies strongly suggest that CRP is a better predictive marker of CVD than LDL-C in women. Individuals with CRP greater than 3 mg/L with LDL cholesterol below 130 mg/dL are at a higher risk for

CVD. CRP is associated with the progression of carotid atherosclerosis and enhances inflammatory process of atherosclerosis.

CRP AND CARDIOVASCULAR DISEASE

Most studies support that higher levels of (high sensitivity) hs-CRP are associated with increased risk for cardiovascular disease. High levels of hs-CRP consistently predict new coronary events in patients with unstable angina and acute myocardial infarction (heart attack). CRP binds to a variety of molecules, particularly liposomes and lipoproteins, such as low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL) cholesterol, and is a powerful activator of the classic complement system.

High levels of hs-CRP in the blood seem to predict prognosis and recurrent events in patients with stroke and peripheral arterial disease. An association between sudden cardiac death, peripheral arterial disease, and hs-CRP was observed. A positive significant correlation of CRP with other coronary risk factors including age, number of cigarettes smoked per day, body mass index, systolic and diastolic blood pressure, total cholesterol, triglycerides, homocysteine, fibrinogen, and D-dimers were reported in several studies. An inverse association of CRP and exercise frequency and HDL-C (high density lipoprotein) were reported. CRP was also a risk factor for fatal and nonfatal myocardial infarction, CHD death ischemic stroke and peripheral vascular disease.

CRP predicts risk of asymptomatic cardiovascular events in women. Research is showing promising results for testing hs-CRP (along with other risk factors) to determine heart disease risk in those with undetected heart disease and risk of complications for those who have already had a heart event (such as a heart attack).

CRP AND OBESITY

Elevated CRP (a state of chronic inflammation) has also been found to be positively correlated with adiposity and with leptin levels (a hormonal correlate of higher levels of body fat). CRP concentration was strongly related to surrogate and direct measures of body fat, diastolic blood pressure, and lipid and apolipoprotein levels CRP was strongly related to all anthropometric and direct measures of total and central abdominal obesity. Higher BMI is associated with higher CRP concentrations, even among young adults aged 17–39 years. These findings suggest a state of low-grade systemic inflammation in overweight and obese persons. In a recent review of 33 articles, weight loss was associated with a decline in CRP level. For each 1 kg of weight loss, the mean change in CRP level was −0.13 mg/L. This suggests that weight loss may be an effective therapeutic strategy for lowering CRP level.

CRP AND DIABETES

The cytokine proteins, also associated with states of chronic inflammation, can influence insulin resistance and glucose uptake, and thus can be associated with Type 2 diabetes. Cytokines can influence insulin resistance and glucose uptake promotes hepatic fatty acid synthesis. Upregulation of receptors for advanced glycation end products has been associated with enhanced inflammatory reactions. Increased expression of these receptors has been found to be associated with impaired glycemic control and may be a contributory factor in the complex array of mechanisms that leads to accelerated atherosclerosis in patients with diabetes. In a national survey study, respondents with hemoglobin A1c (A1C) levels ≥9 percent had a significantly higher rate of elevated CRP than those with A1C levels < 7 percent. A 2.7-fold greater risk of development of diabetes was observed in the highest quartile CRP than those in the lowest quartile. In the Cardiovascular Heart Study, the Women's Health Study, the West of Scotland Coronary Prevention Study, and the Nurses' Health Studies, CRP is significantly associated with incident diabetes.

CRP AND METABOLIC SYNDROME (METS)

The Metabolic Syndrome (MetS) is a group of risk factors that often co-occur with obesity, including inflammation, insulin resistance, high blood pressure, dyslipidemia, and abdominal obesity. CRP levels were more strongly related to insulin resistance and other features of the MetS in women in the Mexico City Diabetes Study. Cross-sectional studies have found associations of CRP with metabolic syndrome and its components, including obesity, insulin resistance, dyslipidemia, elevated blood pressure, and endothelial dysfunction. Increased serum CRP levels predict the development of metabolic syndrome. In the 12-year follow-up study, even a slight increment in serum CRP level was associated with an increased risk of developing metabolic syndrome in elderly women.

CRP AND HYPERTENSION

Elevated CRP may increase blood pressure due to endothelial dysfunction which results in vasoconstriction, and ultimately increases blood pressure. Cross-sectional studies demonstrate that CRP is associated with hypertension, even among those with low initial systolic and diastolic BPs

SEE ALSO: Cardiovascular Disease in African Americans; Cardiovascular Disease in Asian Americans.

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Cushing Syndrome

THIS DISEASE, FIRST described by Harvey Cushing in 1932, refers to any clinical entity due to excessive and prolonged secretion and action of glucocorticoids, although the cause of the disease may relate to any one of a number of origins.

Primary hypercortisolism may be due to an adrenal cortical tumor (adenoma or carcinoma). Cushing Syndrome of secondary origin may derive from a pituitary tumor secreting an excess of adrenocorticotropic hormone (ACTH), or to ectopic ACTH production, or to pituitary corticotroph hyperplasia. The simultaneous measurement of plasma ACTH and cortisol is useful in differentiating between adrenal hyperplasia (due to excess pituitary ACTH stimulation) and adrenal tumors as the cause of Cushing Syndrome.

In patients with adrenal tumors, plasma cortisol levels are high but the concentration of ACTH is low or undetectable. In contrast, both cortisol and ACTH plasma levels are elevated in bilateral adrenal hyperplasia, and both can be lowered by exogenous dexamethasone (a synthetic glucocorticoid) administration.

Some symptoms of cortisol excess are central (truncal obesity), hypertension, glucose intolerance, hirsutism, osteoporosis, polyuria, and polydipsia. The hyperglycemia resulting from excess glucocorticoids leads to so-called steroid diabetes, where prolonged elevated levels of glucose may, in time, lead to pancreatic beta-cell exhaustion (diabetes mellitus). The change in fat distribution is due to the lipolytic action of ACTH and glucocorticoids on the normal fat depots.

The redistribution of fat may not be due to the direct actions of glucocorticoids, but rather to the insulin that is secreted in response to increased hepatic glucose formation. Although the actions of insulin on the normal depots are apparently antagonized by cortisol, insulin appears able to exert a lipogenic effect in other areas of the body, such as the face, upper back, and supraclavicular fat pads.

The catabolic actions of glucocorticoids on skeletal muscle cause thinning of the extremities. Loss of protein matrix of bones causes severe osteoporosis, which may severely affect the spinal column. The excess androgens produced are the cause of hirsutism in the female. Again, the hyperpigmentation that may be present in Cushing Syndrome of secondary origin may be due to the presence of increased circulating levels of ACTH. Polyuria and polydipsia are due to the loss of large volumes of water as a result of solvent drag during the process of excessive glucose excretion by the kidneys.

A number of treatments are possible depending mostly on identifying the cause and removing it. If it is being caused by medications, it should be discontinued when feasible. In most cases of Cushing Syndrome, the treatment is to find the direct or indirect cause (e.g., adrenal gland tumor, pituitary tumor, etc.) and treat it surgically, medically, or by radiation. If the cause cannot be found, the adrenal glands can be removed surgically or suppressed with medications that reduce the glands' production of cortisol.

SEE ALSO: Cortisol; Glucocorticoids.

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nosis of Cushing's Syndrome," *Experimental and Clinical Endocrinology and Diabetes* (v.4, 2005).

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Cytokines

CYTOKINES (formerly known as lymphokines, interleukins, and chemokines) are a group of signaling compounds that enable cells to communicate with each other. These compounds are water-soluble and made of proteins whose mass ranges between 8 to 30 kDa.

Cytokines are released by many cells within the body and are not limited to a single organ system. Because so many different types of cells use cytokines to communicate, their functions are as diverse as the cells that use them. They were originally identified within the immune system where they were found to mediate many immunological and inflammatory functions in response to trauma and disease.

All cytokines have receptors on cells. These receptors cause upregulation or downregulation of particular cellular functions (usually gene expression). Most cytokines are also pleiotropic (i.e., they cause different reactions in different cell types).

The endocrine functions of cytokines are particularly relevant in obesity. The excess adipose tissue, characteristic of obesity, releases large amounts of cytokines that affect almost every organ system within the body. Obesity-associated cytokine production has been thought to play a role in diabetes, hypertension, insulin resistance, arteriosclerosis, vascular endothelial dysfunction, triglyceride overproduction, very-low-density lipid overproduction, and a fall in high-density lipoproteins.

Cytokines that affect these diseases are believed to be derived from immune cells, such as macrophages, which accumulate in large numbers within the adipose tissue of obese individuals. These cells release large amounts of cytokines into the circulation and have numerous deleterious endocrine effects as outlined above.

Adipocytes themselves release cytokines such as leptin, which affect appetite, fat storage, insulin resistance, and reproductive functions. The exact interplay between the cytokines secreted by adipocytes, immune cells, and the rest of the body is incompletely understood and is a fertile ground for research as new cytokines with new functions are being discovered and characterized almost daily.

SEE ALSO: Fertility; Interleukins.

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Db/Db Mouse

THE DB/DB MOUSE is a genetically mutated mouse in which leptin receptors do not function properly despite the mouse still being able to produce leptin; This breed of mouse is sometimes referred to as the diabetes mouse. This mouse was first discovered by Jackson Laboratories in 1966 as an obese mouse that ate excessively. There is a single gene that is mutated which is responsible for the defective leptin receptor. This mouse is different from the ob/ob mouse; in these, the leptin receptors function, but the mouse does not produce leptin. Leptin is a hormone produced in fat cells that plays a role in eating and energy utilization. The db/db mouse is extremely obese and has many of the metabolic defects found in the ob/ob where leptin is not present. These include overeating, decreased physical activity, elevated plasma leptin (db/db mouse only), elevated glucose and insulin levels, insulin resistance, Type 2 diabetes mellitus, fatty liver and liver disease, low body temperature, and kidney disease. The defective db/db gene can appear on several different strains (types) of mice and the deleterious effects from the lack of functioning leptin receptors varies from strain to strain. Some strains of mice have moderately elevated glucose, while other strains may have severely elevated glucose.

The db/db mouse has been utilized in numerous scientific studies. It is very useful in elucidating the function of leptin and understanding the leptin signaling pathway. It is also useful to study many different diseases from which the db/db suffers. For example, some researchers view the db/db mouse as a better model in which to study kidney disease. The symptoms observed in the kidney disease of a db/db mouse are very similar to the symptoms observed in humans. The studies in the db/db mouse have led to change in the clinical treatment of humans who suffer with kidney disease. The db/db mouse has also been useful in studies of insulin resistance and diabetes mellitus. Brain-derived neurotrophic factor (BDNF) is a compound that plays an important role in the synthesis of neurons from both the central and peripheral nervous system. Young db/db mice that were treated with BDNF had improvements in their blood sugar and insulin levels (both decreased) and increased their physical activity and body temperature; this was not observed in older db/db mice.

Unlike ob/ob mice, giving db/db mice leptin will not improve their condition. Db/db mice will remain obese for their entire life; however, they will begin to rapidly lose weight before they die as their pancreas begins to fail. Thiazolidinediones (TZD) are a class of compounds that are used as diabetic medication that functions by activating peroxisome proliferators-activated receptors (PPAR). Db/db mice that are treated with TZDs have had improvement in some of their diabetes complications. Muraglitazar is a new drug

that activates two different types of PPARs and preliminary studies have shown that it has been highly effectively in ameliorating the development of diabetes in young db/db mice and the worsening of diabetes in older db/db mice. The db/db mouse is a useful animal model for studying many metabolic disorders.

SEE ALSO: Leptin; Ob/Ob Mouse.

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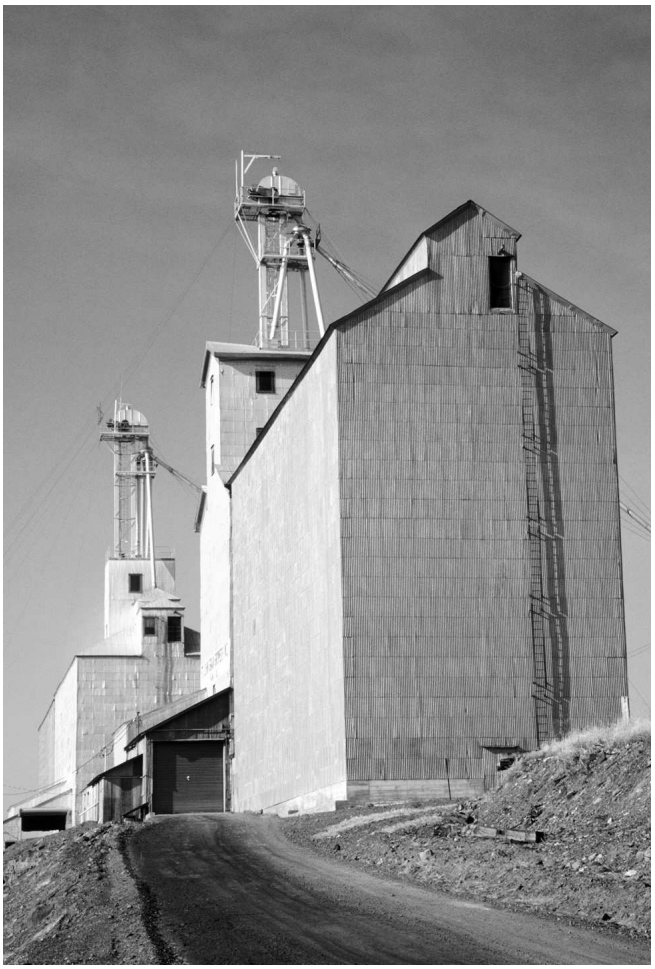

Working with American farms, the Department of Agriculture seeks to improve the health of citizens through assistance and education.

Department of Agriculture

THE UNITED STATES Department of Agriculture (USDA) was created in 1889 as the 6th Executive Department (Cabinet Department) within the U.S. federal government, alongside other Executive Departments such as the Departments of State, Treasury, and Defense.

The USDA provides leadership on food, agriculture, and natural resources issues, including the following key activities as specified in the USDA Strategic Plan for 2002–07: expanding markets for agricultural products, developing alternative markets for agricultural products, expanding job opportunities in rural America, reducing the prevalence of foodborne hazards, improving the nutrition and health of Americans through food assistance and education, and managing and protecting public and private lands.

The USDA budget for 2008 is estimated at approximately $89 billion, of which about 75 percent goes to provide services as required by law, including programs focused on nutrition assistance, farm commodities, export promotion, and conservation. The remaining 25 percent is allocated by discretionary programs, including those to address pest and disease threats, the WIC program (Special Supplemental Nutrition Program for Women, Infants, and Children), rural development loans and grants, research, education, technical assistance for soil and water conservation, marketing assistance, and Forest Service activities including management of National Forests.

Most of the USDA activities relevant to obesity are concentrated in the area of Food and Nutrition. The food pyramid, currently known as MyPyramid, is both a graphical method of displaying the proportion of different foods and nutrients recommended for a balanced diet, and an online interface which allows anyone to calculate the specific daily num-

ber of servings from different food groups which are recommended for them, based on the individual age, sex, and activity level. The food pyramid classifies foods into six groups: grains, vegetables, fruits, milk, meat and beans, and oils. For a 50-year-old female, the food pyramid recommends 2,000 calories per day including 6 ounces of grains, 2.5 cups of vegetables, 2 cups of fruits, 3 cups of milk, and 5.5 ounces of meat and beans. For a 25-year-old male with the same activity level, 2,800 calories are recommended including 10 ounces of grains, 3.5 cups of vegetables, 2.5 cups of fruit, 3 cups of milk, and 7 ounces of meat and beans.

The Food Stamp program provides low-income families in the 48 contiguous states and the District of Columbia with electronic benefits transfer (EBT) cards which they may use to buy eligible food in authorized food retail stores. Food stamp eligibility is contingent on the individual or family meeting certain income and resource tests, which are summarized at the USDA website: they include having $2,000 or less in countable resources such as a bank account, and gross monthly income of $2,167 or less for a family of four; less stringent restrictions apply in certain circumstances, including households with a disabled or elderly member.

Food stamps may be used to buy foods for the household and food-producing seeds and plants; they may not be used for alcoholic beverages, tobacco, or nonfood items such as household supplies or pet food. In some circumstances (for instance, for homeless, elderly, or disabled persons), food stamps can be exchanged for restaurant meals. Fifty-one percent of all food stamp recipients are children age 18 or younger; 9 percent are adults over age 60. Forty-one percent of participants in the food stamp program are Caucasian, 36 percent are African American, 18 percent are Hispanic, and the remainder are Asian, Native American, or of unknown race or ethnicity.

The WIC program provides supplemental foods, nutrition education and counseling, and screening and referrals to other services for pregnant, postpartum, and breastfeeding women, infants and children up to their fifth birthday.

WIC benefits are offered to those who meet income guidelines (in 2006–07, the maximum gross annual income allowed for a family of four was $37,000) and are judged to be at nutritional risk, meaning that a health professional such as a physician or nutritionist has determined that the individual has an inadequate diet or a medical condition such as anemia or a history of poor pregnancy outcomes. Recipients receive WIC foods, either through direct delivery, by access to warehouses, or by receiving a subsidy to purchase WIC foods, which are defined as foods high in protein, calcium, iron, or vitamins A or C. Examples of WIC foods include iron-fortified infant formula and cereal, eggs, milk, cheese, peanut butter, and dried beans or peas.

The National School Lunch Program provides free or low-cost lunches to children attending school; participating schools receive cash subsidies and donated commodities from the USDA in return for offering free or reduced-price lunches to children from low-income families.

The School Breakfast Program was established in 1966 to serve breakfast to "nutritionally needy" children, which was operationalized as children in low-income areas or who had to travel a long distance to school, and in 1975 received permanent authorization. The Special Milk Program provides milk to children in schools, camps, and childcare institutions that do not participate in the School Lunch or School Breakfast programs.

SEE ALSO: Food Guide Pyramid; Food Stamp Nutrition Education Program; Government Agencies; Pregnancy; School Lunch Programs.

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Department of Health and Human Services

THE DEPARTMENT OF Health and Human Services (DHHS) is an Executive Department within the United States federal government. The DHHS was created in 1979 when the Department of Health, Education,

and Welfare was split into the Department of Education and the DHHS. Agencies included in DHHS include the National Institutes of Health, the Food and Drug Administration, Centers for Disease Control and Prevention, the Indian Health Service, the Health Resources and Services Administration, the Substance Abuse and Mental Health Services Administration, the Agency for Healthcare Research and Quality, the U.S. Public Health Service Commissioned Corps, and the Centers for Medicare and Medicaid Services.

The purpose of DHHS is to protect the health of Americans and provide essential human services. DHHS includes more than 300 programs and represents almost a quarter of all federal outlays. It also works closely with state and local governments, and provides funding for many programs provided by state or county agencies or through private sector grantees. The DHHS in 2007 had over 66,000 employees and a budget of almost $700 billion. Priorities of the DHHS, as outlined by Secretary Mike Leavitt in 2005, include value-driven healthcare, improved health information technology, improved access to prescription drugs for Medicare recipients, increased Medicaid benefits, improvement of healthcare in Louisiana, more personalized healthcare including an emphasis on prevention, influenza pandemic preparedness, and increase in the size and training of the Public Health Service Commissioned Corps.

Many agencies within the DHHS have obesity-related initiatives, including the Centers for Disease Control and Prevention and the National Institutes of Health. In addition, information relevant to the prevention aspect of DHHS goals is collected and presented at the website HealthierUS.gov. This website is part of a national initiative to promote community health and wellness, prevent and reduce the costs of disease, and improve people's lives; the major aspects emphasized are physical activity, nutrition, preventive screenings, and avoidance of injurious behaviors such as tobacco smoking, alcohol and drug abuse, and risk-reduction behaviors such as the use of child car seats. Within each subsection, links to relevant programs and further information, often from other agencies within the federal government, are provided.

HealthierUS.gov refers to obesity as "epidemic" in the United States. In the area of physical activity, HealthierUS.gov recommends 30 to 60 minutes of moderate activity on most days, which it links to benefits including relaxation and stress reduction, greater energy, improved sleep, and reduced risk for heart disease, colon cancer, diabetes, osteoporosis, and high blood pressure. In the area of nutrition, HealtherUS.gov advice is fairly nonspecific, but recommends moderate food consumption which includes a variety of fruits of vegetables, and low consumption of fat, sugar, and salt. Healthier eating is linked to reduced risk for heart disease, diabetes, some cancers, strokes, and osteoporosis. Information relevant to obesity research includes links discussing the body mass index (BMI), food portions, reading food labels, information about what constitutes a portion of food, and healthy recipes. The Health Screenings portion of HealtherUS.gov includes guidance on what is a healthy weight, for children and adults, and includes overweight as a risk factor for many diseases; weight is also included in the Checkups and Tests Record which is available for download and encourages individuals to regularly record heath information including blood pressure, blood sugar, dental visits, and hearing tests.

SEE ALSO: Fitness; Government Agencies; Nutrition Education; Physical Activity and Obesity.

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Depression

A DEPRESSIVE DISORDER is an illness that involves the body, mood, and thoughts. Some 20.9 million adults, suffer from a depressive illness in United States. Depression interferes with the ability to work, study, sleep, eat, and enjoy pleasurable activities.

TYPES OF DEPRESSION

Depressive disorders differ based on the number of symptoms, severity, and persistence. Major depres-

Depression exhibits a variety of symptoms; sufferers can experience either dramatic weight gain or weight loss.

sion is manifested by a combination of symptoms that interfere with the ability to work, study, sleep, eat, and enjoy once pleasurable activities. The symptoms of major depression are persistent sad, anxious, or "empty" mood; feelings of hopelessness and/or pessimism; feelings of guilt, worthlessness, and helplessness; loss of interest or pleasure in hobbies and activities that were once enjoyed, including sex; decreased energy, fatigue, or feeling "slowed down"; difficulty concentrating, remembering, and making decisions; insomnia, early-morning awakening, or oversleeping; appetite and/or weight loss or overeating and weight gain; thoughts of death or suicide; suicide attempts; restlessness and irritability; and persistent physical symptoms that do not respond to treatment, such as headaches, digestive disorders, and chronic pain.

DYSTHYMIA

Dysthymia is a less severe type of depression. Dysthymic disorder affects approximately 1.5 percent of the U.S. population age 18 and older in a given year (3.3 million).

BIPOLAR DISORDER

Bipolar disorder is also called manic-depressive illness. Bipolar disorder affects approximately 5.7 million American adults, or about 2.6 percent of the U.S. population age 18 and older in a given year. Bipolar disorder is characterized by cycling mood changes: severe highs (mania) and lows (depression). Mania, left untreated, may worsen to a psychotic state.

SEASONAL AFFECTIVE DISORDER

Seasonal affective disorder (SAD) is a pattern of depression related to changes in seasons and a lack of exposure to sunlight. It may cause headaches, irritability, and a low energy level.

CAUSES OF DEPRESSION

Bipolar disorder runs in families. The causes of depression may be due to stress, physical changes, and medical illnesses such as stroke, heart attack, cancer, Parkinson's disease, and hormonal disorders. A sick person is apathetic and unwilling to care for his or her physical needs, thus prolonging the recovery period. A combination of genetic, psychological, and environmental factors and change in life patterns involved in the onset of a depressive disorder. An increased or reduced appetite and unexplained weight gain or loss may also indicate depression.

ANTIDEPRESSANTS AND OBESITY

Weight gain is a reported side effect of nearly to all antidepressant medications. Tricyclic antidepressants and monoamine oxidase inhibitors (MAOIs) are more likely to be associated with weight gain than are selective serotonin reuptake inhibitors (SSRIs). Improvement in appetite is associated with improved mood may result in increased weight. The antidepressant therapy with mirtazapine was associated with a significant increase in body weight, body fat mass, and leptin concentration. Venlafaxine (Effexor) has been shown to cause weight gain but not as severe as has been reported with the SSRIs paroxetine (Paxil), fuoxetine (Prozac), and sertraline (Zoloft). Mitrazapine (Remeron) has been associated with significant weight gain, possibly secondary to interactions with the histamine (H1) receptor. Trazadone (Desyrel) is an antidepressant with sedative properties that is frequently used as a sleep aid as well as treatment for depression. It appears

to cause less weight gain than amitriptyline (Elavil®) but more than buproprion HCL (Wellbutrin®).

Anticonvulsants tend to cause hyperinsulinemia (elevated insulin in the blood) and increased appetite leading to weight gain. Hyperinsulinemia also results in increased testosterone, which causes a risk to women on these medications for development of Polycystic Ovary Syndrome (POS). Polycystic ovary syndrome can cause weight gain, male pattern baldness, increased facial hair, skin tags, acne, infertility, high blood pressure, abnormal lipid levels, and heart disease. One-third to two-thirds of the patients treated with lithium gain weight. Of those, 25 percent gain enough weight to be classified as obese. Weight gain is dose dependent. Haloperidol (Haldol, Peridol) is a conventional antipsychotic with a lower incidence of weight gain than the newer agents clozapine (Clozaril), olanzapine (Zyprexa), and sertindole (Serlect).

A retrospective study showed that clozapine (Clozaril) and olanzapine (Zyprexa) had the greatest associated weight gain, followed by intermediate weight gain with risperidone (Risperdal). Patients treated with sertindole (Serlect) had less weight gain than those treated with haloperidol. Clozapine (Clozaril) linked to significant weight gain and lipid abnormalities, suggesting increased risk for diabetes. Among the conventional antipsychotics, thioridazine and chlorpromazine have greater potential for weight gain, while molindone (Moban) is the only antipsychotic shown to not increase weight on a consistent basis.

DEPRESSION AND CHRONIC CONDITION

Depression is one of the most common complications of chronic illness. It is estimated that up to one-third of individuals with a serious medical condition experience symptoms of depression. The rate for depression occurring with other medical illnesses is quite high: The rate of depression varies in heart attack patients (40 -65 percent), 18-20 percent with coronary artery disease (without heart attack), 40 percent with Parkinson's disease, 40 percent with multiple sclerosis, 10-27 percent with stroke, 25 percent with cancer and 25 percent of patients with diabetes experience depression.

DEPRESSION AND OBESITY

Epidemiologic data suggest an association between obesity and depression. Extreme obesity was associated with the increased risk for depression across gender and racial groups, even after controlling for chronic physical disease, familial depression, and demographic risk factors. Adult obesity has been associated with depression, especially in women. Studies have also suggested an association between depression in adolescence and higher body mass index (BMI) in adulthood.

Whether depression leads to obesity or obesity causes depression is not clear. Obesity and depression are two major diseases which are associated with many other health problems such as hypertension, dyslipidemia, diabetes mellitus, coronary heart disease, stroke, myocardial infarction, heart failure in patients with systolic hypertension, low bone mineral density, and increased mortality. Goodman and Whitaker showed that depressed adolescents are at increased risk for the development and persistence of obesity later in their life and Pine et al. concluded that depression in childhood was positively associated with BMI during adulthood. In spite of the inconsistent findings overall it is believed that psychological distresses caused by obesity may lead to depression. Serotonin is believed to be involved in the complex process of integrating physiological and behavioral systems geared towards energy balance. An insufficient serotoninergic neuronal function in the central nervous system has been shown in many studies to occur in patents with depression. The Third National Health and Nutrition Examination Survey (1988–94) findings suggest that obesity is associated with depression mainly among persons with severe obesity.

In a recent prospective study, childhood depression was associated with an increased body mass index in adulthood. Chronic stress is also believed to increase abdominal obesity and its related adverse metabolic consequences, such as hypertension, insulin resistance, and dyslipidemia. This may lead to changes in brain morphology and the neuroendocrine axis that can cause both obesity and depression. Obesity in adolescence may be associated with later depression in young adulthood, abdominal obesity among male subjects may be closely related to concomitant depression, and being overweight/obese both in adolescence and adulthood may be a risk for depression among female subjects. Anxiety disorders and depression were associated with higher BMIs in females, whereas these disorders in males were not

associated with higher BMIs. Depression is ten times more frequent in obese patients undergoing the obesity treatment vs. general population. When people gain weight, their risk of developing diabetes mellitus, coronary artery disease, and other health problems increases. The drug's mechanism of action involves blocking activity at serotonergic 5-HT2, muscarinic, cholinergic, histaminic, alpha-adrenergic, and dopamine D1 and D2 receptors.

DEPRESSION AND CARDIOVASCULAR DISEASE

Depression significantly impacts morbidity and mortality in cardiovascular disease patients. Depression is may be an independent risk factor for the onset of coronary heart disease. The risk of depressed patients dying in the 2 years after the initial assessment is two times higher than that of nondepressed patients. Depression may lead to dysregulation of immunologic mechanisms (eg, proinflammatory cytokines such as interleukins [IL-1, IL-6] or tumor necrosis factor [TNF]), which are associated with an increased risk of CHD. Coagulation abnormalities and vascular endothelial dysfunction are thought to play an etiologic role in the development or the progression of atherosclerosis in depressed people. Psychosocial stressors are known to be predictors of depression in patients with CHD and are also known to be predictors of CHD and the prognosis in CHD patients.

DEPRESSION AND DIABETES

Depressive symptom severity was associated with a nonsignificant increase in HbA1c level. Depressed patients have increased insulin resistance after oral glucose testing compared with nondepressed patients. In a meta-analysis of 27 studies found a statistically significant association between depression and hyperglycemia in both Type 1 and Type 2 diabetes.

DEPRESSION AND CANCER

Depression is also found in about 15 percent to 25 percent of cancer patients. Breast cancer survivors who have finished treatment experienced more depression and far higher levels of fatigue, sleep problems, and difficulty working and concentrating than healthy subjects. The depression rate was twice as high as patients with head and neck cancer. The most common type of depression in people with cancer is called reactive depression. The symptoms of feeling moody and unable to perform usual activities last longer and are more pronounced than a normal and expected reaction but do not meet the criteria for major depression.

SEE ALSO: Antidepressants; Drugs and Food.

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DEXA (Dual Energy X-Ray Absorptiometry)

RECENT ADVANCEMENT IN the investigation of body composition and its relation with the onset of diseases has fostered the importance of using the dual energy X-ray absorptiometry (DEXA). The use of DEXA is an established method to measure bone mineral density (BMD) and mass, but is also used to assess lean body mass and whole body fat. DEXA measurements of body composition have previously been validated against a number of criterion methods including carcass analysis in animal studies. In general, the concept of the DEXA is to quantify the attenuated radiation subsequent to its passage through bone and tissue. This occurs by emitting two low-dosage X-ray beams at a patient's bones,

which are calibrated at two energy levels. One beam absorbs mainly soft tissue, while the other concentrates on bone. The BMD can then be determined by subtracting the soft tissue from the total. Currently, DEXA systems are based on either pencil beam or fan beam systems, where the fan beam is the newer generation with faster scan times. Also, DEXA measurements are associated with low-dose radiation (2 microSieverts, :Sv, at the spine or total body measurement and 4 :Sv at the hip) compared to a chest X-ray (~60 :Sv). DEXA is one of the most precise measurements for bone densitometry (i.e., spine, hip, radius) and body composition (whole-body scan), with coefficient of variations between 1 to 2 percent. However, its accuracy is still questionable in the severely obese and very lean. For example, estimates of bone density decrease in accuracy with increases in tissue thickness. Changes in individuals' hydration levels may influence calculations for soft tissue estimates. Correction algorithms are included in operating software to account for varying effects of tissue thickness, but estimates may differ with different manufacturers. Measurements with DEXA have weight limitations from 114–160 kg, which also depend on the instrument manufacturer and model. Despite certain limitations, DEXA methodology is currently the best research tool to estimate body composition in the clinical setting in adults and the pediatric population to better understand the physiology and pathophysiology in obese individuals, and is important in the diagnosis of osteoporosis and low bone density.

SEE ALSO: Osteoporosis.

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Dexatrim

DEXATRIM IS THE brand name used for a number of commercial dietary supplements manufactured by Chattem, Inc., and sold as appetite suppressants and aids to weight loss. The products used in Dexatrim have changed over the years and at times have included phenylpropanolamine (PPA) and ephedra. Dexatrim, along with many other dietary supplements, was subject to recall in 2002 when the United States Food and Drug Administration (FDA) established that ephedra was associated with increased risk of stroke. No Dexatrim product sold today contains ephedra, according to the company website. It is more difficult to determine if any Dexatrim products contain PPA, because (again according to the company website) Chattem disputes that PPA has been established as dangerous, but also states that no Dexatrim products containing PPA will be advertised or shipped, at the request on the Food and Drug Administration (FDA).

The contents of different Dexatrim products currently include common vitamins and minerals such as vitamins C, E, calcium and zinc; herbs such as yohimbe bark and licorice root, and other ingredients such as caffeine and green tea leaf extract. Chattem claims that Dexatrim helps people lose weight in two ways: by acting as an appetite suppressant so people eat less, and by increasing the base metabolic rate so people burn calories faster. It is important to note that in the United States, the FDA does not evaluate the safety or effectiveness of products such as Dexatrim that are marketed as dietary supplements, except for products established as hazardous such as ephedra. Therefore, although the FDA may ban products such as ephedra which they feel have been established as dangerous, neither they nor the manufacturers are required to subject nutritional supplements to the type of testing required of prescription drugs, nor to establish the truth of claims regarding such products (for instance, that Dexatrim helps individuals lose weight). Although surveys have found Dexatrim to be a commonly used herbal weight loss product, no studies of the current formulation have established its efficacy.

Dexatrim is sold by Chattem, Inc., a company which produces or distributes many well-known over-the-counter (nonprescription) pharmaceutical and drugstore products, including ACT mouthwash, Cortisone-10 ointment, Flexall topical analgesis,

Garlique dietary supplement, Gold Bond medicated powders, Selsun Blue dandruff shampoo, and Unisom sleep aids. Chattem began as a family business under the name of the Chattanooga Medicine Company in Chattanooga, Tennessee in 1879, and pioneered direct-marketing and advertising techniques in the pharmaceutical business, in particular the use of outdoor advertising on barns and storefronts.

SEE ALSO: Drug Targets that Decrease Food Intake/Appetite; Ephedra; Food and Drug Administration; Stroke

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Showing restraint by controlling food intake is one of the most significant ways to reduce and maintain weight.

Dietary Restraint

ORIGINALLY CONCEPTUALIZED BY C. Peter Herman and Deborah Mack, dietary restraint is defined as the conscious restriction of food intake to control body weight. Controlling food intake is important for the prevention and treatment of obesity. Several investigators have shown that higher dietary restraint is associated with a healthier body weight and greater success during weight loss. Yet, others show no relationship or negative associations between dietary restraint and body weight or weight-loss success. Recent findings suggest that further refinement of the construct using subscales to define flexible and rigid styles of dietary restraint may reconcile previously conflicting evidence and fuel new advances in the development behavioral approaches for the treatment and prevention of obesity.

Three self-rating questionnaires have been developed to assess dietary restraint. The Three Factor Eating Questionnaire (TFEQ), also known as the Eating Inventory, is the most extensively studied and most commonly used scale.

It is comprised of three factors—dietary restraint, disinhibition, and hunger—and was developed to improve upon the validity of the original Restraint Scale, introduced by C. Peter Herman and Deborah Mack. Another scale, the Dutch Eating Behavior Questionnaire (DEBQ), assesses dietary restraint as well as emotional and external eating behavior. Findings on the relationship between measures of dietary restraint and obesity are equivocal, with some showing no relationship, while others find significant positive or negative correlations between dietary restraint scores and body weight or measures of adiposity. The later refinement of the restraint construct into two distinct subscales helps to reconcile the seemingly contradictory research findings.

The subscales distinguish between flexible restraint, defined as a graduated, adaptive approach to dieting,

and rigid restraint, an all-or-nothing approach that is often maladaptive. Results from a series of studies conducted in Germany showed negative correlations between body mass index and flexible restraint and positive correlations between body mass index and rigid restraint.

Thus, men and women with higher scores on the flexible restraint scale were found to have lower body mass indices, whereas men and women with higher rigid restraint scores had higher indices. Furthermore, increases in flexible restraint during weight loss were associated with greater success whereas increases in rigid restraint were not associated with weight-loss success. Items on the DEBQ reflect a flexible restraint style; thus, it is not unexpected that increases in scores on this scale have been associated with more successful weight loss, whereas in studies where restraint scores from the TFEQ were used, no relationships were found.

These findings illustrate that flexible but not rigid restraint is associated with weight-loss success. Further research is needed to determine if dietary restraint style can be modified. Specifically, the findings suggest that intervention designed to promote the substitution of flexible for rigid restraint behaviors may enhance the efficacy of current behavioral approaches to the treatment and prevention of obesity.

SEE ALSO: Body Mass Index; Cognitive-Behavior Therapy; Disinhibited Eating; Women and Dieting.

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Dieting: Good or Bad?

GIVEN THE CURRENT obesity epidemic, it is important to determine the extent to which individuals should be encouraged to diet. Dieting is already quite common in the general population, but it has variable effectiveness, depending on the weight-control behaviors that are used. Questions abound about the usefulness of dieting as well as its influence on medical and psychiatric health. The controversy that surrounds dieting may in part be due to confusion between dieting and the construct of restraint. Dieting is not considered to be synonymous with restraint, as described by Peter Herman and Janet Polivy. (Recent research indicates that items on their Restraint Scale do not measure dietary behaviors or restrained eating per se, and most "restrained eaters" who the scale identifies do not report currently dieting to lose weight.) Determining how "good" or "bad" dieting is involves three central issues: the effectiveness of dieting, its physical or biological effects, and its psychological effects. The effectiveness and consequences of dieting depend largely on the type of dieting behaviors that are being used and who is using them. Context is critical for understanding how helpful or harmful dieting is.

EFFECTIVENESS OF DIETING

Information is available about the effectiveness of several types of dieting: dieting programs that are researched at university clinics, commercial dieting programs, and self-guided (i.e., independent) dieting. The most information is available about dieting programs that are researched at university clinics. However, the participants who enroll in these programs likely differ in significant ways from typical dieters; individuals who seek treatment from university-based clinics have more psychiatric problems (e.g., depression or binge eating) and a longer history of unsuccessful dieting.

University clinics often offer comprehensive weight-control programs that promote weight loss through nutrition change (e.g., a calorie-restricted, low-fat diet) and physical activity. Behavioral treatment (e.g., teaching techniques such as self-monitoring, goal setting, and stimulus control) can facilitate these changes in diet and exercise. Group-based behavioral treatment is the gold standard for weight-loss dieting. This approach is effective in the short term. On average, it results in a loss of approximately 10 percent of initial body weight in approximately 30 weeks of treatment, which is considered a medically significant weight loss. However, weight-loss maintenance remains a challenge for virtually all approaches, including be-

havioral treatment: the majority of dieters regain lost weight within 3–5 years. As a consequence, obesity is increasingly being conceptualized as a chronic disorder that requires long-term care. When contact with treatment providers is continued (e.g., through group meetings or phone calls) weight regain is slowed. Development of dieting programs that have promising long-term results is a priority for future research.

Some research has also been conducted on the effectiveness of commercial dieting programs, which millions of Americans enroll in each year. Very-low-calorie diets (e.g., Optifast) can promote large weight losses (e.g., 15 to 20 percent of body weight), but weight-loss maintenance is poor. Purchasing meal replacements (e.g., SlimFast) for use in a balanced-deficit diet also can facilitate 5 to 10 percent weight losses; continued use of meal replacements also may facilitate weight-loss maintenance. Finally, group-based approaches such as Weight Watchers can be effective at producing weight loss of approximately 5 percent of initial weight.

Although self-guided dieting is probably the most common approach for weight loss, there is little information available about its effectiveness. Assessment of self-guided dieting is difficult because there is substantial variability in the dieting methods that individuals use, the length of time for which they diet, and the weight-loss goals they have (in fact, it is common for individuals to report that they diet not for weight loss but for weight gain prevention).

In the general population, individuals who identify themselves as dieters or restrained eaters are more likely than nondieters to gain weight over time. One hypothesis for this phenomenon is that ignoring hunger signals by restraining eating can disrupt the ability to regulate appetite and intake. Another hypothesis is that individuals who diet have a propensity toward weight gain that precedes dieting, and that most individuals who diet are simply not using strategies that are effective enough to prevent this weight gain. Only one-third of self-guided dieters report engaging in both caloric restriction and physical activity, and they often do not decrease their caloric intake or increase their caloric expenditure in a manner substantial enough to produce weight loss. Without the structure, support, and guidance of a weight-loss program (such as the behavioral treatment offered by university clinics), many individuals appear to have a difficult time adopting the weight-control behaviors that are necessary for lasting weight loss.

Nonetheless, a variety of approaches, including self-guided dieting, have the potential to produce positive results. The National Weight Control Registry is a database of over 5,000 individuals who have lost approximately 66 pounds and maintained that lost for approximately five years. Many of these individuals lost weight using self-guided or commercial programs. Regardless of weight-loss method, these individuals consistently report that to maintain their weight loss, they eat a low-fat, low-calorie diet and engage in high levels of physical activity. Although this database is clearly not representative of the general population of dieters, it demonstrates that long-term success is possible and provides information on the behaviors that are associated with success (though demonstrating this degree of weight-loss maintenance success in randomized clinical trials remains rare).

PHYSICAL EFFECTS OF DIETING

There are numerous health-related benefits from weight-loss dieting in overweight individuals. Weight losses of 5 to 10 percent usually produce significant improvements in blood sugar control, hypertension, blood lipids, and other health indices. These improvements deteriorate if lost weight is regained.

Weight loss tends to produce changes in bodily functions that encourage weight regain (e.g., heightened levels of ghrelin and greater insulin sensitivity). Weight loss tends to produce a slowing of metabolic rate for two reasons. One is that some of the lost weight is muscle, which unlike fat, is metabolically active. The other is that the body can react to weight loss as if it is starving and therefore show an exaggerated metabolic slowdown, especially if weight losses are large. For those who are able to maintain a weight loss, evidence suggests that this "defensive" component of the metabolic slowdown dissipates over time. Therefore, for most people who lose and maintain a weight loss, their metabolism at their lower weight will be comparable to someone of equal size who never lost weight.

Because weight lost on diets is frequently regained, many people acquire a history of weight loss and regain (or weight cycling). Although initial research on weight cycling raised concerns that weight loss became more difficult and weight regain more

likely with each weight cycle, subsequent research has mostly concluded that cycling does not have significant adverse effects on physiological parameters. There is some evidence that a history of weight cycling is associated with arteriosclerosis and early mortality. However, it is not clear whether any such associations might be due to volitional weight loss or to weight losses due to other factors. Finally, it is important to remember that individuals prone to weight gain who do *not* engage in weight-loss efforts are likely to continue gaining weight, which definitely has negative health consequences.

A reasonable conclusion from this research is that the health benefits of diet-induced weight loss appear to outweigh any concerns about the possible deleterious effects of repeated bouts of weight loss and regain.

PSYCHOLOGICAL EFFECTS OF DIETING

The psychological effects of dieting are also important to consider. Concern about the negative consequences of weight loss was sparked, in part, by an experimental study conducted with normal-weight men who were rapidly reduced to 75 percent of their initial body weight. After weight loss, many of these participants exhibited difficulty concentrating, binge eating, and irritable and depressed mood. Although this type of dieting clearly has negative consequences, the relevance of these phenomena to overweight and obese individuals is questionable.

Dieting has been frequently implicated in the etiology of eating disorders. Young females who diet are at increased risk of developing an eating disorder, compared to nondieters. However, while dieting may be a necessary precursor to an eating disorder, it is probably not a sufficient cause of one. Experimental studies suggest that a third variable may explain the relationship between dieting and eating disorder onset in some women. In fact, when normal-weight young women are placed on a short, moderate weight-loss diet, their disordered eating decreases, rather than increases. Similarly, patients with bulimia nervosa who report frequent dieting actually binge and purge significantly less than patients who infrequently diet, raising further questions about the relationship between dietary restriction and eating disorder symptoms. It is possible that dieting is more likely to increase risk for an eating disorder when it is done by young women, when weight is lost rapidly or through unhealthy methods, or when the dieter is normal weight or underweight when they begin dieting. In contrast, research has shown that in overweight youth, professionally administered weight-loss programs pose minimal risk of precipitating eating disorders and actually result in significant improvements in psychological status. Similarly, dieting and weight loss in overweight adults is not associated with the development of eating disorders.

Although some studies have identified a link between dieting and disinhibited eating, more consistent evidence indicates that weight-loss programs that prescribe modest caloric restriction do not precipitate binge eating. In fact, for individuals who experience binge eating, traditional behavioral weight-loss programs typically result in significant reductions in binge eating. Even more promising, when obese binge eaters lose weight, some improvements in their binge eating are sustained even when weight is regained.

Several studies have found that weight-loss dieting in obese adults is associated with improvements in depression, anxiety, and related outcomes. However, because most of these data were collected from studies of behavioral weight-loss treatment, it is difficult to determine whether these benefits are a consequence of dieting, of weight loss, or of behavioral treatment. Nonetheless, iatrogenic effects of dieting have not typically been observed; even when periods of weight regain follow dieting, it does not appear to have any significant, negative impact on mood, weight-related cognitions, or other psychological outcomes.

AREAS FOR FUTURE RESEARCH

Additional research must examine the long-term effects of dieting on mental and physical health. The benefits and costs of weight-loss dieting that is followed by weight gain also must be compared to the benefits and costs of *not* dieting. Given the current obesogenic environment, the consequences of abstaining from dieting should be studied as carefully as those of engaging in dieting.

SEE ALSO: Dietary Restraint; Eating Disorders and Obesity; Women and Dieting.

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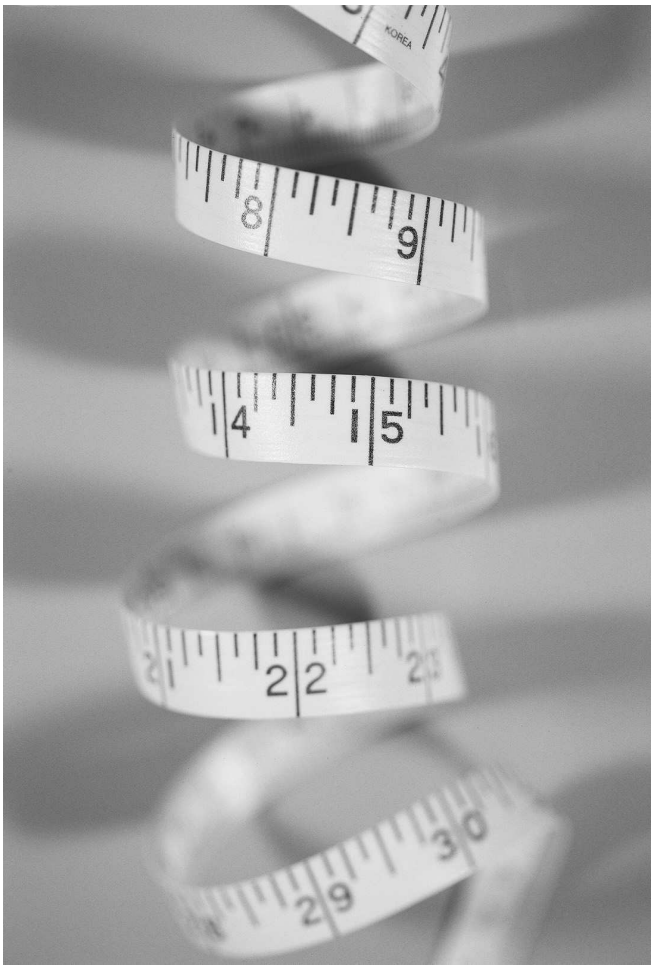
Diet Myths

MAINTAINING A HEALTHY diet involves a balance of behavioral, psychological, environmental, and biological factors. An overwhelming amount of dieting information is available through the internet, television, magazines, and books. Some of this information is good, some bad, and some potentially harmful to dieting individuals. Surprisingly, little research has been done on dieting misconceptions and their effects on weight-loss efforts. Several of these weight-loss dieting myths should be debunked.



While some dieting myths may be innocuous, others can present potential health risks to dieters.

THE "MAGIC BULLET" SYNDROME

This is the belief that a pill, drink, special diet, or exercise machine will enable individuals to lose large amounts of weight rapidly and with little change in lifestyle. Individuals are continually bombarded with misleading advertisements (e.g., "Lose 30 pounds in 30 days!", "Eat as much as you want and still lose weight!"). Such claims take advantage of people's faith in modern science and instill false beliefs that excess weight can be shed quickly and effortlessly. These claims may lead individuals to underestimate the medical severity of obesity and perceive the time and effort healthy dieting requires as unnecessary. Data from a large study in 1999 reported that most dieters are not using the recommended combination of reducing caloric intake and increasing physical activity. When these remedies do not live up to their claims, individuals may become discouraged and less likely to seek legitimate weight-loss alternatives.

DIETING IS BAD

In past decades, there was a fear that dieting would lead to eating disorders, binge eating, and/or further weight gain. Current research, however, does not support this contention. While improper weigh-loss techniques (e.g., skipping meals, starvation/fasting, purging, compulsive overexercise, amphetamine use) can be associated with disordered eating, evidence demonstrates that proper dieting (e.g., limiting portion sizes, consuming foods low in caloric density, limiting fat intake, balancing nutrients, avoiding excess sugar) actually reduces the incidence of eating disorders and binge eating in overweight individuals.

CARBOHYDRATES ARE BAD

Fad diets, such as the low-carbohydrate diet, are notorious for gaining immense popularity, failing to live up

to their hype, and fading away until the next fad arises promising more weight loss with less effort. Ironically, in the early 1980s, the high-carbohydrate diet was touted as the answer to obesity. In the 2000s, America was in the midst of a low-carbohydrate craze. However, research demonstrates that low-carbohydrate diets work if they reduce caloric intake and do not work if they do not reduce caloric intake. Carbohydrates are not necessarily good or bad; weight loss is determined by calories regardless of macronutrient makeup.

SNACKING IS BAD

Frequency of snacking is not consistently related to weight gain. In fact, recent evidence suggests that individuals who eat more often tend to weigh less when controlling for the number of calories eaten. Snacking can help individuals avoid feeling that they need to starve themselves to lose weight and can help curb strong cravings that may otherwise lead to ingesting a large amount of calories. Snacking can also be used strategically before meals (e.g., fruit or salad before a main course) in order to reduce intake of a calorie-dense plate. However, the caloric content of snacks is important. Continually snacking on high-calorie foods will likely lead to weight gain, while an increase in snacking on low-calorie foods may actually result in weight loss.

OBESITY IS INDICATIVE OF AN UNDERACTIVE THYROID

An underactive thyroid can lead to almost unavoidable weight gain despite good nutritional practices. However, underactive thyroid accounts for less than 1 percent of obese individuals. The Third National Health and Nutrition Examination Survey of 17,353 patients reflecting the U.S. population reported a prevalence of 0.3 percent overt hypothyroidism. In fact, aside from this small percentage of individuals, heavier individuals actually have a higher metabolic rate than do lean individuals. Thus, consistent reduction in caloric intake and increase in caloric expenditure will result in decreased body weight in over 99 percent of obese individuals.

REDUCED-FAT OR ORGANIC PRODUCTS ARE NECESSARILY LOW CALORIE AND HEALTHY

"Low fat" or "fat free" does not necessarily mean low calorie. Extra sugars and thickeners are often added to boost flavor and texture, so calorie content may actually be similar to standard products. Individuals may mistakenly think that fat-free products can be eaten in large quantities without contributing to their body weight. This may lead to higher caloric intake than would be associated with full-fat products. Additional products simply cut calories and fat by lowering the serving size of standard recipe items, which may also lead to increased consumption of these foods under the guise of being a healthier alternative. Similarly, many believe organic foods to be necessarily healthier alternatives. The term *organic* refers to methods used to produce foods, not their nutrient content. Organic products often contain just as much fat, sugar, and calories as their nonorganic counterparts, and are just as likely to lead to weight gain when eaten in excess.

EATING AT NIGHT IS WORSE THAN EATING DURING THE DAY

Many diets direct individuals not to eat after a certain time of night in the belief that the body will store more fat because the calories are not burned off with activity. However, studies conducted in whole-body calorimeters (which measure calories burned and stored) reveal that large meals eaten late at night do not make the body store more fat. It is the total calories ingested per 24-hour period that is important. When fewer calories are ingested than burned, weight loss occurs (and vice versa) regardless of what time the calories are ingested.

EXERCISE ALONE IS ENOUGH

A common assumption is that increasing exercise is as effective for weight loss as limiting food intake. To a large extent, individuals compensate for caloric expenditure due to exercise. As exercise increases, caloric intake tends to increase as well.

This is not to say that exercise should not be included in a diet program. Studies from the National Weight Control Registry (individuals who have maintained significant weight loss) reveal that exercise is the single best predictor of weight-loss maintenance. Although exercise has not been shown to significantly increase weight loss when added to lifestyle change diets, it may play an important role in the more difficult task of weight-loss maintenance.

DRINKING WATER INCREASES WEIGHT LOSS

Water consumption that replaces caloric beverages may aid weight loss due to a decrease in caloric intake. However, water consumption itself does not result in weight loss. Drinking water may temporarily increase the feeling of fullness however, studies show that individuals quickly overcome this feeling. Choosing foods with a higher water content (lower in caloric density), however, has been shown to reduce overall caloric intake and cause weight loss. Therefore, it may be more important for individuals to increase water consumption in the foods they eat as opposed to from the tap or bottle.

DIETING HAS TO BE COMPLICATED

Many individuals mistakenly believe they have to measure everything they eat and perform complex calculations using nutrition labels. For the willing participant, these methods can be effective; however, there are numerous dieting options for individuals. A meal-replacement diet is very simple to follow and has been proven as effective as other diets. Sustainability is the key to successful weight loss.

ONCE A "FORBIDDEN FOOD" IS EATEN, THE DIET IS OVER

Dieting individuals often think in "all or nothing" terms; they are either on or off the diet. Breaches in a diet regimen (e.g., eating a dessert not on the diet) occur in everyone. Studies show that these individual lapses do not cause major problems for weight-loss efforts if the person resumes with his or her diet plan. However, once a "forbidden food" is eaten, many individuals assume that the diet has been ruined. This leads many to then consume large quantities of unhealthy foods and sometimes even full-blown binge episodes. Thus eating a "forbidden food" will not hurt the diet if the person accepts it; however, overreacting to such an event may lead an individual to abandon the diet altogether.

CONCLUSION

Research has shown that people who lose weight on a diet consistently show an increase in mood, even individuals on an all-liquid diet. Unfortunately, the diet myths discussed here cover only a fraction of the misguided beliefs held by dieters, many of which are perpetuated by companies who profit by instilling the public with false hopes of effortless weight loss. Weight loss is achieved by ingesting fewer calories than one expends and typically requires time, effort, and consistency. Regardless of the source, claims that weight loss can be achieved through means other than lowering caloric intake and/or raising caloric expenditure are misleading. Diet advice should be sought through individuals with formal training (R.D. or Ph.D.) in nutrition.

SEE ALSO: Dieting: Good or Bad?; Energy Density; Nutrition Fads.

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Dilution Techniques

DILUTION TECHNIQUES ARE methods used to determine the amount of a particular nutrient or other substance in a person's body. They are more consistently accurate than simply measuring a person's serum levels of a particular nutrient, because these serum levels may fluctuate greatly, depending on the person's state of stress, infection, fatigue, or general nutrition. These techniques can be used to assay the nutritional status of a person and therefore screen for any potential malnutrition that may lead to or already be causing an illness.

One example of a dilution technique is the isotope dilution technique (IDT), an excellent method for determining a person's nutritional status. The principle behind the IDT is simple: A person ingests a known amount of a nutrient that has been labeled with an isotope such as deuterium (hydrogen with one proton and one neutron in its nucleus compared to the

typical neutronless nucleus). Deuterium is stable and not radioactive. Sufficient time is allowed so that the labeled isotope can diffuse throughout the body such that it is not concentrated in any particular area. Then a blood sample is taken and the ratio of labeled to unlabeled nutrient in the serum is analyzed. Because the total amount of labeled nutrient in the body is known, the clinician can then calculate how much unlabeled nutrient is present in the person's body, and thus, determine the nutritional status of the person.

This technique works best when used to measure nutrients that are readily absorbed by the body and relatively stable over periods of time. If, for example, the labeled nutrient were barely absorbed, then the ratio would be misleading. Similarly, if the body metabolized most of the labeled nutrient before diffusing to equilibrium, the ratio would again be misleading.

One major drawback to the IDT method is that labeled nutrients can be expensive; therefore, screening an entire population of people with this technique may be prevented by financial considerations.

Too low a level of nutrient in the body may result in serious illnesses; in some cases, too much a nutrient is not healthy either. Therefore, another important application for the dilution technique is to measure a person's body water or other substance status after nutritional or hormonal therapies. Endocrinology, immunology, neural signaling, and nutrition are all tightly linked in the body; therefore, after applying any therapy to a patient, clinicians must monitor the patient's nutritional state to ensure that there is not a malnutrition side effect.

Dilution techniques can be used in concert with bioelectrical impedance analysis as well.

SEE ALSO: Bioelectrical Impedance Analysis; Neuropeptides; Neurotransmitters; Nutrition and Nutritionists.

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Disinhibited Eating

DISINHIBITED EATING OCCURS when an individual temporarily loses control over his or her eating behavior. Common examples of disinhibited eating are eating when one is not hungry, overeating, and engaging in a binge-eating episode. Disinhibited eating is common in individuals who chronically diet as well as those with eating disorders such as bulimia nervosa and binge-eating disorder.

The term *disinhibited eating* was coined by researchers studying dieting behavior. These researchers hypothesized that individuals could be classified by the extent of restraint or control that they chronically exercise over their eating behavior. According to Dietary Restraint Theory, individuals who exhibit a large amount of control over their eating (such as chronic dieters) generally eat in a very systematic way, successfully adhering to a specific dietary regimen. However, the self-control of such individuals is fragile and, under certain circumstances, they are likely to engage in disinhibited eating. Consequently, a pattern of eating characterized by strictly controlled food intake followed by disinhibited, overindulgent eating is common in individuals trying to diet or high in dietary restraint.

Disinhibited eating is likely to occur more frequently in specific situations and under certain internal psychological states. The most common empirically supported factors that lead to disinhibited eating are eating "forbidden foods," believing one has overeaten, distorted thinking, taking tranquilizing substances (such as alcohol), and experiencing strong emotional states.

Eating foods deemed to be dietarily unacceptable or "forbidden," such as chocolate bars, ice cream, and fried foods, or believing one has overeaten can lead to disinhibited eating. One reason for this is that eating forbidden foods or overeating is often associated with distorted and unhelpful thinking focused on the fact that one has made a dietary violation. For example, after eating something deemed to be a dietary violation, an individual may think "I already ruined my diet today, so I might as well eat whatever I want." Such distorted, all-or-nothing thinking leads individuals to engage in disinhibited eating.

Strong mood states are also associated with and predictive of disinhibited eating. Numerous studies

have exposed individuals with varied levels of dietary restraint to mood inducing laboratory situations and found that, when feeling strongly sad, anxious, or depressed, individuals are more likely to engage in disinhibited eating. Additionally, under the influence of alcohol and some drugs, such as marijuana, individuals are more likely to engage in disinhibited eating.

Cognitive-behavioral treatment strategies can be helpful to ward against disinhibited eating. For example, individuals can be trained to refute distorted thoughts, eat "forbidden foods" in moderation, and gain awareness of mood states that would ordinarily lead them to engage in disinhibited eating.

SEE ALSO: Binge Eating; Bulimia Nervosa; Dietary Restraint; Disordered Eating.

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Disordered Eating

THERE ARE MANY times when the act of consuming of food varies from the norm; this variation can take place in the form of excessive consumption of food or a complete reduction in consumption to the point where an individual starves. Disordered eating patterns can occur due to genetic (e.g., obesity has some genetic causes), environmental, psychiatric, or as an effect of other diseases present in the individual.

There is a general consensus that one should attempt to adhere to the guidelines of food consumption to avoid complications. An attempt to eat three meals a day with a substantial breakfast and two smaller meals with healthy snacks such as yogurt is recommended. Meals should include a variety of foods and should include at least five fruits and vegetables, averaging out to 500 g a day. This enables one to consume the essential vitamins, minerals, fibers, and antioxidants. The consumption of oily fish once a week, which includes omega-3 polyunsaturated fatty acids, is also recommended. Eating foods with low glycemic index is also recommended as these foods increase the time it takes for blood sugar levels to rise. Normal eating is, however, flexible in that a slight alteration in patterns over time or a greater alteration within a short period is considered normal and will not prove to be problematic.

Disordered eating appears in many different forms. The different types of disordered eating are discussed below.

ANOREXIA NERVOSA

Anorexia nervosa is a psychiatric eating disorder. The disorder has a prevalence rate in women of 1 to 2 percent and 0.1 to 0.2 percent of males. However, 5 percent of women may experience many of the symptoms of anorexia nervosa without developing the full disorder. Most of the sufferers of the disorder are in their adolescence; however, the disorder has been known to occur in elderly people. The onset of anorexia nervosa during childhood can affect the timing of puberty and adult height. The intensity of the disease affects the timing of menarche in most patients.

Anorexia nervosa is prevalent in Caucasian women, particularly those from high academic backgrounds. Studies have shown that the disorder can be seen in those with family and marital problems. Patients often have certain characteristics such as obsessive compulsion for perfection and age-inappropriate engagement is sexual activity.

Although anorexia nervosa is a psychiatric disorder, the causes are often due to interplay between other factors, such as social and genetic factors; the latter is demonstrated by the fact that monozygotic twins have a higher occurrence rate of the disorder than dizygotic twins. There are many other risk factors for developing anorexia nervosa. The disorder is seen in higher rates among athletes, students who have heavy workloads, and those with previous exposure to traumatic life experiences (e.g., abuse).

Anorexia sufferers have a weight loss of 15 percent below the expected weight. This occurs due to individuals using various measures to reduce weight such as adhering to a strict diet which may comprise of avoiding the main meals in a day. These individuals may further perfom vigorous exercise to lose weight which they perceive as unhealthy, although

to others they appear fragile and very slim. Hyperactivity is observed in approximately 80 percent of patients. These individuals may also use laxatives and diuretics to further lose weight. Patients may become severely depressed as a result of their constant dissatisfaction with their appearance.

As these patients are practicing almost ritualistic eating behaviors and restricting their diet to a dangerous level, they are also limiting their intake of essential vitamins and minerals. These patients often have a deficiency of protein and hence have muscle wasting. They also have hypoglycemia and depressed thyroid fucntion with low levels of thryoid hormone T3 (triiodothyronine). Reproductive abnormalities such as low estrogen levels, anovulation, and eventually the complete cessation of menstruation (amennorhea) may develop. Patients may also have decreased IGF-1 (insulin like growth factor-1) levels, reflecting decreased growth hormone action and hence retarded growth. Patients may have several cardiovascular abnormalities such as hypotension, bradycardia, mitral valve prolapse, dysrhythmias, and shock due to congestive heart failure. Gastrointestinal problems include delayed gastric emptying, resulting in constipation. Patients will also have dental enamel erosion as a result of the excessive emesis (vomit is acidic). Patients also have enlarged parotid (salivary) glands. Renal disturbances include decreased glomerular filtration rate, edema, acidosis, hyperaldosteronism, and hypokalemia.

The unbalanced electrolytes in the body may lead to seizures. Patients may have decreased antidiuretic hormone and present with diabetes insipidus. Patients also have dermatological abnormalities such as lanugo, brittle hair and nails, hair loss, and dry skin. Hematological abnormalities include anemia, leukopenia, thrombocytopenia, and impaired immunity. Patients may also suffer from hypothermia.Patients often have abnormal bone remodeling and osteopenia with a decrease in osteoprotegerin (OPG) and receptor activator of nuclear factor-kappaB ligand (RANKL), which could partly explain the increase in bone loss that occurs in these patients. Family therapy is an effective treatment for anorexia nervosa. Those who respond well to outpatient family intervention generally remain well. In general, conjoint family meetings early on in treatment when raised levels of parental criticism are evident are avoided. The process of refeeding is undertaken slowly and ideal weight gain should occur at a rate of 1–2 lb per week.

Anorexia nervosa if untreated has a poor prognosis. It has a very high mortality rate, which may be up to 20 percent. This is, however, mostly due to suicide and less commonly due to the main complications of the disorder. Half of treated patients will recover fully.

BULIMIA NERVOSA

Bulimia nervosa is an eating disorder where the individual experiences episodes of bingeing and purging. The incidence of the disorder is approximately 0.5 to 1 percent of women. Many of these women are in their adolescent years. The condition may also occur in men; 10 percent of bulimics are male. There are two types of bulimia nervosa: the purging type and the non-purging type. The former is associated with a excessive eating within a short period of time followed by acts of self-induced vomiting; the latter is associated excessive eating within a short period of time followed by other activities such as vigorous exercise, fasting, or inappropriate intake of laxatives to compensate for the excessive intake of food. These patients experience a sense of lack of control over eating during the episode.

The bulimic patient may engage in self-starvation between binge-purge episodes, thus presenting the same dangers as the anorexic patients such as osteopenia, in addition to the ones presented by the binging and purging. Approximately 25 percent of bulimics have had an episode of anorexia nervosa.

Many causes of the disorder are suggested; however, the exact etiology is unknown. Social factors may increase the risk of developing the disorder such as previous exposure to a traumatic/disturbing experience, abuse (mental, physical, and sexual), and family problems. Patients may also suffer as a result of bullying leading to low self-esteem. Social factors such as the Western perception of thin models representing the epitome of beauty and the general Western culture of thinness has been linked to bulimia nervosa. Research has linked low levels of the neurotransmitter serotonin (5-hydroxytryptamine) to bulimia nervosa and the development of depressed mood in these patients.

Bulimic patients may be of normal weight or slightly above average weight and hence the presence of other symptoms such as enlarged parotid glands and scarring on the back of the hands are important

in characterising the disorder. Dental complications frequently occur and include erosion of enamel and periodontal disease. Rarely seen complications are esophageal tears caused by purging and acute dilation of the stomach, occur after very large binges. Other symptoms include peripheral edema.

Often traits of childhood overeating are found to be more common in bulimic women compared to their unaffected siblings. The increased risk of the disorder due to being overweight during childhood suggests that prevention strategies for childhood obesity are also useful for bulimia nervosa. Young women with Type 1 diabetes mellitus have a higher incidence of eating disorders than young women in the general population. These diabetic adolescents often lose weight before being diagnosed with diabetes mellitus; when they begin having insulin treatment, they gain weight and as a result become apprehensive and try to use various measures such as bingeing and purging to reduce this weight. These patients may eventually discover another method of losing weight; by omitting doses of insulin, they can reduce their weight and hence these patients undertake this dangerous behavior to maintain weight. As a result, these patients are at an increased risk of developing other conditions such as retinopathy and also have increase risk of mortality from uncontrolled diabetes.

To treat the bulimia nervosa, the patient is generally properly assessed and subsequently administered various self-help guidelines and possibly prescribed antidepressant (e.g., fluoxetine) which selectively inhibits serotonin reuptake and hence increases the levels of serotonin. Patients may also be provided with cognitive behavior therapy. Patients who have strong support and encouragement from families and friends tend to have a greater chance of recovery.

Bulimia nervosa is more common than anorexic nervosa, and the prognosis is more favorable. Approximately 70 percent of patients have been known to recover. After recovery, however, many patients develop other problems such as anxiety and depression. Unexpected deaths may occur in patients, as women with bulimia nervosa are at risk to attempt suicide and many medical complications do exist due to the purging nature of this condition. Bulimic patients who have low weight also have an increase risk of death due to low blood potassium levels and cardiac arrhythmia.

BINGE-EATING DISORDER

Binge-eating disorder is a disorder with similar characteristics to bulimia nervosa; however, the former is defined as acts of frequent overconsumption of food without the subsequent purging often seen in bulimia nervosa. This disorder has a prevalence of 1 to 3 percent and affects males and females equally. Peak age of onset is 13–15; however, the disorder does occur during middle age and in the elderly.

These patients have a lack of control and hence cannot stop eating; they will continue to eat even when they feel full or not hungry. This will be followed by guilt or embarrassment and hence these patients will often eat alone particularly at night. The patient will eat (particularly fatty foods) excessively at least two days a week for six months. As a result, they are overweight or obese resulting in drastic measures to avoid other people and social situations. They also have difficulties with impulse control in other areas of their lives. Risk factors for the disorder are typically the same as bulimia nervosa; additional risk factors include, for example, elevated levels of perceived stress, which may precede the onset of the disorders. Studies suggest that approximately 25 to 50 percent of people suffering from obesity suffer from binge-eating disorder. Binge-eating disorder often results in some of the health risks associated with clinical obesity. Binge-eating disorder has, however, a more favorable prognosis than other eating disorders with estimates of a recovery rate of 80 percent after five years and a low relapse rate.

There are several other eating disorders such as eating disorders not otherwise specified (EDNOS) which includes the disorders mentioned above presenting in atypical form, for example, anorexic patients who menstruate normally. EDNOS develop in 4 to 6 percent of the general population. Other eating disorders include Pica, a disorder found most frequently in children who persistently eat inappropriate material such as clay, stones, feces, and lightbulbs. It is often seen in those with developmental disabilities. Prader-Willi syndrome is a childhood disorder characterized by children overeating; this is an inherited disorder and these children may develop life-threatening obesity. These patients also have mental retardation. Nocturnal sleep-related eating disorder is another eating disorder where the person

eats excessively while asleep; these patients are not conscious and this act is often associated with sleepwalking. These individuals are not aware of what is happening and have no recollection of what has happened the previous night. This disorder occurs in 1 to 3 percent of the general population. Another eating disorder where the patients eat during the night is night-eating syndrome; this is where the individual has increased appetite during the night and almost no desire to eat during morning. These individuals will frequently eat different snacks during the night but do not binge eat.

Eating disorders have multiple and complex etiologies. They present an increasing problem to society and are responsible for more loss of life than any other type of psychological illness. Eating disorders are common, especially in adolescents, and their worldwide prevalence is increasing. Approximately 72 percent of alcoholic women below 30 years of age have an eating disorder. Only 10 percent of men and women with eating disorders obtain treatment and thus many instances of eating disorders remain undiagnosed. Interventions for patients with eating disorders have traditionally been offered on an outpatient or inpatient basis.

However, there are now day hospital programs that offer the possibility of relapse prevention in these patients. Significant progress has also been achieved in the development of psychological treatments for eating disorders; cognitive behavior therapy is currently the treatment of choice for bulimia nervosa and binge-eating disorder, and the use of a specific form of family therapy for adolescents with anorexia nervosa is also useful. Eating disorders, however, cannot be prevented and many obstacles remain in developing the optimum treatment. Effective interventions for the main types of eating disorders and also the other disorders that have not met diagnostic criteria need to be made to reduce the growing trend of eating disorders among many societies.

SEE ALSO: Anorexia Nervosa; Binge Eating; Bulimia Nervosa.

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Dominican Americans

DOMINICAN AMERICANS ARE residents of the United States who have emigrated from or have ancestry from the Dominican Republic. The Dominican Republic is a Spanish-speaking nation of approximately 5.5 million people located on the western half of the island of Hispaniola in the Caribbean Sea; the remainder of the island consists of the nation of Haiti. In the 2000 U.S. Census, about 765,000 individuals identified themselves as being Dominican, constituting about 2.2 percent of the total identifying as Hispanic. Dominican Americans are an ethnically diverse group whose racial ancestry includes the Spanish colonizers of the Caribbean, other Europeans, African slaves, Haitians and migrants from other Caribbean countries (who themselves may be a mix of ethnicities), and people from the Middle East and Asia. Most Dominican Americans are recent migrants to the United States; most arrived after 1960, and immigration greatly increased in the 1980s.

Because they are a relatively small and recently arrived ethnic group in the United States, few studies have been done specifically of Dominican Americans and obesity, and most United States government surveys do not collect information on Dominican Americans as a separate ethnic group. Therefore, information about the health of Dominican Americans must be gleaned primarily from studies of Hispanic Americans (with the proviso that Mexican Americans are the largest Hispanic-American group and thus dominate any statistics on that group), but also from studies of African Americans, other communities of migrants from the Caribbean or Central and South America, and of Dominicans still residing in the Dominican Republic.

Obesity is higher among Hispanic Americans than among Caucasian Americans, among both

children and adults, and rates of obesity and obesity-related diseases such as hypertension and type 2 diabetes also occur at a higher rate. Various explanations have been proffered, including differing dietary choices and rates of physical activity, cultural acceptance of larger body types, poverty, inequality in healthcare due to language barriers or other reasons, and genetic differences.

Despite the importance of ethnically specific studies, it is important to remember that in many ways, health risks for Hispanics are similar to those for the American population as a whole. For instance, overweight and obesity have been rising in the American population as a whole, and leading causes of death for Hispanics are similar for Hispanics as for Caucasians, with the exception that diabetes is a much greater cause of death for Hispanic women where it ranks fourth overall rather than eighth. Hispanics are intermediate among ethnic groups in overall health; they have higher mortality rates than Caucasian Americans or Asian and Pacific Islanders at most or all age groups, and mortality rates than American Indians/Alaska Natives and African Americans.

SEE ALSO: African Americans; Central America and Caribbean; Hispanic Americans; Immigration and Obesity.

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Dopamine

DOPAMINE IS A catecholamine neurotransmitter that is involved in many aspects of food intake, feeding behavior, food motivation, and appetite. Whole forebrain dopamine depletions are known to produce aphagia (i.e., lack of eating). Although this effect has been erroneously attributed to actions on the so-called "reward" system, considerable evidence indicates that dopamine depletions that are restricted to the nucleus accumbens do not generally impair food intake. In fact, the critical dopamine terminal area at which dopamine depletions or dopamine antagonism impairs food intake is the ventrolateral neostriatum or ventral putamen. This region is generally thought to be somotatopically organized and appears to regulate specific aspects of food-intake behavior. Dopmaine depletions in this region impair the motor components of feeding, as demonstrated by impairments in feeding rate (in grams/min), food handling, and oral motor dysfunctions. In contrast to these effects of neostriatal dopamine depletions, accumbens dopamine depletions or dopamine antagonism leave appetite intact but appear to reduce

While all of the effects of dopamine have not been determined, this neurotransmitter is involved in controlling appetite.

behavioral activation and the tendency to work for food; these manipulations reduce the exertion of effort in food-seeking behavior, and alter effort-related choice by shifting organisms toward food sources that do not require much work to obtain.

Dopamine in the hypothalamus is also involved in the regulation of food intake. In this case, dopaminergic involvement appears to be more important for features of appetite regulation. Many well-known appetite suppressants, such as amphetamines, mazindol, and phenylpropanolamine, facilitate dopamine transmission by increasing release, blocking uptake, or both.

Several drugs that are known to be appetite suppressants act as dopamine agonists. The antidepressant buproprion, which blocks dopamine uptake, can suppress appetite, while dopamine antagonists have been shown to increase body weight gain. There is evidence that enhancement of dopamine transmission in perifornical/lateral hypothalamus can act to suppress appetite, and this is at least one of the mechanisms that is thought to be involved in the effects of appetite suppressants that act on dopamine. In addition, there is evidence that alterations in dopamine function are related to aspects of obesity.

Dopamine is a neurotransmitter that is involved in a wide variety of functions related to food intake. Each distinct dopamine terminal area has specific functions related to eating (i.e., appetite regulation, behavioral activation, effort processes, decision making, compulsiveness, motoric functions), and for that reason, dopamine appears to be involved in so many different aspects of normal and pathological features of food consumption.

SEE ALSO: Neurotransmitters.

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Doubly Labeled Water

THE ABILITY TO measure free-living energy expenditure is important given that most precise techniques require an individual to remain in a clinical setting for hours at a time. Therefore, the emergence of a novel technique that is noninvasive and allows a person to go about his or her normal daily activities moved the field of energy metabolism into a new era. This technique, the doubly labeled water method (DLW), is safe and straightforward, but technically complex.

Basically, DLW uses water labeled with two stable isotopes—oxygen-18 (H218O) and deuterium (D2O)—hence, the "double label." These isotopes are safe and mix with the total body water pool just as any other type of water. After a baseline urine or saliva sample is taken, a measured dose of the water, containing a specific amount of each isotope relative to body weight or total fat-free mass (e.g., 0.1 g H218O/kg body weight and 0.08 g D2O/kg body weight) is given to the volunteer. Approximately five hours post dose, the DLW has equilibrated throughout the total body water pool and a urine or saliva sample is collected. Then, on any number of days following the dose, subsequent samples are collected and all samples are analyzed for the ratio of DLW to unlabeled water present in the urine or saliva using a mass spectrometer. A computer program is then used to analyze the rate of loss of each isotope from the total body water pool and the difference in the two rates is used to estimate the CO2 production, allowing for a precise estimate of total energy expenditure to be made.

The DLW method has been found to be accurate to within 3 to 6 percent of total energy expenditure measured using direct calorimetry, the most precise technique available. Thus, not only is it extremely accurate, but it also allows for individuals to carry on with their normal daily activities and does not require them to change their lifestyles in any real way. The fact that free-living energy expenditure is captured improves the accuracy of dietary measures by allowing for a comparison to be made between total energy expenditure and estimated energy intake. Unfortunately, the technical expertise required to analyze the samples as well as the cost of the DLW (upward of $200 per subject) makes the DLW method prohibitively expensive for many investigators in developing countries. Alternatives to the DLW method do exist,

but what is gained in technical ease and cost is balanced by a loss in precision.

The main uses of the DLW are to measure energy expenditure in free-living persons, children or adults. It can also be used to more accurately assess body composition, by providing an estimate of total body water which can then be extrapolated to the amount of fat mass and lean body mass. A novel use of these isotopes is to more accurately assess breast milk intake by delivering a dose of the water to a breast-feeding mother and collecting urine samples from the infant. Finally, DLW has been used to compare food intake measures using the assumption that when a person is in energy balance, his or her energy intake equals his or her energy expenditure, so a measure of energy expenditure using DLW should indicate his or her usual energy intake.

SEE ALSO: Metabolic Rate.

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Down's Syndrome

DOWN'S SYNDROME WAS described by John Langdon Down in 1862. It is a genetic disorder, most commonly the result of an extra chromosome 21, also known as trisomy 21. The incidence of Down's syndrome is approximately 1 in 1,000 live births, and occurs in all races and cultures. The most predictive factor is the age of the mother, and when she is 40, the risk is about 1 in 100 or 10-fold higher than usual. For women above 35, an older spouse above 50, may also enhance the risk. Most fetuses with Down's syndrome are naturally aborted. Down's syndrome can be reliably detected by amniocentesis and genetic chromosomal tests, and in most such cases, the mother will choose to have an abortion. A chromosomal test can also be performed after the infant is born to confirm the syndrome.

Down's syndrome is characterized by short stature, rounded face with flattened features, and reduced muscular coordination. Mild to severe cognitive disability and developmental delays are also characteristic. Heart defects, breathing problems and obstructive sleep apnea, hearing problems, low thyroid hormone levels, as well as celiac disease are common. Psychologically, depression, obsessive-compulsive disorder, and conduct disorder are also more common. Antidepressant medications, such as the selective serotonin reuptake inhibitors (SSRIs), can be helpful in treating depression. DS children have significantly lower resting metabolic rate (RMR) possibly related to the reduced thyroid hormone, but in adults with DS, RMR does not differ. It is possible that the lower RMR in childhood contributes to weight gain later in life. Reduced physical activity in DS may also be a predisposing factor to obesity, which is much more common in women than in men. Food intake, however, does not appear to be greater than expected. Body composition also does not differ, but bone density of the pelvis and spine are lower. Although life span is reduced, it has continued to increase to around 60 currently.

Depending on the degree of mental disability, which varies considerably, children with Down's syndrome can attend mainstream schools, receive specialized education within such schools, or be placed in specialized schools. Early intervention programs that provide intellectual stimulation as well as early walking practice on a treadmill may help Down's syndrome children reach their potential. As adults, some are able to hold jobs and become fairly independent and many reside in adult group homes and still others remain institutionalized. More emphasis should be placed on encouraging healthy lifestyles with nutritious meals and opportunities for regular exercise in an appropriate measured manner to help reduce the likelihood of obesity. National organizations, such as the National Down Syndrome Society, can be valuable resources for information and to help locate local parent-support groups.

SEE ALSO: Depression; Obsessive Compulsive Disorder; Sleep Apnea.

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Drugs and Food

ALTHOUGH THE WAR on drugs has been fought since the 1980s, drug use, abuse, and dependence is still a part of our society. The *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition Revised, outlines eleven classes of drugs with which people experience significant problems. Although many drugs are illegal, legal substances continue to have a significant public health impact. Tobacco use in the United States is still the number one cause of preventable death in the United States. Alcohol is one of the most commonly used mind-altering drugs in American culture, with up to 90 percent of people consuming it at some point in their lifetime. The lifetime risk of alcohol dependence is at approximately 15 percent, with an overall rate of current alcohol dependence on an annual basis of approximately 5 percent. The prolific use of alcohol, tobacco, and other substances in American society has illuminated the indelible link between drug and food consumption, which spans the emotional, social, and biological realms.

Perhaps the most apparent connection between food and drugs is emotional and social. As alcohol is the one of the most commonly used and socially acceptable addictive drug in American culture it provides the clearest examples. Socially, many get-togethers are centered on a good meal, a strong drink, or a combination of the two, as evidenced by the prolific variety of bars and restaurants available. Additionally, both food and alcohol are woven into the culture of celebration and commiseration.

Birthday parties begin with cake as the main event, but in later years, 21st birthday shots take center stage. Similarly, cultural representations of consoling oneself with a pint of ice cream are as well known as crying into one's beer. The special ability of food and alcohol to regulate emotion has been associated with problematic use. People who are motivated to consume alcohol or food to alter or cope with negative emotions are more likely to experience out of control eating and problem drinking.

Advances in scientific methodology have also begun to shed light on the neurobiological links between food and drugs. Imaging studies of the brain have found a similar pattern of brain activation when participants have been presented with either a palatable food or a drug of abuse.

More specifically, food and alcohol both produce part of their behavioral reinforcement by releasing endogenous opioids and dopamine in the limbic system. Depending on how rewarding the food is to the participant, the dopamine system reacts in a similar manner as it does with drugs of abuse, by releasing dopamine in the nucleus accumbens. Additionally, other brain imaging studies in humans have suggested that areas of the brain involved with drug consumption are also involved in food consumption and the neurobiological processes that drive emotional eating.

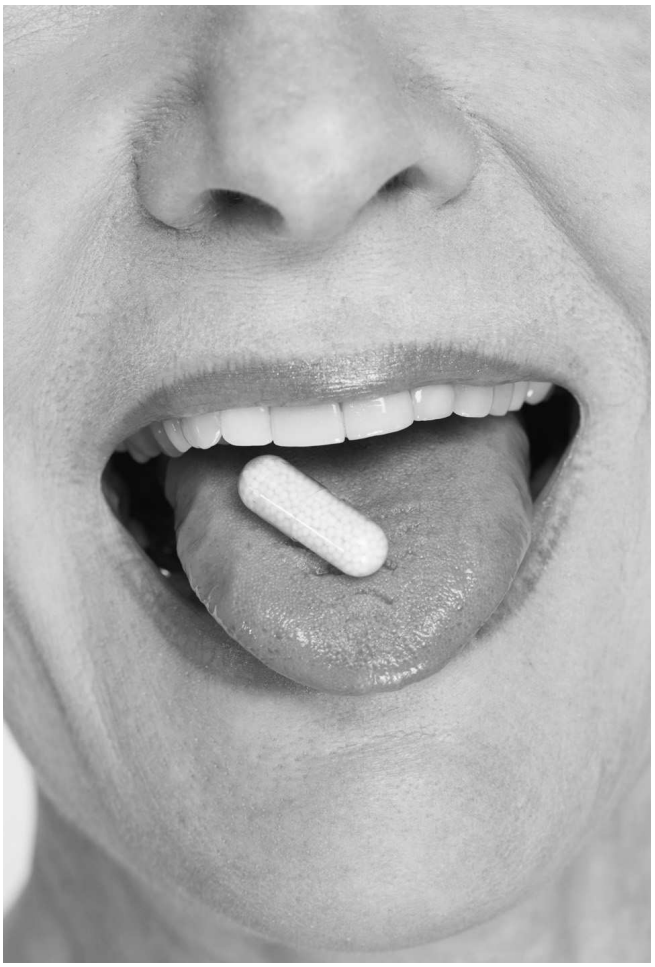
Further, positron emission tomographic imaging studies have shown that obese individuals and individuals who are substance dependent both have significantly lower dopamine D2 receptor levels in the brain, with the lowest D2 receptors in the participants with the highest BMIs. Lower dopamine D2 receptors in obese individuals would make them less sensitive to rewarding experiences, thus they may be vulnerable to excessive food intake as a mechanism to temporarily compensate for this deficiency.

Additionally, pharmacotherapy traditionally used to prevent alcohol relapse by blocking the opiate system, such as naloxone, is also capable of reducing preferences for high sugar foods and inducing opiate withdrawal in animals that demonstrate signs of dependence with sugar consumption. Naloxone is also

capable of reducing the consumption of high fat/high sugar foods in obese and normal weight binge eaters. Similarly, blocking dopamine receptors with medication also reduces the reward value of both sweet foods and drugs of abuse.

The eating patterns of individuals with substance use disorders have also made the link between food and alcohol more apparent. Clinical observations of drug-dependent individuals have suggested that drug use may dampen the desire to eat. Some drug addicts stop consuming food and begin to rapidly drop weight. For example, cocaine addicts, heroine addicts, and alcoholics are at a greater risk for malnourishment, as they focus their energy and attention on consuming their respective substances rather than consuming food. People may begin drug use, especially tobacco and stimulants, specifically for their appetite-suppressing effects. On the other hand, food preferences and consumption patterns also appear to be affected by drug use and dependence. Although food consumption may drop, heroin addicts appear to have an increased preference for sweets. The well-known link between marijuana and the consumption of craving for large amounts of food is known as the "munchies" in popular culture. Alcoholics demonstrate a stronger preference for higher concentrations of sweeteners than non-alcoholics. This increased preference for sweet foods also extends to those with a family member with alcoholism, suggesting a possible genetic link for sweet food and alcohol preferences.

The relationship between food and drugs is also demonstrated in the treatment of drug and alcohol dependence. Clinical observations and empirical evidence suggests that many clients struggling with addiction being to consume more food during periods of abstinence from drugs. The resulting weight gain may provide a major obstacle in helping people stay in recovery, especially with those addicted to cigarettes. This increase in appetite may be due to the reemergence of an appetite that was previously suppressed or may be the result of food consumption replacing drug consumption as a means to achieve pleasurable and soothing feelings. Alcoholics Anonymous (AA) has identified food as an especially important part of recovery, as demonstrated by their relapse prevention mantra, H.A.L.T. AA recommends that recovering addicts avoid being Hungry, Angry, Lonely, or Tired, as these states can lead to an increased risk of relapse.



Clinical observations of drug-dependent individuals have suggested that drug use may dampen the desire to eat.

Not only does Alcoholics Anonymous identify an empty stomach as a risk factor for relapse, but it also suggests that if a recovering addict is tempted to use drugs or alcohol again, they should turn to a sweet food instead. The consumption of a highly palatable food, such as a milkshake, is thought to reduce the urge to use alcohol and thus, helps keep the alcoholic on the road to recovery.

Compulsive eating and compulsive alcohol use also have an increased probability of occurring together. Researchers and clinicians have found increased drug and alcohol abuse among bulimic participants. Similarly, patients in treatment for drug and alcohol use disorders have a higher prevalence rate of compulsive eating behaviors than other clinical patients. There have also been reports of an increased risk for alcohol problems following weight-

loss surgery, such as gastric bypass, that is performed to reduce the amount of food that can be physically consumed. Some patients have reported that following their drastic weight loss they began to consume alcohol compulsively, in the same manner that they previously consumed food. Some have suggested that these patients may be replacing food with alcohol to deal with an underlying emotional problem.

Other research has also pointed to the physiological effects of gastric bypass surgery on alcohol metabolism. Gastric bypass patients have a more rapid absorption of alcohol and experience more inebriation and impairment on less alcohol than someone who has not undergone the procedure. Also, the reduced amount of food consumed by these patients may also contribute to the increased effect of alcohol, as food consumed before drinking reduces the degree of impairment by slowing alcohol metabolism. Interestingly, patients applying for gastric bypass surgery are rarely turned away due to excess alcohol or drug use. Further exploration of this clinical observation has revealed that alcohol consumption is significantly less frequent for obese participants than normal weight or overweight participants. Explanations of this effect have encompassed both the social and biological realms. Obese individuals are at increased risk for weight discrimination, such as taunts and jokes about their size or bullying. Being subjected to this bias can result in low self-esteem and depression. Although negative emotion and depression has been linked to increased substance use, this does not appear to be the case for obese people. Negative social attitudes about obese individuals may prevent them from frequenting places, such as bars and clubs, where alcohol is frequently consumed. Other research has suggested that because alcohol and food both exploit similar areas of the brain, namely the dopamine and opiate system, excess food consumption may occupy biological mechanisms commonly used by alcohol, thus preventing excess alcohol consumption.

There is significant behavioral and biological evidence that food and drugs have a relationship with one another. Not only are food and drugs frequently consumed for similar emotional and social motivations, but they also share similar neurobiological underpinnings. Clinically, problems with food and alcohol consumption are more likely to occur together. Additionally, substance use, abuse, and dependence also alter food consumption patterns and preferences. Evidence also suggests that while obesity may be associated with less alcohol consumption, surgical treatments for obesity may actually increase risk for alcohol use problems. Although many interesting links between food and drugs have been illuminated, future research is still needed to fully understand the relationship between the two. Having a more fully developed understanding of the link between food and drugs may result in better prevention, treatment, and public health efforts for both substance use and eating problems.

SEE ALSO: Alcohol; Disinhibited Eating; Food "Addictions"; Neurotransmitters.

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Drugs that Block Fat Cell Formation

THE ADIPOSE TISSUE is a specialized conjunctive tissue with a predominance of the cell type called adipocyte. Their main function is to store energy as triacylglycerides (TGs). The energy-storing function of adipose tissue is highly efficient due to the low density and high caloric value of TGs. The adipose tissue is also important for temperature isolation, for struc-

tural support of organs, and for a recently discovered endocrine function. Adipose tissue metabolism is very active, involving multiple processes including lipogenesis, lipolysis, and adipogenesis, which are under dynamic equilibrium. Adipogenesis is the differentiation process by which preadipocyte cells mature into adipocytes. Lipolysis is a catabolic pathway, whereby stored TGs are metabolized to yield free fatty acids (FFAs) and glycerol with subsequent oxidation of FFAs for energy production.

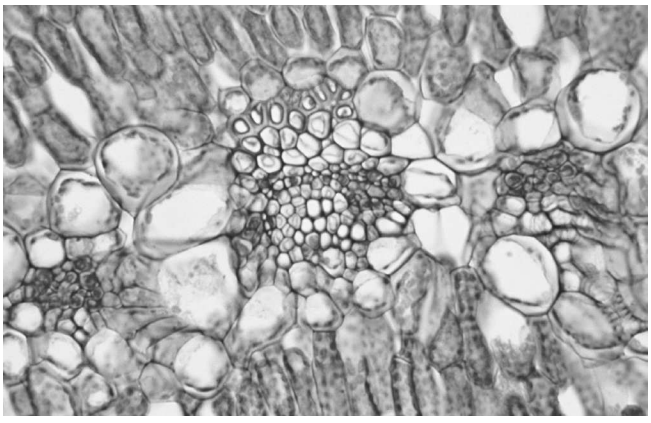
Lipogenesis is the process of storing energy in the form of TGs into the adipocyte. In this case, FFAs are re-esterified to yield TGs. The occurrence of lipogenesis in the adipocytes requires glucose intake, which can be transformed into glycerol phosphate. The necessary FFAs for lipogenesis usually come from the diet. However, they can also be synthesized from glucose incoming into the adipocyte, with the participation of the enzyme fatty acid synthase (FAS) in a process called lipogenesis de novo. Although not a major process, it also takes place in the adipocyte. Lipid metabolism of adipose cells is modulated by the intracellular protein kinases pathway, which is initiated by activation of membrane receptors, inducing internal signaling cascades that ultimately result in the activation or inhibition of catabolic and anabolic pathways.

Because obesity is the storage of excess energy, reduction of body fat can be achieved either by reducing food (energy) intake or by increasing energy expenditure. A new approach for the treatment and prophylaxis of obesity may be based on the inhibition of carbonic anhydrase (CA), enzymes involved in several steps of de novo lipogenesis, both in the mitochondria and the cytosol of cells. Topiramate and zonisamide, clinically used antiepileptic drugs also showing strong CA inhibitory properties, possess a very much desired side effect in obese patients, that is, induction of weight loss. Indeed, it has been recently proven that both topiramate as well as zonisamide act as very potent in vitro inhibitors of several CA isozymes (including CA II, CA VA, and CA VB), and that this might explain their antiobesity effects. Furthermore, it was also demonstrated that inhibition of CA by trifluoromethanesulfonamide (TFM) or acetazolamide can decrease lipogenesis in adipocytes in cell culture. Acetazolamide is a clinically used drug that has been shown to reduce intraocular pressure and is used as an adjunct to other agents in the treatment of glaucoma. This seems to be the beginning of a very new and promising approach for the treatment of obesity, with the hope that more compounds showing this property will be soon developed and available for clinical use.

A number of in vitro studies have also examined the effects of human immunodeficiency virus (HIV) protease inhibitors (PIs) on murine and human adipocyte differentiation. These studies indicate that different PIs have different effects on adipocyte function and differentiation. Adipogenesis of human mesenchymal stem cells (hMSCs) was strongly inhibited by saquinavir and nelfinavir ex vivo.

Administration of the hormone leptin produces weight loss by decreasing appetite, while at the same time increasing the rate of fat metabolism. Leptin activates 5'-AMP-activated protein kinase (AMPK) in muscle, inhibiting acetyl coenzyme A, an enzyme that catalyzes a key step in fat synthesis. As a result, the energy sources that go into fat formation are shifted into an oxidative pathway providing energy for muscle cells. In the liver, leptin turns down the activity of the gene for stearoyl-CoA desaturase-1 (SCD-1), which has a similar role to acetyl CoA. FAS inhibitors such as FAS-89B have been found to stimulate the rate of fatty acid oxidation in peripheral tissues, particularly adipose tissue.

Recently, the use of naturaceuticals in the management of obesity has gained more attention. Conjugated linoleic acid (CLA) is a unique lipid that elicits dramatic reductions in adiposity in several animal models when included at 1 percent of the diet. In vivo and in vitro analyses of physiological modifications



Adipose tissue stores fat; drugs that prevent the formation of fat cells (above) may help prevent some weight gain.

imparted by conjugated linoleic acid on protein and gene expression suggest that conjugated linoleic acid exerts its delipidating effects by increasing expression of genes associated with modulating energy expenditure, apoptosis, fatty acid oxidation, lipolysis and inflammation, as well as decreasing stromal vascular cell differentiation, and lipogenesis. Peroxisome proliferator-activated receptor (PPAR-α) agonists such as oleylethanolamide (OEA), a naturally occurring lipid, induce lipid catabolism through α-oxidation, a well-known pathway for energy expenditure. Myriceline is a natural product that is obtained from the bark, leaves, and flowers of Myrica cerifera, which has been found to reduce lipogenesis and dipogenesis, and promote lipolysis by selective inhibition of tyrosine kinase activity of the β-receptor subunit and other enzymes involved in lipid metabolism pathways of adipocytes.

SEE ALSO: Adipocytes; Leptin.

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Drug Targets that Decrease Food Intake/Appetite

THE REGULATION OF appetite and food intake is a complex process that involves the integration and processing of signals from the gastrointestinal tract, energy stores (such as fat cells and the liver), and areas of the brain. Hormones, neurotransmitters, and other circulating factors mediate these signals; the integration of these signals in the brain leads to sensations of hunger and fullness. There are multiple parallel systems that contribute to controlling food intake and appetite and many of these represent drug targets for weight-loss treatments.

Appetite and food intake are governed by a balance between effects that are classed as orexigenic (pro-hunger signals that induce feeding or food-seeking behavior) and anorexigenic (antihunger signals that suppress feeding). Orexigenic signals also tend to promote the buildup of body weight and energy stores, a term known as anabolism, while anorexigenic signals may do the opposite, a process known as catabolism. A potential drug for weight loss or obesity treatment can, therefore, decrease food intake and promote weight loss by stimulating anorexigenic signals or by blocking orexigenic signals.

Drug targets include compounds designed to antagonize (block) the binding of hormones released by the gastrointestinal tract in response to fasting, to modulate the activity of hormones or neurotransmitters in the brain, or to stimulate the production or activity of signals that promote a sensation of satiety (fullness). These actions may be mediated directly by binding of a hormone or a neurotransmitter to its receptor, or indirectly by modulating the levels of other circulating factors/proteins or nerve signaling that themselves decrease appetite.

Many pharmacotherapies that modulate food intake/appetite are currently under development for weight loss and the treatment of obesity. These include classes of medication that are approved as weight treatments (sibutramine, phentermine, diethylpropion, and in some countries outside the United States, rimonabant). Other potential therapies are at various stages of early and mid-stage development; these drugs may have a defined target on a specific receptor, while others may modulate multiple or as yet poorly defined targets.

SEE ALSO: Future of Medical Treatment of Obesity; Hormones; Humoral Factors and Satiety; Hypothalamus.

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DSM-IV

DIAGNOSTIC AND STATISTICAL Manual of Mental Disorders, Fourth Edition, or DSM-IV, is a manual published by the American Psychiatric Association. DSM-IV categorizes all psychiatric disorders for both children and adults and lists known causes for disorders, epidemiological statistics, prognosis, and treatment approaches. While DSM-IV was created by an American medical society, it is used in many other countries as the standard in mental healthcare and psychiatric diagnosis. In addition, insurance companies, pharmaceutical companies, and health policy makers also use the manual.

The DSM handbook was first published in 1952 and has subsequently gone through five revisions. DSM-IV is considered the last major revision and was published in 1994 with a text revision in 2000 to keep the manual updated with the current literature. DSM-V, the latest incarnation of the handbook, is currently being drafted and due for publication in 2011.

DSM-IV is organized using a multiaxial approach to diagnosis to account for the various ways that mental health can be affected in an individual. As a result, DSM-IV has five levels (axes) that relate to various aspects of disability. Axis I includes clinical disorders, such as major mental disorders, developmental disorders, and learning disorders. Axis II includes underlying pervasive or personality conditions and mental retardation. Axis III incorporates medical conditions that may be relevant to the understanding and treatment of the mental disorder. Axis IV includes psychosocial and environmental factors relating to the disorder. Axis V is a global assessment of functioning measured on a scale of 0 to 100. Common well-known Axis I disorders include depression, bipolar disorder, attention deficit hyperactivity disorder, and schizophrenia. Common Axis II disorders include borderline personality disorder, antisocial personality disorder, and mild mental retardation.

While DSM-IV is widely used in mental healthcare, it also has many limitations. Commonly held criticisms against DSM include the fact that DSM invents illnesses and behaviors using an arbitrary definition for normality. In addition, patients are found to rarely fall into discrete categories of disease according to DSM, further underscoring the criticism of arbitrary definitions of normal. A clear illustration of the arbitrary definition problem can be seen when a well-known fact of the most commonly diagnosed personality disorder is 301.9, Personality Disorder Not Otherwise Specified. Some critics also believe that because treatment is the basis and goal of the psychiatric profession, DSM should be organized according to treatment modalities.

SEE ALSO: Assessment of Obesity and Health Risks; Depression, Disordered Eating; Eating Disorders and Gender; Eating Disorders and Obesity.

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Early Onset Menarche and Obesity in Women

AS A YOUNG woman approaches puberty, the hormones involved in her reproductive system become more active. Two of these hormones, estrogen and progesterone, are responsible for the growth and stabilization of the endometrium, the lining of a woman's uterus. Throughout her life, the levels of these hormones in a woman's bloodstream will rise and fall in a characteristic monthly pattern. When the hormone levels are at their highest, the endometrium is primed to accept a fertilized egg and carry forth a pregnancy. However, when these hormones are at their lowest, the cells of the endometrium will slough off, resulting in menstruation. A woman's first menstrual period is known as menarche. Menarche is one of the final stages in pubertal development and is widely considered a rite of passage into womanhood.

AGE OF ONSET

Menarche typically takes place between the 12th and 13th years of life; one study showed that on average, it occurs at 12.06 years old in African-American girls, 12.25 years old in Mexican-American girls, and 12.55 years old in Caucasian girls. Although menarche is one of the final stages of puberty, researchers often use it as a proxy for determining one's age at puberty; this is because the onset of menarche is far easier for a young woman to recollect than the more subtle pubertal events. Thus, the discussion of early onset menarche is often more accurately a discussion of the early onset of puberty as a whole.

OBESITY AND MENARCHE

The exact relationship between obesity and early onset menarche is unclear. According to the Frisch-Revelle model (developed by two puberty researchers in the 1970s), there is a certain "critical weight" that a young woman must achieve for menarche to take place. This model implies that weight is the driving force of menarche. Other researchers argue a completely reversed direction of causality, citing puberty itself as the cause of weight changes in young women.

PUBERTY AS A CAUSE OF WEIGHT INCREASE

Research indicates that the process of going through puberty causes changes in one's body composition, resulting in increased weight and higher percentage of body fat. Thus, on average, young women who have entered puberty will weigh more than their same-age counterparts who have not yet entered puberty. Research also indicates that early maturing children tend to be more obese in adulthood than their later maturing counterparts. The researchers who argue that puberty drives weight changes point to a mixture of genetics, socioeconomic conditions, nutritional

status, and general health as the driving force behind puberty onset in a young woman. They believe that when the combination of these factors finally results in a young woman entering puberty, it is these pubertal changes that result in her increased weight as compared to her nonpubertal peers.

WEIGHT INCREASE AS A CAUSE OF PUBERTY

There is also a great deal of evidence in support of the Frisch-Revelle model. One such finding is that as obesity in American children has become increasingly more prominent in the past several decades, the age of puberty onset has declined significantly, suggesting that increasing weight may drive earlier puberty. (It must be noted that this association between weight and puberty does not necessarily ensure causation. Indeed, some researchers believe that the increasing use of insecticides, which can break down into estrogen-like compounds, may also explain the earlier onset of puberty.) Studies of body mass index (BMI) and puberty also lend support to the model: one study indicates that those girls who have a higher BMI than their peers at even 3 years of age tend to have earlier onset of puberty (a different study in children aged 5–9 years had similar results). Finally, physiology research also lends support to this model, as adipose (fat) tissue is believed to generate estrogen, which at certain levels helps promote a young woman's progression into puberty. Thus, a greater amount of adipose tissue (which is likely associated with higher weight and BMI) may lead to higher estrogen levels, which may translate into earlier puberty.

CONCLUSION

Despite their different frames of reference, these notions about puberty and obesity are not mutually exclusive. There is strong data to support that obesity is a factor in promoting early puberty and that puberty itself is a promoter of weight gain. In this way, obesity has the potential to have a significant effect on the development of young women, both as they progress toward puberty and as they graduate from it.

SEE ALSO: Estrogen Levels; Hormones.

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Eating Disorders and Athletes

PARTICIPATION IN ATHLETICS may put some individuals at an increased risk for the development of eating disorders such as anorexia nervosa or bulimia nervosa. These disorders are characterized by an overvaluation of weight and shape. The hallmark feature of anorexia nervosa is a refusal to maintain a normal weight, while individuals with bulimia nervosa maintain a normal weight but engage in binge eating and purging. Athletes are faced with unique concerns regarding body shape, weight, and nutrition in relation to athletic performance. However, research investigating whether athletes are at an increased risk for developing eating disorders has yielded mixed results.

While some studies indicate that participation in athletics may serve as a protective factor against disturbed eating attitudes and behaviors, other research has found that athletes endorse more difficulties with eating, dieting, and body image than nonathletes. There is a general consensus that participation in weight-class sports (i.e., wrestling, weightlifting, lightweight football, and tae kwon do), aesthetic sports (i.e., gymnastics, figure skating, diving, and synchronized swimming), and endurance sports (i.e., long-distance running, swimming, and cycling) heightens risk for the development of eating disorders. Weight-class sports encourage unhealthy weight-loss methods for competition, aesthetic sports judge athletes on appearance, and endurance sports generally require a lean physique for optimal performance. However, it is

Because athletes must deal with constant pressures to stay fit and healthy, eating disorders can follow.

difficult to determine whether participation in these sports is fostering disordered eating or if individuals predisposed to develop a disorder gravitate toward certain types of sports.

Beyond the risk associated with participation in specific sports, higher rates of disturbed eating attitudes and behaviors have been observed among other athletic subgroups including dancers, elite athletes, and college athletes. Individuals who are nonelite but high-intensity athletes are also at an increased risk for engaging in eating disorder behaviors. The risk to college athletes may vary as a function of other college-specific factors such as the availability of counseling and academic support, attitudes of coaches and trainers, and pressures to win. The initiation of sport-specific training at a young age also makes individuals vulnerable to the development of eating disorders. Prepubertal athletes may begin a sport that ultimately is not suitable for their adult body type, causing them to engage in pathological weight-loss methods in an effort to attain the necessary build for the sport.

There are a number of theories that seek to explain why these subgroups of athletes may be particularly vulnerable to the development of eating pathology. In general, athletes have additional pressures beyond societal expectations to attain a certain weight or body shape in order to optimize performance. Coaches frequently recommend weight loss to remedy subpar performances, but these recommendations are often made without guidance about proper nutrition and are not necessarily based on fact. Athletes are then left to their own devices, which may lead to experimentation with unhealthy weight-management techniques, such as extreme dieting or purging. The athletic environment can also lead to distorted perceptions of what is normal and healthy.

Coaches and athletes may begin to develop unhealthy expectations of body shape and weight, overlooking emaciated individuals who are performing well. They may be blinded by improved performances that accompany weight loss and view the shedding of pounds as a commitment to the sport. In this way, the athletic environment may "legitimize" both extreme weight loss and excessive exercise, thus masking a problem. A person who excessively exercises can appear disciplined and dedicated rather than "disordered." In addition, because individuals with eating disorders are able to achieve a high level of performance for a long time in spite of energy deprivation, overexercising is a way to validate their behaviors and convince themselves and others their disorder is not a problem. Athletes may also be at high risk for disordered eating because of personality traits commonly shared by individuals with eating disorders, such as perfectionism, drive, conscientiousness, high personal expectations, obsessionality, and self-denial.

Once an individual has developed an eating disorder, athletic involvement may create a barrier to treatment, because athletes often fear that treatment aimed at altering eating disorder characteristics may reduce an athlete's ability and competitiveness. Eventually, though, eating disorders can compromise athletic performance because of physical problems such as fatigue, dehydration, heart problems, or stress

fractures. Athletes with eating disorders confer an elevated risk of physical complications related to the disorders because of the additional stress athletic activity places on their bodies. However, once performance deteriorates, athletes are often more amenable to treatment aimed at recovery and a return to their sport.

While these risk factors may paint a worrisome picture of sports participation, athletics have been shown to provide a number of benefits and potential protective factors against developing eating disorders. Sports can help an individual diversify his or her self-concept and provide a source of pride outside of one's looks. Sports can also help an individual focus on what the body can do, rather than how the body looks. In addition, research indicates that nonelite, nonlean high school athletes do not appear to be at an elevated risk for development of eating disorders and may even have fewer eating problems than high school nonathletes.

SEE ALSO: Disordered Eating; Eating Disorders and Gender; EDNOS; Exercise.

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Eating disorders like anorexia nervosa and bulimia nervosa are far more predominant in women.

Eating Disorders and Gender

RESEARCH ON THE prevalence of eating disorders has identified distinct gender differences among those diagnosed. Specifically, many studies have documented that anorexia nervosa, bulimia nervosa, and, to a lesser extent, binge-eating disorder are more common among women. Anorexia and bulimia are found primarily in women, while men make up a more substantial, though not equal, portion of those diagnosed with binge-eating disorder. While the precise reason for this discrepancy is unknown, many believe that societal pressure on women to be thin and the general value placed on appearance in society play an important role.

A recent survey examining the prevalence of eating disorders in the United States reported that the lifetime prevalence estimate of anorexia is 0.9 percent among women and 0.3 percent among men, of bulimia is 1.5 percent among women and 0.5 percent among men, and of binge-eating disorder is 3.5 percent among women and 2 percent among men. Thus, the lifetime prevalence for any of the three eating disorders, according to this study, was 1¾ to 3 times as high among women compared to men. The prevalence of bulimia and anorexia in men reported by this study is actually high compared to other studies. For example, clinical and case registry studies by Fairburn and Beglin and Hoek and van Hoeken reported that men make up less than 10 percent of those diagnosed with an eating disorder. Population-based studies by Garfinkel et al. reported that less than 15 percent of those diagnosed with anorexia and less than 10 percent of those diagnosed with bulimia are men. Spitzer

and colleagues found that in patient samples, the ratio of women to men with binge-eating disorder was 3:2; however, in community samples, binge-eating disorder was found to be equally prevalent in men and women.

A study conducted by Lewinsohn and colleagues looked at disordered eating behaviors and attitudes in a community sample of young adults. They reported greater eating disorder symptomatology among women compared to men. The biggest gender differences were in body dissatisfaction and drive for thinness. Women were more likely to endorse both of these symptoms as well as to have much higher scores on scales that measure these two traits, both of which were identified as anorexic-related dimensions. Men scored slightly higher on measures of excessive exercise. Lewinsohn et al. also reported roughly equal rates of overeating in the past three months for men and women. With respect to overeating episodes, however, women were more likely to report experiencing a sense of loss of control, feeling bad about these episodes, and considering their eating habits abnormal. In addition, men were both less likely to report wanting treatment and having been treated for their eating disorder. Many believe that there are gender differences with regard to the expression of specific eating disorder symptoms, and these findings encourage future research to look at specific eating disorder symptoms and behaviors as well as subthreshold eating disorders to better understand and identify these gender differences.

According to research findings, women are more likely to develop an eating disorder,and thus being female is a risk factor for eating disorders in general. While the precise reason for this gender difference is unknown, many charge the societal pressure on women to be thin. Hsu suggests that eating disorders are more prevalent among women because more women are dieting in order to lose or control their weight. He bases this contention on research that has shown that the prevalence of eating disorders increases in environments that emphasize thinness and that those who are most concerned with dieting are also more likely to have an eating disorder.

An interesting study by Hepp et al. looked at the relationship between gender role orientation and eating disorder behaviors and found that among women diagnosed with anorexia or bulimia, androgyny tended to be associated with lower levels of eating disorder symptomatology. Thus, according to their findings, higher levels of masculinity and femininity had the strongest protective effect in regards to disordered eating.

SEE ALSO: Anorexia Nervosa; Binge Eating; Bulimia Nervosa; Families of Eating Disorder Patients.

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Eating Disorders and Obesity

EATING DISORDERS AND obesity make up two of the most frequently battled clinical disorders and have morbidity and mortality rates that are currently among the highest of any psychological or health-related conditions. Although eating disorders and obesity are often seen as two different issues, they actually share many connections. Eating disorders, obesity, and other weight-related disorders can overlap as individuals move from unhealthy dieting to obesity. It

is not uncommon for an individual with eating issues to suffer from more than one eating disorder and/or to flip from one to another in varying degrees of severity. An individual can move from anorexia to bulimia to binge-eating disorder. People suffering from binge-eating disorder often are overweight or obese. Research suggests unhealthy dieting rituals as a result of the media's role along with outside influences may be contributing to eating disorders and obesity. People suffering from eating disorders may show signs of body image distortion or body dysmorphic disorder.

BODY IMAGE DISSATISFACTION AND UNHEALTHY DIETING PRACTICES

Body image dissatisfaction and unhealthy dieting practices may be at the helm of the development of eating disorders and obesity. It is suggested through some historical and cultural evidence that social and cultural contexts are contributors to risk for eating disorders as a result of the cultural valuation of thinness internalized in some individuals. In some vulnerable individuals, this thin internalized ideal leads to body weight dissatisfaction, and potentially, the development of full fledge eating disorders. Investigators posit many eating and weight issues start with individuals who are unhappy with their bodies and turn to unhealthy weight-loss strategies such as smoking cigarettes, using nonprescription drugs, skipping meals, fasting, and other dysfunctional rituals.

Body image disorder and body dysmorphic disorder (BDD) may play a role in eating disorders. Body image is defined as an internal view of one's own appearance. Overestimating certain parts of the body such as the waist or thighs when compared to unbiased measurements is often used as a tool to indicate body image disturbance such as dislike, disparaged, or unacceptable. As related to eating disorders, disordered image abnormality is one of the essential diagnostic criteria for anorexia nervosa and bulimia nervosa. There is often an over concern with weight or body shape. Although the criteria for people suffering from binge-eating disorder do not include the criteria of abnormality of body image, symptoms of significant negative body image are present.

BDD is similar to body image disorder; however, it is an *extreme* dislike, disparaged, or unacceptable body image. The individual is obsessively focused on the disliked body feature. This focus interferes with everyday function of life. Certainly, the general population wishes to change some part of their body, but to those suffering from BDD, their body is unspeakably hideous, yet to onlookers, it is considered normal. It is difficult to interact with others or to function normally due to a tremendous fear of ridicule and humiliation as a result of their appearance. Throughout the day every day, both body image disorder and BDD people invest continuous thoughts about their body, compulsive mirror checking or mirror avoidance, while asking others for assurance.

MEDIA INVOLVEMENT

With the influx of eating disorders, it is suggested that mixed messages from Western media plays a role in disordered eating. The media often portrays the "super thin" body type as the preferred one, and some researchers believe that the inability for most to attain this can lead to feelings of inadequacy, which can in turn promote some individuals to binge. The super-slender body shapes that fill magazines, billboards, movie screens, and television screens fail to represent a realistic diversity of body shapes, particularly ones that are not slender.

On the one hand, foods are being pushed continuously on billboards, magazines, television, and the radio. The traditional American diet is rich in saturated fats. Study after study reveals our children are becoming overweight and obese in perilously high numbers. It is believed these alarmingly high numbers are partially due to the rapid and often excessive increase to supersized meals, boundless fast-food restaurants, and less time and fewer options for physical activity. On the other hand, those same media channels are pushing thin, svelte bodies. Thrown into the mix is the idea that we need to be healthy. Consequently, it is not unusual to see overly thin individuals or overly obese individuals, neither group healthy.

Individual vulnerability and general cultural values may be subscribing to an association of thinness with social prestige. In fact, it has been learned a child who has a self-schema as being overweight can project that schema into what might be the future. In other words, a child could harbor the fear of being obese as an adult. Although these fears are only possibilities, they could influence current behavior and the choices we make. In the 30-year span, from the 1980s and 2000s, the fad diets were all about "carb loading." The

idea was to fill up on tremendous amounts of low-fat starches adding small portions of protein. On the flip side, the recent fad has been to load up on protein and fat and stay away from the carbohydrates.

Modern advertising implores females to become slimmer and just more attractive; this is not an easy task given the limits of heredity, physiology, and the cost of cosmetic surgery. It is important to note that the male population is not excluded. The advertising directed at them is to have a muscular body, lots of hair on their head, and exercise equipment to achieve a more attractive body to be accepted by society at large.

This craving to become super thin is not just an American idea. In fact, it is spreading from one end of the globe to another. As a result of unprecedented advances in technology, globalization increasingly allows diverse cultures to interact and converge. Moreover, commerce, immigration, and shared resources, information, and ideas are bringing us closer together, resulting in persons around the globe being roped into these impossible standards.

As a result, extreme dieting often comes into play. Unfortunately, there can be negative associations to extreme dieting. In fact, dieters are food preoccupied, distractible, emotional, binge prone, and unhappy. Studies conclude eating disorders are more inevitable in groups involved in extreme dieting and weight loss. More specifically, a general parallel is present between cultural pressure to be thin and eating disorders, which is widely and commonly occurring both across and within different ethnic groups.

EATING DISORDERS AND BEHAVIOR

Eating disorders are characterized by severe disturbances in eating behavior and come in many different guises. Eating disorders affect five million individuals and poses serious medical and psychological risks. Eating disorders and obesity both contribute to a host of weight-related issues. Some of these problems include disorders such as bulimia nervosa, anorexia nervosa, anorexic and bulimic behaviors, binge-eating disorder, and unhealthy dieting practices. Individuals can experience more than one disorder or swing from one problem to another at different degrees of severity.

The two most widely recognized eating disorders are anorexia nervosa and bulimia nervosa. In addition, many patients present with atypical eating disorders, classified in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV TR) as eating disorder not otherwise specified (EDNO). The most notable atypical eating disorder is binge-eating disorder (BED), which is characterized by recurrent binge eating without the compensatory weight-control methods, such as purging, which defines bulimia nervosa. Binge-eating disorder is considered the most common of all the eating disorders.

A person suffering from bulimia nervosa eats a large amount of food in a small period of time (bingeing) utilizing inappropriate compensatory measures to rid themselves (purging) of the excess intake of food and/or to avoid weight gain. The disproportionate compensation includes throwing up recently eaten food, laxatives, enemas, overexercising, or other medication to aid in ridding the self of food. Often, diuretics are used to rid the self of excess fluids to feel lighter and more completely emptied. A person suffering from this disorder may also pass through bouts of not eating or eating very little.

Anorexia nervosa is an eating disorder of intentional starvation despite hunger. Research suggests this disorder often begins in the young population and involves extreme weight loss: at least 15 percent below the person's normal body weight. It is not uncommon for a person suffering from this disorder to look emaciated and yet be terrified of gaining weight. In fact, food and weight become obsessions and take up a great deal of the individual's energy. Some anorexics have strict exercise routines to keep off the weight. It is not unusual for the female anorexic population to lack monthly menstrual periods. Men who suffer from anorexia often become impotent.

Common medical complications of anorexia nervosa include: cyanosis (purple or bluish coloring on hands and feet due to reduced blood flow), lanugo (a soft downy hair covering the body), dry skin and brittle hair, low blood pressure, cardiac arrhythmias, and loss of menstrual periods. In addition, anorexics can have many hormonal changes due to loss of body fat that revert them to pre-puberty levels, they have gastrointestinal problems (severe constipation, and slowed gastric motility), and reduced bone mass and density, putting them at risk for osteoporosis. Anorexic and bulimic behaviors combined are another eating disorder often referred to as binge

eating/purging anorexia, which is a combination of bulimia and anorexia. People who suffer from this disorder will not only cut the intake of calories but also binge and purge through self-induced vomiting, misuse of laxatives, diuretics, or enemas. In essence, the person has symptoms of both anorexia and bulimia. Research indicates approximately 50 percent of individuals with anorexia also develop bulimia.

Binge eating (sometimes referred to as compulsive overeating) is a common problem among overweight people. This is a distinct, recently recognized eating disorder without inappropriate compensatory behavior and has been proposed for further study in an appendix of the fourth edition of the DSM-IV-TR.

Binge eating involves taking in abnormally large amounts of food in a relatively short period of time, accompanied by a sense of loss of control over eating followed by depression, moodiness, and unhappiness. Often, the person eats alone and is unable to stop eating although full. Feelings of being out of control followed by feelings of guilt, disgust, and/or depression are often accompanied with overeating and bingeing.

Unhealthy dieting practices include restricting calories or eating only certain foods at certain times with the goal of losing weight or staying thin. Unhealthy dieting practices are just that—unhealthy. Meals are not balanced resulting in a lack of nutrients needed by the body to operate at an optimal level. It is believed that unhealthy dieting practices can lead to eating disorders.

Another type of eating disorder is known as disordered eating, which refers to troublesome eating behaviors such as restrictive dieting, bingeing, or purging. The difference in disordered eating and eating disorders is in the severity. Disordered eating occurs less frequently or is less severe than those required to meet the full criteria for the diagnosis of an eating disorder.

WORLDWIDE EPIDEMIC

Obesity is not only a serious and increasingly prevalent condition in the United States, but it is now a worldwide epidemic and has recently been declared one of the major concerns for global health by the World Health Organization (WHO). The WHO estimates that there are nearly 180 million obese adults, and most likely, in addition to that number, there are at least twice as many adults who are overweight. Currently, evidence suggests that 65 percent of adult Americans are overweight and 31 percent are obese and this number is growing at an alarming rate. In fact, it is suggested that 60 to 70 million people weigh above the statistical average.

Obesity is associated with, or a precursor state to, a plethora of serious diseases, including Type 2 diabetes mellitus, cardiovascular disease, stroke, and certain carcinomas. Although the percentage of children and adolescents suffering from obesity is not as high as adults, the current movement of obesity in our young population is staggering. It is reported that 13 percent of children aged 6–11 and 14 percent of adolescents are obese and larger numbers are overweight.

It is questioned whether media involvement may be leading to body dissatisfaction and feeding into the eating disorders. This year, more than 60,000 Americans will struggle with morbid obesity (100 lb or more overweight), not to mention those who are simply obese. In fact, more than one billion people worldwide, including 22 million children younger than five years of age, are now overweight or obese. This fact is continuously increasing. The likelihood that obesity present during childhood will persist into adulthood rises with the age of the child, independent of the effect of parental obesity. In fact, several studies have indicated that approximately 70 percent of overweight adolescents become obese adults.

EATING DISORDERS AND OBESITY CONNECTIONS

Unfortunately, the individual's self-worth is overly determined by body weight, shape, and appearance. It is believed that the person suffering from eating disorders overvalue their weight and shape to compensate for low self-esteem, which involves the individual's attempt to self-regulate his or her emotional world and manage his or her stressful interpersonal relationships with food. There are different factors that can predispose, precipitate, and perpetuate the individual's eating disorder.

Contributing factors to eating disorders focus on interpersonal difficulties, which include role disputes, role transitions, interpersonal deficits, and unresolved grief. Interestingly, an individual's eating disorder is placed within this interpersonal framework (e.g., a specific role dispute as a trigger for binge eating). Research strongly implies that certain environmental and personality variables lay the foundation for its de-

velopment years before the disturbed eating becomes apparent, regardless of whether an eating disorder has an early or a later onset. In fact, eating disorders are caused by factors beyond a simple preoccupation with food and weight. It is believed an eating disorder is caused by a complex interplay of sociocultural, environmental, and individual factors.

A person with an eating disorder will attempt to self-regulate the emotional world they live in as well as attempt to manage stressful interpersonal situations with food. The self-worth of the individual with an eating disorder is overly determined by body weight, shape, and appearance. Extreme weight fluctuations, low self-esteem, cognitive distortions such as dichotomous thinking, and increased thoughts about food and eating, increased emotional liability, an increased vulnerability to social and environmental influences are part of the psychological makeup in the eating disordered. Dysfunctions in interpersonal functioning and self-worth are two psychological theories that are ever present in eating disorders.

Although each disorder is distinct on a number of different dimensions, they all harbor issues of self-worth and interpersonal functioning. It is not uncommon for an individual in any of the eating disorders to have aloofness in his or her mannerism, along with a tendency to push people away. The media's emphasis on appearance, the central role of beauty in femininity, physical changes in a lifespan, and personality traits such as anxiety avoidance and perfectionism have all been impressed in the development of eating disorders. There is a fear of relationships and the inability to handle feelings, especially when social interaction is difficult, leaving a constant self-conscious, insecure feeling with undertones of depression.

CONCLUSION

Perhaps the answer for those suffering from eating disorders and obesity is in sound therapeutic relationships, self-monitoring, education on weight and eating, and establishing healthy eating patterns. The goal is working toward eliminating dieting, teaching problem-solving skills, cognitive restructuring, and working through strategies for addressing shape and weight concerns. There is no doubt interest in eating disorders and obesity is on the rise with clinicians and researchers. The connections between eating disorders and obesity often cross paths as they share many connections. Eating disorders, obesity, along with other weight-related disorders can overlap as individuals move from unhealthy dieting to obesity.

SEE ALSO: Anorexia Nervosa; Binge Eating; Body Dysmorphic Disorder; Body Image Disorders; Bulimia Nervosa.

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Eating Disorders in Schoolchildren

EATING DISORDERS ARE observed with increasing frequency in children and adolescents. Although food restriction is the most commonly reported symptom, children with eating disorders may have varying presentations and may meet only partial criteria for anorexia nervosa or bulimia nervosa.

GENERAL DIAGNOSTIC CRITERIA

The *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (DSM-IV), published by the American Psychiatric Association, establishes criteria used in diagnosing and distinguishing eating disorders.

Anorexia nervosa is diagnosed according to three diagnostic criteria: a refusal to maintain body weight within a normal range for the person's age and height, an intense fear of gaining weight, and a severe disturbance of body image in which body image becomes responsible for self-worth accompanied by a denial of the gravity of the illness. The illness may take the form of one of two subtypes: restricting (ANR), or bingeing/purging (ANBP). Individuals with ANR use constraint in their eating to reduce their weight and are, in general, more perfectionistic in personality and

Formerly seen only in young and older adults, eating disorders are becoming far more prevalent in younger children.

restrictive in their eating behaviors. In contrast, individuals with ANBP may binge and use purging methods (i.e., vomiting, laxatives) to control their weight.

The DSM-IV criteria for bulimia nervosa include recurrent episodes of binge eating accompanied by a feeling of a loss of control (binge eating constitutes a consumption of larger-than-normal quantities of food in a discrete period); recurrent compensatory measures to avoid gaining weight postbinge that either involve purging (e.g., self-induced vomiting) or nonpurging activities (e.g., excessive exercise or fasting); the bingeing and purging behaviors occur a minimum of two times a week for a duration of 3 months; self-evaluation is disproportionately influenced by body shape and weight. Also, these disturbances do not occur during episodes of anorexia nervosa.

Also included in the DSM-IV is the category Eating Disorder Not Otherwise Specified (EDNOS), which includes all clinically significant eating behaviors that do not fit into the other categories.

EATING DISORDERS IN CHILDREN AND ADOLESCENTS: UNIQUE FEATURES

Diagnosing eating disorders in children and adolescents poses a difficult challenge as children and adolescents tend to present atypically and carry unusual features with their illness. Consequently, children with eating disorders may pass undetected by medical providers, as they may not meet the full criteria for anorexia nervosa or bulimia nervosa. For example, a child who has not yet reached menarche may "miss" the anorexia nervosa criterion of absent periods.

Research on eating disorders in children and young adolescents suggests that they are most likely to be given a diagnosis of EDNOS as these younger patients are less likely to engage in typical binge/purge behavior and are likely to meet only partial criteria for anorexia nervosa or bulimia nervosa. Restriction of food is identified as a common presenting feature in children and adolescents. Furthermore, younger patients with eating disorders are more likely to be male and to have a high rate of comorbid psychiatric illness such as obsessive-compulsive disorder. Additionally, younger patients are less likely to admit to the use of diet pills or laxatives as methods of weight control. Of particular concern is the finding that younger patients experience a more rapid rate of weight loss than older patients, thereby increasing the risk of bone loss and abnormal growth and development. Other possible medical complications in younger patients include damage to reproductive organs and decreased brain cortical mass.

There is a concerted effort to uncover etiological factors that contribute to the onset of eating disorders in children and adolescents. Environmental factors such as family dynamics or dysfunction have been implicated, as well as biological factors such as the onset of puberty. A history of sexual abuse or other traumatic event has been associated with binge/purge behaviors. Other risk factors currently being researched include the effect of Western culture, female gender, dieting history, body image, media exposure, and other sociocultural factors.

TREATMENT OPTIONS AND OUTCOMES

Early intervention in children and adolescents with eating disorders is of paramount importance as medical complications can occur even before significant weight loss is remarked. Early treatment may prevent

irreversible effects of the eating disorder that may damage normal growth and development.

Treatment of eating disorders in children places unique demands on caregivers due to children's tendencies to meet treatment with severe resistance and behavioral tantrums. In general, a multidisciplinary treatment approach is recommended. A treatment team may be comprised of a medical provider, a mental health provider, a therapist, and a registered dietician, preferably one who specializes in eating disorders. Weight management, restoration, and nutritional guidance are fundamental to successful treatment. Currently, there is no medical approved by the Food and Drug Administration for treatment of eating disorders in children. However, selective serotonin reuptake inhibitors, antihistamines, and atypical antipsychotics such as olanzapine are useful for managing concomitant symptoms relating to anxiety, insomnia, or depression. It is important for the prescribing medical provider to consider the differences in pharmacotherapy for children and adolescents; children and adolescents may require more of the drug per unit of body weight due to a faster metabolism and clearance.

As the etiology of eating disorders in general is considered multifactorial in nature, so are the variations in outcomes studies. One study with children and adolescents cited a full recovery achieved in 75 percent of patients and partial recovery in 86 percent after follow-up of 10 to 15 years. Mortality from eating disorders was estimated to be between 0 and 18 percent. Poor outcomes are associated with an early age of onset, comorbid obsessive-compulsive disorder, high serum creatine levels, and treatment dropout for anorexia nervosa and higher rates of bingeing and purging and co-occurring substance abuse for bulimia nervosa. Having experienced a negative life event is associated with a good outcome in adolescents with anorexia nervosa, while higher social class, younger age of onset, and a shorter duration of the eating disorder are associated with a good prognosis in patients with bulimia nervosa.

SEE ALSO: Anorexia Nervosa; Body Dysmorphic Disorder; Bulimia Nervosa; Eating Disorders and Obesity.

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Eating Out in the United States

ACCORDING TO THE National Restaurant Association (NRA), people in the United States ate out for around fifty-four billion meals in the year 1998. That year, each person spent just over eight hundred dollars on eating out. There were approximately two hundred seventy million people in the United Sates; therefore, the annual revenue in 1998 was about two hundred sixteen billion dollars. As of the year 2007, approximately four hundred thousand restaurants are in operation in the United States. Combined, these restaurants make revenue of nearly two hundred forty billion dollars annually. One estimate of the revenue for 2007 doubles those numbers—both for restaurants and revenue.

Around the turn of the 21st century, consumers began showing a preference for healthier food options. Restaurants began regularly offering multiple portion sizes as well as more natural ingredients. Health-conscious food chains sprang up serving juices (Jamba Juice, for example), nutritious smoothies (such as Smoothie King), etc. Subway restaurants began an intensive marketing campaign featuring Jared, a formerly obese person, who lost 235 pounds after a year-long diet eating only food from Subway. The problem for the consumer became filtering through the healthy-sounding names and advertising campaigns and finding the truly healthy meal options.

It is difficult to maintain a health diet when eating out. Many foods come packed with sugars, fat, and calories. The NRA offers an eating out guide on its website that allows consumers to search for restaurants that feature healthy menu items. Search criteria include zip code, price range, and additional options

Larger portion sizes and a lack of nutritional information in restaurants have added to the obesity problem.

such as take-out, delivery, or catering. In order for a restaurant to be featured on this website, the restaurant must provide nutritional information for all its menu options. Any deep-fried foods will not be considered unless they are a small garnish, such as a wonton strip on a salad. To be considered, a full meal must include either fruits and/or vegetables, lean protein (and two or fewer red meat dishes per restaurant), or one hundred percent whole grains (a whole grain includes all three parts of the grain: the outer bran, the inner germ, and the endosperm which is the starchy product in refined grains).

Additionally, full meals and appetizers are on a strict allowance of calories, fat grams, and saturated fat grams. Full meals are allowed 750 calories (37 percent of the United States Food and Drug Administration [FDA] recommended 2,000 calories), 25 fat grams (the FDA recommends 44–78 grams per day), and eight saturated grams (the FDA recommends 22 grams of saturated fat per day); appetizers and side dishes can have 250 calories, eight grams of fat, and three grams of saturated fat. If a menu item fulfills two of these three criteria, and overshoots the third by less than 10 percent, it can still be featured.

It is important to note, however, that these meals often do not mean the FDA definition of "healthy," which also includes sodium and cholesterol. Additionally, other nutritional information such as fiber or sugar is not considered. It is important for the consumer to choose a meal based on its entire nutritional content, not just the calories and fat grams. The NRA highlights meals that are low in sodium and cholesterol, but does not include these values in its selection criteria. Restaurants are included in this listing if they submit information; therefore, smaller independent restaurants may not know of the listing and will subsequently be unmentioned. Most of the search results turn out to be fast food or restaurant chains; therefore, the site could be useful for travelers looking for familiar meal options.

Many nutritional resources offer tips for staying health-conscious when eating out. These tips are often similar and typically include requesting oil-based dressings over cream- or cheese-based dressing, and always requesting the dressing or sauce on the side. Additionally, avoiding deep-fried foods as well as all-you-can-eat buffets are good choices. At a location that offers larger-than-necessary portions, consumers can eat only half the meal and take the second half home. It may be easier to do so if the consumer requests a take-home box and immediately removes half the portion from sight. Finally, a consumer should select a dessert that includes fresh fruit rather than one that has a high content of corn syrup.

High fructose corn syrup entered the fast-food market in the 1970s; since then, its consumption has spiked, and so has the rate of obesity. Studies have not shown a definite link, however. Some scientists theorize that fructose and specifically high fructose corn syrup is not as satiating as glucose. Glucose levels in the body, after a meal, are used by the body to monitor secretion of other peptides and hormones that control our feelings of hunger. One theory argues that if two meals contained either a certain amount of glucose or the same amount of fructose, the glucose-containing meal would make a consumer feel full before the fructose-containing meal. Therefore, meals rich in high fructose corn syrup might circumvent our satiation feelings and keep us in a hungry state well after we have consumed enough food. Many items at fast food restaurants contain a large proportion of high fructose corn syrup.

Another dimension to eating out is that large restaurants often buy food in bulk. Many foods have added sugars, salts, and preservatives. Coffee shops, which often sell food items high in sugar and fat, number about 20,000 in the United States; coffee shop combine revenue is about eleven billion dollars

annually. Additional sources for food when eating out include doughnut shops, gas stations and other convenience stores, grocery stores which offer pre-made items, and school or work cafeterias.

SEE ALSO: Access to Nutritious Foods; Accessibility of Foods; Appetite Signals; Department of Agriculture; Economics of Food; Fast Food; Food and Drug Administration; Hunger; Income Level; Increasing Portion Sizes; School Lunch Programs; Supersizing; Taxation of "Unhealthy" Foods; Western Diet.

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Economic Disparities among Obesity in Women

OBESITY AND THE sex of an individual have a strong relationship with economic status. While the prevalence of obesity has increased in the United States and around the world, it is well known that obesity in affluent societies is more common in lower socioeconomic groups and in women. In fact, the disparity that currently exists based on socioeconomic status and sex continues to grow and may indicate a relationship whereby obesity affects social and economic status, and social and economic status affects likelihood of obesity.

Much of the relationship between socioeconomic status, women, and obesity can be explained by the fact that low socioeconomic status and economic constraints may lead to decreased access to healthy foods and proper exercise. In addition, low income, unemployment, and social isolation may also lead to variations in weight. Obesity, which is viewed poorly by affluent societies in a social context, may also lead to lower salaries and disadvantages for social and economic advancement. Women are both more likely to be obese, and more likely to be in a lower socioeconomic group, compared to men. For this reason, obese women are often economically worse off compared to the rest of society. Current studies demonstrate that women being overweight was associated with unemployment, and obesity with long-term unemployment and absence of a strong social network. In addition, obese women were more likely to have low individual earnings, low household disposable income, and low individual incomes.

While obese women in general face growing economic disparities in the United States, obese women who are from certain minority groups face even

Lack of affordable, nutritious food has cause obesity to become more prevalent in lower economic classes.

more of a divide. In fact, African-American, American Indian, and Hispanic-American women have the highest risk of becoming overweight. As an example, a recent study demonstrated that 37 percent of African-American women are obese (BMI >25), 33 percent of Mexican-American women are obese and this is in comparison to the 24 percent of Caucasian women who are obese. Asian Americans are the only minority group that has lower rates of obesity than the general population.

Currently, a large debate concerning obesity and economic disparities among women is related to the concept of whether genetics or socioeconomic factors play a larger role in causing obesity. Although it was found in heritability studies that 70 percent of body weight can be tied to genetics, proponents of an environmental socioeconomic cause of obesity say that genetics alone cannot account for the 50 percent increase in the number of Americans who are currently obese in the past 20 years. These same proponents argue that social class and economic ability can shape the environment to promote weight gain, while genetics often predispose certain individuals to respond to this environment by eventually developing obesity.

SEE ALSO: Economics of Food; Economics of Obesity; Ethnic Disparities among Obesity in Women; Health Disparities—NIH Strategic Plan; Obesity and Socioeconomic Status; Prevalence of Obesity in U.S. Women.

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Economics of Food

THE ECONOMICS OF food is a broad, complex topic involving many issues such as commodities, food markets and prices, international trade, security, consumption, assistance programs, safety, biotechnology, labeling, and organic agriculture. U.S. and global economic polices concerning agriculture, trade, investment, and marketing of food affect what the world eats and therefore can be regarded as global health policies. Research has shown that the economics of food can be directly implicated as one of the causes of obesity. Since the mid-1980s, the incidence of obesity in the United States has been rising as the cost of food has dropped below the rate of inflation. A better understanding of the economics of food may therefore provide insight into economic causes of obesity.

COMMODITIES, FOOD MARKETS AND PRICING

The United States produces many agricultural commodities for domestic consumption and export. According to the United Nations Food and Agriculture Organization, the United States is the number one producer of several globally important crops including corn, soybeans, chicken, pork, beef, turkey, and milk. Their top ten highest grossing commodities (in order by dollar value) are: corn, beef, milk, chicken, soybeans, pork, wheat, eggs, tomatoes, and grapes. Corn and soybeans make up a large portion of the American agriculture and food sector. Soybeans are used to make cattle feed, vegetable oils, tofu, and protein isolates. Corn is even more versatile. Aside from being used as cattle feed, vegetable oil, and a major ingredient in many food products, there is a rapidly expanding industry converting corn into ethanol for fuel. The United States is also the number one producer of high fructose corn syrup, which now replaces sugar in many convenience foods like soft drinks and confections.

The U.S. food marketing system is an important part of the economy, accounting for 12.3 percent of the U.S. gross domestic product (GDP) in 2001. It has five main stages: production, processing and manufacturing, wholesaling, retailing, and consumption. Farmers produce food crops and sell them to manufacturers or processors. The processed or packaged products are then sold to wholesalers that in turn sell them to retailers. Consumers buy products from retail food stores (e.g., grocery stores or super centers), or food service distributors (e.g., restaurants). An increasing amount of what consumers pay for food goes towards advertising, transportation, packaging, distribution, and labor, rather than going to the farm

sector. Food technology has made food production increasingly efficient in the United States. Food industry income growth outpaces any increases in food expenditures, resulting in a reduction of the share of income Americans spend on food. In 2001, consumers spent 10 percent of their income on food, compared to 18 percent in 1960. Recent consolidation in the retail food sector and food service sector has also helped keep food prices low. These large companies frequently have contracts directly with the farm sector, instead of buying through wholesalers, making the whole process even more efficient.

The consumer price index (CPI) is a statistical estimate of the price of goods and services consumed by U.S. households. The CPI for food measures the amount of money U.S. households spend on food and is broken down into several categories including food bought away from home, food bought for the home, alcohol purchases, etc. The change in CPI for food over time can be compared to the overall rate of inflation as a way to determine if food is getting relatively more or less expensive. For example, from 1960 through 1980, average food prices rose slightly faster than the overall inflation rate (5.5 percent versus 5.3 percent per year). However, between 1985 and 2000, when obesity rates more than doubled, food prices rose 3.4 percent per year from 1980 to 2000, which is slower than the 3.8 percent average rise in the inflation rate over the same period. So in actuality, relative food prices during this period fell 14 percent. Retail food price inflation has accelerated in 2007; the CPI for all food products is predicted to increase from 3.5 percent to 4.5 percent due to increased commodity and energy costs, partly in response to the expanding corn ethanol industry.

GLOBAL FOOD MARKETS AND INTERNATIONAL TRADE

As incomes have risen in many countries around the world, the demand for food has shifted from staples like rice and wheat to processed, value-added products like meat, dairy, pasta, and processed vegetables. There are four main categories of foods traded on the global market: traditional bulk commodities (e.g., rice, wheat, soy, and corn), horticultural products (e.g. fresh fruits and vegetables), semi-processed foods (e.g. flour, cooking oils), and processed food products (e.g. pasta, prepared meats). Horticulture, semi-processed, and processed foods are all considered high-value products because they are either ready to eat or very perishable. In 1991, U.S. exports of high-value products surpassed exports of bulk commodities. Imports of high-value products are also on the rise, giving greater food choices to Americans and adding competition for U.S. food processors.

Since 2002, the United States has imported more processed foods than it exports. About 25 percent of all imports are non-competitive food crops such as tropical fruits that cannot be grown in the United States. As a way to offset this processed food trade deficit, American food manufacturers are building manufacturing plants abroad. This reduces transportation costs, gives better insight to local markets, and eliminates costly tariffs. Foreign food companies are also investing in U.S. retail stores and manufacturing facilities. While sales by foreign food companies in the United States is about equal to sales by U.S. companies abroad (roughly $200 billion in 2001), foreign retail companies in the United States have more than 10 times greater sales than US retailers abroad. The difference is largely made up by U.S. manufacturing abroad.

International food trade faces many financial barriers, but none as universal as import tariffs. Agricultural tariffs are generally higher than other import tariffs, and vary greatly by import and importing country. Countries that are members of the World Trade Organization (WTO) that participated in the Uruguay Round Agreement on Agriculture negotiate to reduce import tariffs, set sanitary standards, and set special safeguards to regulate the flow of agricultural commodities. Antidumping laws (AD) and countervailing duties (CVD) are set up to ensure foreign companies cannot flood the international market with their goods (AD) or unfairly subsidize their exports (CVD).

FOOD SECURITY

Food security is the ability of all people to have access to food at all times in order to maintain an active and healthy life. There are three conditions that must be fulfilled in order to maintain food security. First, there must be sufficient food production to ensure that food is available for people to lead healthy, active lives. On a national level, this means there must be sufficient domestic food production and imports

to meet a country's food demands. Second, people must be able to access food. This is determined by household income and geographic proximity to food sources. Finally, people must be able to use the food that is available. They must have access to clean drinking water, safe food, and basic health care in order to make best use of the available food.

In wealthy countries like the United States, food production is more than adequate to make food available to the entire population. However, low-income families have limited access to and limited utilization of food.

Worldwide, over 1 billion people are hungry. Global food production is not the issue, since the growth in production has out-paced population growth in recent years. Even though there is more food available per person, access and utilization are the key issues. Some countries are food insecure on a national level, but countries can have national food security with uneven food distribution.

Many starving people live in rural areas where food is produced, but do not have enough income to purchase and utilize food. The United States provides food aid to food insecure countries in the form of commodities or cash. They maintain a food security commodity reserve by purchasing grains when the price is low and putting them into storage until needed for a food donation. From 1991 to 2001, the effectiveness of U.S. food aid was 66 percent, meaning 34 percent either went to countries that were not in need or the food was not properly distributed to hungry people.

FOOD CONSUMPTION

The types and amounts of foods that people choose to eat have an influence on global markets and society in general. Food choices are highly individualized and vary depending on income, family structure, time constraints, etc. If Americans followed the USDA's Food Guide Pyramid dietary recommendations, consumer demand would change U.S. agricultural production, trade, and food prices.

To do this, Americans would have to change their diet considerably. Although per capita consumption of all fruits and vegetables (fresh and processed) has risen slowly since 1975, there is a large gap between recommended consumption and actual consumption. To meet these dietary changes, fruit production and imports would have to double. Production of dark-green leafy and deep-yellow vegetables, dry beans, peas, and lentils would have to increase by four times as the production of starchy vegetable crops is decreased. In addition, production of sweeteners, fats, and oils would be considerably reduced, since Americans currently over consume these products. A 60 percent reduction in sweetener consumption would be necessary to meet pyramid recommendations, reducing production of sugar beets, sugar cane, and corn for HFCS.

Fat intake in the United States is among the highest in the world. A 36 percent reduction would be necessary to meet pyramid recommendations, which would have serious impacts on the soybean industry since soybeans currently dominate the added fat and oil market. Consumption of dairy would have to be increased by 22 percent and meats by 5 percent to meet pyramid guidelines, however this increase would have to be low-fat choices such as skim milk, chicken, and fish. American grain consumption is on par with pyramid recommendations, but there would still be a large shift in the grain industry due to an increased demand for dairy cattle feed and decreased demand for oil and HFCS uses.

GOVERNMENT FOOD ASSISTANCE PROGRAMS

Many food assistance programs are offered by the U.S. government to ensure those in poverty to not suffer from starvation. These programs include the Food Stamp Program, National School Lunch Program, School Breakfast Program, the Child and Adult Care Food Program, and the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) Program. Food stamps account for 62 percent of federal spending on food assistance. They are available monthly to low-income households. The National School Lunch Program provides nutritious, low-cost or free lunches to students from low-income families. The School Breakfast Program is essentially the same, except low-cost or free breakfast is provided instead of lunch.

The Child and Adult Care Program provides food subsidies for participating day-care or adult-care centers. The WIC program assists low-income pregnant, breast-feeding, or postpartum women and children up to 5 years old who are at nutritional risk. Although the original intention of food assistance programs

was to keep people from starving, there have been unintentional consequences. A recent USDA report found that long term participation in the Food Stamp Program increased the incidence of obesity. Another report found that both WIC program participants and non-participants at the same income level have similar nutritional intakes. Since WIC is intended to help people at nutritional risk, these findings call into question the level of nutritional risk that actually exists in the United States.

Another type of government assistance for food is agricultural subsidies to farmers. An agricultural subsidy is monetary assistance granted by the government to farmers to supplement their income and manage the supply and influence the cost of agricultural commodities on international markets. The USDA is required by U.S. farm bills to subsidize the production of over two dozen commodities. In 2004, the most subsidized crops in the United States were grains, cotton, wheat, rice, soybeans and soy products, dairy, peanuts, sugar, minor oilseeds, tobacco, wool and mohair, vegetable oil products, and honey. Subsidies give farmers extra money for the crops they grow but also set a price floor to protect them against financial loss when there is an oversupply of a certain commodity and the price drops below what it costs to produce the crop.

As agriculture in the United States has shifted from small family farms to large corporate farms, so have the beneficiaries of farm subsidies. According to the USDA, real farm income from 1929 to 2004 has remained stagnant mainly from low crop prices and high operating costs. Even though there is much debate about whether farm subsidies are the cause or effect of low farm prices, there is wide agreement that US farm policy contributes to the lowering of agricultural prices worldwide.

SEE ALSO: Economics of Obesity; Food Guide Pyramid; Food Labeling; Food Technology.

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Economics of Obesity

SINCE THE 1970S, obesity in the United States has dramatically increased, with nearly two out of every three adult Americans currently classified as obese or overweight. The prevalence of overweight children has increased from 5 percent in 1970 to 15 percent in 2000.

The mechanisms driving this trend are unclear, multivariate, and controversial. At a basic level, however, the trend is a product of decisions made by the individual over the course of a lifetime. Economists study these decisions made by individuals under constraints of cost, time, income, and other resources. Because weight gain can be viewed as a product of such decisions, economic analysis can provide unique insights as such constrained choices are decomposed and analyzed.

At the same time, obesity has health consequences and, in turn, economic sequelae that should be understood and considered. Only through understanding the causes and effects of obesity can we effectively hope to design appropriate interventions to stem the growth in obesity.

DRIVING FACTORS

While popular media might place blame on a multitude of factors such as the fast-food industry, economists have attempted to build basic models to understand the weight trends. In simple terms, weight gain is the result of an increase in calories ingested over calories expended. An increase in calories can result from greater food intake at each meal, more meals of equal caloric intake, or changes in food preparation to include more calories.

However, a decrease in calorie expenditure can be the product of a more sedentary lifestyle through a

decrease in energy expenditure at work or even in leisure activities. The challenge lies not in just documenting the changes in the above in relation to the changes in weight, but to pin down the causal link between these myriad factors in the observed trend in increased obesity. Despite the paucity of necessary data tracking the daily caloric intake and expenditure values for large groups of individuals over long periods of time, economists have used clever models with limited data to uncover some of these underlying relationships.

ROLE OF TECHNOLOGY

Technological change has impacted both the supply and demand for obesity. On the supply side, technology has made the agricultural production process more efficient, such that the price per calorie has become much cheaper, increasing the demand for calories. On the demand side, technology has made the time spent at work more sedentary. For example, because work was more strenuous in more agrarian and early industrial societies, laborers were essentially paid to exercise. However, post-Industrial Revolution, the nature of work itself has changed; individuals are essentially paid in terms of foregone leisure. In essence, individuals are paid to substitute out-of-work leisure exercise for exercise typically undertaken at work. Empirical estimates by RAND economist Darius Lakdawalla and University of Chicago economist Tomas Philipson show that 40 percent of the recent increase in weight is due to the lower food prices from agricultural innovation, while 60 percent is a product of declining physical activity due to technological improvements in home and market production.

While this model does explain the long-run changes in obesity trends, it falls short of explaining the large increase in obesity in the United States starting in the 1980s, a time during which commensurate changes in technology do not exist. Therefore, Harvard economists David Cutler, Edward Glaeser, and Jesse Shapiro study the role of technology in increasing caloric intake, as it has decreased the time costs of food preparation. Economists use a broad definition of price that includes the monetary amount paid in addition to the time needed to use the product. In 1965, nonworking woman spent two hours preparing a meal and cleaning thereafter, while 30 years later, the same tasks require less than an hour. This causes a fall in price paid in terms of time, thereby, increasing both the quantity and variety of food prepared and consumed. Cutler, et al., observe that the increased calories were primarily attributable to higher consumption of snacks, driven by technology improvements in the making of prepared foods. They also find that technology lowered the fixed and variable costs of meal preparation and led to greater variety and frequency of meals. This was especially true for women because they experienced the largest savings of time and energy; and therefore as expected, women showed the most rapid average weight gain.

ECONOMIC COSTS OF OBESITY

Such decisions made in the present by individuals have long and lasting effects on their own health and productivity in the future. The rapid rise in obesity has been associated with a corresponding rise in healthcare costs related specifically to obesity. Obese individuals have a 14 to 25 percent increase in the number of physician visits and 24 to 74 percent increase in the number of days spent in the hospital than similar, normal-weight individuals. In terms of annual medical expenditures, analysis of U.S. national survey data from 1997–98 found that obese adults aged 18–65 incur a 36 percent higher cost of medical treatment than normal-weight individuals. Furthermore, it has been estimated that 300,000 Americans die annually from obesity and a sedentary lifestyle through an increased risk of coronary artery disease, stroke, high blood pressure, cancer, and diabetes. In 1995 dollars, researchers estimated that the direct costs of obesity amounted to $99.2 billion, or 10 percent of total U.S. healthcare expenditures. In 2003 dollars, the 2001 U.S. Surgeon General report on obesity stated that annual indirect costs of obesity total $64 billion, suggesting that the indirect and direct costs combined may be as high as $139 billion per year.

SOCIETAL RESPONSE

Despite full information on the health consequences of obesity, benefits of physical activity, and the nutritional content of food, a certain subset of the population will likely continue to engage in lifestyles that promote weight gain. The reason is that the high personal cost of maintaining normal weight is not completely borne by individuals themselves and the cost of maintaining healthier weights is indeed ex-

pensive. Because taxpayers, instead of individuals themselves, bear a significant portion of the current and future costs of an individual's added weight, the decisions that individuals make regarding weight gain are likely not to be optimal from a societal perspective. Calculations by nutritional scientists at the University of Wisconsin bring to light the added cost of much-scrutinized "supersize" option, finding that paying 67 cents to supersize an order is paying only 17 percent more for 73 percent more calories, which would result in an average increase of 36 g of adipose tissue. The future medical costs for the supersize option would be $6.64 for an obese man and $3.46 for an obese woman. These numbers are far greater than the price initially paid for the supersized meal. In addition, this later cost of the present-time decision to supersize is not fully borne by the individual, because the future healthcare cost bill is partially borne by the taxpayer.

WEIGHT LOSS

The decisions made in the present need not create future healthcare costs because obesity is a medical condition that is treatable and manageable for most people in the same manner as any other chronic condition. Diet, medication, or bariatric surgery are interventions of differing intensity and cost that provide weight-loss options to the individual, resulting not just in decreased future medical costs, but also increased nonmedical benefits to the individual. Using a large U.S. longitudinal survey, economist Jay Zagorsky at the University of Ohio has found a significant negative relationship between body mass index (BMI) and a Caucasian woman's net worth. Specifically, after controlling for confounding factors such as age, sex, marital status, and previous net worth, he found that a one-unit increase in BMI for a young woman is associated with roughly a $1,300 or 8 percent reduction in wealth. So while some of these high-tech weight-reduction strategies might seem prohibitively expensive, the health and financial benefits from the loss in pounds might just provide the impetus for more widespread adoption.

NEXT GENERATION

If perhaps the obesity trend affected only adults, the trend may have ended with this generation. Unfortunately, children have increasingly become more overweight and obese. Some of the rationales proposed include an increased availability and usage of television and video games, too much homework, less time to exercise, more fast food, and more usage of cars. However, economists have found that the overall changes are a decline in free time for children, as it is occupied by day care and after-school programs, and a decline in appropriate nutrition. On the bright side, though, as there is increased time in day care, school, and after-school programs, there is a greater opportunity for intervention through education about obesity, better nutrition, and greater time allotment for physical activity.

SEE ALSO: Economic Disparities among Obesity in Women; Economics of Food; Obesity as a Public Health Crisis.

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EDNOS

INDIVIDUALS WITH CLINICALLY significant eating disorders who do not meet the full diagnostic criteria for either anorexia nervosa or bulimia nervosa are currently diagnosed with Eating Disorder Not Otherwise Specified (EDNOS). Individuals with EDNOS may exhibit partial syndromes of anorexia nervosa or bulimia nervosa, show mixed features of both disorders, or have extremely atypical eating behaviors that are not captured by either disorder.

Examples of individuals who should be diagnosed with EDNOS are those who: (1) meet all criteria for anorexia nervosa except have regular menstrual cycles, (2) meet all criteria for anorexia nervosa except their weight falls within normal range, (3) meet all criteria for bulimia nervosa except they engage in binge

eating or purging behaviors less than twice per week or for fewer than three months, (4) purge after eating small amounts of food while retaining a normal body weight (i.e., "purging disorder"), (5) repeatedly chew and spit out large amounts of food without swallowing, or (6) meet criteria for binge eating disorder. The body weight of individuals with EDNOS may vary, but those with the binge-eating-disorder subtype (who regularly eat large amounts of food while feeling out of control) are typically overweight or obese.

The EDNOS diagnosis was initially introduced in the 1987 edition of the *Diagnostic and Statistical Manual of Mental Disorders* as a catchall category for unusual eating disorder presentations. However, data from multiple clinical and nonclinical samples indicate that EDNOS is more prevalent than anorexia nervosa and bulimia nervosa combined. In a community-based sample of approximately 2,000 young females, EDNOS was the most common eating disorder, comprising 2.37 percent of the sample. EDNOS is especially prevalent in populations that have received less research attention such as young children, males, and older adults.

EATING PATHOLOGY

Although some forms of EDNOS may represent relatively mild subclinical, prodromal, or residual variants of anorexia nervosa or bulimia nervosa, most individuals with EDNOS exhibit levels of eating pathology and functional impairment commensurate to those with anorexia nervosa and bulimia nervosa. In addition, there is substantial diagnostic crossover between EDNOS and the main eating disorders over time, and rates of EDNOS are elevated in the family members of people with anorexia nervosa and bulimia nervosa.

The high prevalence of EDNOS poses a significant challenge to clinical practice, because to date, no evidence-based treatment has been developed specifically for this disorder. In addition, behavioral geneticists may have difficulty identifying specific genes associated with EDNOS due to the heterogeneity of the disease. In order to ameliorate these concerns, investigators have proposed revising the diagnostic criteria for eating disorders by relaxing the criteria for anorexia nervosa and bulimia nervosa, extracting new diagnoses from the EDNOS category (e.g., binge eating disorder), or collapsing all three disorders (anorexia nervosa, bulimia nervosa, and EDNOS) into a single transdiagnostic category.

SEE ALSO: Anorexia Nervosa; Binge Eating; Bulimia Nervosa; Disordered Eating.

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Elevated Cholesterol

CHOLESTEROL IS A sterol (steroid alcohol) that is essential in cell membrane, steroid hormone, and bile synthesis. Cholesterol is transported via lipoproteins. Obesity is a risk factor for hypercholesterolemia (elevated cholesterol). Hypercholesterolemia increases the risk of atherosclerosis, which is the main cause of cardiovascular disease and stroke. While there is no strong evidence that targeting obesity improves coronary heart disease (CHD) outcome, exercise, diet, and lifestyle modifications effectively treat hypercholesterolemia.

Low-density lipoproteins (LDL) carry cholesterol absorbed by the gut or produced by the liver to the tissues. Excess LDL can lead to vascular damage including atherosclerosis, thrombi, and emboli that can cause stroke or death. LDL levels increase due to increased saturated fat intake or hereditary conditions. High-density lipoproteins (HDL) remove excess cholesterol from the blood. Increased HDL levels protect against CHD. Hypercholesterolemia allows damaging reactions with free cholesterol to occur within vessel walls. Excess LDL forms atheromas and can be taken up by white blood cells, causing them to form foam cells, leading to further damage. Oxidizing reactions, atheromas and foam cells increase blood pressure, the formation of thrombi and emboli, and the risk of atherosclerosis.

Cholesterol screening is recommended for men older than 35 years and women older than 45 years. If other risk factors such as diabetes, high blood pressure, smoking, or a family history of premature cardiovascular disease exist, screening should begin after 20 years of age.

Fasting measurement of total cholesterol, triglycerides, and HDL allows LDL calculation. Optimally, total cholesterol should be under 200 mg/dL, LDL should be under 100 mg/dL, HDL should be over 41 mg/dL, triglycerides should be under 150 mg/dL, and the total cholesterol to HDL ratio should be under 4. The Framingham score considers total cholesterol, HDL, smoking history, blood pressure, and age to predict the risk of developing coronary heart disease.

Treatment of hypercholesterolemia involves lifestyle modifications including diet and exercise. Several classes of medication, including statins, bile-acid–binding resins, fibric acid derivatives, cholesterol absorption inhibitors, and niacin, can be used if lifestyle modifications are unsuccessful. Only statins, which reduce the rate of de novo cholesterol synthesis, have been shown to reduce overall mortality due to hypercholesterolemia complications.

SEE ALSO: Atherosclerosis; Blood Lipids; Exercise; Fat Intake; High Density Lipoproteins; Low Density Lipoproteins; Stroke.

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Endometrial and Uterine Cancers

THE UTERUS CONSISTS of smooth muscle (the myometrium) overlaid by an endothelial gland-studded mucosal stroma that makes up the endometrial lining of the uterine cavity. The endometrial lining undergoes cyclic response to hormonal stimulation during monthly menstrual cycles and throughout pregnancy. Cyclic proliferation and shedding is stimulated indirectly by the pituitary gonadotropins, luteinizing hormone (LH), and follicle-stimulating hormone (FSH) and directly by the ovarian estrogens and progestins.

The endometrial lining begins each menstrual cycle as a thin stromal layer. As estrogen secretion increases, stromal and endometrial cells proliferate rapidly and the stromal layer thickens markedly. At ovulation, progestins augment estrogen secretions, causing the endometrial glands to accumulate glycogen and lipids in their cytoplasm. This "uterine milk" will provide a nutrient supply to the implanted ovum. If fertilization does not occur, both estrogen and progestin secretion declines, causing rapid involution of the endometrium and vasospasm in the uterine blood vessels. The endothelial lining, deprived of both blood nutriture and hormonal signaling, becomes hemorrhagic, undergoes necrosis, gradually separates from the uterus, and is expelled.

Subtle abnormalities in hormonal signals can interrupt the normal endometrial cycling, causing pain and bleeding. Anovulatory cycles result from excessive estrogen stimulation of the endometrium with absent or inadequate progestin influence. This pattern of hormonal imbalance can result from metabolic abnormalities such as marked obesity and malnutrition. Because they reflect hormonal imbalance, anovulatory cycles are also common at menarche and in the perimenopausal period and are not associated with endometrial cancer risk.

In contrast, endometrial hyperplasia, also called endometrial intraepithelial neoplasia, is strongly implicated as a precancerous lesion. Endometrial hyperplasia differs from benign anovulation in that the endometrium has a higher ratio of glands to stroma and glands exhibit crowding, enlargement, and shape irregularity. Two types of endometrial cancer have been characterized. Type I tumors represent up to 80 percent of cases and are associated with endometrial hyperplasia together with mutations in the *ras* protooncogene and the PTEN tumor suppressor gene. Decreased activity of the mutant PTEN gene increases sensitivity of endometrial cells to estrogen stimulation. Less common type 2 tumors develop in a background of atrophic endometrial tissue in older women and often have mutations in the p53 tumor suppressor gene. Some epidemiological studies suggest that obesity and lifestyle factors modify risk for

type I endometrial cancer to a greater extent than for type 2 cancers.

The incidence of endometrial cancer is up to 10 fold higher in industrialized than rural countries and increases as populations migrate from less to more developed areas. Strong environmental influences have been identified, including estrogenic environmental pollutants, reduced parity, sedentary life style, use of exogenous estrogens for oral contraception or postmenopausal replacement therapy and increased body mass index (BMI). Unopposed estrogen it thought to be the link between risk factors and endometrial cancer. Mitogenic effects of estrogens, when insufficiently counterbalanced by progesterone can result in growth promotion and hyperproliferation in hormone dependent tissues.

Obesity is associated with increased total and bioavailable plasma sex steroid levels in both pre- and postmenopausal women. In postmenopausal women, estrogens are derived almost entirely by extraglandular aromatization of androgens in adipose tissue. Hyperinsulinemia, also associated with obesity, inhibits the hepatic synthesis of sex-hormone-binding globulin (SHBG) and can enhance the synthesis of androgens by the gonads and adrenal glands.

In noncycling postmenopausal women, the body mass index is positively related to plasma levels of estrogen metabolites and to levels of bioavailable estrogen not bound to SHBG. Investigations are underway to assess the influence of other mediators associated with increased adipose tissue mass such as leptin and adiponectin.

Additionally, it is possible that estrogen also can act as a "complete" carcinogen, capable of inflicting DNA damage by oxidative mechanisms.

SEE ALSO: Estrogen Levels; Hormones; Menopause.

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Energy Density

ENERGY DENSITY REFERS to the amount of calories in a given weight of food (calories per gram; kcal/g). A food that is high in energy density provides a large amount of calories in a small portion of food, while a food that is low in energy density has fewer calories for the same weight of food. Eating foods of lower energy density has been associated with reduced short-term energy intake and, in some studies, reduced body weight. Thus, consuming a diet that is lower in energy density may be an important strategy to enhance satiety and help with weight management.

The macronutrient and the water content of a food determine the energy density of a food. Fat is the most energy-dense nutrient, which provides 9 calories per gram, followed by carbohydrate and protein which both provide 4 calories per gram. Alcohol provides 7 calories per gram. Water has the greatest impact on energy density because it adds weight to food but no calories. Thus, foods that are higher in energy density typically have a high fat content (e.g., butter, mayonnaise, nuts, chocolate) and/or a low moisture content (e.g., crackers, chips). Conversely, foods that are lower in energy density typically have a low fat content and/or a higher water content (e.g., fruits, vegetables, soup). There are exceptions, however, in that not all high-fat foods are necessarily higher in energy density and all low-fat foods are lower in energy density. For example, cheese, due to its high moisture content, can have the same energy density as fat-free but dry pretzels.

Lowering the energy density of a mixed meal can be achieved in various ways. One strategy is to add water-rich ingredients to a dish, such as water-rich fruits or vegetables, which add weight but not many calories and therefore reduce the overall energy density of the dish. Another strategy is to cut out some fat from the dish, which can be achieved by substituting lower fat ingredients for their full-fat counterparts (e.g., use low-fat cheese instead of full-fat cheese).

The combination of these two strategies can greatly lower the energy density of a meal while increasing its overall volume. Eating a larger volume of food without ingesting many calories can greatly enhance short-term satiety (i.e., the feeling of fullness). For example, studies by Barbara J. Rolls and colleagues have shown that eating a salad or soup, which are both very

low in energy density, as a first course reduces intake during the main course of the meal.

In conclusion, the energy density of a food or mixed dish greatly influences the amount of calories it provides for a given weight of food. Eating foods with a lower energy density has been suggested to enhance fullness and reduce caloric intake.

SEE ALSO: Fruits and Vegetables; Low-Fat Diets; Volumetrics.

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Energy Expenditure Technologies

THE MEASUREMENT OF energy expenditure (EE) is valuable in understanding the etiology of obesity and in the prevention and treatment of excess body weight. There are several methods that have been developed to quantify the various components of EE in as precise and accurate a manner as possible.

Two fundamental approaches to the objective measurement of EE are direct and indirect calorimetry. Direct calorimetry measures EE as the rate at which heat is lost from the body to the environment, including the nonevaporative heat losses (conduction, convection, and radiation) and evaporative heat loss in the form of water vapor.

Direct calorimetry usually involves whole body measurements in an enclosed chamber. Today, very few calorimetric chambers work on the principle of direct calorimetry because it is technically much more difficult than indirect calorimetry. Indirect calorimetry predicts heat production (EE) from rates of respiratory gas exchange, that is, oxygen (O2) consumption and carbon dioxide (CO2) production. There are several indirect calorimetric methods to measure EE. In the closed-circuit method, the subject is kept in a sealed room (calorimeter room and respiration chamber), and this chamber is ventilated with a constant supply of fresh air. The subject's respiratory gas exchange is measured by comparing the composition of well-mixed air in the chamber with the composition of air entering the chamber, together with the flow rate of air. Most chamber calorimeters are furnished and include television, radio, telephone, some exercise equipment, and toilet and washing facilities, thus permitting measurements that approximate sedentary existence with tight control on intake and activity.

Careful monitoring of the chamber and gases is required to ensure accurate measurements. In the open circuit system, a hood or canopy is worn over the subject's head and is ventilated with room air that enters the hood. As the subjects breathes under this hood or canopy, the airflow and the percentage of O2 and CO2 are measured using a metabolic cart consisting of gas analyzers for O2 and CO2. The openness and portability of the metabolic cart make it the method of choice for measuring basal EE and particularly for exercise-related EE. The doubly labeled water method is a form of indirect calorimetry because it measures CO2 production. One advantage of this method over the chamber is that it does not restrict physical activity and is currently the best objective method to measure free-living EE over a longer period of time, for example, 7 to 14 days.

There are several technologically advanced devices that allow continuous monitoring of the various components of EE with little interference to the subject's activity. These include movement assessment devices such as pedometers, which are either clipped to a belt or worn on the ankle and are designed primarily to count specific movements such as steps while walking or running. Some pedometers adjust for stride length to estimate the distance walked while the more sophisticated battery-operated ones also have a sensitivity adjustment.

Portable accelerometers work on the principle that when an individual moves, the limbs and body are accelerated, theoretically in proportion to the muscular forces responsible for the accelerations and thus to EE. Advances in global positioning systems and radar technology have provided opportunities for remote monitoring of activity EE, and devices have now

been developed to map and measure this component of EE. Physiological measurements such as heart rate, core body temperature, blood pressure, oxygen uptake while performing an activity, or a combination of such measures can also be obtained using various modern-day monitors. These measures are subject to limitations and should be interpreted with caution when used to quantify EE.

SEE ALSO: Bod Pod and Pea Pod; Doubly Labeled Water; Pima Indians.

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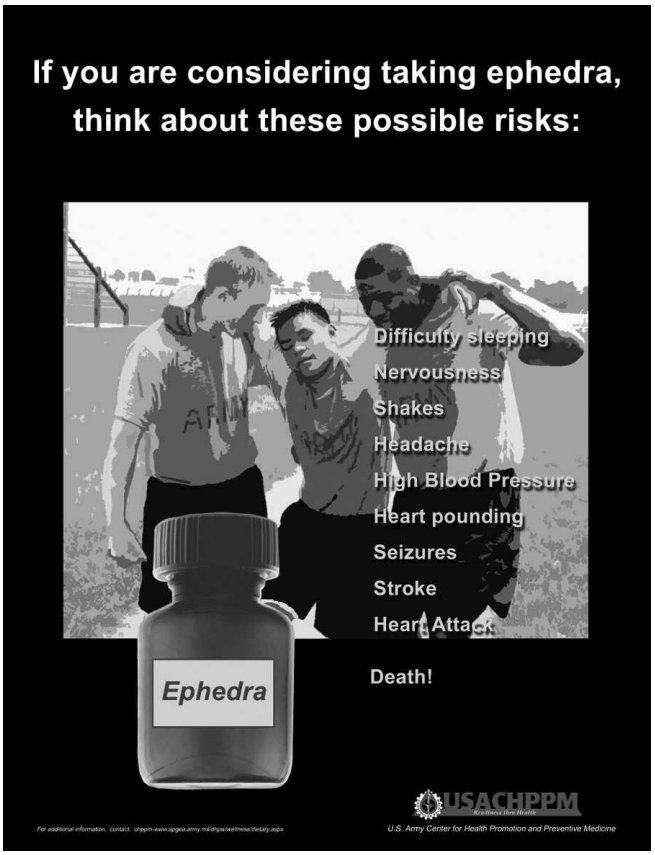

Once a legal substance used in weight loss, ephedra has been banned due to potentially serious side effects.

Ephedra

EPHEDRA IS AN alkaloid compound which was frequently used in dietary supplements and over-the-counter medications sold in the United States before being banned by the Food and Drug Administration (FDA) in 2004. Ephedra was traditionally obtained from plants in the genus *Ephedra.* The active constituents of plants in the genus *Ephedra* include the compounds ephedrine and pseudoephedrine, which are still produced and sold legally in the United States because they are classified as drugs rather than dietary supplements.

Ephedra has a number of biological effects, and it has been used for many years as a stimulant, treatment for colds, influenza and asthma, and weight-loss aid. The herbal drug *ma huang*, of the Ephedra family, has been known in traditional Chinese medicine for at least 5,000 years. Effects of ephedra include increased heart rate, increased blood pressure, increase in body heat and metabolism, and expansion of the bronchial tubes. Side effects of ephedra include nervousness, dizziness, profuse perspiration and dehydration, and irregular heartbeat, and it is associated with increased risk of heart attack and stroke. Before being banned by the FDA, ephedra was commonly used in the United States as a weight-loss aid because it increases both metabolic rate and acts as an appetite suppressant.

Because products sold as nutritional supplements in the United States are not required to provide evidence of either their safety or efficacy, ephedra and related products were available for many years in the United States without being subject to the kind of testing required of prescription drugs.

However, in the late 1990s, concerns mounted over the side effects of ephedra use, and the FDA proposed requiring warning labels and outlawing high dosages in supplements. The Ephedra Education Council, a

public relations group funded by the supplement industry, was created to battle these proposed changes. However, review of mounting evidence against ephedra, coupled with the possibly ephedra-related death of professional athletes Korey Stringer and Steve Bechler, led to further evidence reviews, and in 2004, the FDA banned the sale of dietary supplements containing ephedra in the United States. Many sporting bodies also ban Ephedra, including the National Football League, the International Olympic Committee, and the National Basketball Association.

A meta-analysis conducted in 2003 by Shekelle and colleagues found that ephedrine and ephedra-based products produce modest gains in short-term weight loss relative to placebo, that there were no data regarding long-term weight loss, and that use of ephedra and ephedrine is associated with increased risk of psychiatric, autonomic, and gastrointestinal symptoms.

SEE ALSO: Dexatrim; Drug Targets that Decrease Food Intake/Appetite; Food and Drug Administration; Stroke.

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Epistatic Effects of Genes on Obesity

THERE ARE MANY complex human diseases and traits that are determined by multiple genetic, environmental, and behavioral influences. The global epidemic of obesity results from a combination of genetic susceptibility, increased availability of high-energy foods, and decreased requirement for physical activity in modern society. The increasing prevalence of obesity is associated with conditions such as Type 2 diabetes mellitus, hypertension, cardiovascular disease, dyslipidemia, osteoarthritis, lower quality of life, some cancers, and premature death.

A multigenic trait, obesity has a substantial genetic component involving multiple genes and gene–gene and gene–environment interactions that contribute to its pathogenesis.

Epistasis, or the interactions among genes, contributes to explain obesity onset and severity. The genetic basis of variation in obesity is thought to be due to many genes of relatively small effect and their interactions. In addition, growing evidence suggests that epistasis plays an important role in the genetic control and evolution of complex traits

Genomics is the study of the entire human genome and involves the actions of single genes and the interactions of multiple genes with each other and with the environment. In humans, there are complex interactions among multiple genes and environmental factors that play an important role in controlling obesity traits. For example, variations at multiple genetic loci contribute to the etiology of typical obesity, and it is probable that allelic effects at some loci may be amplified in the presence of variants at other loci.

The different strategies to study the genetic background is based on linkage analysis and candidate gene approach. However, the genetic background remains stable over many generations and genes have not changed substantially over this time period.

Consequently, the rising prevalence of obesity in developed and developing societies reflects lifestyle changes even though multiple studies show that the genetic contribution to obesity is significant. For example, only 1 to 5 percent of obesity cases can be explained by a single gene mutation, even though a family history of obesity is a strong predictor of the condition.

It seems that numerous genes with modest effect contribute to an individual's predisposition toward the more common forms of obesity. Heritabilities for obesity-related phenotypes vary from 6 to 85 percent among various populations. Although obesity is partly determined by genetic factors, an "obesity-promoting environment" is typically necessary for the phenotypic expression of obesity.

Obesity may be derived from a failure on the homeostasis systems, as a consequence of a dysfunction

at the genetic level, which may be affected by changing environmental exposure (dietary habits, sedentarism, etc.). Variants in several candidate genes have been identified and association analyses and functional studies have shown that they contribute to modest obesity and related phenotypes.

In addition, nutrition and physical activity are environmental factors which both influence gene expression. For example, different individuals may respond differently to the same nutritional stimulus and cycles of physical activity and inactivity interact with genes resulting in a functional outcome appropriate for the environment.

The genetically mediated susceptibility to environmental exposure is referred to as gene–environment interaction. Most cases of human obesity probably result from subtle interactions of susceptibility genes with environmental factors favoring deposition of excess calories as fat.

The recent surge of obesity may relate to past evolutionary pressure that favored selection of mechanisms defending body weight against caloric restriction rather than against caloric excess.

Gene-interaction networks may contain modules of co-regulated or interacting genes with distinct biological functions. Such modules may be linked to specific gene polymorphisms, transcription factors, cellular functions, and disease mechanisms. There are genes with large effects that are independent of environment and epistasis, and genes whose alleles interact with the environment to produce obesity in some individuals but not others, and genes that interact with each other.

R. J. Loos and T. Rankinen suggest four levels of genetic contribution to obesity: (1) genetic obesity—genetic mutation in a single gene leads to obesity despite environment (1 to 5 percent of cases); (2) strong predisposition—overweight in nonobesigenic environment and obese in obesigenic environment; (3) slight predisposition—normal weight in nonobesigenic environment and overweight in obesigenic environment; and (4) genetically resistant—normal weight in obesigenic environment. In practice, obesity risk at least depends on two important factors, which mutually interact: (1) genetic variants and gene expression changes in candidate genes and (2) exposure to environmental risk factors. To understand the overall biological etiology of obesity, scientists must identify the genes responsible for each of these different mechanisms and the gene–gene and gene–environment interactions.

SEE ALSO: Genetic Influences on Eating Disorders; Genetic Mapping of Obesity elated Genes; Genetic Taste Factors; Genetics.

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Estrogen Levels

ESTROGEN IS A hormone produced primarily in the ovaries. A significant amount is also in body fat. Estrogen is a specific hormone involved in the development of female sexual characteristics such as breasts, uterine, and ovarian tissue. Estrogen also plays a role in regulating a woman's menstrual cycle. Estrogen is made from cholesterol in the ovaries and the adrenal glands.

There are three types of estrogen in women. Estrone (E1) and estradiol (E2) are produced in the ovaries. Estrone is dominant in postmenopausal women. Estrone can be converted back from the conversion of estradiol to estrone. Estradiol is dominant in postmenopausal women and is the most potent form of estrogen. Estriol (E3) is highest during pregnancy, being produced by the placenta.

Estrogen increases collagen production in the skin. In the liver, estrogen increases lipoprotein receptors, which elevate high-density lipoprotein (HDL), the good cholesterol. Estrogen helps with prevention of bone loss and maintains the integrity of the vaginal wall.

The onset of menopause causes a distinct decline in estrogen production, instigating a decline of its positive attributes. Ovaries cease estrogen production at

menopause. Small amounts of estrogen are then produced from prehormones of the adrenal glands that transform to estrogen from fat tissue. Bone is broken down and the vaginal wall becomes thinner and less flexible.

Hormone therapy may be warranted during the years prior to menopause. This time is referred to as perimenopause. Estrogen therapy may be prescribed for menopausal symptoms, contraception, irregular menstrual cycles, or delayed puberty in a young girl. Estrogen replacement is controversial as to its effects on heart disease in women. A discussion with a qualified physician may offer appropriate guidance as to which course of action an individual may want to pursue.

The amount of estrogen in the blood may have a direct correlation to obesity as fat cells produce estrogen. Prior to menopause, this increased estrogen decreases the risk of breast cancer. Research suggests that following menopause, the high level of estrogen produced in obese women from the fat tissue may increase risk for breast cancer and osteoporosis.

It is known that men have about half the amount of estrogen as women. In men, testosterone and androsteredione are converted to estrogen in individual organs. The male supply of estrogen is never depleted. An excess of estrogen in men may be the result of liver disease, testosterone deficiency (hypogonadism), chronic alcoholism, estrogen-producing tumors, or congenital adrenal hyperplasia. As yet, it is unclear as to the exact role estrogen plays in men.

Compounds similar to estrogen, phytoestrogens, are found in some plant compounds. They are present in soy, clover, green tea, flaxseed, and some legumes. They occupy the same receptor sites as estrogen and have many similarities.

SEE ALSO: Breast Cancer; Estrogen-Related Receptor; Hormones; Menopause.

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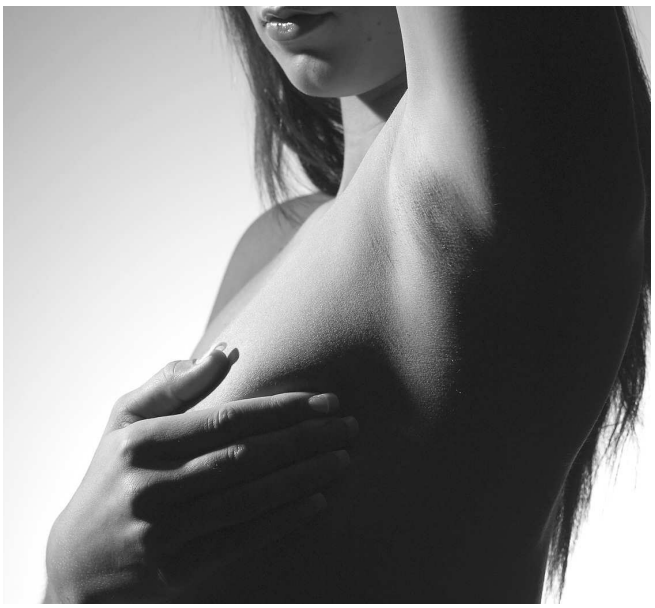
Estrogen-Related Receptor

ESTROGEN RECEPTORS OPERATE similarly to a lock and key. Estrogen works as the key to a receptor (lock) on the outside of a specific cell. These locks are not the same in every organ. Estrogen stimulates the growth of breast cells and may also stimulate the growth of breast cancer. Drugs referred to as selective estrogen receptor modulators (SERMs) were developed to prevent estrogen from stimulating the growth of breast cells that could become cancerous by blocking the estrogen receptors in the breast.

Estrogen-related receptors (ERRs), specifically ERR α, are an orphan member of the superfamily of nuclear receptors. An orphan receptor is one that has no known ligands (proteins that bind to it). ER and ERR α are related in that they bond to DNA sites. ERR α binds to DNA targets by the process of transcription.

Research has been conducted with mice as to the role ERR α may play in the body. This research hypothesizes a possible connection to intestinal fat transport and may play a role in bone remodeling. It is also thought that ERR α is present at high levels in tissues having high oxidative properties. It is theorized that there may be a linkage to estrogen signaling and oxidative metabolism An increase in energy demand such as during fasting, exercise, or cold exposure re-



Estrogen, the female hormone produced by the ovaries, has been linked to both breast cancer and obesity.

quires an elevated level of ERR α. In studies on mice, ERR α was removed, creating a defect in thermogenic function. ERR α appears to be high in tissue which has high oxidative capacity.

It is theorized that ERR α plays a role during times of metabolic stress and dietary lipid digestion and absorption. There may be a link to high expression of ERR α in brown fat by lipid metabolism and transport, which are significant, factors in energy balance.

Research is continuing to determine the role ERR plays in thermogenic function and homeostasis. ERR may play a role in lipid metabolism and energy balance.

SEE ALSO: Breast Cancer; Estrogen; Hormones.

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Ethnic Disparities in Obesity among Women

THERE HAS BEEN a significant rise in the prevalence of obesity in the United States during the last 50 years and this has become a public health concern of epidemic proportions. Body weight is often classified into four categories: underweight, normal weight, overweight, and obesity. To determine into which category a person should be placed, his or her body weight needs to be converted into another value known as body mass index (BMI). This number is obtained by dividing a person's weight in kilograms by the square of their height in meters. A person with a BMI of less than 18.5 is considered underweight. A person with a BMI between 18.5–24.9 is considered to be of normal weight. A person with a BMI between 25.0–29.9 is considered to be overweight. Finally, a person with a BMI greater than 30 is considered clinically obese. These classifications for BMI ranges are based upon the risk of developing certain diseases with each BMI range. In 2000, it was estimated that more than half of the adults residing in the United States could be defined as clinically overweight (having a BMI between 25.0–29.9). Furthermore, one-fifth of the U.S. adult population was clinically obese (having a BMI >30) and 2 percent of the U.S. adult population was classified as morbid obesity (having a BMI >40). Some researchers consider these estimates to be conservative and that the prevalence of obesity is much greater in this country.

Many diseases have an increased prevalence when a person is overweight or obese. These include heart disease; high blood pressure; Type 2 diabetes mellitus; several cancers including, but not limited to, breast, colon, and prostate; stroke; metabolic syndrome; gallstones; and arthritis.

While the prevalence of obesity has increased for the entire U.S. population, disparities still remain in the prevalence of obesity based upon gender and race or ethnicity. Females are more likely to be overweight and obese compared to males for all races and ethnicities studied.

Within the female population, African-American women are more likely to be overweight and obese compared to Caucasian, Hispanic, or Asian women. According to data collected for the National Health and Nutrition Education Survey (NHANES; a large, national epidemiological study), half of the Caucasian women who were between 20–39 years old were considered overweight (body mass index [BMI] >25), while 70 percent of African-American women in this age group were overweight.

Furthermore, half of the African-American females in this age group were clinically obese (BMI >30), while only one-quarter of the Caucasian women in this age group were clinically obese. Differences in the prevalence of obesity among races are not limited to adult populations as African-American children have a greater prevalence of obesity compared to Caucasian children.

Along with the increased prevalence of obesity in African-American women, there is an increased

prevalence of many diseases such as insulin resistance and Type 2 diabetes mellitus. In addition, because of the greater prevalence of insulin resistance in African-American women, they also have increased insulin secretion from their pancreas and decreased insulin clearance in their body. These clinical symptoms may lead to a greater percentage of African-American women developing Type 2 diabetes mellitus from insulin resistance in a shorter period of time compared to Caucasian women.

There are many other differences between African-American and Caucasian women relating to the difference in their prevalence of obesity. African-American women have a lower metabolic rate compared to Caucasian women. This means that for the same amount of body weight, African-American women need to consume fewer calories to maintain that body weight. As portion sizes continue to grow in this country, African-American women may be more susceptible to weight gain compared to Caucasian women because of their lower metabolic rates. Several differences also exist in the body composition of African-American women compared to Caucasian women. African-American women tend to have a greater bone density as well as longer bones compared to Caucasian women.

This may explain the decreased prevalence of bone injuries observed in African-American women. African-American women also tend to have more muscle compared to Caucasian women. In addition, in regard to the distribution of fat in the body, African-American women have a greater amount of fat in their muscles, but less fat around their abdomens. The extra fat in the muscle may be related to the increased prevalence of insulin resistance and Type 2 diabetes mellitus observed in African-American women. African-American women also tend to have a better lipid profile compared to Caucasian women; they tend to have lower triglycerides, a marker of the risk for developing heart disease. African-American females have a lower risk of developing heart disease in an obese state compared to Caucasian females.

Another ethnic group with elevated obesity rates in the female population is the Hispanic population. The term *Hispanic* encompasses a diverse group of people who have a very heterogeneous mixture of genetic information. People of Hispanic origin tend to be more overweight and obese than Caucasians and less overweight and obese than African Americans. In the 20–39 year old age range, over 60 percent of Mexican-American women were clinically overweight and nearly one-third of Mexican American women were clinically obese.

Hispanic women have an increased prevalence of insulin resistance and Type 2 diabetes mellitus compared to Caucasian women but a lower prevalence of these disorders compared to African-American women.

Another ethnic group that has seen an increase in their prevalence of obesity rates is Asian-American females. Asian-American females have a lower prevalence of being overweight and obese compared to Caucasian, Hispanic, or African-American females. However, the use of the cutoff values for the BMI scale has often been criticized for Asian Americans.

Several researchers have suggested that Asian Americans should be considered overweight at a BMI of 23. This is because Asian Americans have an increased risk of developing certain diseases with smaller weight gains.

Asian-American females are more likely to gain weight around their stomach. The excess weight gain around the stomach is associated with an increased risk in developing many of the diseases that are mentioned above. However, other researchers have argued that the current BMI numbers are appropriate for the Asian-American female population.

The rise in obesity along with the concurrent rise in metabolic disorders has been hypothesized to shorten the life expectancy of the population in the United States for the first time since the mid-1800s. The estimated number of excess deaths in the United States that are attributable to obesity range from 112,000 to 325,000 deaths every year and the number of years of life lost (YLL) attributed to obesity range from 5 to 20 years based on age, gender, and race.

The cost of obesity on society has been estimated to be $117 billion per year. This estimate includes both direct and indirect costs such as medical expenses and lost productivity. Ethnicities that have an increased prevalence of obesity are more likely to suffer from a poorer quality of life and shorter lifespan.

SEE ALSO: Ethnic Disparities in the Prevalence of Childhood Obesity; Ethnic Variations in Body Fat Storage; Ethnic Variations in Obesity-Related Health Risks.

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Ethnic Disparities in the Prevalence of Childhood Obesity

THE PREVALENCE OF obesity has increased dramatically over the past three decades. This increase has been noted in both adults and children. However, the increase in children is a major concern among healthcare professionals and policymakers because obese children have an increased risk for becoming obese adults. Currently, approximately 15.3 percent of children (ages 6–11) and 15.5 percent of adolescents (ages 12–19) are obese. The concern about the increased prevalence of childhood obesity is heightened regarding ethnic minority and low-income communities.

The Institute of Medicine (IOM) has reported that childhood obesity has become a national priority. This characterization of childhood obesity by the IOM and others has directed more attention to a health problem that is seen as an epidemic. Despite conflicting views about what has caused obesity to increase in the first place, the obesity epidemic can be attributed to multiple factors. For example, environmental, biological, and behavioral factors all play significant roles in contributing to obesity in children.

Multiple sources provide data that demonstrate an upward trend in obesity rates since the early 1960s to the present. The major source of data comes from the National Health and Nutrition Examination Survey (NHANES), a nationally representative survey that has been conducted periodically since the early 1970s. Other data sources include, but are not limited to, the Pediatric Nutrition Surveillance System (PedNSS), which gathers annual nutritional risk data from more that 8 million children participating in public assistance programs; and the National Longitudinal Study of Adolescent Health (Add Health Study), a nationally representative survey of teenagers.

Notwithstanding the source of data, the results continue to reflect that the prevalence of childhood obesity is greatest within ethnic minority and low-income communities. As reflected by the data, obesity rates are generally higher for Native-American and Hispanic children of both sexes and for African-American girls. Because more data are available regarding African Americans and Hispanics, most of the historical as well as current literature focuses on these two populations. Additionally, low-income children are at greater risk of obesity regardless of ethnicity.

Compared to other ethnic groups, relatively few U.S. data regarding health and weight status of Asian Americans are available. Unlike other ethnic minority groups, Asian Americans have not been oversampled in national studies. Additionally, some studies have combined obesity prevalence rates for Asian Americans with Pacific Islanders. Overall, Asian-American children do not fit the general pattern of higher obesity rates similar to children in other ethnic minority groups.

Most of the data regarding the prevalence of obesity on Native-American children have been collected since 1990. Despite a great deal of variation among tribes, the data indicate a widespread prevalence of obesity with Native-American youth. The data indicate that 12 percent of Native-American children aged 2–4 years are overweight. A similar trend has been found among Hispanic children in the same age group. Overall, the data reveal that obesity among Native-American children may begin in early childhood. Moreover, the prevalence of overweight and

obesity continues to increase in Native-American schoolchildren aged 5–17 years. In two large population-based studies, 39 percent of Native-American youth were overweight.

Hispanic-American children have also seen a substantial increase in the prevalence of obesity. Similar to Asian Americans and Native Americans, there is a great deal of variation among Hispanic groups. For example, one Hispanic HANES study reported that obesity was more common among Puerto Rican adolescents compared to Mexican Americans and Cubans. Another study revealed similar obesity prevalences (32.2 to 33.1 percent) among Mexican American, Puerto Rican, and Cuban adolescents aged 13–18 years. In contrast, Central/South American Hispanics have a lower rate of obesity (25.7 percent). Generational differences have also been observed among Hispanics, with only 26 percent of first-generation Hispanic adolescents being obese compared with 33 percent of second- and third-generation Hispanics. Overall, current data show that approximately 23.7 percent of Hispanic-American children (ages 6–11) are obese and 23.4 percent of Hispanic-American adolescents (ages 12–19) are obese.

African-American children have experienced some of the greatest increases in the prevalence of obesity. Some of the research has revealed a twofold increase in the prevalence of obesity among children aged 4–5 years from 1976–80 to 1988–94 (3 to 8.7 percent for boys, and 6.5 to 12.6 percent for girls). It has also been reported that for children aged 6–11 years, the prevalence of obesity increased from 11.6 (1976–80) to 17.7 percent (1988–94) for girls, and from 8.6 to 14.7 percent for boys. Consistent with younger African-American children, there has been an increase in the prevalence of obesity among adolescents aged 12–17 years (10 to 16.0 percent for girls, and 6.1 to 12.5 percent for boys). Overall, current data reveal that approximately 19.5 percent of African-American children (ages 6–11) are obese and 23.6 percent of adolescents (ages 12–19) are obese.

The obesity epidemic has taken a heavy toll on the health of the nation's children. Many obesity-related illnesses that were once primarily seen in adults, such as Type 2 diabetes and high blood pressure, are now being seen more frequently in children. These obesity-related illnesses are especially a concern for minority children. Overall, minority children, especially African Americans, Hispanic Americans, and Native Americans, tend to have a higher prevalence of childhood obesity than their Caucasian peers. Several factors have been found to have a significant influence on ethnic disparities of childhood obesity: environment, biology, and behavior. These factors tend to interact with low-income status, which places minority children at greater risk for becoming obese and ultimately obesity-related illnesses.

SEE ALSO: Ethnic Disparities among Obesity in Women; Ethnic Variations in Body Fat Storage; Ethnic Variations in Obesity-Related Health Risks.

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Ethnic Variations in Body Fat Storage

RESEARCH SHOWS THAT ethnic groups exhibit varying patterns of fat distribution. Humans have two general patterns of fat distribution: androidal (commonly referred to as central or abdominal/truncal adiposity) and gynoidal (lower-body/peripheral adiposity), or described more colloquially by Marie Savard as "apple-shaped" or "pear-shaped," respectively. While both patterns of fat distribution have potential health risks, research shows that abdominal (especially vis-

ceral fat) and truncal adiposity carry an increased risk for obesity-related complications such as glucose intolerance, hyperinsulinemia and the development of Type 2 diabetes, cardiovascular disease (CVD), and some types of cancer. Increased levels of central adiposity observed in some ethnic groups may explain the increased incidence of metabolic complications and CVD in these groups.

Ethnic groups, including non-Hispanic blacks, Mexican Americans, and some Native American groups in the United States, and black Caribbean and Irish groups in Britain, consistently show a higher prevalence of overweight and obesity according to body mass index (BMI) than the general population of the country. However, a growing body of evidence suggests that BMI alone may not adequately define overweight and obesity in adults or children for several reasons.

First, BMI does not distinguish between fat mass and lean (nonfat) mass. Therefore, a definition of obesity based on BMI alone might classify individuals with large central fat deposits as "normal" (defined as within the BMI range of 25–29.9), when that individual may be at greater risk for metabolic and cardiovascular complications.

Second, BMI also gives no indication of the distribution of body fat, and as mentioned above, fat distribution has been linked to several comorbidities. Simple anthropometric measures including waist circumference (WC), waist-hip ratio (WHR), and subscapular to triceps (STR) skinfold thickness ratio have been established as reliable measures of central adiposity. Among these, WC has been endorsed as the best surrogate measure of abdominal adiposity and cutoff points of over 102 centimeters in men and of over 88 centimeters in women are widely accepted as values representing risk for the development of obesity-related comorbidities. More sophisticated measurements such as computed tomography (CT) scans or magnetic resonance imaging (MRI) have also been used by some investigators because they can accurately distinguish between subcutaneous and visceral fat accumulation, but their usage is limited by their expense.

Certain ethnic groups exhibit a high prevalence of central obesity. When South Asians gain weight, they tend to accumulate fat in the abdominal and truncal regions of the body. This has been documented in several studies showing that they have larger waist circumferences, larger abdominal diameters, and thicker trunk skin folds for a given weight compared to other groups. South Asians also show a relationship between the onset of comorbidities such as Type 2 diabetes and CVD at much lower levels of BMI than has been documented in other groups. Japanese and Taiwanese Americans exhibit similar fat distribution as South Asians. Research in the United States shows that Mexican-American men and women have higher WC and STR than non-Hispanic Whites. Several studies demonstrate that abdominal adiposity in African Americans is characterized by a greater accumulation of subcutaneous fat than visceral fat, compared with other ethnic groups. Ethnic groups such as Pima Indians and Naruans do not show central fat distribution.

Why certain groups exhibit central adiposity and others exhibit lower-body adiposity is not clear. As far back as 1956, Jean Vague recognized that visceral fat accumulation was greater in men than women for a given weight, and suggested that central fat distribution may be related to cardiovascular risk. In human females, lower-body fat has been evolutionarily advantageous for fertility and reproduction, which may explain why lower-body fat is difficult to lose. Central fat distribution may be related to the thrifty genotype and evolutionary biology, which may now have been rendered maladaptive in the context of improved food security and decreased physical activity. Research shows that fat distribution is to a large extent genetic, with a calculated heritability of 0.36–0.6; however, environmental factors such as education, physical activity, and parity (in women) play a role in fat distribution.

Given that central adiposity is recognized as a risk factor for metabolic complications and CVD, it is important that those groups exhibiting this form of fat distribution be targeted for health interventions, which may help to limit the accumulation of fat and reduce morbidity.

SEE ALSO: Asia, South; Assessment of Obesity and Health Risks; Body Fat Distribution in African Americans; Ethnic Variations in Obesity-Related Health Risks.

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Ethnic Variations in Obesity-Related Health Risks

A LARGE BODY of literature demonstrates an association between obesity (specifically central obesity), insulin resistance, and risk factors for chronic diseases such as impaired glucose tolerance, Type 2 diabetes, cardiovascular disease (CVD), hypertension, and certain cancers, including breast, prostate, and colon cancers. These associated conditions have been clustered together and described in the literature under the names of metabolic syndrome, insulin-resistance syndrome, syndrome X, plurimetabolic syndrome, and cardiometabolic syndrome. Ethnic variations in obesity-related health risks have been explored in detail, with particular attention given to the relationship between adiposity and insulin resistance, Type 2 diabetes, hypertension, and CVD in certain groups. Research shows that obesity is more strongly correlated with Type 2 diabetes in South Asian, Native American, and Mexican American populations; with hypertension (and stroke) in individuals with African ancestry (in African Americans in the United States, black Africans and Afro-Caribbeans in the United Kingdom, as well as in the Caribbean and Africa); and with CVD among Caucasian Americans and South Asians (from the Indian subcontinent and from East Africa).

In addition to traditional lifestyle risk factors for the development of metabolic complications and CVD such as diet, physical activity, and smoking patterns, research shows that percentage of body fat and distribution of fat are related to disease risk, and ethnic groups show variation both in percentage of body fat and body fat distribution. Some populations (namely mainland Chinese, Chinese from Hong Kong and Taiwan, Filipinos, Koreans, and South Asians) have higher body fat percentage at a given body mass index (BMI) than Caucasian or European populations. Some Pacific populations also have a lower percentage of body fat for a given BMI than Caucasian or European populations. In one study, Paul Deurenberg and colleagues show that for the same percentage of body fat, age, and gender, American blacks have a 1.3 kg/m2 and Polynesians have a 4.5 kg/m2 higher BMI compared to Caucasians. By contrast, the typical BMI of Chinese, Ethiopians, Indonesians, and Thais are 1.9, 4.6, 3.2, and 2.9 kg/m2 lower compared to Caucasians, respectively. This is cause for concern as a higher percentage of body fat at a lower BMI reflects increased risk of disease and may explain why some Asian populations show a high prevalence of the comorbidities of obesity at comparatively lower levels of BMI than Caucasian or European populations. As a result, some argue that the cutoff points for overweight and obesity for Asians should be lower than the World Health Organization (WHO) cutoff points, but this continues to be widely disputed.

Central fat distribution, which has been observed in some ethnic groups such as South Asians and Mexican, Japanese, Taiwanese, and Native Americans, also increases the risk for metabolic complications and CVD. The role of visceral and subcutaneous abdominal fat in metabolic complications and CVD risk is not clear, but it has become a question of great interest. Two potential mechanisms have been suggested: the highly lipolytic visceral depot releases fatty acids into the portal vein that travel to the liver, which can lead to glucose intolerance and insulin resistance; and elevated free testosterone and reduced sex-hormone-binding globulin may promote increased central adiposity and reduce fractional hepatic extraction of insulin.

In all populations, obesity shows a strong correlation with insulin resistance and Type 2 diabetes and the global prevalence of Type 2 diabetes is rising, reflecting increasing weight gain worldwide. The regions with the greatest potential increases of diabetes are Asia and Africa where the rates are predicted

to rise to two or three times those experienced today. Recent research shows that factors acting early in life may impact risk for disease later in life. The Barker hypothesis, also referred to as the thrifty phenotype hypothesis or the developmental origins hypothesis, suggests that poor fetal and early postnatal nutrition imposes mechanisms of nutritional thrift on a growing individual which may result in reduced capacity for insulin secretion, insulin resistance, and impaired glucose tolerance as adaptive strategies to cope with its frugal early nutrient supply. The resulting metabolic programming may lead to a greater risk for developing diabetes later in life. High levels of low birth weight observed in South Asians, coupled with good postnatal food security, may explain the increasing prevalence of Type 2 diabetes in this group.

While research demonstrates that ethnicity influences the prevalence of obesity-related health risks, it remains unclear why certain ethnic groups show a higher incidence of metabolic disorders and CVD. Extensive literature shows that it is not merely a function of genetics and, therefore, environmental, social, cultural, and intergenerational factors must be considered in the appropriate treatment and prevention in all ethnic groups worldwide.

SEE ALSO: Barker Hypothesis; Ethnic Differences in Body Fat Storage; Hypertension in African Americans; Native Americans.

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Europe, Eastern

ALTHOUGH THE ANCIENT Greeks generally portrayed athletic males on their friezes and figures shown on their jars, there is some evidence of female obesity in surviving imagery. Certainly, there are many figurines of females, often described as "Venuses" which show obese people. In a recent study on a clay figurine found at Farsala, Thessaly, now held at the Athanassakeion Archaeological Museum of Volos, there have been attempts to evaluate views on ancient feminine obesity in connection with the concepts of "Mother Earth." Other studies have shown that obesity was a major problem in the Byzantine Empire, as increasing prosperity and sedentary lifestyles among some people in cities and towns led to a high body mass index (BMI), and subsequent medical complications.

In Russia, there has long been a problem with obesity rising from overeating and drinking, lack of exercise, and some genetic and inherited traits. Many of these problems affected the rich urban elite. The Russian playwright Anton Chekhov (1860–1904) in his notebook wrote of a problem in which a doctor asked the maid of an obese man to put his slippers as far as possible under the bed to remind her master of his obesity at every possible opportunity. Political cartoonists in Russian history often associated obesity with financial or political corruption, with many of these during the Brezhnev period in the Soviet Union careful to show a much slimmer version of Leonid Brezhnev than actually was the case.

Since the end of the Communist rule, obesity has also been associated, in the public mind, with political corruption, with caricaturists and cartoonists overemphasizing the weight of some politicians. Although obesity has long been noticeable in European Russia, and also in Ukraine, Belarus, and Moldova, it is now a major health concern throughout Siberia as well where the main Siberian populations have massively reduced their amount of physical activity, as well as increasing their consumption of foods that are likely to lead to obesity.

In Finland, the prosperity of the country since the mid-19th century has seen a gradual rise in the BMI, which has become particularly noticeable in the later 20th century. This led to the creation of the Finnish Association for the Study of Obesity (FASO) with Dr. Kirsi Pietiläinen of Helsinki being the national rep-

resentative on the International Association for the Study of Obesity (IASO).

In Poland, Hungary, Czechoslovakia, Romania, and Bulgaria, until World War II, obesity was associated with wealth, and during the period of Communist rule came to embody, in the public mind if nowhere else, political corruption, although some came from genetic and inherited factors, and smoking, as well as a more sedentary lifestyle. The end of Communism and the massive rise in affluence in East Central and Eastern Europe has seen the establishment of a large number of associations to improve access to information on the reduction of obesity. In Poland, the Polish Scientific Association of Obesity and Metabolism has Professor Barbara Zahorska-Markiewicz, Department of Pathophysiology, Medical University of Silesia, Katowice, as the national representative on the IASO. In recent years, L. Szponar, J. Ciok, A. Dolna, and M. Oltarzewski from the Department of Food and Nutrition Safety, National Food and Nutrition Institute, Warsaw, have been involved in a number of studies on obesity in Poland.

The Hungarian Society for the Study of Obesity (HSSO) has Professor Csaba Nyakas of the Institute of Sports Sciences, Semmelweis University, Budapest, as their national representative on the IASO. In the neighboring Czech Republic, the Czech Society for the Study of Obesity (CSSO) is responsible for raising awareness of obesity in its country, with Dr. Vladimir Stich of the Department of Sports Medicine, Faculty of Medicine, Charles University, Prague, being the national representative on the IASO. In Romania, the Romanian Association for the Study of Obesity (RASO) has Professor Nicolae Hancu of Cluy-Napoca as the national representative on the IASO; and in Bulgaria, the Bulgarian Association for Study of Obesity and Related Diseases (BASORD) has Professor Handjiev Svetoslav of the Department of Nutritionietetics and

A town square in Eastern Europe. Due in part to an increase in the number of people following a modern, sedentary lifestyle, the obesity problem is growing much more severe throughout Eastern Europe.

Metabolic Diseases, Sofia, as its national representative on the IASO.

In Albania, there was traditionally a much lower rate of obesity than in other parts of Eastern Europe. However, in period since the end of Communist rule, increased prosperity has led to some cases of acute obesity, and a higher rate of risk factors connected with acute coronary syndrome, and also Type 2 diabetes. A study in 2007 involving 1,120 in the Albanian capital, Tirana, found that over 75 percent had excess body weight, and 22 percent of men and 30.9 percent of women were found to be obese—figures which are very high given the previously low prevalence rates of obesity in the country.

There has long been an obesity problem in Greece with the diet contributing to the problem. About 35 percent of men in Greece being overweight. Curiously, the problem has not adversely affected some other health indicators in the country, with Greece being 7th (of 191) in terms of its average life expectancy, and Greece also having one of the lowest rates of heart disease in the region. The Hellenic Medical Association for Obesity (HMAO) was founded in 1990 as a nongovernmental scientific association, with Dr. Ioannis Kaklamanos of Athens as Greece's national representative on the IASO.

In the former Yugoslavia, the Croatian Obesity Association (COA) has Professor Dr. Mirko Korsic of the Department of Endocrinology, Clinical Hospital Zagreb, as the national representative on the IASO; the Macedonian Association for Obesity (MAO) has Professor Dr. Gordana Pemovska of the Clinic of Endocrinolgy, Diabetes and Metabolic Disorders, Skopje, as its national representative on the IASO; the Serbian Association for the Study of Obesity (SASO) has Professor Dr. Jagoda Jorga of the Department of Nutrition, School of Medicine, Pasterova, Belgrade, as the national representative on the IASO; and in Slovenia, the Professional Section of the Slovenian Association for Obesity has Dr. Tina Sentocnik of Ljubljana as its national representative on the IASO. All these groups have been instrumental in raising the awareness of obesity in their respective countries.

SEE ALSO: Europe, Western.

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Europe, Western

OBESITY HAS INCREASINGLY become a major problem in Western Europe owing to excessive eating, eating the wrong foods, some inherited genetic traits, and also for medical reasons, or as a side effect from medicines. Although it is clearly a much greater problem nowadays than it has been previously, there is much evidence of obesity in the history of the region since classical times.

Gluttony as a problem is more associated with the Roman Empire as it became wealthier and lazier, and some of its citizens greatly indulged themselves. The Roman actor Aesopus was well known for the ostentatious vulgarity of his enormous eating habits, and Pliny the Elder was certainly very large. Other obese Romans included the writer Horace and the Emperor Vitellius who was emperor from April to December 69. The Roman physician Galen wrote that there was a man called Nichomachus of Smyrna (modern-day Izmir, Turkey) who was so large that he could not get out of bed. Traditionally, obesity for men tends to be associated with political corruption. In the Celtic world, obesity appears to have been very rare, although rulers such as King Louernius of Gaul are recorded to have indulged themselves and their close supporters with vast feasts, with heavy consumption of meat.

The first king of England who can definitely be said to have suffered from obesity was Henry VIII, evident from contemporary accounts and the size of his armor. One of Henry VIII's ministers, Cardinal Thomas Wolsey, also became obese in his later years. John Marriott, a public glutton from the reign of James I, became the subject of a pamphlet *The Great Eater of Graye's Inn, or the Life of Mr. Marriott, the Cormorant*. It claimed that he had himself once eaten a banquet prepared for 20 people. Samuel Pepys, in his diary, refers to various obese people, including Mr. Mills, the parson, whose size is equated with his laziness. Mention should also be made of the siege of Londonderry in 1688–89 when it was recorded that one extremely fat man refused to go into the streets during the 105-day siege because his neighbors often licked their lips when they saw him. The term "Fat Man in Londonderry" soon became an expression to describe people in similar situations. George Cheyne, a London medical doctor, promoted vegetarianism as a way of controlling body weight and as a possible cure for obesity.

By the 18th century, body size became associated with wealth, although it is clear from paintings that some people were what would now be regarded as obese. Sir Robert Walpole, the first British Prime Minister, became increasingly corpulent in his later years, with some cartoonists considerably exaggerating his size, as they seem to have done with other political and social figures of the period. James Boswell, the biographer and diarist; Samuel Johnson, the lexicographer and author; Frederick, Lord North, prime minister from 1770–82; and Edward Gibbon, the author of *The Decline and Fall of the Roman Empire*, in his later years, were four well-known individuals of the period who were all extremely corpulent. Although as Prince Regent, George, son of George III, was a handsome man, by the time he acceded to the throne as George IV in 1820, he was extremely overweight.

French delicacy: A variety of causes has increased the number of obese and overweight people throughout Western Europe.

There were also details of many cases of obesity coming solely from excessive eating. William Douglas, the fourth Duke of Queensberry and third Earl of March, is said to have regularly eaten two breakfasts and two lunches, ending the day with five full dinners consumed from between 5 p.m. and 3 a.m. It was during this period that it was recorded that Jack Biggers ate a meal consisting of 6 pounds of bacon, a green salad, a loaf of bread, and a dozen suet dumplings,

washed down by a gallon of beer. The *Annual Register* (November 1765) reported that Walter Willey, the obscure servant of a London brewer ate a 6 pounds of roast goose, about 4 pounds of bread, and 3 quarts of port. Nicholas Wood of Kent is said to have eaten an entire sheep at one sitting, leaving only the bones, horns, wool, and skin, and at another sitting, it was claimed that he ate 400 pigeons.

By the end of the 18th century in Britain, obesity was once again heavily associated with decadence and also with political corruption. British caricatures of Indian middlemen and some "Nabobs" (Britons who returned after a successful business career in India) often showed them as obese, and cartoons and drawings of Lord Macartney's mission to China in 1792 similarly showed Chinese officials as grotesquely obese.

During Victorian England, in spite of a healthier diet, there were still many prominent examples of obesity, perhaps the most famous being that of the "Tichborne Claimant." Following the death of James Tichborne, his French-born widow started to advertise for news of her son who had disappeared in 1854 in a shipwreck off the coast of Chile. A butcher in the Australian town of Wagga Wagga came forward claiming to be the missing Sir Roger Tichborne. Coming to London in 1866, only Roger's mother supported his claim, but this enabled him to live a wanton lifestyle, eating voraciously and his weight quickly rose to 27 stones (378 pounds). In a subsequent court case, he was found guilty of perjury and jailed for 14 years, his obesity in court being the subject of many caricatures and much ridicule.

France also had a number of prominent obese people. Following the end of the Roman Empire, Gaul was taken over by the Franks whose king from 751 until 768 was Pepin the Younger, who is sometimes described in history books as being Pepin "The Fat." Pepin, the son of Charles Martel, is most famous for being the father of Charlemagne. Another Frankish king was also known for his size. Charles "The Fat" became Holy Roman Emperor in 879 and the king of the East Franks in 884. With a reputation for being lethargic and inept, although he managed to accumulate a very large number of titles concurrently becoming king of Alemannia and Rhaetia, king of Italy, king of Upper Burgundy, king of East Frankia, king of West Francia (France), and king of Aquitaine.

There were two subsequent French kings of the Capetian dynasty whose size earned them the title "The Fat." The first of these, Philip the Fat, ruled from 1059 until 1108. Becoming king at the age of 7, he went with Richard the Lionheart on the Third Crusade, with his size contrasted by English chroniclers with athletic prowess of his English counterpart. U.S. Historian Joseph Dahmus described him as undistinguished and recorded that he spent most of his energy eating, "wenching," and sleeping. His son, Louis VI of France, was also given the title "The Fat," succeeding his father to the throne in 1108. Louis "The Fat" managed to consolidate his kingdom, but died in 1139 from excessive eating, which seems to have followed his contracting dysentery. The penultimate Capetian king of France, Louis XVIII, was also a large man whose size was rarely reflected in contemporary paintings and images of him, although a profile drawing from 1814 shows his obesity clearly.

During the 19th century, the increased prosperity of France led to a development of gluttony, and inevitably, increased obesity. Honoré de Balzac was said to have eaten 12 cutlets, 110 oysters, one duck, and two partridges at a single sitting. Although the theologian John Calvin was overweight, historians doubt he was as obese as Balzac and other critics made out in *Catherine de Medici*. Balzac's contemporary, The socialist politician Jean Jaurès was short and obese, something that his political critics used to parody him as "a fat merchant who overeats."

French research into obesity has helped achieve a greater understanding of the symptoms connected with it. In 1920, a French physician Georges Bardet studied a disease that was characterized by obesity and various other problems. Two years later, the Hungarian Arthur Biedl added to Bardet's work, and the rare genetic disease became known as the Bardet-Biedl syndrome. Within France, the Association Française d'Etudes et de Recherché sur l'Obésité works on promoting awareness of obesity, with Dr. Yannick Lemarchand-Brustel of the Faculty of Medicine, Nice, being the national representative on the IASO. In Belgium, the Belgian Association for the Study of Obesity operates for coordination of research into obesity, with Dr. Maximilien Kutnowski of the Department of Internal Medicine at the University Hospital Brugmann being the Belgian national representative on the IASO. The Netherlands

Association for the Study of Obesity operates in the Netherlands and the Netherlands West Indies, with Professor Ellen Blaak of the Department of Human Biology, Maastricht University, being the national representative on the IASO.

In Spain and Portugal, several rulers were also well known for their obesity. Sancho I of Leon, Spain was given the title "The Fat" (*El Craso*). He ruled the state of Leon, taking over after the death of his older brother Ordono III. He reigned for three years before being forced into temporary exile, but regained control of the kingdom in 960. However, his obesity gave him so much trouble that he sought the help of some Moorish physicians from the court of Abdul-Rahman III of Cordoba. They suggested a strict diet and also the use of a distillation of herbs, which helped him, shed some of his weight. However, diet was to be a major problem, and legend has that he died eating a poisoned apple given to him by a subject.

The third king of Portugal, Afonso II, also gained the title "The Fat" (*O Gordo*). He was born just before the death of his grandfather, the founder of Christian Portugal, after whom he was named, and when he was a young man, he rapidly became obese, which was said to have developed from an affliction he had in his youth. This prevented him from leading his soldiers into battle against the Muslim Almohads, but this did not prevent his armies considerably enlarging the kingdom. The third king in the Iberian peninsula who had the title "The Fat" was Henry I, king of Navarre in northern Spain, and also Henry III, Count of Champagne.

Obesity in the general Spanish population came much later, with the introduction of sugar into the diet of many Spaniards in the early 16th century with sugar plantations using slave labor appearing in many parts of the Americas. To promote awareness, the Sociedad Española para el Estudio de la Obesidad operates in Spain and the Sociedade Portuguesa para o Estudo da Obesidade in Portugal. Dr. Jordi Salas of Reus is the Spanish representative on the IASO and Professor Galvão-Teles of Lisbon is the Portuguese representative on the IASO.

In Italy, there had been a long tradition of gluttony going back to the Roman Empire, with many later examples such as Rodrigo Borgia who became Pope Alexander VI in 1492. The only obese pope in recent times has been John XXIII, pope from 1958 until 1963, who has always been renowned for his personal kindness and sincerity, perhaps emphasizing the image of obesity as representing being jolly, friendly, and affable. Research in Italy is promoted by the Socita Italiana dell'Obesita with Professor Carlo Mario Rotella of the Dipartamento di Fisipatologia Clinica, Polivalente dell'Università, Florence, being the national representative on the IASO.

German history has seen a number of portly rulers, but few became as well known as Frederick I, king of Würtemberg, who was known as the "Great Belly-Gerant." The French Emperor Napoleon Bonaparte was said to have remarked that God had created Prince Frederick to show the extent to which the human skin could be stretched without. Fitz, the powerful German Jewish banker and art connoisseur, suffered greatly from obesity. Herman Goering was always portly, becoming obese during World War II. Helmut Kohl, chancellor of Germany from 1982 until 1998, and the man who presided over the reunification of Germany, was tall and physically large, occasionally being nicknamed the "pear" because of his shape, or appearing as a pear in cartoons.

In Germany, the Deutsche Adipostas Gesellschaft promotes obesity awareness, with the Swiss Association for the Study of Obesity running in Switzerland and the Austrian Obesity Association in Austria. The respective national representatives on the IASO are Professor Alfred Wirth of Teutoburger-Wald-Klinik, Bad Rothenfelde, Germany; Professor Yves Schutz, Institute of Physiology, Faculty of Medicine, Lausanne, Switzerland; and Professor Dr. Monika Lechleitner of the Department of Internal Medicine, University of Innsbruck, Austria.

Since World War II, the increasing prosperity in Western Europe, combined with more people taking up sedentary occupations and less physical exercise, has seen a rise in obesity in men, women, and more worryingly, in children. Other contributing factors have been genetic as well as certain medications especially steroids which have the side effect of considerably increasing body weight. Some have seen the prevalence of smoking, and indeed the various antismoking medications, as also causing a problem in weight gain. The rise in the obesity levels have resulted in an increase in Type 2 diabetes, heart disease, and many other problems associated with obesity, which has placed an added burden on the health

services in Western Europe. In some countries, the rise in obesity levels has been very marked. In the Netherlands, the obesity levels of boys and girls doubled between 1980 and 1997, and again from 1997 until 2004, with a threefold increase for some ages. In Portugal, a survey has shown that in the period 2003–05, some 38.6 percent of the population surveyed was overweight, and 13.8 percent were obese, with the result that the overweight and obese people make up over half the population.

To counter this, there has been the introduction of many forms of dieting, promoted on television, in newspapers, and in magazines. These have led to many people losing weight, others not putting on as much as they otherwise would have done, and a problem with eating disorders such as anorexia nervosa and bulimia nervosa. As well as diets, there have also been regular campaigns to encourage physical fitness in children, with compulsory school sports and physical activity. Some schools have also encouraged healthier diets, and a few in Britain and France have sought to prevent children bringing unhealthy food to school. With adults, food manufacturers have been keener to label goods as being "fat free" or "99 percent fat free" as a way of encouraging more people to eat healthier food. Many "fast-food" restaurants and cafés have also created healthier food options, with the "slow food" movement doing much to try to reduce the amount of fatty food such as french fries, potato crisps, soft drinks such as colas, and the like.

SEE ALSO: Europe, Eastern.

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Exercise

THE ENERGY COST of exercise is supported by one or more of three energy systems. Each of these is distinguishable from the other by the rate at which it can take food energy and convert it into usable chemical energy to fuel muscle contraction during exercise. Physical fitness and health are both related to the functional capacity of each of the energy systems. Improvements in health and fitness can be achieved only by improving the working capacity of the energy systems; this is accomplished by specifically designing exercise programs that target each energy system so that it is forced to work harder than accustomed. Weight control is best achieved through low-to-moderate intensity exercise training that can be sustained for 30–60 minutes or more on most days of the week. Factors that facilitate or impede newly established exercise behaviors should also be considered when planning for long-term success.

ENERGY SYSTEMS

To understand how exercise influences the energy stores (body weight and fat) of the body, one must understand how the energy demand of the body is met through the utilization of dietary nutrients. Most people are familiar with calories, or more appropriately kilocalories (kcal), the unit of energy. People also understand that the kcal we derive from food is utilized in the body to do whatever the body needs to do that requires energy, whether it be replacement of cell constituents, growth, or physical work. Our body transforms the chemical energy of food into electrical, mechanical, and heat energy to do what it needs to do. However, the fundamental form of energy the body deals with is not heat (kcal) but chemical. So, to understand how exercise affects energy balance in the body, it is easier to talk in terms of the chemical energy currency of the body, adenosine triphosphate (ATP), rather than heat (kcal). However, because food energy is usually thought of in terms of kcal, it often becomes confusing going back and forth between kcal and ATP. With this in mind, remember that all energy requirements of the body can be converted into ATP equivalents (e.g., 7.3 kcal = 6.022 x 1023 ATP).

ATP is the chemical compound that supplies the muscles with the energy they need to perform physical movement. Fats, proteins, and carbohydrates are

Regular exercise can not only help reduce weight, it is linked to a variety of other positive health benefits.

the food sources that supply the body with the needed energy to make ATP required by the muscles. Thus, we eat food that contains energy (usually measured in kcal), that energy is transferred to form ATP, and that same ATP is used in the muscles to give them the energy for muscle contraction.

The body has three basic chemical pathways in which energy from food can be converted into ATP: the ATP-phosphocreatine (ATP-PC) system, the lactic acid system, and aerobics. Each of these pathways is distinct from the other, but there are several crossover points where the pathways share chemical compounds, and all three pathways can be functioning at the same time in the same cell. The distinguishing characteristic for each of these ATP producing pathways is their power output, or rate of ATP production. The ATP-PC system can produce ATP at a rate approximately twice that of the lactic acid system and three to five times the rate of aerobics.

The link between exercise and the ATP producing pathways, then, is the demand for ATP, which is determined by exercise intensity. If exercise intensity is extremely high, the ATP-PC system will supply the needed energy. Conversely, if the demand for ATP energy in the working muscles is relatively low, the aerobic energy system will be used to support the energy needs of the exercise. If the energy demand is moderate, then the supporting energy system is the lactic acid system.

A second factor that determines which energy system will be used to support the demands of exercise is the duration of the exercise. The ATP-PC system has the capacity to support any and all intensities of exercise, but the resources available for the production of ATP through the ATP-PC system are limited to 15–20 continuous seconds. At high exercise intensities, once the ATP-PC resources become depleted, exercise intensity has to be reduced to continue exercise. At this point, the major responsibility for supporting the exercise power output demand shifts to the lactic acid system. This system also has limited resources, which will become exhausted after four to six minutes of continuous exercise at moderate-to-high intensity. At this point, the exercise intensity must be downshifted again to a low-to-moderate level for the aerobic system to supply the needed ATP to sustain the continued exercise. The aerobic energy system is the only system of the three that, for all practical purposes, has an unlimited source of energy resources from which to draw. In other words, fatigue during low-to-moderate intensity exercise is caused by factors other than running out of fuel. Furthermore, as the term implies, aerobic production of ATP requires oxygen, which must be delivered to the working muscles by the heart, lungs, and circulatory system. Consequently, lack of oxygen, not fuel, is often the limiting factor in prolonged exercise performance.

Protein is not used extensively to support the direct needs of exercise, and is therefore, generally ignored as a significant contributor in the scheme of the energetics of exercise. The ATP-PC system is fueled by fat or carbohydrate, but the mechanism for replenishing the energy stores in the ATP-PC system is through aerobics, thus making the "recharging" of the ATP-PC system resources impossible during intense exercise. Hence, once the stored ATP-PC is used during exercise (15–20 seconds) the ATP-PC system is inoperable until exercise ceases or exercise intensity is reduced to a level where aerobics can generate enough ATP to support the exercise demand as well as replenish the ATP-PC system.

Carbohydrate can be the fuel source for both the lactic acid system and aerobics, but if the exercise intensity demand for ATP is higher than that which aerobics can supply, exercise will last only as long as there

is carbohydrate available to fuel the lactic acid system (five to seven minutes). On the other hand, if the exercise intensity is low enough for the aerobic energy system to support the exercise energy demand, a mix of fat and carbohydrate will be used to fuel the working muscles. Fat will become the dominant fuel source as exercise duration extends beyond an hour and carbohydrate stores become scarce. Because body fat stores are abundant, even in the normal-weight person, low-to-moderate exercise can continue for hours.

EXERCISE, FITNESS, AND HEALTH

Exercise training can increase physical fitness as well as improve health or reduce disease risk. Physical fitness and health are often viewed as equivalent, but they are not. Fitness is a measure of the body's ability to perform, whereas health is a measure of the body's status of well-being.

There are four components of physical fitness: cardiovascular or cardiorespiratory capacity, muscular strength, muscular endurance, and flexibility. Other than flexibility, the components of fitness are directly related to the energy systems previously discussed. Cardiovascular or cardiorespiratory capacity is a measure of the body's ability to deliver oxygen to the muscles and utilize that oxygen, with the residuals of dietary fat and carbohydrate, to form ATP. Muscular strength is a measure of the muscle's ability to exert maximal force, whereas muscular endurance is a measure of the muscle's capacity to exert a submaximal force repeatedly. Cardiovascular or cardiorespiratory fitness relates directly to the aerobic energy system, muscular strength to the ATP-PC system, and muscular endurance to the lactic acid system.

Physical health is also related to the three energy systems. Chronic diseases such as heart disease, peripheral vascular disease, hypertension, and diabetes are related to cardiorespiratory capacity or functional aerobic capacity. Conditions such as osteoporosis and diabetes are related to muscular strength and muscular endurance. Consequently, exercise training, targeting a specific component of physical fitness or health, really consists of training or improving the capacity of one or more of the energy systems.

Aerobic exercise is any type of exercise that uses oxygen in the production of energy (ATP) to support the exercise. Because oxygen is transported to the exercising muscles through the cardiovascular system, aerobic exercise is also termed *cardiovascular* or *cardiorespiratory exercise*. Although other types of exercise have positive effects on health, aerobic exercise has proved to be the safest and have the greatest impact on health and disease risk reduction. Furthermore, because obesity is associated with many diseases (comorbidities) such as heart disease, diabetes, and hypertension, aerobic exercise is the best form of exercise for the obese person.

Aerobic exercise must meet certain criteria for the training effect to occur in the aerobic energy system. The type of exercise needs to be continuous, rhythmic, and use the large muscle groups. This type of exercise will ensure that a large volume of blood carrying oxygen will be continuously delivered to the exercising muscles so ATP can be produced through the aerobic energy system. Examples are walking, jogging, swimming, hiking, cycling, and rowing. Strength training exercises do not work for aerobic training because they are not continuous and the power output required for a strength training is too great to be met by the aerobic energy system.

For improvement in the energy system to occur, exercise training must be repeated at intervals for several weeks. The minimal guideline for the frequency of exercise to show a training effect is every other day, or three times a week. Each exercise session must last a certain length of time for the heart, lungs, and circulatory system to coordinate with the aerobic energy system in delivering newly formed ATP to the exercising muscles. The minimal length is 15–20 minutes. Because aerobic energy production has the slowest rate of ATP production (compared to the ATP-PC and lactic acid systems), the intensity of exercise must be low-to-moderate, otherwise the other systems will be used to support the exercise.

There have been several ways physiologists have designed to monitor aerobic exercise intensity, all of which are focused on having the cardiorespiratory system work at 40 to 80 percent capacity. Most often, exercise intensity is monitored by exercising at a level that elevates the heart rate to a predetermined level that correlates to 40 to 80 percent of cardiorespiratory capacity. A simpler method of monitoring exercise intensity is to exercise at a level just below that at which you cannot hold a conversation. Increasing the length, frequency, and intensity of exercise will ensure that adaptations in the aerobic energy system continue to occur.

Training of the ATP-PC energy system happens when repeated short bursts of muscle contraction near maximal output occur, and there is a rest interval long enough for the aerobic energy system to replenish the ATP-PC constituents in between bursts of energy production. Examples of exercises that train the ATP-PC system are short sprints of 100–200 meters at maximal pace, strength training to fatigue the muscle within several seconds, and explosive exercises like jumping. The lactic acid system is trained similarly to the ATP-PC system, but the exercise bursts should be a few to several minutes with the same amount of rest time in between repeated sets. An example would be repeatedly running 800 meters with a two-minute rest interval between sets.

EXERCISE AND WEIGHT CONTROL

Aerobic exercise is one of the few factors that are positively correlated with successful long-term reduced body weight maintenance in the previously obese. Previously obese individuals who maintain a reduced body weight report that regular exercise is the greatest factor responsible for their prolonged weight maintenance. Research has determined that for a previously obese person to maintain a reduced body weight through exercise alone, he or she needs to perform moderate intensity exercise for 80–90 minutes a day. This translates into burning approximately 700 kilocalories each day in exercise. Unfortunately, to expend 700 kilocalories in exercise, a person needs to walk or jog about seven miles. This amount of exercise is beyond the reach of most people and presents a tremendous barrier to long-term weight control. Moreover, many previously obese individuals do not have a history of being physically active or may have medical conditions that preclude or limit exercise participation. Thus, the expectation of people exercising enough to obtain and maintain a normal body weight seems unrealistic.

On the other hand, obese individuals can receive the benefits of regular exercise, even if their body weight does not normalize. Exercise lowers blood pressure, lowers cholesterol, and improves blood glucose control in the obese, even without weight loss. Furthermore, these health benefits of exercise are manifest in as few as seven days with only 30–60 minutes a day of moderate-intensity exercise.

The effects of exercise on body weight loss itself are not as great as one would expect. Weight loss of only about 0.2 kilograms (0.5 pounds) per week is typical of most exercise-only weight-loss programs. Furthermore, when calculated exercise energy expenditure is taken into account, people do not lose as much weight as was predicted through exercise programming. For these reasons, the suggestion for exercise prescription for weight loss is to focus primarily on promoting high-energy expenditure through a large volume of exercise. On the other hand, exercise intensity and duration are often reduced to levels below those recommended for improvement in physical fitness because of the orthopedic risks and limited functional capacities in the obese population. Obese individuals are therefore faced with the dilemma of how to optimize exercise-induced fat loss in the midst of limited exercise tolerance.

In an effort to solve this dilemma, many have used unproven assumptions about exercise and fuel use to promote what they claim to be optimal fat-burning exercise protocols. The biggest exercise prescription debate centers on exercise intensity. However, as explained above, exercise intensity and duration are closely related. As one increases exercise intensity, the capacity for exercise duration decreases, and as one diminishes exercise intensity, the capacity for exercise duration is increased. Parallel to this association between exercise intensity and duration is the association between exercise intensity and energy system or fuel use. High-intensity exercise derives a greater portion of the fuel source from carbohydrate, while low-intensity exercise derives a greater portion of the fuel source from fat. High-intensity exercise relies on the lactic acid and ATP-PC energy systems, whereas low-intensity exercise relies upon the aerobic energy system.

Because the biochemistry of the body dictates that fat can only be burned through the aerobic energy system, the logical choice for exercise programming is low-intensity exercise for extended duration. The traditional recommendation for weight-loss exercise is for the individual to exercise at 60 percent or below maximal aerobic capacity for as long as possible, and as frequently as possible. Evidence over the last several years, however, has shown that weight loss can result from nonaerobic exercise such as strength training. Nonetheless, the amount of weight lost through strength training is less than that achieved through aerobic exercise.

EXERCISE RECOMMENDATIONS

Because physical fitness is evaluated in terms of cardiorespiratory capacity, muscular strength, muscular endurance, and flexibility, the ideal would be for all people to participate in an exercise-training program to improve or maintain fitness in each of these categories. However, the majority of Americans do not participate in any type of regular exercise program. To promote the message of increased physical activity for all Americans, the American College of Sports Medicine and the Centers for Disease Control and Prevention have recommended that every adult should accumulate 30–60 minutes or more of moderate-intensity physical activity on most, and preferably all, days of the week. Moderate activity in this recommendation is defined as activity that elicits an energy expenditure of three to six times resting rate. In layman's terms, this means simple activities such as walking, gardening, playing golf, walking the dog, as well as incorporating more activity into one's lifestyle, such as using the stairs instead of the elevator, parking the car at the far end of the lot, and so forth. Those who follow these recommendations for activity will experience many of the health-related benefits of physical activity, but may not improve their fitness level or lose weight. Physical fitness, however, is not a dichotomy, but a continuum. Once physical activity becomes a part of one's lifestyle, higher levels of fitness than recommended above can be achieved by participating in a more structured exercise program.

Improvements in cardiorespiratory fitness, or aerobic capacity, are directly related to two principles of exercise training: overload and specificity. The overload principle states that an organ, tissue, or system will improve its functional capacity only if it is exposed to a load to which it is not normally accustomed. Repeated exposure to this overload, or exercise stress, causes an adaptation, which improves functional capacity. The principle of specificity states that the training effects derived from an exercise program are specific to the organ, tissue, or system being overloaded. The application of these two principles means that specific muscles, tissues, or energy systems will improve their functional capacity only if the exercise prescription is structured to overload those very same muscles, tissues, or energy systems. The achievement of the desired training effect is therefore accomplished through an exercise prescription that specifies the proper type, frequency, duration, and intensity of exercise required to overload the muscles, tissues, and/or energy system wherein the specific training adaptation is desired.

The overall objective of exercise participation for the overweight person is to bring about a physiological change that will improve the health status of the individual as well as reduce weight and prevent future disease. Most people must overcome several obstacles or barriers to exercise before exercise participation becomes part of their lifestyle. Thus, the art of exercise prescription is the successful integration of exercise science with behavioral techniques that result in long-term program adherence and attainment of the individual's goals.

The overload principle can be applied to the cardiorespiratory system in two ways: pressure overload and volume overload. Pressure overload is found when peripheral resistance in the circulatory system is increased and the heart has to beat harder to overcome this resistance. This condition causes hypertension or high blood pressure and is an unhealthy and dangerous way to overload the cardiorespiratory system. Strength training or weight lifting also causes pressure overload, which is another reason why weight lifting is not the preferred exercise choice for the obese person. The healthy way to overload the system is through volume overload. In volume overloading, the heart muscle is overloaded by pumping a larger volume of blood than that to which it is accustomed. Volume overloading is achieved best when the exercise uses large muscle groups over prolonged periods of time in activities that are rhythmic and continuous. Thus, the type of exercise necessary for improving cardiovascular function or aerobic fitness is one that is continuous, rhythmic, and uses the large muscle groups. Examples of good "aerobic exercises" are walking, jogging, cycling, dancing, or endurance games.

The duration of exercise should be 20–60 minutes of continuous or intermittent (10 minutes minimum) exercise accumulated throughout the day. As discussed previously, the duration of the exercise that can be sustained is dependent upon exercise intensity. Low-to-moderate intensity exercises can be maintained for a longer period of time than high-intensity exercise. The best exercise for the overweight person attempting weight loss is an exercise of low-to-moderate intensity and long duration.

The exercise stimulus must be applied repeatedly to achieve and maintain the desired adaptation in the car-

diorespiratory system. The minimal frequency of activity for improving fitness is three times a week. However, for weight loss, daily exercise is recommended.

The final criteria to meet for overloading the cardiorespiratory system during exercise training relates to the intensity of the exercise. To achieve the benefits of an aerobic training, one needs to exercise at an intensity of 40 to 80 percent of maximal aerobic capacity. Individuals who are severely obese and unconditioned should exercise at the lower end of the intensity range (40 to 60 percent), while those who are accustomed to exercise can exercise at the higher intensity of the range (60 to 80 percent). The best indicator available outside the laboratory for estimating aerobic intensity during exercise is heart rate. However, most laypeople and some exercise professionals do not understand the principles underlying monitoring heart rate during exercise and make erroneous assumptions about heart rate and the aerobic energy system. An easier way to monitor exercise intensity is to use rating of perceived exertion. It must be remembered, though, that the relationship between rating of perceived exertion and the aerobic energy system is valid only if the exercise is prolonged and aerobic in nature—meaning that the type of exercise is continuous, rhythmic, and uses the large muscle groups.

The ideal level of intensity for aerobic exercise training and weight loss is where the rating of perceived exertion describes the exercise stress on the whole body as fairly light to somewhat hard. The unconditioned individual who is unaccustomed to physical activity should start at an intensity that is fairly light. As the person becomes more accustomed to physical activity, the intensity can be elevated, the duration enlarged, and the frequency increased. Making these adjustments in the exercise program will increase the volume of exercise, increase the energy expenditure, and increase the likelihood of weight loss.

Adjustments in the exercise prescription should be made according to individual needs and any existing comorbidities or relative contraindications. If vigorous activity is planned, the exercise session should be preceded by a warm-up period and followed by a cool-down period, each consisting of several minutes of low-intensity aerobic activity (e.g., walking). Medical parameters such as heart rate, blood pressure, or blood glucose may need to be monitored for patients with existing comorbidities.

EXERCISE ADHERENCE

The two most frequently cited reasons that people give for not being able to maintain a healthy weight after a weight-loss attempt is that they returned to their old eating behaviors and discontinued exercise. Maintaining an exercise program is even more difficult than dieting in that most people will stop exercising before they give up their diet attempts. Thus, steps should be taken to dismantle the barriers to exercise and support newly established behaviors. Areas in which barriers to exercise should be explored and support systems designed include tangible barriers such as time, scheduling, and a place to exercise; emotional barriers such as using food as comfort or to soothe emotional distress; and developing social support networks. Last, an individualized system of self-monitoring should be designed. Self-monitoring might include such things as keeping an exercise log, recording feelings after exercise, and evaluating progress through prevalence of disease symptoms or reduction in disease risks.

SEE ALSO: American College of Sports Medicine; Fitness.

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Expanded Food and Nutrition Education Program

THE EXPANDED FOOD and Nutrition Education Program (EFNEP) is a federally funded program designed to provide nutrition education to low-income youth and families with young children. EFNEP's

aim is to improve the following for its target population: diets and nutritional welfare; nutrition knowledge; ability to select healthful food choices; food production, preparation, storage, safety, and sanitation practices; and financial and resource management to assist them in the achievement of healthy, active lifestyles.

Thus, nutrition education provided via EFNEP is defined in a broad sense, that is, in addition to teaching topics traditionally considered to be nutrition education, programs may provide participants with educational opportunities to assist them in more effectively managing their resources, gardening, food preservation, personal development, and/or weight management. Local EFNEP programs are granted considerable autonomy in their approaches used to accomplish the program's aims. This allows for ample consideration of cultural factors that may be apparent both in the teaching and learning styles of people hailing from varied backgrounds.

Created in response to President Lyndon B. Johnson's 1964 War on Poverty, EFNEP is the oldest federally supported nutrition education program in existence in the United States. Support for EFNEP's development and implementation included legislation to initiate the program via a pilot program, provide national funding on an ongoing basis, and train EFNEP nutrition education staff.

EFNEP is administered by U.S. Department of Agriculture's Cooperative State Research, Extension, and Education Service (CSREES) through funding distributed to U.S. Land-Grant Universities/Institutions in all 50 states, both of those funded in 1862, as well as those funded in 1890, and the following U.S. territories: American Samoa, Guam, Micronesia, Northern Marianas, Puerto Rico, and the Virgin Islands.

Federal legislation mandates that "to the maximum extent practicable" EFNEP be delivered via paraprofessional staff "hired from the indigenous target population." The employment of paraprofessional staff as peer nutrition educators has been an integral part of EFNEP since its inception. Paraprofessionals are those who lack a baccalaureate degree in the subject matter under which their work falls and who provide services under the supervision of a degreed professional. In the 1960s, paraprofessional staff lines were developed in the social, educational, and health fields as part of the "New Careers" movement, a movement whose goals were to provide the unemployed and underemployed career training and jobs as a means to help them out of poverty, to use a cost-effective strategy to meet the increased demand for human services workers that had been created by the "War on Poverty," and to enhance service delivery by utilizing the inherent skills and life experience knowledge of those residing in the community.

EFNEP paraprofessional educators remain peer educators who share ethnicities, languages, food customs, food access, and values, as well as common understandings of community beliefs and barriers to nutrition education. They demonstrate an empathy, sensitivity, and responsibility for the needs of those living in their communities. Thus, paraprofessional–participant trust, caring, reciprocity, and respect characterize EFNEP education.

These factors enhance emotional support and assist in the identification of participant problems, particularly with regard to sensitive participant issues, for example, their inability to maintain food security for their families. Program participants perceive their peer educators as being like themselves and therefore consider them to be credible information sources and persons in whom they are willing to confide information needed for effective program delivery.

In EFNEP, the paraprofessional educational model, sometimes referred to as the EFNEP model, has accomplished its aims. Many EFNEP paraprofessionals have moved beyond paraprofessional roles to assume professional positions. The program has consistently run on a cost-efficient basis and EFNEP is noted for its high degree of cultural competence due to the strong rapport that exists between its educators and their program participants.

EFNEP impact evaluation has been an important component of the program since its inception. Initially, dietary impact assessment was necessary to justify the experimental use of Section 32 funds. Legislators relied on data to demonstrate the ability of the program to effectively change its participants' dietary quality. This accountability proved worthwhile as has been demonstrated through EFNEP's continued funding.

Data collected to demonstrate dietary change included a survey of demographic characteristics and

a 24-hour food recall. Although no nutrient analyses were initially performed, the 24-hour food recalls were assessed under the "professional eye of the supervisor" to reflect the number of servings participants consumed from each of the four food groups before and after EFNEP intervention. Later, a Behavior Checklist was added to capture those behaviors not evidenced via dietary assessment, that is, food safety practices, and so forth. Initially, EFNEP programming with adults was performed via individualized, in-home learning. However, beginning in the mid- to late 1980s, due to economic considerations as well as safety concerns, group presentations became the most widely used method of program delivery, particularly in urban areas.

In 1980, the Progression Model, which progresses participants through the program to "graduating" from the EFNEP, was implemented. The model uses the results of the 24-hour pre- and postdiet recall and Behavior Checklists to assess the learning needs and progress through the program. Although revised and updated since its initiation, this now-computerized program evaluation model remains in place. Graduation standards, including the number of classes that must be completed by each graduating program participant, are set at the local program level.

Throughout the years, the EFNEP model has embraced collaborations with community agencies and researchers. Through collaborative efforts, local programs have participated in many research endeavors.

SEE ALSO: Department of Agriculture; Department of Health and Social Services; Federal Initiatives to Prevent Obesity; Food Stamp Nutrition Education Program.

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External Controls

THE CONCEPT OF external control is in comparison to that of self-control. Often external control is used in the context of the equation: (Internal) Self-Control + External Control = Weight Control.

People with an inability to self-regulate an action such as smoking, consuming alcohol, or overeating often turn to external controls for assistance. An external control is something that is out of the person's control that directly affects their particular action. For example, a family member throwing away cigarettes is an external control for smoking. An external control for over-eating may be having access to a limited amount of food. Several popular weight-loss programs use just such an external control.

An early example of external controls can be found in a group of Portuguese monks of the Middle Ages who consciously built a kitchen door narrow enough to keep out all but the slender monks.

A chief external control for youths is a parent figure dictating how much food the child will consume. Some data hints that people whose portions were decided by parents, i.e., children who grew up being told to "finish their plates," have more trouble aligning adult food intake with hunger than people who were allowed to determine their food doses as youths.

Imposing external controls requires determination. Some people move to metropolitan locations that force them to walk more, rather than dwell in suburbs. Others take activity-focused vacations. Recruiting family members and spouses to help limit temptations and access to food has repeatedly been shown to aid people in reducing their over-eating. Individuals who are supported in their endeavors to control their eating are less likely to feel depressed about their actions and also less likely to relapse into overeating.

With recent interest in overeating, obesity, and dieting, the desire for a solution to overeating has inspired entrepreneurs. For example, it is commonly known that the body takes fifteen to twenty minutes to recognize a state of satiety; in 1987 a patent was filed for a device that is effectively a microphone to amplify sounds made in the gastrointestinal tract when enough food has been consumed, to warn the eater before the standard physiological fifteen to twenty minutes. Another device filed under the same patent was to be internally embedded and would send a radio

signal to an external processor once enough nutrients have been consumed. These two devices would act as an external control to alert the eater when enough food has been eaten, thus avoiding over-eating.

External controls can range from subtle to extreme. For example, limiting the stress in one's life might relax the need to find solace in food. Extreme cases of imposing external controls include wiring one's teeth shut or undergoing gastric bypass surgery, colloquially known as "stomach stapling".

Gastric bypass surgery shunts food from a smaller-sized stomach to further down the small intestine than normal (the shunt joins the jejunum). In this way, the stomach is much smaller and therefore fills up more easily (an individual who has undergone gastric bypass surgery can consume about the amount of food that would fit in a shot glass). Additionally, the first part of the small intestine (duodenum) is circumvented, limiting caloric absorption. Downsides to this surgery include extreme pain and nausea if too much food has been consumed, and possible under-absorption of vital nutrients such as iron and vitamin B-12. Deficiency in vitamin B-12 is particularly frequent in individuals with gastric bypass because a gastric secretion is necessary for absorption of this vitamin. Therefore, not only smaller portions of food must be eaten, but specific foods and supplements must be consumed in order to obtain proper nutrition.

Although an extreme external control, gastric bypass has helped many morbidly obese people take control over their eating. It is meant for only those individuals who have been obese for extended periods of time. Individuals cannot have a drinking problem because the excessive liquid intake would cause extreme pain.

Finally, it is imperative that individuals undergoing gastric bypass not have uncontrolled depression, as often the psychological strain of such a surgery and the immediate physiological results are a burden. Alongside the fact that individuals will no longer be able to turn to food for comfort, they will initially feel low in energy due to limited energy intake as well as muscle weakness; depression is therefore a common side effect of the surgery.

Although also a drastic external control, jaw wiring is a much less invasive procedure than gastric bypass because there is no surgery and therefore no anesthesia involved. Jaw wiring involves bonding brackets onto the top and bottom teeth, on both sides of the mouth, and then wiring those brackets together. An individual's teeth do not touch, but the jaw cannot be opened to allow food intake. Therefore, a strictly liquid diet is imposed. As with gastric bypass surgery, care must be taken to ensure proper nutrition; an additional side effect to jaw wiring is difficulty with speech. People usually keep their jaw wired shut for approximately six months. Individuals must also face social reactions to the wires, which can be difficult.

SEE ALSO: Food Intake Patterns; Jenny Craig; Self-Esteem and Obesity; Sensory-Specific Satiety; Stress; Weight Watchers.

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Familial Lipodystrophies

LIPODYSTROPHY, OR ABNORMAL distribution of body fat including subcutaneous lipoatrophy, which is the loss of fat located under the outermost layer of skin on limbs or the trunk, and fat accumulation in the face, back, or intraabdominal area were observed in some patients and their families in the 1970s. The inherited lipodystrophy was referred to as Kobberling-Dunnigan syndrome or familial lipodystrophy. Various metabolic abnormalities and comorbidities of obesity were observed in familial lipodystrophy.

Type 1 familial partial lipodystrophy (FPLD1), Kobberling type, causes lipoatrophy in the limbs, with a normal or increased fat distribution on the face, neck, and trunk. FPLD2, Dunnigan type, causes lipoatrophy in the limbs and trunk, with fat accumulation in the neck and labia. FPLD3 has similar clinical phenotypes as the other familial lipodystrophies, but is due to a mutation on a different gene than FPLD1 and FPLD2.

Familial lipodystrophy is considered an autosomal-dominant disorder, which means that one nonsex gene from a parent carrying the mutated gene is required to show the physiological features of familial lipodystrophy.

Metabolic abnormalities, also seen in obesity, in familial lipodystrophy include diabetes mellitus, insulin resistance, hypertriglyceridemia, low plasma high-density lipoprotein, acanthosis nigricans, hypertension, dyslipidemia, and cardiovascular disease. Diabetes mellitus and insulin resistance are conditions that affect the individual's body to utilize glucose as energy. Acanthosis nigricans is a brown overpigmentation of the skin, due to excessive production of insulin.

Magnetic resonance imaging (MRI) can show the distribution of fat in familial lipodystrophy. Genetic tests can be used to examine the gene that is mutated. A pedigree chart, which is a chart mapping the genetic traits of a family, may also be helpful in the assessment of the condition.

The treatment of metabolic conditions of familial lipodystrophy has been similar to the metabolic conditions of obese individuals. Peroxisome proliferator-activated receptor (PPAR) gamma agonists are used for insulin resistance and diabetes in both familial lipodystrophy and obesity. Leptin treatment, a protein that influences energy metabolism and appetite, and gastric bypass surgery, which manipulates the nutrient intake from the gastrointestinal tract, are being investigated for the treatment of familial lipodystrophy. Both treatments are also being tested on the treatment of obesity.

SEE ALSO: Genetics; Genomics.

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Families of Eating Disorder Patients

THE FAMILIES OF patients with eating disorders share several characteristics; some characteristics are present before the eating disorder onset and others are a consequence of it. Twin and family studies show that eating disorders run in families. Below, the family characteristics, consequences, and aggregation of anorexia nervosa (AN), bulimia nervosa (BN), binge-eating disorder (BED), and night eating syndrome (NES) are discussed.

FAMILIES OF PATIENTS WITH ANOREXIA NERVOSA

Research on the families of patients with AN has focused on several factors, such as the interpersonal family dynamics, siblings, and the impact of AN on caregivers. In general, families of people with AN have been characterized as rigid, avoidant of disagreement, and sometimes lacking in interpersonal boundaries.

Researchers have noted that the presence of siblings can affect the experience of a person with AN, either by contributing to the illness or by helping the patient cope with his or her disorder. Siblings can influence people with AN directly and also indirectly through parents.

The impact of AN on families appears to be profound. Several studies have shown that the eating disorder becomes the central focus of family life. Parents feel that they must put their life on hold to care for their child with AN, and they worry about the negative impact of the eating disorder on their child's heath and social functioning.

While an individual suffers from an eating disorder, the interpersonal dynamics within the patient's family are also important. There is also evidence suggesting that several eating disorders may have a genetic link.

Caregivers of patients with AN have reported a number of negative emotions that are often centered around feelings of helplessness. Parents often report guilt, as they believe they have contributed to their child's disorder. The effect of guilt and worry on the caregiver, often the parent, has been shown to lead to physical and mental exhaustion and sleep deprivation.

Several studies have shown that AN runs in families, and researchers are beginning to discover the genes, especially those related to serotonin functioning, that put one at risk for the development of this disorder.

FAMILIES OF PATIENTS WITH BN

Families of patients with BN have been characterized somewhat differently than families of patients with AN. In general, research suggests that there is less rigidity, less stability, and more disturbance in family functioning among families of patients with BN.

The consequences of BN for families are, in many ways, similar to those of AN. Like AN, BN can dominate family life. Caretakers of patients with BN report powerful negative emotions centering on feelings of helplessness, as well as a sense of responsibility for the development of the disorder. Caregiving for BN takes a toll on physical health and raises many practical concerns such as difficulties at mealtime, financial burden, and loss of social opportunities for both the carer and the individual with BN.

BN also affects family life when the individual with BN is the parent. Research has shown that depressive symptoms both during pregnancy and postpartum are common among women with BN.

Research from family, twin, and adoption studies shows that BN runs in families. Genetic and environmental influences on temperament, such as thrill seeking and impulsivity have been the focus of much research, as have the familial coaggregation of BN, substance use disorders, and mood disorders.

FAMILIES OF PATIENTS WITH BED AND NES

Less is known about the families of patients with BED and NES. Research has suggested that adolescents with BED experience disruptions in family cohesion; no studies to date have examined the family functioning of adolescents with NES.

Both conditions appear to run in families. Twin and family studies suggest that the heritability of BED is significant, at around 40 percent. The first-degree relatives of NES patients are five times more likely than the first-degree relatives of healthy controls to have NES.

In summary, eating disorders are influenced by both genetic and environmental family factors, and they significantly impact family functioning and caregiver well-being.

SEE ALSO: Anorexia Nervosa, Bulimia Nervosa, Binge Eating, Night Eating Syndrome

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Family Behavioral Interventions

OBESITY REMAINS A complex disorder requiring a multifaceted treatment approach. Behavioral intervention is frequently referred to as behavioral weight control, behavioral treatment, and/or lifestyle

modification. Behavioral intervention is a comprehensive approach to weight management that combines education and behavioral techniques. The Diabetes Prevention Program, one of the most impressive behavioral intervention studies, demonstrated a 7 percent decrease of initial weight. This amount concurs with the 5 to 10 percent recommended weight loss that the National Heart, Lung, and Blood Institute and the World Health Organization put forth. When treating overweight children, a family-based approach is critical.

Behavioral intervention for obesity is the first line of treatment preceding more aggressive methods such as pharmacotherapy or surgery. This method of treatment emphasizes a reduced calorie diet (e.g., 1,200 to 1,500 kilocalories per day), increased physical activity (e.g., 180 minutes per week), and modification in thoughts and behaviors. Psychoeducation is an essential adjunct in which patients are educated on the causes of overweight, energy balance, nutrition, and physical activity. The typical structure of behavioral interventions consists of 16 to 26 weeks of group or individual treatment lasting 60 to 90 minutes. Formally trained healthcare providers (e.g., dieticians, behavioral psychologists) deliver the intervention.

Key behavioral strategies include the following:

Self-monitoring: being aware of one's own behavior. For example, patients are asked to record meals, times of eating, physical activity, and weight so that they can identify difficulties or patterns and modify behavior. This is often acknowledged as the most integral technique.

Goal setting: patients learn to articulate specific, measurable, and realistic goals. Although weight loss is the primary objective, behavior goals (e.g., eating 1,300 calories/day, walking briskly for 60 minutes five times per week) are emphasized.

Stimulus control: patients learn to identify cues or triggers that are often associated with unhealthy eating habits. A classic example is going to the movies and eating popcorn or eating a snack when watching television.

Slowing the rate of eating: patients are encouraged to enjoy their food and eat less. Patients are encouraged to put their fork down or take sips of water while eating to allow their body to recognize satiation.

Regular eating: patients are instructed to eat at least three meals a day at regular times (e.g., breakfast, lunch, and dinner) as opposed to skipping meals and overeating at a later time.

Problem solving: the patient identifies difficulties and generates feasible solutions.

Relapse prevention: the patient prepares for situations that may come up in the future. The patient learns to identify high-risk situations to avoid, but is also equipped with skills to handle unavoidable difficult situations.

Nutrition education: patients are educated on healthy, well-balanced diets including items from the five basic food groups as outlined in MyPyramid.

Physical activity education: patients are educated on the benefits of exercise as well as how to increase both lifestyle activity and structured exercise.

Cognitive restructuring: patients learn to modify irrational, unrealistic, or self-deprecating thoughts. Patients identify negative cognitions and work to reframe them and adopt more positive thinking. For example, a patient may overeat and think, "I'll never be able to do this, I might as well just give up." The patient could reframe such a thought to be more positive: "I had a lapse today, but if I keep trying, I will improve."

Family behavioral intervention, or family-based treatment (FBT), is comprised of the aforementioned skills in addition to family involvement, parental control of the home environment (e.g., shopping for and preparing healthier foods, while eliminating junk food from the home), parental support, positive reinforcement, and modeling of desired eating and activity behaviors. Parents learn behavior management strategies to assist their children in adopting healthier behaviors. FBT can be delivered in a variety of methods targeting (1) the parent and child together in a session, (2) the parent and child in separate sessions that occur simultaneously, or (3) the parent alone without the child involved in treatment. A plethora of evidence exists for FBT in the treatment of childhood obesity. Len Epstein's group from the University of Buffalo has successfully demonstrated short- and long-term results of FBT for child obesity for three decades.

SEE ALSO: Behavioral Treatment of Child Obesity.

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Family Therapy in the Treatment of Overweight Children

IT IS COMMONLY recognized that the intervention of choice for overweight children is a change in lifestyle. As family environment serves as the main context of change in children's weight-related behaviors, family-based intervention is an integral part of treatment and prevention. While the goal for any overweight child is undoubtedly behavioral modifications in dietary and exercise behavior, it is also essential that families are equipped to facilitate such changes. Thus, family-focused treatments should incorporate components of behavioral parent training, but the efficacy of behavioral parent training can be enhanced with a broader focus that includes the family environment. Some families may thus benefit from family therapy, focused not on changing specific behaviors related to eating or exercise, but rather with an emphasis on basic parenting skills, the family's emotional climate, conflict resolution, and overall motivation in order to prepare parents to influence a change in their children's behavior.

Modification of eating behavior and reduction of sedentary behavior are the cornerstone of intervention for both achieving and maintaining weight loss in overweight children. Many have demonstrated that behavioral treatment is most beneficial when the family is involved as family behavior plays an important part in childhood obesity. In fact, in programs such as those developed by Maria Golan and colleagues, parents are viewed as the primary change mediator in weight-related interventions because their involvement is crucial for the induction of a healthy environment, modeling of healthy eating and activity patters, and improvement in the child's behavior and weight in the long term. While parents are often instructed to implement specific behavioral treatments and preventative measures designed to effect children's eating and exercise habits, the effectiveness of these practices is likely to vary depending on whether they're in the context of a healthy family environment.

Parenting skills have been shown to be particularly important determinants of behavioral outcome. Family therapy interventions targeting weak parenting skills in the treatment of childhood obesity are widely supported by existing research. Improvement in authoritative parenting style (parents are assertive, but not restrictive) might contribute to parents' ability to maintain a healthier environment and thus support healthy behavioral changes. Professionals caution that parents must learn to regulate but not control the environment, as parental control can interfere with a child's ability to attend to internal signals of hunger and satiety that serve self-regulation.

Solution-focused brief therapy (SFBT) and systematic family therapy have also been found to be useful in a wide range of contexts for overweight children. A central belief in SFBT is that children and their families may actually have most of the resources and strengths they need to solve problems. Therapy is utilized to highlight individual and family resources by solution building and to create goals in order to empower the family unit to change behavior toward a healthier lifestyle. Carl-Erik Flodmark and colleagues demonstrated that using family therapy focused not on changing specific behaviors related to eating or exercise but rather focused on the family's emotional climate and resources for coping still showed significant weight loss relative to controls. Moreover, they found that family therapy actually prevented the progression of obesity in older teenagers if treatment is started early.

A change in the entire family is believed to create more lasting change for a wide range of child and adolescent problems because every family member is involved in the change process and all members continue to exert synchronous influence on one another. However, some have cautioned that increasing the family focus beyond teaching parents to use behavior modification to change children's eating and exercise behaviors may actually reduce effectiveness, if parents are distracted from learning new eating and exercise

habits that have proven effective. While discrepancies remain regarding the most effective family-based intervention, it is evident that the family is in the strongest position to help or hinder healthy behaviors in the overweight child and must be utilized to facilitate challenges of treatment.

SEE ALSO: Family Behavioral Interventions; Parental and Home Environments.

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Fast Food

EATING OUT IS on the rise. The United States Department of Agriculture's (USDA's) food intake surveys indicate that between 1977 and 1996, daily caloric intake from food eaten outside of the home increased from 18 to 32 percent, with a 40-percent increase between 1987 and 2000 in the proportion of the population that reported eating three or more commercially prepared meals per week. Food-away-from-home expenditures have increased to account for about half of total food expenditures, and fast-food restaurants (defined by the North American Industry Classification System as restaurants offering limited waitperson service) account for nearly 40 percent of the away-from-home market. Furthermore, the rising consumption of restaurant fare has coincided with an increased prevalence of obesity, and several studies have reported a positive association between the frequency of eating at fast-food restaurants and body weight. In addition, a higher per capita number of restaurants has been positively associated with a higher body mass index (BMI), particularly among populations of lower socioeconomic status, who are already at an increased risk for obesity and its related complications. Although these associations do not prove causality, they raise important questions. Customers eating away from home are often confronted with large portions of foods that are higher in calories, saturated fat, and cholesterol compared to foods prepared at home and that may be more likely to contribute to weight gain or exacerbate obesity-related complications. Lack of knowledge about appropriate serving sizes and inadequate nutrition information may further complicate matters and lead to overconsumption of unhealthy foods. Spurred by public interest in health and nutrition, some segments of the fast-food restaurant industry have increased the number of healthy food options on their menus. Moreover, legislation has been proposed to require more extensive nutrition labeling of restaurant foods so that customers will be able to make informed choices about the nutrient content of the foods they select and learn how to incorporate away-from-home eating into a healthy diet that does not contribute to weight gain.

The rise in foods consumed away from home has been attributed to the increase in dual-income households in which both spouses work long hours and do not have time to prepare home-cooked meals. Fast-food restaurants provide a quick and inexpensive alternative. Indeed, by the late 1990s, dollar sales per fast-food outlet were comparable to those per full-service restaurants, and take-out and drive-through traffic now account for more than 60 percent of all fast-food sales, underscoring the importance of convenience. Further, several studies have reported a decline in cooking skills among younger generations, suggesting that even if time were available for food preparation, these individuals might still rely heavily on the restaurant industry. However, foods prepared

away from home tend to contain more calories per meal and are higher in saturated fat and cholesterol and lower in dietary fiber, calcium, and iron compared to foods prepared at home. Fast-food consumption has been inversely associated with dietary quality measures such as the USDA's Healthy Eating Index, a scoring system measuring how closely a diet meets USDA Food Guide Pyramid recommendations for grains, vegetables, fruits, milk, and meat/meat alternatives. One study found that individuals who obtained a higher percentage of food from fast-food restaurants had lower Healthy Eating Index scores for fruit, total fat, and saturated fat compared to those who obtained a smaller percentage of their meals from fast-food restaurants.

Further, the size of restaurant and fast-food meals has increased dramatically over the past few decades so that patrons who eat out frequently have increased access to low-cost, energy-dense foods. Portion sizes began to grow in the 1970s and have continued to increase, prompting restaurants to replace standard 10.5-inch dinner plates with 12.5-inch versions and automobile manufacturers to install larger cup holders to accommodate "supersized" beverages and the "grab-and-go" lifestyle that has many Americans consuming fast-food meals purchased at a drive-through window in their cars. Many of these larger portion sizes far exceed standard serving sizes, which are developed by the USDA and the U.S. Food and Drug Administration (FDA) based on the amount of a food believed to be customarily consumed. For example, a sampling of foods from popular fast-food restaurants found that chocolate chip cookies exceeded USDA serving size standards by 700 percent. Similarly, cooked pasta, muffins, steaks, and bagels exceeded the USDA standards by 480 percent, 333 percent, 224 percent, and 195 percent, respectively. In comparison, just a few decades ago, at the dawn of the fast-food era, portion sizes were considerably smaller: current portions of hamburgers, french fries, and soda are two to five times larger than the portions offered when these items were first introduced to the public. These original sizes have become today's "small" portions, with "large" and "supersize" options often providing double or triple the calories found in the smallest size. In many fast-food restaurants, meal combinations offer customers a discount for selecting the largest portions of an entrée ("supersizing" the meal) in combination with a large beverage and side item. For example, in 2002, at one fast-food restaurant chain, an additional $1.57 could buy a supersized cheeseburger meal with 600 calories more than the next largest size. An additional $0.64 could buy 330 more calories of french fries, and an extra $0.37 could buy about 450 more calories of some soft drinks. In contrast, few, if any, fast-food restaurants offer a reduced price for smaller portions of the same entrée. Thus, in many fast-food restaurants, choosing a healthy meal based on pricing and available nutrient information is difficult and at times even seems irrational when considering price alone. Moreover, the exact size of these "supersized" choices may vary by region; in Europe, portions served at chain fast-food restaurants are markedly smaller than those served in the United States. For instance, in 1998, the largest soda served by a major fast-food restaurant in London, Rome, and Dublin weighed the same as the second-to-largest size served by the same franchise in the United States.

Large portions and low prices make fast food attractive to consumers, but the health costs may be much greater.

These products are attractive to both the food industry and to customers: larger packages draw attention, increase sales, and cause profits to rise, and consumers believe that they are getting a "bargain." Of course, these portions also contribute additional calories, causing concern that increased caloric intake coupled with factors such as physical inactivity may be contributing to the obesity epidemic in the United States. Research indicates that on-the-spot portion size estimation is difficult for many people and that when served a larger portion, people often eat more. In one study, portions of baked pasta served in a cafeteria-style fast-food restaurant were varied on different days; customer intake was determined by discreetly weighing each dish before and after the meal, and customers were asked to evaluate the appropriateness of the portion size they were served and compare the amount of food they consumed to their usual intake. Those served a portion of baked pasta that was 50-percent larger than the standard portion consumed 43 percent more energy than those who were served the smaller portion. Further, customers who were served the larger portion consumed more of the side dishes accompanying the entrée and overall, consumed an additional 159 calories during the meal compared to those consuming the standard portion. When asked to rate whether the portion size was appropriate and to compare the amount of food they had eaten with the amount they usually consumed at lunch, customers responded similarly regardless of which portion they had consumed. Similar findings have been reported with other foods (e.g., popcorn, sandwiches, packaged snacks—when served a larger portion, consumers tend to eat more and often do not realize that the portion is larger.

Thus, portion size can affect intake, and lack of knowledge or misconceptions of appropriate serving sizes may lead customers to consume more food than usual when served a larger portion at a restaurant. In fact, a survey of more than 1,000 Americans conducted by the American Institute for Cancer Research (AICR) found that 30 percent of those responding said that the amount of food they eat is based on the amount of food they are served, and 43 percent of those who reported being above their ideal weight determined portion size this way. Moreover, 69 percent of respondents reported finishing their entrée when dining out, and 60 percent of those who reported "cleaning their plates" said they believed the portion they were served was "just right." However, not everyone is satisfied with oversized portions: 71 percent of men and 66 percent of women who reported leaving food behind when dining out described the portion as being too large, and almost seven out of 10 Americans who regularly leave some of their restaurant entrée uneaten take the leftovers home. Yet, even though many Americans are dissatisfied with these larger portions, only 1 percent of those surveyed by AICR were able to correctly identify standard serving sizes for common foods, and others have reported that consumers typically underestimate the fat, saturated fat, and calories provided by a restaurant meal and that actual amounts of these are often double what consumers predict.

Other factors, including race/ethnicity, age, and income, may also influence possible associations between fast-food restaurant use and obesity. This is of particular interest to health professionals because the prevalence of obesity and its associated complications is higher among African Americans and Latinos than in the general United States population. For instance, a survey of African Americans living in North Carolina reported a mean BMI of 31.3 kgm-2 for those who usually/often consumed fast food versus a mean BMI 28.6 kgm-2 for those who rarely/never consumed fast food. In addition, those who reported consuming fast food usually/often were less physically active than those who rarely/never consumed fast food. Those who ate at fast-food restaurants more frequently also had higher intakes of total and saturated fat and lower intakes of vegetables.

Frequent fast-food restaurant use was also positively related to perceived difficulty in ordering healthy foods at restaurants and preparing healthy meals. Moreover, a weight-maintenance study of women aged 20–45 reported that an increase of one fast-food meal per week was associated with an average weight gain of 1.5 pounds above the average weight gain of 3.75 pounds that occurred during the three-year study. Further, more frequent fast-food restaurant use was significantly associated with younger age, lower income, and non-Caucasian ethnicity. Emerging evidence suggests that fast-food intake and insulin resistance may be related. For example, a 15-year follow-up study of 18- to 30-year-olds reported that participants who used fast-food restaurants more

than two times per week at baseline and at the 15-year follow-up gained an extra 10 pounds of weight and had a 104 percent greater increase in insulin resistance, measured by fasting serum insulin.

Access to fast-food restaurants affects neighborhoods disproportionately and may be greatest among populations with the greatest risk of obesity and related chronic diseases. In both the United States and England, fast-food restaurants are more prevalent in lower-income neighborhoods. Specifically, one analysis revealed significant correlations between the number of residents per fast-food restaurant and obesity prevalence, reporting that states with a lower prevalence of obesity tended to have more residents per fast-food restaurant and that income, physical inactivity, and the percentage of African Americans and males were also significant predictors of obesity. Others have found that predominantly black neighborhoods and neighborhoods with a lower socioeconomic status have more than double the exposure to fast-food restaurants and fewer full-service establishments compared to neighborhoods that are predominantly

Caucasian or have a higher socioeconomic status. Moreover, among Latino women, fast-food restaurant use has been positively associated with higher acculturation, which has been positively associated with less healthy diets that are high in total and saturated fats and lower in total fiber. The types of foods served at fast-food restaurants may also vary by neighborhood socioeconomic status. Several studies evaluating the availability of "healthy" menu options such as fruit, salads, or lean meats that are baked or broiled instead of fried reported that lower income neighborhoods were less likely to have fast-food restaurants that served these healthier choices. Thus, in communities for which the risk of obesity may already be high, environmental factors such as a high density of fast-food restaurants may make following a healthy lifestyle and maintaining a healthy weight even more difficult.

Possible associations between fast-food consumption and obesity among young people are also a concern. BMI has been positively associated with fast-food restaurant use in adolescents, and obese adolescents are more likely to remain obese as adults. Several mechanisms may be responsible. First, as with adults, intake of energy from fat and saturated fat is often higher among children and adolescents when eating at a restaurant than when eating at other locations (e.g., home, school, day care, or friends' homes). Second, television advertising of soft drinks, fried foods, and snacks may contribute to overconsumption of foods that are high in calories and total fat and low in healthy nutrients. Among adolescents, each one-hour increase in television viewing corresponded with an increase of about 0.2 to 1.4 servings per week of commonly advertised foods, including fast-food meals, sugar-sweetened beverages, and baked sweet snacks. Perceived barriers to healthy eating have also been positively linked to fast-food consumption among adolescents. In a study of students attending a large metropolitan high school, adolescent males and females who reported frequenting fast-food restaurants more than three times during the past week were significantly more likely to report that healthy foods tasted bad and that they did not have the time to eat healthy foods and were not interested in doing so.

Consumer demand is a key factor in determining which foods remain on the menu, but attempts by fast-food restaurants to introduce new choices have not always been successful, despite consumer requests for healthier options. Slow sales of lower-fat versions of hamburgers and other fast-food items caused several major fast-food chains to remove these items from the menus soon after their introduction in the 1990s. However, some changes, such as the switch to vegetable oils for deep-frying in response to consumer concern over the health effects of saturated fat, have been successful. Further, one study evaluating a restaurant-based nutrition program to substitute lower-fat menu items for traditional higher-fat options found that customers (n = 686) were significantly more satisfied with the lower-fat items than with the regular versions.

Others have found that many fast-food restaurant customers would support legislation requiring nutrition labeling on restaurant menus. However, although many fast-food restaurant chains have compiled nutrition information about their foods to distribute to patrons, the information is not always readily available in stores, limiting consumer access to point-of-purchase nutrition facts that might help them make healthy choices. Paradoxically, providing additional nutrient information does not always lead to healthier choices. Providing college students with information

on fat content caused students in one school to select items with less fat from the campus cafeteria but to ignore the amounts and types of foods they were eating. Further, because every consumer has different nutrient needs and because restaurant meals are often customized to each consumer's preferences, precise nutrition information is difficult to provide. However, nutrient information appears to have a strong influence on the attitudes, intentions, and choices of customers ordering food, and easily-accessible nutrient information may help fast-food restaurant patrons navigate the range of choices presented to them and select options that will help them maintain a healthy weight.

The search for ways to increase the number of healthy menu options in fast-food restaurants has led some to consider the factors that motivate marketing executives and leaders in the fast-food industry. Clearly, sales and profit are key issues. Further, menu items that are easy to prepare have an additional advantage in that they can be made by less-skilled employees and do not require restaurants to hire workers with advanced culinary training, thereby reducing costs and increasing profit margins. Thus, fast-food restaurants that offer healthier menu items marketed toward a health-conscious clientele do so only when they believe it is worthwhile: A sufficient number of diners must order healthier fare and influence friends and family to do the same for these items to be attractive for a restaurant operator to sell. However, many restaurant operators feel that diners want to indulge rather than choose healthy fare when eating out and, consequently, choose to emphasize healthy items only as they fit with the menu (e.g., serving a meat entrée advertised as "low carbohydrate" without potatoes). Even if they believe there is sufficient demand for healthier foods, other barriers aside from cost may stand in the way. Some are concerned that marketing foods as "healthy" will scare customers, and instead use words such as "fresh" or "flavorful" to emphasize that taste is not compromised. Others face obstacles to storing fruits and vegetables, limited shelf-life, lack of storage space, and the seasonality of items.

Despite these concerns, many fast-food restaurant operators are open to new ideas for preparing and marketing healthy foods and to collaboration with the produce industry in the development of methods for packing and distributing high-quality fresh produce that will ensure a longer shelf-life and reduce spoilage. In addition, incentives, such as tax breaks or price supports might encourage fast-food restaurants to serve healthy options at the same or a lower price than less healthy foods, and some have also recommended legislation requiring fast-food restaurants to include point-of-purchase nutrition information in an effort to motivate customers to choose healthier options. At the federal level, the proposed Menu Education and Labeling Act would require restaurant chains with more than 20 outlets to have nutrient information available for customers, and several states are considering legislation that would require restaurant franchises with 10 or more outlets nationwide to provide information on the number of calories and the amount of key nutrients in their foods. In response to these initiatives, the FDA has also begun considering national standards for the provision of nutrition information by restaurants. Labeling of fast-food and full-service restaurant menus might inform customers about the nutrient contents of the foods for sale and help them to choose foods that are healthy. Further, nutrient labeling might prompt restaurants selling foods that are high in total fat and calories to alter their products so that they are more nutritious and competitive with restaurants that already sell healthier options.

Customers may wonder how fast-food restaurants compare nutritionally to full-service establishments. Although full-service restaurants may be more likely to offer healthy options such as plain vegetables and salad bars, fast-food restaurants are more likely to display nutrient information, but in some establishments, it is not posted near the menus and is difficult to find. Compared to sit-down restaurants, fast-food establishments often encourage patrons to "upgrade" their meal to a larger portion for greater value, although "all-you-can-eat" availability, another potential barrier to healthy eating, is more likely to occur at full-service restaurants. Sharing a meal typically does not cost extra at a fast-food restaurant; however, some full-service restaurants have a plate charge for shared entrées, although others offer customers less expensive, smaller portions for certain menu items. Finally, although fast-food restaurants may be more likely to offer a children's menu, full-service restaurants are more likely to serve 100 percent fruit juices and 1 percent or nonfat milk. Thus, both types of restaurants can provide some healthy choices to the savvy customer. Clearly, there is a need for health education that will equip consum-

ers with the knowledge and skills needed to evaluate the options that confront them when eating out and enable them to correctly distinguish healthier choices from less healthy options.

SEE ALSO: Eating Out in the United States; Food Labeling; Increasing Portion Sizes.

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Fat Acceptance

FAT ACCEPTANCE, ALSO known as size acceptance, fights discrimination based on weight, works to improve the self-esteem of heavy people, counters negative media stereotypes, strives to change limited notions of what body types are considered attractive, and encourages doctors to provide care focused on health rather than weight.

WEIGHT DISCRIMINATION

Weight discrimination is widespread in our culture. Prospective employers often refuse to hire large-size people, especially in physical jobs or jobs where employees interact with the public. Size acceptance advocates argue that skills and performance should be the only criteria for evaluating applicants and employees. Large people are subject to harassment by their employers, are kept in jobs beneath their abilities, and are often demoted or fired because of weight prejudice. They are systematically denied health and life insurance or forced to pay higher premiums, and are often turned down as applicants to educational institutions.

Fat acceptance advocates suggest that discrimination based on weight should be eliminated.

Public facilities are inaccessible to many large people because of turnstiles, narrow armchairs, and small bathrooms. Airplanes, trains, and buses often have seating that is uncomfortably tight for larger-than-average people. Plus-size people suffer bullying, harassment, and derision from strangers, and insults, criticism, and more subtle forms of disrespect from friends and family. The size acceptance movement encourages fat people to stand up for themselves, and works toward laws that protect people from discrimination based on weight.

HEALTH ISSUES

Much of the bias against fat people is based on the idea that weight is within a person's control. Size acceptance advocates counter this misconception by pointing to the long-term ineffectiveness of all currently known methods of weight loss. The vast majority of dieters will regain the weight they lost over time, so it is rational to conclude that the diet is failing, not the people following the diet. The size acceptance movement also questions the premises that being heavy is always unhealthy and that weight loss always makes you healthier.

Some of the health risks of overweight, advocates say, are due to the stress of prejudice. Biased attitudes on the part of healthcare practitioners also contribute to health problems, especially when plus-size people delay care for their medical problems out of fear of diet lectures and judgment. Medical offices often lack armless chairs, larger gowns, bigger blood pressure cuffs, and other equipment that would make the facility size friendly. In addition, larger patients are often unable to obtain diagnostic tests such as magnetic resonance images (MRIs), computerized tomography (CAT) scans, X-rays, and bone density scans because few facilities have the necessary medical equipment to fit larger bodies or accommodate higher weights.

Advocates teach people to communicate with their doctors and to insist on respectful, weight-neutral healthcare. The fat acceptance movement also joins with the health at every size movement to support a nondiet approach to health and healthcare, advocating eating according to internal cues of hunger and satiety, finding forms of physical movement that are pleasurable, and working for health improvement without focusing on weight loss.

MEDIA IMAGES

The size acceptance movement works to change societal attitudes about body image and attractiveness. The movement celebrates size diversity, noting that ideal body type is a concept that changes over time and across cultures. In many cultures, a woman's desirability increases with her weight. Artists such as Auguste Renoir, Gaston Lachaise, and Fernando Botero chose heavy women as their models. Lillian Russell, who weighed 240 pounds, was considered the most beautiful woman in the world at the turn of the 19th century.

Size acceptance teaches people to look at media images and advertising critically. Quick weight-loss schemes are very likely to fail over time, despite the alluring before-and-after pictures. Instead of emulating the unrealistically thin media models, fat acceptance advises people to overcome their internalized oppression and improve their self-esteem through support, education, and camaraderie with other self-accepting people.

ADVOCACY

While fat acceptance is a grassroots movement, there are several organizations that represent the goals of the movement, including National Association for the Advancement of Fat Acceptance (NAAFA), International Size Acceptance Association (ISAA), and Council on Size & Weight Discrimination (CSWD). Social and support groups are found in various regions as well as on the internet, including many Big Beautiful Woman (BBW) dating services. Fat lesbians come together in NOLOSE, and fat gay men gather in Girth and Mirth. Professionals who use the size acceptance, nondiet approach, are represented by Association for Size Diversity and Health (ASDH).

Activist groups and individuals have protested discriminatory policies through the use of books, videos, letter-writing campaigns, letters to the editor, e-mail lists, and websites. Advocates say that helping individuals cope with instances of unfair treatment, or simply living as a proud fat person, can make the point that worthy people come in all sizes.

Detractors say that encouraging fat people to feel better about themselves will keep them from trying to lose weight. Implicit in this theory is the erroneous notion that stigma and blaming will create behavioral change. Fat acceptance advocates see antiobesity initiatives as an attempt to eradicate fat people as a class. Size diversity is natural to human beings, and should be accepted and celebrated. Treating people as second-class citizens because they do not conform to society's ideals is wrongful discrimination.

SEE ALSO: Council on Size and Weight Discrimination; National Association to Advance Fat Acceptance; Non-Diet Approaches; Weight Cycling and Yo-Yo Dieting.

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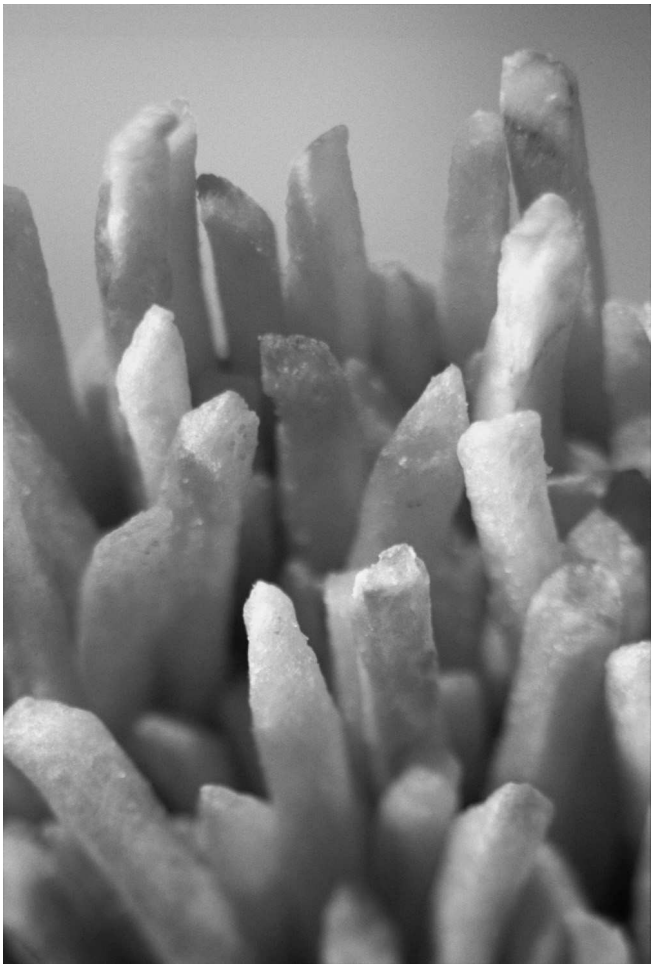
Fat Intake

THE ROLE OF dietary fat and its effect on human adiposity has been controversial for some time. Low-fat and very-low-fat diets were thought to reduce the percentage of body fat in the past. More recently, popular high-fat diets were found to have a similar effect. The growing obesity epidemic along with confusing information regarding fat intake recommendations for weight loss and weight maintenance has led to a renewed interest into the role that dietary fat plays. Recent research suggests that a low fat (about 20 percent of calories from fat) to a moderate-fat diet (about 35 percent of calories from fat), has little to no effect on the percentage of body fat as has been observed long term.

Dietary fat supplies about nine calories per gram, which is more than twice the amount of energy that is found in carbohydrates and proteins. Low-fat diets have been recommended, because of fat's higher energy density, as a preferred way to reduce the amount of total calories consumed. However, many foods containing moderate to high amounts of fat may lead to greater satiety, and consequently reduced food consumption.

Dietary fats are essential to many biological functions and are an integral part of cell membranes. In addition, essential fatty acids from dietary fats are needed in the absorption of the fat-soluble vitamins A, D, E, and K as well as in the absorption of carotenoids. Dietary fats occur naturally as saturated, monounsaturated, and polyunsaturated fatty acids.



While fat is important to a balanced diet, ingesting too much fat can have dramatic and serious health effects.

Trans fatty acids are unsaturated fatty acids, which have been chemically altered to become partially hydrogenated. Cholesterol, even though not a fatty acid, is a fatty substance essential in many biological pathways. Cholesterol is synthesized in the liver as well as obtained through animal sources in the diet.

Saturated fat, which is predominantly found in animal products, and *trans* fats, which are frequently used in vegetable shortenings, cookies, and snack foods, has been linked to an increase in LDL-cholesterol levels. Elevated LDL cholesterol is frequently associated with obesity, and may increase the potential for coronary heart disease. As a general guideline, the fat intake from saturated fat should be less than 10 percent of the total caloric intake, with energy from *trans* fats reduced to a minimum and a dietary cholesterol intake of less than 300 milligrams/day. Some

research suggests even lower intakes of saturated fat and cholesterol to treat adults with elevated LDL cholesterol levels. Foods high in saturated fats are usually solid at room temperature and include, for example, cheese, whole-milk products, beef, as well as fried foods, butter, ice cream, and desserts.

The main source of calories from fat should come from polyunsaturated and monounsaturated fatty acids, which are generally liquid at room temperature. Good sources of monounsaturated fatty acids include vegetable oils, such as olive and canola oil, as well as nuts. Evidence exists that a Mediterranean-style diet, which is high in monounsaturated fatty acids, may be effective in reducing the prevalence of the metabolic syndrome and its associated cardiovascular risk.

Polyunsaturated fatty acids can be subdivided into omega-3 and omega-6 fatty acids. Omega-3 and omega-6 fatty acids are essential fatty acids that differ in their chemical structure with regard to the position of the first chemical double bond. Omega-6 fatty acids are found in vegetable oils including soybean, corn, and safflower oil. Good sources of omega-3 fatty acids include flaxseed, walnuts, shellfish, and fish high in fat, such as salmon, mackerel, and trout.

The ratio of omega-6 to omega-3 fatty acids is another key factor when discussing fat intake. The typical Western diet is lower in omega-3 fatty acids, and studies have shown that the ratio between omega-6 and omega-3 fatty acids is as high as 16:1. Some research is suggesting that a ratio of 1:1 to 4:1 of omega-6 to omega-3 fatty acids may be beneficial in the treatment of chronic conditions such as cardiovascular disease or rheumatoid arthritis.

A diet for weight loss and weight maintenance should have about 20 to 35 percent of calories from fat. The total intake of saturated fatty acids should be no more than 10 percent of the daily caloric intake, with the remainder coming from mono- and polyunsaturated fatty acids. Of the polyunsaturated fatty acids, an increased intake of omega-3 fatty acids from fish sources and flaxseed is recommended.

SEE ALSO: Carbohydrate and Protein Intake; Overall Diet Quality.

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Fat Taste

TRADITIONALLY, FAT IN foods was thought to be perceived through textural cues, such as creaminess and oiliness. The thought that fat might have a detectable taste was only recognized in the past few years, first in animal studies, and then more recently in humans. Previous literature identified only five basic taste sensations: sweet, sour, bitter, salty, and umami (L–amino acid). However, recent studies point to the possibility of a receptor system for fat, hence a fat taste. Dietary fat was thought to be perceived exclusively by trigeminal (textural) and olfactory cues, but it seems that taste plays an important role in fat sensing as well. Fat taste may be one mechanism that has developed to promote increased consumption of the nutrient. This could lead to health problems, since preference for high-fat foods and excessive fat intake in humans increases risk for numerous diseases, such as obesity, diabetes, heart disease, hypertension, and cancer.

Taste perception occurs when molecules of food or drink contact and activate taste cells, which are assembled into buds and located at various sites, such as the tongue, throat, and upper esophagus. Fats or their constituents may stimulate some or all of these buds. Although most dietary fat consists of triacylglycerol (TAG), long-chain fatty acids (LCFAs), which are released from TAG by the enzyme lingual lipase, allow for some free fatty acids to be liberated in the oral cavity where they can activate taste cells. In fact, when lingual lipase is blocked, rodents show a considerable reduction in there fat preferences, presumably because the ability to taste fats has been blocked.

One protein that has been implicated as a potential fat taste receptor is CD36, also known as FAT for fatty acid translocase. CD36 is expressed in human and rodent taste papillae, as well as a variety of other tissues, and it functions to transport long chain fatty acids (LCFA) across the cell membrane. Once LCFAs are delivered inside the

cell, they are able to transmit information about fat taste stimuli to the brain. When rodents were made CD36-deficient, the high palatability of LCFA-enriched solutions was completely abolished, so in a sense, the absence of CD36 made these animals unable to form fat preferences. This finding has yet to be replicated in humans.

Fatty acid taste may involve more than the oral cavity. Research suggests that fat is tasted in postoral regions of the gastrointestinal tract, such as the intestine, and other areas of the body, such as adipose tissue, skeletal muscle, heart, and brain. The expression *fat taste* is used to define the detection of fatty acids even beyond the oral cavity because the CD36 taste transduction mechanism seems to be universal and apply to the other fat-sensing tissues besides the GI tract.

Fat taste may activate the metabolic response to dietary fat. In humans and animals, sham studies following oil capsule ingestion demonstrated that oral fat exposure stimulated larger postprandial elevations in circulating TAG than exposure to nonfat stimuli or no oral stimulation. Follow-up studies revealed that the taste component, and not the olfactory stimulation, from the oral exposure of fat was the metabolic cue for the rise in circulating TAG.

SEE ALSO: Blood Lipids; CD36 and FAT (Fatty Acid Transporters); Fat Intake; Palatability.

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Fatty Acid Transport Proteins

FATTY ACID TRANSPORT proteins (FATPs) are a group of proteins that play a critical role in moving fatty acids throughout the body. Some FATPs deliver fatty acids to cells of organs such as muscle or heart, while other FATPs move fatty acids around within the cell to specific organelles such as the mitochondria or the nucleus. Once inside the cell, fatty acids may be used for many cellular activities including making energy or affecting gene expression. Traditionally, it was believed that fatty acids entered a cell by diffusion (without any help). However, over the course of the last decade, FATPs were discovered and their critical role is beginning to be fully understood. They are often considered to be a rate-limiting step in fatty acid metabolism. Only the liver and fat tissue can synthesize fatty acids, a process known as *de novo synthesis*, and thus all other organs, such as the heart, kidneys and muscle depend on fatty acids to be delivered which highlight the critical role that FATPs play in metabolism.

There are many different types of FATPs. Some of these include fatty acid translocase (FAT/CD36), FATP, fatty acid binding protein-plasma membrane (FATPpm), fatty acid-binding protein-cellular (FATPc), and Acyl CoA synthase. FAT/CD36 is located within the cell membrane and plays a critical role in transporting fatty acids inside a cell. It is necessary for normal fat and sugar metabolism in the body. There have been six FATPs discovered to date in humans that are known as FATP1–FATP6, respectively. They also are responsible for bring fatty acids inside a cell. It is believed that they change the structure of the fatty acid, which allows it to pass through the cell membrane easier. FATPpm helps facilitate the movement of fatty acids through the cell membrane, while FATPc helps move fatty acids within the cell to their target (i.e., the nucleus or mitochondria). Acyl-CoA synthase is an enzyme that was primarily believed to play a role in making fatty acids. Recent studies have found that it may also have a role in transporting fatty acids within the cell.

There are some known medical conditions that may be related to abnormal FATPs. Some studies that have removed FATPs in mice have been used to identify several metabolic defects. Mice that do not have any FAT/CD36 cannot perform fat oxidation and tire out very quickly during exercise. Conversely, mice that have extra FAT/CD36 levels (through genetic alterations) can sustain exercise for longer time than mice that have normal FAT/CD36 levels. Mice that lack FAT/CD36 are also less likely to become obese while consuming a high-fat diet. In humans, FAT/CD36 deficiencies exist at high levels in some populations. Three to 10 percent of Asians and 5 to 18.5 percent of African Americans have a deficiency of FAT/CD36, while only 0.3 percent of Caucasians are

deficient. The lack of FAT/CD36 is associated with abnormal fat metabolism in these patients and increase risk for heart disease (including enlarged heart), insulin resistance, and Type 2 diabetes mellitus.

SEE ALSO: Metabolic Disorders and Childhood Obesity.

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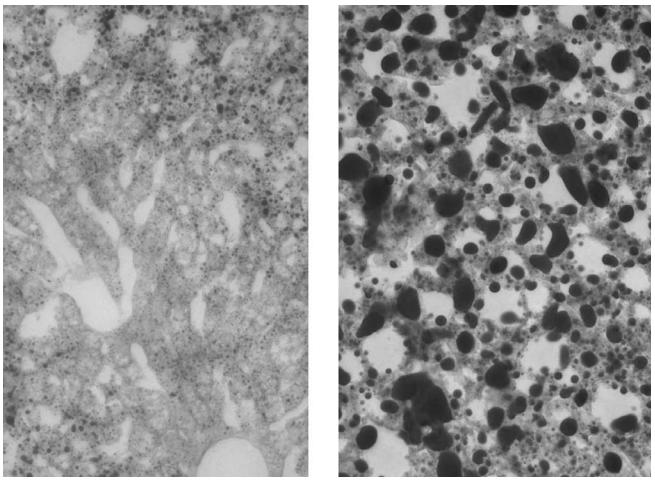
Fatty Liver

FATTY LIVER DISEASE (FLD) results from an accumulation of lipids within liver cells and ranges from fatty liver alone (steatosis) to fatty liver associated with inflammation (steatohepatitis). FLD may be related to alcohol (generally above 10 grams per day) or not (nonalcoholic fatty liver disease [NAFLD]). If steatohepatitis is present without a history of alcohol use, the condition is termed *nonalcoholic steatohepatitis* (NASH). Steatohepatitis may progress to cirrhosis as scar tissue and regenerative nodules, with accompanying loss of liver function, replace normal liver cells.

FLD is now the most common cause of abnormal liver function blood tests in the United States. Approximately 25 to 35 percent of the general population has fatty liver changes with a higher incidence among females. NAFLD is found in over 80 percent of people who are obese. NASH is the most common cause of liver disease among U.S. adolescents, and is identified in over 50 percent of patients undergoing bariatric surgery.

The most common association with FLD is metabolic syndrome, a combination of insulin resistance, central obesity, high blood pressure, and/or abnormal cholesterol. Other factors, such as medications, alcohol, pregnancy, and inborn metabolic abnormalities due to enzyme deficiencies, may also contribute.



A normal mouse liver at left, compared to a mouse with a large accumulation of fat in the right panel, seen in the dark droplets.

Most people with FLD have no symptoms and are diagnosed only after having an abnormal blood test. Others may experience persistent fatigue, upper abdominal discomfort, or in cases of cirrhosis, jaundice, abdominal distension due to fluid, swelling of the feet, breast enlargement in males, or menstrual disorders in women.

Physical examination often reveals enlargement of the liver and sometimes the spleen. Patients with alcohol-related FLD may have other problems such as muscle wasting, pancreatic inflammation, or nerve damage. Laboratory studies should include blood tests for liver function, lipid levels, and viral hepatitis. Radiological studies such as ultrasound or computed tomography (CT) may help identify FLD, but a liver biopsy is required to establish the diagnosis.

Studies of patients with NAFLD have shown that 30 percent progress to cirrhosis, 30 percent remain stable, and 30 percent improve over a three-year period without treatment. Abstinence from alcohol is the key to therapy in alcohol-related FLD. A low fat, American Diabetes Association diet as well as a weight-loss goal of one to two pounds per week helps reverse changes in NAFLD. No established treatment is available for NASH although several treatment strategies have been suggested which include exercise, weight training, lipid-lowering medications, and weight loss.

SEE ALSO: Gastrointestinal Disorders.

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Federal Initiatives to Prevent Obesity

FEDERAL INITIATIVES TO prevent obesity represent collaborative and integrated efforts to address overweight and obesity as major public health issues within the United States. Key initiatives include those from the Surgeon General, Department of Health and Human Services (HHS), National Institutes of Health (NIH), and the Office of the President. The initiatives are designed to accomplish their objectives via mechanisms such as research, education, or funding of other programs, and are often designed to be integrated with state and local government initiatives and community-based programs. Many initiatives focus on improving nutrition and increasing physical activity based on scientific evidence that too many calories and too little exercise are major contributing factors to overweight and obesity. In addition, the majority of the initiatives contain objectives for reducing and preventing obesity-related conditions such as cardiovascular disease, diabetes, and hypertension.

THE SURGEON GENERAL'S CALL TO ACTION TO PREVENT AND DECREASE OVERWEIGHT AND OBESITY 2001

This Call to Action from the Surgeon General summarizes the health risks, epidemiology, economic consequences, and disparities in prevalence of overweight and obesity, and emphasizes the benefits of weight loss. The five key principles of the plan are: (1) promoting recognition of overweight and obesity as major public health problems, (2) encouraging healthy nutrition choices and physical activity, (3) identifying effective and culturally appropriate interventions, (4) encouraging environmental changes to help prevent overweight and obesity, and (5) developing and enhancing relationships between public and private sectors for plan implementation. The Call to Action focuses on five key settings (families and communities, schools, healthcare, media and communications, and the workplace) and utilizes the CARE framework (Communication, Action, Research, and Evaluation) to accomplish objectives within each key setting.

STRATEGIC PLAN FOR NIH OBESITY RESEARCH

The *Strategic Plan for NIH Obesity Research*, published in 2004, is a multidimensional plan for addressing obesity and represents a collaborative effort between the NIH, other federal agencies, public and private organizations, and community members. It provides a guide for coordinating clinical and population-based obesity-related research activities across NIH institutes. The plan was developed by the NIH Obesity Research Task Force (cochaired by directors from the National Institute of Diabetes and Digestive and Kidney Diseases [NIDDK] and the National Heart, Blood, and Lung Institute [NHBLI]) and is organized into four research areas. The first, research toward preventing and treating obesity through lifestyle modification, is intended to identify behavioral and environmental factors contributing to childhood and adult obesity and to test potential intervention strategies.

The second, research on preventing and treating obesity through pharmacologic, surgical, and other medical approaches, focuses on biological factors responsible for regulating fat storage, appetite, and the balance between energy intake and expenditure. The third area, breaking links between obesity and associated health conditions, uses research to investigate biological connections between obesity and related diseases. The fourth area, crosscutting research topics, includes technology, translational research (applying research results in the real world), education and outreach efforts, and research on obesity-related health disparities.

HEALTHY PEOPLE 2010

Healthy People 2010, issued by HHS, is a set of 10-year evidence-based national health objectives focused on increasing quality and years of happy life and eliminating health disparities. The initiative is supported by partnerships with other federal agencies, businesses, communities, tribal organizations, and state and local governments. The Healthy People 2010 plan identifies 10 leading health indicators, including the prevalence of overweight and obesity and physical activity levels, which will be used to measure the nation's health over time. Specific goals of Healthy People 2010 include increasing the proportion of adults at a healthy weight and reducing the proportion of children and adolescents who are overweight or obese. Healthy People 2010 objectives related to nutrition and weight will also be used to measure progress in implementing recommendations from the Dietary Guidelines for Americans.

DIETARY GUIDELINES FOR AMERICANS

Produced by a partnership between HHS and the U.S. Department of Agriculture (USDA), the dietary guidelines are a set of nutritional recommendations intended for use by educators, nutritionists, healthcare providers, and policy makers. The guidelines, updated every five years, are intended to promote health and prevent disease by improving nutrition and increasing physical activity. Scientific evidence demonstrates that poor diet and lack of physical activity are the main contributors to overweight and obesity. Key recommendations in the guidelines are grouped into nine interrelated focus areas that when utilized together are designed to help Americans make healthier food choices, consume fewer calories, and become more physically active.

REPORT OF THE FOOD AND DRUG ADMINISTRATION (FDA) WORKING GROUP ON OBESITY

The mission of the FDA is to promote and protect public health. The agency is addressing the obesity epidemic via recommendations made by its Obesity Working Group in March 2004 targeting food labeling, enforcement, education, restaurants and industry, therapeutics, and research. The FDA is promoting the message that "calories count." Specifically, the FDA will evaluate plans for revising food labels (to more clearly highlight calories and serving size), enforce accuracy in serving size information, and educate consumers about using label information when making food choices. Restaurants will be encouraged to provide nutritional information for consumers. In addition, the FDA plans to enhance research coordination with public and private sector partners to study food consumption patterns related to overweight and obesity, and to develop interventions influencing consumers to make healthier food choices.

OBESITY EDUCATION INITIATIVE (OEI)

The OEI was implemented by the NHLBI in 1991 and serves as an educational tool for professionals and the general public. The overall goal of this initiative is to reduce coronary heart disease risk (and associated morbidity and mortality) by reducing the prevalence of overweight and obesity and increasing physical activity (in alignment with objectives from Healthy People 2010). The initiative employs two strategies. The first, a population approach, targets the general public and includes the Hearts N' Parks community-based program encouraging healthy eating and physical activity (in partnership with the National Recreation and Park Association). The second, a high-risk targeted approach, focuses on individuals who are at high risk for, or are experiencing, adverse health effects or medical complications from overweight or obesity. To help health professionals manage weight loss and weight maintenance in their high-risk patients, NHLBI and the North American Association for the Study of Obesity (NAASO) developed *The Practical Guide: Identification, Evaluation, and Treatment of Overweight & Obesity in Adults*.

WEIGHT-CONTROL INFORMATION NETWORK (WIN)

WIN is an information service established in 1994 by the NIDDK. WIN provides science-based information on obesity, weight control, physical activity, and nutrition via fact sheets, electronic newsletters (*WIN Notes*), and other publications distributed to health professionals, members of the public, the media, and Congress. Statistics on overweight and obesity, including definitions, prevalence, and economic costs, are available through WIN. As a part of WIN, the NIDDK developed Sisters Together: Move More, Eat Better, an initiative encouraging African-American women to maintain a healthy weight via diet and exercise.

WE CAN! WAYS TO ENHANCE CHILDREN'S ACTIVITY AND NUTRITION

We Can! is a national education program for preventing overweight and obesity in children aged 8–13 by emphasizing improved food choices, increased physical activity, and reduced screen time (the amount of time spent watching television, playing video games, and sitting at the computer). The program helps parents and caregivers, the primary influencers of this age group, to understand the risks of obesity-related health conditions, choose healthy foods to limit calories from fat and sugar, and encourage at least 60 minutes of daily physical activity for children. We Can! offers resources for home and community settings including posters, advertisements, parent handbooks, and community tool kits. We Can! represents a collaboration between NHBLI, NIDDK, National Cancer Institute (NCI), and National Institute of Child Health and Human Development (NICHD).

THE PRESIDENT'S HEALTHIER US INITIATIVE

This broad federal initiative (with several subinitiatives) is designed to help Americans live longer and healthier lives by eating a nutritious diet, making healthy choices, and being physically active every day. Through a partnership with HHS and the Centers for Disease Control and Prevention (CDC), the Steps to a Healthier US Cooperative Agreement Program (Steps Program) provides funding to states, city, and tribal entities to implement health-promotion and disease-prevention programs targeted specifically toward reducing obesity, diabetes, and asthma. These health-promotion and disease-prevention action plans are required to be aligned with state-level plans, other CDC programs, and federal initiatives. The President's Challenge encourages individuals of all ages to become more physically active. The program provides materials specifically tailored to educators, advocates, communities, and corporate groups. The President's Council on Physical Fitness and Sports (PCPFS), composed of volunteer citizen advisers to the President and HHS, has set a goal of getting 20 million more Americans to exercise and become physically fit.

SEE ALSO: National Heart, Lung, and Blood Institute; National Institutes of Health; NIDDK; State and Local Initiatives to Prevent Obesity.

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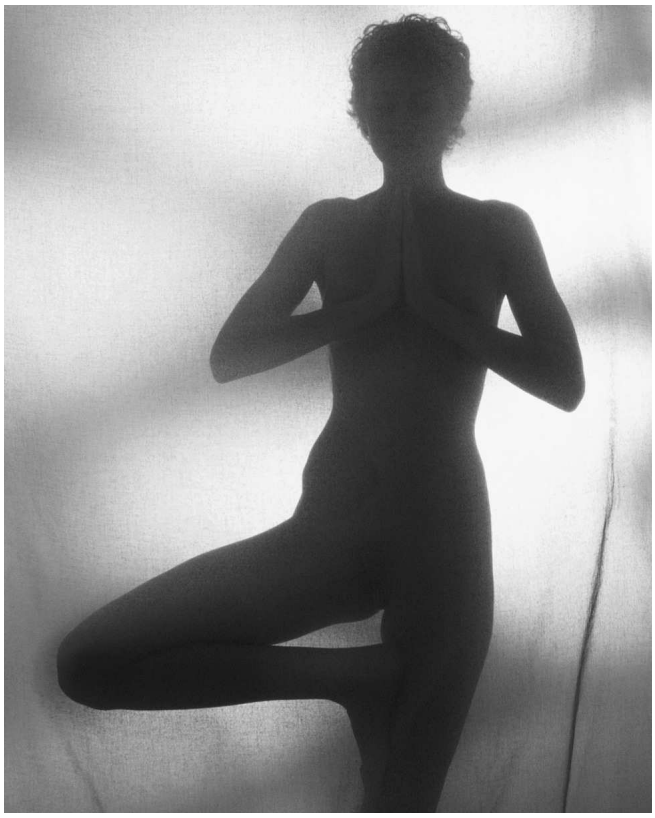
Feminist Perspective and Body Image Disorders

BODY IMAGE DISORDERS (BIDs) encompass the clinical diagnoses of anorexia nervosa, bulimia nervosa, and body dysmorphic disorder, all of which feature significant distress surrounding the way in which the body is experienced. Prior to discussing feminist theory and BIDs, it is important to note that traditionally the term *disorder* is underemphasized in feminist literature. Feminists often argue against focusing on diagnostic categories used to label individuals because it is viewed as "pathologizing," or assuming behavior is deviant. This matter has been the subject of extreme controversy between proponents of the medical model and those adhering to feminist interpretations. Feminists contend that when the beauty ideal compels women to feel shame instead of self-acceptance of the curves that make up the natural female form, body distress (however extreme) should not be



Feminists believe that the obsession with the female body has played a role in preventing women from becoming powerful.

interpreted as pathological. In fact, it is a normative and culturally sanctioned response to an unrealistic thin ideal that has persisted since the second wave of the women's movement. This is not suggesting that feminists do not view BIDs as debilitating; instead they are seen as representations of a very problematic cultural phenomenon. Hence, this body of literature emphasizes decoding the meaning underlying BIDs as opposed to simply treating the symptoms.

The core feature of a feminist perspective is its consideration of social context and the critical examination of oppressive social structures that influence an individual's experience of his or her body. Because problems with body image and eating are about nine times more likely to occur in women than men (making eating disorders the most gendered psychiatric diagnoses in the *Diagnostic and Statistical Manual of Mental Disorders* [DSM-IV]), the focus in the feminist literature is generally on the experiences of women. Feminists view the body as a cultural text on which messages about social norms, power structures, and systems of control and privilege are inscribed. Feminists recognize that despite two women's movements, the position of women in our culture remains secondary to that of men. That is, men still largely control decision making and the allocation of resources. Many feminists view the cultivation of an unnaturally thin ideal body for women as a means of maintaining this power differential between sexes. History has shown that during times when the social roles of women were changing and new opportunities were afforded to them, beauty ideals became increasingly thin. For instance, in the 1920s, during the women's suffrage movement, the rail-thin boyish figure glamorized by the famous flapper became the ideal for female beauty. Given this evidence, some feminists believe that an insidious obsession with the female body arose to prevent women from becoming too strong and influential.

Feminists decry the dieting industry and the objectification of the female body in Western culture as culprits in women's battles with their weight. Ubiquitous messages mandating weight loss if one's body does not fit the ideal causes an erosion of a woman's sense of self-worth and confidence. Furthermore, the energy spent buying into the mass media's "cult of thinness" costs women an enormous amount of productivity. In many cases, when a dieting woman reaches the slender body ideal, maintaining it requires hours of daily exercise and existence in a constant state of hunger. This makes it less likely that she will have time or energy to make a significant contribution in whatever roles she is upholding. Additionally, the woman who diets is often overwhelmed by irritability as she fights the physical weakness resulting from denying her body the food it needs to function optimally. Eventually, the body's propensity to return to its natural size wins, and if she has internalized the media-driven thin ideal, her self-esteem consequently plummets. This is a vicious cycle that occurs frequently among women. It allows for the perpetuation of the status quo with regard to gendered power structures and represents the primary contention of the feminist perspective and body image disturbance.

Several theories have arisen out of the feminist literature on BIDs. One of these theories involves the notion of the superwoman ideal. It was recognized that after large numbers of women developed careers and professional identities, their roles as housewives,

mothers, and caretakers in general were not lightened in response. Negotiating the demands of added responsibilities in their careers to their already full-time jobs in the domestic domain place many women in situations where they were leading superhuman lives. Juggling these multiple roles successfully involves letting go of the need to be perfect and asking for (as well as receiving) help. However, women who endorse the superwomen ideal require that they have the *perfect* job, husband, children, home, and appearance. Besides holding exceedingly high standards for herself, the woman endorsing the superwoman ideal is fiercely independent and refuses help in accomplishing her multiple tasks. Furthermore, she equates the attainment of a thin body as a representation of her autonomy. Both theoretical work and empirical research have underscored endorsement of the superwomen ideal as a characteristic of women who struggle with BIDs. Perfectionism and an unrelenting need for independence are the cardinal problems leading these women down the path toward an eating disorder.

Feminist theory should not be misunderstood as *blaming* men for women's problems with their bodies. While the feminist perspective places the sociocultural context at the root cause of BIDs, this fact does not preclude the plausibility of the biological argument. Many feminist researchers view biological and environmental factors as inextricably linked in the cause of eating disorders. A common etiological metaphor is that genetics load the gun and environment pulls the trigger. In short, a woman with biological vulnerability in a culture that objectifies females bodies and experiences that lead her to internalize the thin beauty ideal is a recipe for the development of extreme forms of otherwise sanctioned weight-related distress.

SEE ALSO: Body Dysmorphic Disorder; Body Image; Eating Disorders and Gender.

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Fenfluramine

FENFLURAMINE IS AN antiobesity medication that came into the U.S. market in the early 1970s. Fenfluramine is the racemic mixture of dexofenfluramine and levofenfluramine. Dexfenfluramine is the active form and is effective in one-half the dose of the racemic mixture. Fenfluramine is currently available in many countries throughout the world. Although structurally very similar to the noradrenergic drugs, these agents act by different mechanisms. Fenfluramine and dexfenfluramine are highly selective serotonin agonists that enhance serotonin release into nerve synapses and inhibit its reuptake. In a major study by the National Heart, Lung, and Blood Institute, the noradrenergic agent phentermine was combined with the serotonergic drug fenfluramine.

The concept of this medication was to increase the levels of serotonin, an important neurotransmitter in food intake regulation, in the brain. Serotonin is a monoamine neurotransmitter that is synthesized in serotonergic neurons in the central nervous system (CNS). Serotinon is believed to play an important role in the regulation of anger, aggression, body temperature, mood, sleep, vomiting, sexuality, and appetite. An increase of serotonin levels in the brain are thought to depresses the part of the CNS that regulates mood and appetite, leading to a decrease in food intake and subsequent weight loss.

However, this medication was withdrawn from the U.S. market in 1997 after reports of heart valve disease and pulmonary hypertension, a high pressure of blood moving into the lungs. Heart valves also have serotonin receptors, which regulate their growth. The distinctive valvular abnormality seen with fenfluramine is a thickening of the chordae tendinae in the heart. These are the thin, fibrous chords that lie within the heart muscle wall and contribute to the support of the tricuspid and mitral valves. These abnormalities seen in patients who take the medication have led it to be removed from use.

Consequently, the Food and Drug Administration (FDA), acting on new evidence about significant side effects associated with fenfluramine has asked the manufacturers to voluntarily withdraw both treatments for obesity from the market. The FDA recommended that patients using fenfluramine products stop taking them. Users of these products should contact their doctors to discuss their treatment. There is

currently a seemingly endless pursuit for a medicinal cure to the drastic obesity epidemic in the United States and abroad. Further research with other serotonergic medications is under way and may change treatment measures for obesity in years to come.

SEE ALSO: Central Nervous System; Dexatrim; Drug Targets that Decrease Food Intake/Appetite; Drugs and Food; Food and Drug Administration; Serotonergic Medications.

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Fertility

Fertility can be affected by a variety of factors. Included in these are both obesity and eating disorders.

FERTILITY IS DEFINED as the ability to conceive after one year of unprotected coitus. Fertility is a multifactorial process. Researchers have linked impaired fecundity in women with a body mass index (BMI) in excess of 30kg/m2. Obesity is now the number one cause of infertility in the United States.

Obesity is associated with three alterations that interfere with normal ovulation and weight loss improves all three: (1) increased peripheral aromatization of androgens to estrogens; (2) decreased levels of sex hormone binding globulin (SHBG) resulting in increased levels of free estradiol and testosterone; and (3) increased insulin levels that can stimulate ovarian stromal tissue production of androgens.

Polycystic ovarian syndrome (PCOS) is the most common cause of anovulatory infertility in the United States, affecting 5 to 10 percent of women of reproductive age. PCOS is characterized by chronic anovulation with biochemical and/or clinical evidence of androgen excess and without evidence of adrenal, thyroid, or pituitary glands diseases. PCOS is not only a disease of infertility but is also associated with an increased risk of diabetes mellitus, stroke, hyperlipidemia, coronary artery disease, and endometrial carcinoma. PCOS is associated with, and aggravated by, obesity. Seventy-five percent of women with PCOS suffer from infertility, which is primarily related to anovulation and a high rate of early miscarriage. In obese women with PCOS, weight loss as a means of improving insulin sensitivity is often a primary goal, because loss of 5 to 10 percent of body weight over six months is sufficient to reestablish ovarian function in more than 50 percent of patients. Aside from weight loss, several treatment options exist including those that target insulin resistance (metformin, thiazolidinediones).

The phenomenon of resumed menstruation after surgically induced weight loss was observed initially in the era of jejunoileal bypass. The current mainline surgical options of Roux-en-Y gastric bypass (RYGP) and the adjustable silicone gastric band have fewer metabolic side effects and have been shown to be

highly effective in reversing the insulin resistance associated with Type 2 diabetes mellitus. Pregnancy following weight loss surgery, among women who have been previously diagnosed with infertility, has proven to be safe for both mother and child.

In the many overweight and obese women who achieve pregnancy, there are a number of increased and interrelated adverse perinatal outcomes. Maternal morbidity includes increased incidences of a number of disorders including chronic hypertension, gestational diabetes, preeclampsia, fetal macrosomia, as well as higher rates of cesarean delivery and postpartum complications. Obese women have a two- to threefold risk for a fetus with a neural-tube defect or other anomalies. There is also a two- to threefold increased incidence in omphalocele, heart defects, and multiple anomalies. An increased incidence of late-pregnancy stillbirths (1.6- to 2.6- fold) has been associated with obesity. A prepregnancy BMI greater than 30kg/m2 is an independent risk factor for stillbirth.

Due to the rapid and dramatic weight loss experienced in the first year after bariatric surgery, pregnancy should be avoided until at least the second year after surgery. The American College of Obstetricians and Gynecologists (ACOG) advises all patients to delay pregnancy for 12 to 18 months after bariatric surgery.

Pregnant women who have undergone bariatric surgery are at risk for specific nutritional deficiencies that could potentially impact both the mother and the infant. In both restrictive and malabsorptive procedures, decreased caloric intake can result in drastic weight reduction and a catabolic state. In addition, small gastric pouch volumes created in restrictive procedures may aggravate hyperemesis. The RYGP potentially has a greater impact on pregnancy due to the added malabsorptive component. Iron deficiency anemia, vitamin B12, folate, calcium, and vitamin D deficiencies can particularly impair fetal growth and development. If pregnancy is planned or desired, a careful screening of compliance with multivitamin supplementation, anemia, vitamin deficiency, and metabolic derangements should be obtained and any deficiencies corrected prior to pregnancy. During pregnancy, prenatal vitamins, oral iron, and calcium should be prescribed. Folate and iron supplementation may need to be higher due to malabsorption. Severe iron deficiency anemia, refractory to oral iron treatment, requiring blood transfusion has been described after gastric bypass surgery. In a recent study, however, pregnancy outcomes within the first year after weight-loss surgery revealed no significant episodes of malnutrition, adverse fetal outcomes, or pregnancy complications. When comparing prior pregnancy outcomes with those after gastric bypass, there is a dramatic reduction in hypertension, diabetes, and infant weight greater than 4,000 grams. Pregnancy outcomes after lap band are consistent with general community outcomes rather than outcomes from severely obese women.

SEE ALSO: Cytokines; Hormones; Impotence; Pituitary Gland; Polycystic Ovary Disease; Pregnancy; Sexual Health.

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Fiber and Obesity

FIBER ENCOMPASSES BOTH dietary fiber and added fiber. Dietary fiber is defined by the National Academy of Sciences as nondigestible carbohydrates and lignin that are intrinsic and intact in plants, while added fiber includes isolated, nondigestible carbohydrates that have beneficial physiological effects in

humans. Sources of dietary fiber include fruits, vegetables, whole grains, and legumes/nuts, while sources of added fiber include fiber supplements found in pill or functional food form. Both types of fiber have beneficial health effects.

Experts suggest that fiber is beneficial in managing chronic diseases such as cardiovascular disease, diabetes, colon cancer, and obesity. The current recommendation for total fiber intake for young men is 38 grams per day and for young women is 25 grams per day. Mean intake for fiber is 16.5 to 17.9 grams per day for men and 12.1 to 13.8 grams per day for women, significantly lower than the recommendations.

There appears to be an association between fiber intake and obesity. Populations that consume higher fiber report lower obesity rates. Additionally, fiber intake is inversely associated with body weight as well as body fat. Controlled studies have examined both individuals consuming whole foods with dietary fiber and individuals on diets with fiber supplements. One study found that supplementing a low-calorie (1,200 kcal/day) diet with four grams of fiber per day produced a significantly greater loss in body weight than individuals without fiber supplements. However, another study using a fiber supplement could not detect any differences in hunger, satiety, or body weight between individuals with or without fiber supplements.

Fiber is thought to reduce body weight through several means. First, fiber-rich foods require increased chewing, which results in saliva and gastric acid production. This saliva and gastric acid production may result in stomach distention (i.e., a feeling of fullness). Additionally, soluble/viscous fibers, which absorb water in the stomach, are thought to delay gastric emptying time and consequently promote satiation and a prolonged feeling of fullness. After a meal, these soluble fibers may suppress glycemic and insulinemic responses, which may reduce the rate of return of hunger. This is also supported by evidence that a large intake of fiber at breakfast results in reduced food intake at lunch.

Fiber may also exert its effect by blocking or limiting the absorption of macronutrients, which would decrease net caloric intake, resulting in weight maintenance. Experts think that fiber effects the secretion of certain gut hormones, such as cholecystokinen (CCK). CCK is secreted after a meal from cells in the upper small intestine, and controls gut motility, gallbladder contraction, and secretion of pancreatic enzymes. Consequently, if fiber can modulate CCK secretion, this will be another means in which fiber promotes weight maintenance.

While evidence suggests that increasing fiber intake is beneficial for individuals seeking to maintain a healthy weight, it is not desirable to consume an excessive amount of fiber. Although no tolerable upper intake level for fiber has been established by the National Academy of Sciences' *Dietary Reference Intakes*, it is important to note that overconsumption of fiber can lead to decreased availability of certain vitamins and minerals.

SEE ALSO: Nutrition and Nutritionists; Nutrition Education.

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Fitness

THE IMPORTANCE OF fitness in promoting health, preventing disease, and managing weight is now well recognized. A considerable body of research exists examining the relationship between various aspects of fitness and obesity as well as related chronic diseases and mortality. Currently, however, there is some debate over whether improved fitness in the presence of obesity leads to a reduced risk for cardiovascular

disease-related mortality. Despite this debate, numerous recommendations have been made regarding level and volume of exercise for improvements in fitness and, concomitantly, health. This entry will discuss components of fitness, improvement of fitness, and research on the link between fitness and obesity.

COMPONENTS OF FITNESS

Fitness is a multidimensional state of being in which the body is able to function efficiently and effectively. The components of fitness can be divided into three areas: health-related fitness, metabolic fitness, and skill-related fitness.

Health-related fitness consists of body composition, cardiorespiratory fitness (CRF), flexibility, strength, and muscular endurance, all of which are directly related to physical health and some of which are directly related to obesity. Body composition refers to relative and absolute amounts of fat, lean tissue (muscle and bone), and water in the body. The fit person has a low, but not too low, level of body fat that is distributed more in the hips and buttocks than the abdomen. Excessive amount of body fat, especially intra-abdominal (belly) fat, is associated with increased risk of disease, while excessively low fat is also detrimental as it causes physiological dysfunction. An adequate amount of muscle mass is important because low levels contribute to metabolic complications and a greater risk of fat gain. Strong, healthy bones are important to optimal health, and low bone mass and density are risk factors for osteoporotic fracture.

There are several different methods for measuring body composition, which can be important in tracking weight gain and obesity at the individual and population levels. Techniques such as hydrodensitometry (water displacement) and plethysmography (air displacement) measure body composition from a ratio of body mass to body volume. A newer technique, dual X-ray absorptiometry (DEXA), is also highly reliable and has the additional ability to determine exactly where fat is located in the body. These techniques are very useful for individual measurements, but cannot be used on a larger scale because they are costly and require highly trained personnel. The anthropomorphic measurement of body mass index (BMI) assesses relative weight and height and is often used in studies of obesity. Circumference measurements of various areas of the body, such as those used for waist-to-hip ratio calculation, and skin-fold measurements, also are used very frequently. Finally, bioelectrical impedance analysis (BIA) is becoming increasingly more common as it assesses body composition by measuring total body electrical conductivity using a specialized household scale.

Physical fitness is a combination of flexibility, body composition, cardiorespiratory health, strength, and endurance.

A second component of health-related fitness is CRF. CRF is the efficiency of the heart, lungs, and vascular system in delivering oxygen and nutrients to the working muscles and the ability of muscles to utilize fuel so that prolonged physical activity can be sustained. CRF is considered health related because low levels are associated with a marked risk of premature death from all causes, and increases in CRF are associated with a reduction in such deaths. The best way to improve CRF is through engaging in exercises that use large muscle groups and require aerobic pathways to sustain the activity (i.e., jogging, cycling, swimming, dancing, rollerblading, or cross-country skiing) for an extended period of time (20–60 minutes). The most common measure of CRF is maximal

oxygen uptake (VO2Max), which is an assessment of the greatest rate at which oxygen can be taken in, distributed, and used during exercise. VO2Max is most commonly assessed by slowly and systematically increasing exercise to exhaustion, usually performed on a stationary bike or treadmill. While this "maximal" exercise testing is considered the gold standard, it requires that the participant exercise to volitional fatigue and often requires medical supervision and emergency equipment. Thus, practitioners commonly rely on "submaximal" tests in which they utilize heart rate as a predictor of VO2Max.

Other components of health-related fitness that are less often correlated with obesity include flexibility, strength, and muscular endurance. Flexibility refers to the ability to maintain optimal movement ranges at a single joint or series of joints. Improvements in flexibility can help reduce muscle tension and reduce the risk of injury. Strength refers to the ability of the muscles to exert force as in controlling one's own body weight or lifting heavy objects. Finally, muscular endurance is the ability of the muscles to exert themselves repeatedly with minimal fatigue. A fit person can repeat movements or engage in physical activity for a long period of time without undue fatigue. In general, only a moderate amount of flexibility, strength, and endurance are required to prevent disease and promote health. High levels of these components relate more to performance than to health benefits. For example, a moderate level of strength is necessary to prevent back injuries and posture problems, whereas a high level of strength improves performance in active sports such as football or hockey.

The second area of fitness, metabolic fitness, is a nonperformance component of physical fitness and is evidenced by healthy cholesterol levels, blood pressure, blood sugar, and insulin levels. Strongly related to obesity, these metabolic factors can be manipulated with weight loss or gain. A person who has a constellation of metabolic risk factors (i.e., high cholesterol, high blood pressure, and excessive body fat) is said to have metabolic syndrome, a serious risk factor for coronary artery disease, noninsulin-dependent diabetes, hypertension, certain types of cancer, and other chronic diseases. Individuals who are overweight or obese have substantially increased risk for metabolic syndrome. The basic therapeutic approach to treating metabolic syndrome is typically an organized program of lifestyle change, focusing on increased physical activity and weight reduction.

The third area of fitness is skill-related fitness, which directly relates to improved physical performance. The components of skill-related fitness are agility, balance, coordination, power, speed, and reaction time. Agility is the ability to change the direction of movement with speed and precision. Balance refers to the maintenance of equilibrium both while stationary and while moving. Coordination is the ability to use the senses to bring together different parts of the body to perform motor tasks with harmony and accuracy. Speed is the ability to perform a movement in a short period of time. Power, essentially a combination of speed and strength, is the ability to transfer energy into force at a fast rate. Finally, reaction time is the time it takes to react to a stimulus. These components are not simply useful for athletes; varying degrees of competencies in these skills are necessary for various motor skills. For example, while agility is useful in sports in which a person needs to move in response to an opposing player, being agile can also help a person to avoid tripping over unexpected objects.

IMPROVING FITNESS

Whereas physical activity is simply any bodily movement that results in energy expenditure beyond resting expenditure, exercise is planned, structured, repetitive, and purposeful activity in the pursuit or maintenance of health and fitness. Improvements in health and fitness can occur in a variety of ways, and numerous health organizations have issued recommendations across the spectrum of physical activity to this end. These guidelines vary in terms of frequency, intensity, and time of activity as well as their intended outcome. For example, both the Surgeon General in 1996 and the Institute of Medicine in 2002 provided comprehensive recommendations for health-related physical activity. Neither of these recommendations, however, addressed improvements in fitness; the Surgeon General's recommendations focus on the health benefits of physical activity, while the Institute of Medicine's recommendations focus on physical activity as a requirement for weight maintenance. On the other hand, a third set of guidelines, issued by the American College of Sports Medicine, was directed at improving fitness through a combina-

tion of frequency, intensity, and duration of chronic exercise. This set of guidelines recommended vigorous exercise for at least 20 minutes a day for three to five days a week. A range of intensities was provided within the guidelines, as low-fit individuals could experience improvements in CRF even at lower intensities. Presumably, adherence to this set of guidelines also would result in health benefits of activity and improved weight management.

FITNESS IN THE CONTEXT OF OBESITY

Fitness and obesity in the United States have been following divergent paths, as rising rates of obesity have been documented along with diminishing levels of fitness. These health-related trends contribute to the overall burden of disease, and they are targets for public health recommendations. The prevalence of obesity among U.S. adults has received the most attention due to its record levels in the population. Rates of overweight children and adolescents also continue to rise, suggesting the likelihood that the trend of excess weight in the United States will continue into the future. Low levels of cardiorespiratory fitness (CRF) also have been noted in a large percentage of adolescents and adults. The health impact of these trends is well-established. Low levels of CRF, in particular, fail to afford the normally protective benefits of fitness against cardiovascular disease and all-cause mortality (i.e., death for any reason). Further, obesity has been shown to be independently associated with increased risk for cardiovascular disease and other health conditions, such as Type 2 diabetes, hypertension, and dyslipidemia.

A common denominator for weight and fitness is physical activity. Low levels of CRF are related to low levels of physical activity, and lack of physical activity is a potential causal factor in the complex etiology of obesity. The present-day environment, filled with laborsaving conveniences and easy transportation, offers few opportunities for physical activity sufficient for weight management or improvements in CRF. Consequently, 40 to 50 million Americans are sedentary and many more are insufficiently active.

There is debate, however, over the exact relationship between fitness and weight. Not all researchers agree on the cardioprotective effect of high levels of CRF in the presence of obesity. This debate has been referred to popularly as an issue of "fitness versus fatness." A large number of studies have been conducted to examine the relationship between CRF and disease or death among normal-weight, overweight, and obese populations. Many of these large, well-controlled studies showed that CRF remained cardioprotective even when individuals were overweight or obese. In fact, obese individuals with high levels of CRF were at reduced risk for death from cardiovascular disease compared to lean, unfit individuals. These studies also showed that high levels of CRF were related to decreased risk for all-cause mortality. Other researchers, however, have found that neither CRF nor physical activity diminishes the relationship between obesity and poor health outcomes. For instance, high levels of CRF might not improve biomarkers of cardiovascular disease risk or protect against Type 2 diabetes. The issue, therefore, is undecided.

The "fitness versus fatness" controversy does not diminish the urgency of establishing public health goals. Engaging in physical activity or exercise at recommended levels is an important aspect of treatment for obesity and is necessary to improve CRF. Improvements in weight and CRF both are independently associated with improved health outcomes. Because obesity is the result of positive energy balance, caloric expenditure due to activity becomes integral to any weight loss plan.

Consistent physical activity, especially that which is sufficient to improve fitness, is also integral to successful weight maintenance, whether as a lifestyle activity or as part of a weight loss plan.

SEE ALSO: American College of Sports Medicine; Exercise; Inaccessibility of Exercise; Physical Activity and Obesity; Physical Activity Patterns in the Obese.

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Flavor: Taste and Smell

THE SENSES OF taste and smell are closely linked with food-intake behavior, particularly because they are responsible for the perception and enjoyment of foods and beverages. In addition to the pleasure attained from food, the chemosensory systems of mammals provide life-preserving signals to the organism about the chemicals present within the immediate environment. Thus, these systems are vital in determining reproductive success of an organism. Taste, also known as gustation, is a commonly misused term, because when referring to the taste of a chocolate, a piece of cake, or a beverage, most people actually mean overall flavor. Only a small portion (around 5 percent) of overall flavor of foods is actually due to the perception of basic tastes (sweet, bitter, salty, sour, and umami). Flavor is defined as the complex effect of basic taste sensations, olfactory (smell) sensations, and trigeminal sensations (carbonation or the hotness of chili pepper), and textural aspects of food (creaminess, oiliness, smoothness, etc.). Flavor is achieved primarily through the olfactory system (about 95 percent), with only 5 percent attributed to basic taste perception, or the gustatory system. A clear example of this fact is that we often lose the ability to experience the flavor of foods when we have a cold or a stuffy nose. Due to an excess of mucous secretions, olfactory receptors are blocked, thus decreasing the functionality of the olfactory system.

Taste, or gustation, is a sense that involves the detection of the five basic tastes: sweet, salty, sour, bitter, and umami. The umami taste is a savory or meaty taste of L-glutamate, the most common amino acid in proteins, which has recently been added as one of the basic tastes. Umami is a familiar taste in Japanese cuisine, and the discovery of this fifth distinct basic taste was made almost a century ago by a chemist at Imperial University. Some researchers have recently debated that fat, or more specifically, polyunsaturated long chain fatty acids (PUFAs), are also detected by receptors on taste cells. Fat was always thought to be tasteless, and only detectable through the textural components (e.g., creaminess, oiliness) it adds to foods. The discovery of a taste component to dietary fat would add a new complexity to the perception of this nutrient. However, until further research confirms the existence of a "fat taste" mechanism in humans, we remain with the ability to taste five basic types chemicals.

The anatomical features of the taste system consist of taste buds, taste papillae, taste pores, and taste cells. Taste buds are groups of anywhere from 30 to 100 epithelial cells (taste cells) that are often embedded within structural units known as the taste papillae. Papillae consist of three primary types: fungiform, foliate, and circumvallate. The fungiform papillae consist of anywhere from one to three taste buds, and are the large, mushroom-shaped papillae on the anterior surface of the tongue that you can see with the naked eye. The chorda tympani branch of the facial nerve innervates these papillae. Foliate papillae appear as three to four small vertical lines on the side of the tongue. Circumvallate papillae are arranged in a V shape at the back of the tongue. Both foliate and circumvallate papillae are innervated by the glossopharyngeal nerve. At the apex of taste buds, a small pore exists (the taste pore) that extends into the oral cavity, and allows access taste stimuli to gain access to the individual taste cells. These individual taste cells often contain either ion channels or specialized receptors that can respond to the five basic tastes.

One of the primary functions of taste receptor cells on the surface of the tongue is to distinguish appealing stimuli, such as sweet, salty, and umami, from aversive taste stimuli, such as sour and bitter. Palatable taste stimuli often signal the presence of calories, and thus the ability to recognize these by taste can help an organism survive. At the same time, the aversive taste of bitter often signals the presence of toxic chemicals in a food source. Each of the basic tastes uses a unique method of detection that utilizes either ion channels or second messenger proteins to ultimately depolarize taste receptor cells, and carry messages to the brain. Salty taste stimuli (NaCl) are detected by diffusion of sodium ions through specialized Na+ channels that are expressed on taste cells. Ultimately, this diffusion of sodium ions through sodium channels results in the depolarization of taste receptor cells, the first step in signaling to the brain that salt is present on the tongue. Sour taste is also perceived through ion channels present on the taste cells, although the ion channels present in this case sense the presence of acids (H+), and are activated by decreases in extracellular pH. The mechanisms of

The senses of taste and smell are closely linked; much of the flavor of foods comes from their smell.

bitter, sweet, and umami perception are beyond the scope of this entry, but common with all three are the binding of tastants to specialized second messenger coupled taste receptors on the surface of cell membranes. Improved and more efficient molecular biology techniques have made the discovery of taste perception mechanisms a fertile ground for discovery over the past few years.

Olfaction, or the sense of smell, is defined as the perception of airborne odorants by the olfactory epithelium, located in the roof of the nasal cavity. The olfactory epithelium is covered by a secretion of mucus, and contains the sensory cells for odor detection. The sense of smell is one of the oldest sensory systems, and its functions are vital to organisms' health and well being. The primary functions of this system are to distinguish both the type (quality) and strength of an odor (used for nonfoods) or aroma (used for foods). The olfactory system is also an important warning system, capable of sensing volatile molecules from some distance, before we are actually close enough to taste something.

Odor determination has been a poorly understood process, with many complexities due mainly to the sheer volume (thousands) of possible odors that the brain must be able to identify. This function is accomplished by a complicated sequence of events that begins with odor molecules traveling into the nasal cavity to gain access to olfactory receptor neurons. Each olfactory neuron is covered with small hairlike fibers called cilia, and these fibers assist in directing odor molecules to the olfactory receptors. After binding to the receptor, the neurons send messages about the specific odorant through axons, and these messages converge into a few larger structures called glomeruli. Based on the pattern of glomeruli that are activated, the brain can determine the specific type of smell that is being experienced in the nose.

Both the senses of taste and smell can have close relationships to overall emotion and well being. Brain areas such as the amygdala hypothalamus (emotion) and the hippocampus (memories) are reactive to taste and smell stimuli. The reactions that these areas of the brain have to foods are not well understood as of yet, but certainly, we have all experienced smells or tastes that have evoked powerful memories. As more sophisticated methods of studying the brain are refined, such as functional magnetic resonance imaging (MRI), scientists will be able to clearly understand what happens to the brain upon exposure to palatable foods. Several preliminary studies in this area have shown that obese and lean individuals might have differences in these brain areas that respond differently to exposure to tasty, high-caloric foods.

SEE ALSO: Magnetic Resonance Imaging Scans; Sweet Taste; Taste Aversion Learning.

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Flavor-Nutrient Learning

BECAUSE WE MUST eat to obtain nutrition, we rapidly learn to associate certain flavors with nutritional rewards as well as nutritional emptiness. For example, if we eat a meal with a certain flavor and

receive a nutritional reward such as high energy, we will learn to associate that "good" flavor with nutrition. If we eat another meal with a different flavor and in turn feel energetically empty afterward, we will learn a negative association between that "bad" flavor and nutrition. This type of learning is important evolutionarily in all animal species. The trouble with modern foods is that often "good" flavors are paired with nutritionally empty meals, thus confusing the consumer. It is difficult for humans to unlearn a flavor-nutrient association; therefore, it is difficult for people to choose nutritionally balanced meals based on flavor alone.

A great number of scientific studies have investigated the phenomenon of flavor-nutrient learning. It has been known for many years that when a laboratory animal such as a rat is given a flavored food after a fast, it will develop an association between that flavor and calories. This effect is a model of flavor-nutrient learning. In 1993, researchers showed that if, however, following a fast that animal is given food and then a flavor, the animal will show reduced preference for the flavor that came after caloric satiation. This "fullness effect" may be the opposite of flavor-nutrient learning. Yet, while rats have been an excellent tool for flavor-nutrient learning studies, this effect has been nearly impossible to show in humans.

A recent study from 2007 showed that human flavor-nutrient learning may have been difficult to show in an investigation because there is a difference between restrained and nonrestrained eaters. This study used women who had been rated on a test for dietary restraint. When given desserts with high- or low-energy content, the women who could exercise dietary restraint preferred the desserts with high-energy content at the end of the study. In contrast, the unrestrained eaters did not develop a preference for either flavor. This study reinforces how in today's time of unrestrained eating, it is difficult, if not impossible, to develop proper flavor-nutrient learning.

Flavor-nutrient learning is not the same phenomenon as taste-nutrient learning. Flavor-nutrient learning is when we learn to associate a flavor with a nutritional reward and therefore prefer that flavor to another one. In contrast, taste-nutrient learning is when we associate a particular taste with a particular nutritional reward. Tastes are qualitative such as sweet, salty, salty-sweet, bittersweet, and so forth. A study published in the *European Journal of Neuroscience* in 2005 shows that a particular brain region, the amygdala, may be involved in flavor-nutrient learning but not taste-nutrient learning. As scientists begin to understand the neural circuitry for flavor-nutrient learning, their knowledge may lead to better tools for nutritional counseling and dietary design.

SEE ALSO: Nutrition and Nutritionists; Nutrition Education; Taste Aversion Learning.

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Flavor Programming and Childhood Food Preferences

PROGRAMMING OF FLAVOR preferences and aversions occurs in childhood through exposure to flavors and conditioning of response to particular flavors through associations with the various internal and external consequences of their consumption. A number of factors contribute to the development of flavor preferences. Certainly, there is an innate competent. Research indicates that sweet flavors are universally preferred by children, even shortly after birth. Further, a common preference for salty flavors tends to develop around 4 months of age. Genetics may also play a role. For instance, children of overweight parents have demonstrated decreased ability to detect sweetness compared to other children and thus may require sweetness in greater intensities amounts in order to be sated. However, environment and early experience play a significant role in the development of flavor preferences, which has an effect on food preferences later in life.

Research demonstrates that flavor learning begins during fetal development. Tastes, smells, and flavors from a mother's diet are transported to a child through amniotic fluid. Researchers have demonstrated that odors from certain foods and flavors in a mother's diet, such as garlic, are detectable to human smell in the amniotic fluid. Through this early exposure, a child habituates to the diet of the mother and learns what foods and flavors are safe and preferable. A connection between mother's diet throughout pregnancy and subsequent child preferences has been established. For instance, infants whose mothers drink carrot juice during pregnancy exhibit more acceptance and enjoyment of carrot-flavored cereal at 6 months. This suggests that mothers should be encouraged to eat a varied diet during pregnancy in order to expose the fetus to various flavors. This may lead to greater acceptance of a wide variety of foods as a child, which is important as greater dietary variety is often an indicator of satisfactory nutrient intake.

Early infancy, during breast- or bottle-feeding, appears to be another critical period for the development of flavor preference. Breastfed infants continue to be exposed to a variety of flavors from mother's diet through breast milk. Flavors transmitted through breast milk affect the infant's suckling patterns and appear to have an effect on food preferences in childhood. Researchers have demonstrated increased suckling in response to the flavor of garlic or vanilla in mother's breast milk. Further, flavors eaten by the mother during the breastfeeding period are typically preferred by the child when exposed to the same flavors in solid food.

Bottle-fed infants do appear to orient to flavors associated with their particular formula as well. For instance, those fed with bitter-tasting formula solutions are more accepting of bitter and sour flavors later in childhood. However, breast-fed infants are more accepting of a wide variety of foods later in life. This is presumably because they are exposed to a greater variety of flavors in breast milk, as opposed to bottle-fed infants, whose exposure is limited to the unique flavors of the formula they are fed. The belief that breastfeeding will expand the child's diet repertoire later in life, coupled with knowledge of the health benefits conferred by breastfeeding, leads many to argue for mothers to breastfeed their children.

Flavor programming continues throughout childhood; however, new flavors are approached more cautiously in early childhood (between the age of 2 and 5) than during infancy. Infancy appears to be a critical period for flavor learning, during which flavors are readily accepted and preferences are quickly learned. During early childhood, however, children grow increasing hesitant to try new foods. From an evolutionary perspective, this tendency seems to make sense. Feeding is largely controlled by the mother during infancy, who presumably knows what foods are safe and nutritious for the child, thus infants are likely primed to be more accepting during this period. Following the weaning period, however, children assume greater responsibility for feeding, thus an innate caution toward new food may guide them to orient only toward foods they know are safe.

Despite this increased caution toward new foods, children do continue to develop flavor and food preferences during this period which will affect later eating habits. As would be expected, the most important determinant of food preference during this age is familiarity. Thus, the key to enhancing the acceptability of a food is to make it more familiar. Research has consistently demonstrated that exposure to the taste of a food in early childhood increases acceptability of and preference for the food. Indeed, mothers who report exposing their children to a variety of foods at a young age tend to have children who are less neophobic and have fewer feeding problems.

Flavor preferences can also be conditioned in early childhood through the postingestive consequences and social context associated with the consumption of the flavor. Through repeated pairings, children can come to prefer flavors associated with food and drinks of higher energy density and fat content, due to the greater sense of satiety associated with their consumption. Additionally, preference for a certain flavor can be learned through repeatedly pairing the flavor with certain social contexts. For instance, studies have indicated that preference for a flavor increases when foods associated with the flavor have been repeatedly used as a reward, paired with adult praise and attention, or withheld from the child. However, if a certain food or flavor is used as a contingency for some other behavior (e.g., "If you eat your vegetables, you can go outside and play"), the food or flavor becomes less preferable to a child.

This research gives insight into the development of preferences for foods and flavors that are likely to promote obesity. Not only do children come to prefer foods of high-fat or high-sugar content because they are more likely to fill them, but such foods are often used as rewards for good behavior and otherwise withheld. Parents often "bribe" or "barter" with their children over the ingestion of healthier foods, such as fruits and vegetables, by promising rewards for their consumption.

Further, foods with higher fat and sugar content and less nutritive value (e.g., cake) are often presented in contexts associated with greater attention and praise (e.g., a child's birthday). However, knowledge of this preference learning may help to promote healthier eating habits. Parents can expose a child to the flavors of healthier foods of higher energy density, which will enhance preference through greater satiety, while providing nutritive value. Greater attention and praise can be given during the ingestion of healthier foods; "junk" foods can be presented in moderation, rather than withheld altogether; and a variety of flavors and healthier foods such as fruits and vegetables can be integrated into celebratory contexts.

Flavor programming continues to some degree throughout childhood; however, preferences tend to become more stable over time. Studies of eating behavior in middle childhood (ages 6–12) indicate that repeated exposure continues to increase the preference for various foods and flavors. Thus, dietary variety continues to be important throughout childhood; however, preferences may be more difficult to alter during this period.

SEE ALSO: Flavor: Taste and Smell; Food Preferences; Genetic Taste Factors; Taste Aversion Learning.

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Folic Acid and Neural Tube Defects

NEURAL TUBE DEFECTS are a common category of congenital anomalies. Neural tube defects are serious and directly affect the developing nervous system of the fetus. These defects include anencephaly, spina bifida, and encephalocele. In the United States, there is an estimated one to two per 1,000 babies born each year with a neural tube defect. Neural tube defects occur in the first month after conception during the critical period when the neural tube is closed in the fetus. Anencephaly and spina bifida are considered the most common neural tube defects. Anencephaly occurs as a partial absence of the skull bones due to inadequate closure of the neural tube. Spina bifida is essentially a failure of the vertebral arches to close over an open neural tube defect, resulting in an exposed spinal cord and nerves at birth.

Folic acid is found naturally in a number of foods, such as beans, lentils, greens, papaya, broccoli, peas, and asparagus. Most folic acid, however, is attained from fortified food sources, mainly cereals and grains. Folic acid supplementation in foods has been shown to reduce the incidence of neural tube defects. Some studies have demonstrated that neural tube defects can be reduced as much as 40 to 100 percent if folic acid is ingested on a daily basis by the pregnant mother. Because the critical period when neural tube defects form is in the first month after conception, neural tube defects are best prevented by adequate daily folic acid in mothers' diet throughout reproductive years. In 1998, the Food and Drug Administration required that enriched cereal grain flours be supplemented with folic acid.

Obesity has been shown to increase risk for having a baby with a neural tube defect. Women with a BMI over 29 have twice the risk of having a child with a neural tube defect, compared to both lean and underweight women. It is unclear why obesity increases risk for NTDs, nor is it known if losing weight prior to pregnancy will improve this risk. Fortifying food in the general diet of women is considered an effective and low-cost alternative to a strategy encouraging women to change their dietary choices to include more folic acid intake. After the mandated fortification, the number of neural tube defects in the United States decreased significantly. The use of folic acid re-

mains one of the best examples of public health intervention and prevention in the United States.

SEE ALSO: Center for Maternal and Child Health; Food and Drug Administration; Office of Dietary Supplements; Prevention.

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Food "Addictions"

LOSS OF CONTROL over intake, constant desire (i.e., craving) for a substance, use-associated guilt, and secretive use of a substance are the hallmarks of drug addiction. Many people report feelings of loss of control over eating and obsessive thoughts and craving for food, as well as guilt, depression, and secretive eating in response to loss of control. The *Diagnostic and Statistical Manual*, 4th edition (DSM-IV) lists provisional criteria for binge eating disorder (which usually occurs in obese individuals, but may occur in lean individuals) and definitive criteria for bulimia nervosa (which usually occurs in lean individuals) that include all of these symptoms. There is no agreement among scientists as to whether the existence of these symptoms in an individual constitutes an addiction to food.

Addiction is a term that really describes the combined physiological, psychological, and social consequences of repeated uncontrolled intake of substances. However, some people use the term *addiction* to refer to the physiological and/or psychological dependence on a substance. Using this definition, the presence of an addiction can thus be demonstrated by the emergence of physiological symptoms (e.g., shaking, teeth chattering, sweating) or psychological symptoms (e.g., irritability, aggression, depression, crying) upon withdrawal of the substance for a period of time. Researchers have demonstrated physiological "addictive" responses (in rats) to sucrose when provided on a limited and unpredictable basis. In fact, all living creatures are physically dependent on food to sustain life.

Because we all need food to survive, it is difficult to ascribe addiction to certain behaviors toward food. However, recent brain imaging data have shown similarities in dopamine functioning and activation of the reward circuitry of the brain between those experiencing "loss of control" over eating and those addicted to drugs. The difference, however, between food and drugs with respect to addiction potential, lies in the *reduced amount of dopamine released* upon food stimulation compared to drug stimulation and the *shorter duration of dopamine release* upon stimulation, which causes a more rapid return of the brain reward circuitry to its normal state compared to that observed with drug use. The different time course for food-induced dopamine effects compared to drug-induced dopamine effects, results in a reduced opportunity for an individual to experience an altered state, or "high," which fuels continued abuse of the substance. In addition to altered activity in the brain reward circuitry, decreased signaling in the "emotion" centers of the brain due to stress and depression can also promote uncontrolled eating. Carbohydrate- and fat-induced dopamine release in the brain can cause

Addiction is defined as a loss of control regarding a substance or action. Many people find themselves "addicted" to particular foods.

a transient and temporary relief of the stress or depression. This temporary relief promotes conditioned learning that underscores the development of a craving for carbohydrate-/fat-containing food during future periods of stress and/or depression.

Besides altered brain responses to food, disorders in peripheral digestion and metabolism can also contribute to a sense of "loss of control" over eating in obese individuals. The peripheral digestive and metabolic systems of the body contribute signals of satiation (stop eating now) and satiety (I've had enough to eat) to the brain. Increased stomach capacity and decreased stimulation of the vagus nerve produce weaker signaling to the brain of satiation ("stop eating" signal) in some obese individual, and insensitivity to hormone-based signals of satiety (I've had enough to eat), such as insulin resistance and leptin resistance, add to reduced "stop eating" signals that are also relayed to the brain.

What predisposes some obese individuals to experience "loss of control" over eating? Brain imaging and genetic testing of components of the brain dopaminergic system are contributing to our understanding of these phenomena and to factors that increase risk of developing "loss of control" over eating. A recent imaging study demonstrated that extremely obese individuals have fewer brain receptors (the signal-receiving sites) for dopamine (the reward-related neurohormone) than lean or less obese individuals. Whether this predisposes an obese individual to focus on food or is the result of excessive focusing on food is not known. However, recent work in drug-naive rats demonstrated that those with fewer dopamine receptors were more likely to become dependent on cocaine, suggesting that a similar situation could also predispose an individual to uncontrolled eating.

SEE ALSO: Addictive Behavior; Carbohydrate "Addictions"; Food and Mood.

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Food and Drug Administration

THE FOOD AND Drug Administration (FDA), an agency within the U.S. Department of Health and Human Services (DHHS), is charged with regulating food, drugs, biological products, cosmetics, and medical and therapeutic devices sold in the United States. The roots of the FDA lie in the Bureau of Chemistry, founded in 1863 within the Department of Agriculture, and which focused on the adulteration of food products. The modern era of the FDA dates from 1906, the year the Pure Food and Drug Act was passed; this law added regulatory functions to the FDA's mission and broadened the agency's powers. The FDA assumed its current name in 1930; it became part of the Federal Security Agency in 1940, was transferred to the Department of Health, Education, and Welfare (HEW) in 1953, became part of the Public Health Service within HEW in 1968, and part of the DHHS in 1980.

The FDA designated an Obesity Working Group to study the problem of obesity in the United States, which the FDA at its Web site calls an "epidemic." The final report of this Working Group focused on the concept of caloric balance, that is, that overweight and obesity are a function of the consumption of more calories than are burned in daily activities. The slogan for the FDA's campaign is "Calories Count." Actions recommended by this committee to combat overweight and obesity include improving food labels to including meaningful serving sizes and to display calorie counts more prominently, initiating a consumer education campaign around the "calories count" concept, encouraging restaurants to provide nutritional information, increasing enforcement actions regarding the accuracy of food labels, revising FDA guidance concerning the development of obesity drugs, and cooperating with other entities, including government agencies, nonprofits, industry, and academia, on obesity research.

The Center for Drug Evaluation and Research (CDER), one of five centers within the FDA, regulates both prescription and over-the-counter drugs. The distinction between prescription and over-the-counter drugs has been made in the United States since the Durham-Humphrey Amendment of 1951. The company marketing the drug performs the actual testing of drugs: CDER reviews the information, including

testing data and proposed labeling provided by the company. Two drugs are currently (2007) approved to treat obesity. Orlistat, manufacture by Roche Laboratories Inc. under the trade name Xenical®, was the first approved drug in the lipase inhibitor class; it prevents fat from being broken down and absorbed in the digestive tract, thus reducing caloric intake. Sibutramine, which suppresses the appetite by inhibiting norepinephrine and serotonin reuptake, is produced under the brand name Meridia® by Knoll Pharmaceutical Company. Fenfluramine and phentermine were approved for single-drug, short-term therapy; however, their long-term use in combination (Fen-Phen) has been linked with valvular heart disease and both drugs were withdrawn from the U.S. market.

SEE ALSO: Drug Targets that Decrease Food Intake/Appetite; Drugs and Food; Drugs that Block Fat Cell Formation; Fenfluramine; Government Agencies; Office of Dietary Supplements.

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Food Guide Pyramid

THE FOOD GUIDE pyramid is a graphic device developed by the U. S. Department of Agriculture (USDA) as an educational tool to illustrate the relative proportion of foods types recommended to compose a healthy diet. The USDA has been issuing dietary recommendations for Americans since 1894, and the best-known food guide pyramid (hereafter "the traditional food pyramid") was developed in 1992 and is still used on food packaging and some official Web sites, including that of the National Agricultural Library of the USDA. The traditional food pyramid was a revision of the "four basic food groups" which had been used in USDA educational materials since 1956: the groups, and recommended minimum servings per day, were meats, poultry, fish, legumes, eggs, and nuts (two servings); dairy products (two servings); grains (four servings); and fruits and vegetables (four servings). The traditional food pyramid was officially replaced in 2005 by MyPyramid, which presented the similar concepts in a different graphical form and includes a computer interface to allow individuals to create individualized nutritional recommendations based on their age, gender, and activity level.

The traditional food pyramid is divided into four horizontal layers, and the middle layers are divided into two sections each: the size of the sections graphically represents the recommended servings of each type of food. Notes to the pyramid include definitions of what constitutes one serving: for instance one cup of milk, two to three ounces of cooked meat, one cup of raw leafy vegetables and one slice of bread all constitute a single serving. The base of the pyramid is the bread, cereal, rice, and pasta group (6–11 servings per day, including several servings per day of whole grains). The second layer of the pyramid is divided into the vegetable group and the fruit group (three to five servings daily, including legumes and dark green leafy vegetables several times per week) and the fruit group (two to four servings daily). The third layer of the pyramid contains the milk, yogurt, and cheese group and the meat, poultry, fish, dry beans, eggs, and nuts group (two to three servings are recommended daily from each group, and in both cases, low-fat options are recommended). The top layer of the food pyramid represents fats, oils, and sweets; no specific consumption recommendations are given other than that these foods should be used sparingly.

The traditional food guide pyramid was replaced in 2005 by MyPyramid, which presents the proportion of different food groups recommended by dividing a triangle into color-coded vertical sections; the width of each section represents the relative proportion recommended for that food group. The MyPyramid.gov Web site, includes the MyPyramid Plan, an interactive tool that allows calculation of personalized dietary recommendations. While the ability to create customized recommendations and the incorporation of considerations such as activity level represent an advance over the traditional food pyramid, the graphical presentation of MyPyramid makes it less useful as an educational tool. The reason is that

while the traditional food pyramid included pictures of food and was labeled with the names of the groups and recommended servings, MyPyramid uses only colored sections whose meaning is not clear without access to a printed key explaining the meaning of each color. In addition, because there are no generalized serving recommendations included in MyPyramid, a person without access to a computer cannot obtain information on the number of servings recommended for each food group.

MyPyramid has six sections: vegetables, grains, fruits, meat and beans, oils, and milk. Specific numbers of servings are not included in MyPyramid graphics but must be calculated depending on the individual's age, gender, and activity level. For instance, for a 50-year-old woman getting 30–60 minutes of moderate or leisure exercise per day, MyPyramid Plan recommends 2,000 calories per day. This includes six ounces of grains, 2.5 cups of vegetables, two cups of fruit, three cups of milk, six teaspoons of oil, and 5.5 ounces of meat and beans. Each recommendation comes with further advice, such as consuming at least three whole grains per day and cups of dark green vegetables weekly, and basic information about the importance of physical activity is also included. For a 21-year-old male getting more than 60 minutes of physical activity per day, MyPyramid Plan recommends 3,000 calories daily, including 10 ounces of grains, four cups of vegetables, 2.5 cups of fruits, three cups of milk, seven ounces of meat and beans, and 10 teaspoons of oil. MyPyramid Plan includes the option of entering weight and height, in which case dietary recommendations may be modified. For example, a 5' 6" 50-year-old woman weighing 130 pounds receives the 2,000-calorie advice mentioned above, while entering the same information with a weight of 180 pounds triggers a message that the weight is not in a healthy range for the height. MyPyramid Plan then presents the user with two options: either generating food recommendations for maintaining the specified weight, or for a diet that would cause gradual weight loss. In the former case, a 2,400-calorie daily diet is recommended; in the latter case, the recommendation is to consume 2,000 calories daily, with a corresponding adjustment in the servings from each food group.

Both incarnations of the food pyramid, and previous USDA dietary recommendations as well, have been criticized as based on shaky science, unduly emphasizing meat and milk products, ignoring food consumptions patterns common to different ethnic groups, and failing to incorporate recent information about nutritional requirements and healthy eating.

The Harvard School of Public Health has created an alternative food guide pyramid called the Healthy Eating Pyramid. It is composed similarly to the traditional food pyramid and is divided into seven horizontal layers including nine food groups. It also incorporates pictures of food, names of the food groups, and serving recommendations, so that it can be used as a stand-alone graphic similar to the traditional food pyramid. The base of the Healthy Eating Pyramid is daily exercise and weight control. The second layer is composed of whole grain foods (recommended to be consumed at most meals) and plant oils, including olive, canola, soy, and peanut oils.

The third layer is split into vegetables (to be consumed "in abundance") and fruits (two to three times per day). The fourth layer is nuts and legumes (one to three times per day). The fifth layer is fish, poultry, and eggs (zero to two times per day), and the sixth layer is dairy or a calcium supplement (one to two times per day). The seventh layer includes two food categories, both of which are to be consumed sparingly: red meat and butter, and white rice, white bread, white pasta, potatoes, soda, and sweets. Notable changes from the USDA pyramid include the emphasis on plant products and the deemphasis on animal products, and the inclusion of weight control and exercise as a basis for a healthy diet. There are also recommendations for most people to take a multivitamin, and for moderate alcohol consumption if appropriate.

SEE ALSO: Department of Agriculture; Government Agencies; Government Policies and Obesity; Nutrition and Nutritionists; Nutrition Education.

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Food Insecurity and Obesity

THE PREVALENCE OF both food insecurity and obesity are increasing in the United States. Food security for a household is defined as access by all members at all times to enough food for an active, healthy life. In contrast, food insecurity describes not having ready availability of nutritionally adequate and safe foods, and an assured ability to acquire acceptable foods in socially acceptable ways, without scavenging, stealing, or resorting to emergency food supplies.

Food insecurity has been identified as a predictor of obesity. Those who are confronted with a threat of food deprivation tend to have higher rates of obesity. A number of potential mechanisms have been suggested to explain this seemingly paradoxical association, including the overconsumption of inexpensive high-energy foods; physiologic adaptation of increased body fat in response to periods of overeating followed by periods of deprivation, commonly known as the "food stamp cycle"; and stress- or anxiety-induced biological changes. Economic variables appear to be the primary link. As such, in the United States, where most people's experience with hunger or food insecurity is sporadic or episodic (as opposed to continuous or chronic), hunger and obesity can and do coexist. However, as might be expected, very low food security often characterized by feelings of hunger is associated with a lower risk of obesity.

Food security is commonly measured using a questionnaire developed by the United States Department of Agriculture (USDA). The USDA measures the nation's food security annually. Despite a general societal perception of food surplus, national statistics related to the prevalence of food insecurity revealed that in 2005, 12 percent of all households were food insecure. This number has risen over the past five years.

Food insecurity interacts with poverty status to influence obesity. As such, weight gain and food insecurity can affect the same individuals. Both food insecurity and obesity rates tend to be highest for limited-resource individuals, particularly women. (Obesity may be linked to women's habits of periodically going without food so that their children can eat.) Limited-resource individuals confront many barriers to the procurement of healthy food, such as high costs of "healthy" foods, limited access to large supermarkets, and prevalence of fast-food restaurants

HIGH COSTS OF "HEALTHY" FOODS

Consuming energy-dense foods contributes to weight gain. Energy density is defined as the quantity of energy per unit of edible weight. The energy density of foods is a function of their water content. Low energy-dense foods are heavily hydrated. High energy-dense foods are dry and contain large quantities of fat and sugar and typically few nutrients. Examples of high energy-dense foods include most fast foods, soft drinks, and snack foods, such as potato chips and cookies. Low energy-dense foods include lean meats, fruits, and vegetables.

A unit of energy from high-fat, high-sugar foods, such as potato chips and cookies, is typically less expensive than a unit of energy from fruit and vegetables. In other words, high energy-dense foods cost less than low energy-dense foods. In relation to this concept, it is important to consider the relationship between energy density (kcal/kg) of selected foods and their "energy cost" as expressed in kcal/$. For example, the energy cost of potato chips is 1,200 kcal/$ and the energy cost of soft drinks is 875 kcal/$. In contrast, the energy cost is approximately 250 kcal/$ for fresh carrots and approximately 170 kcal/$ for frozen orange juice. The difference in cost per calorie between the high energy-dense foods and those of lower density is more than sixfold. High energy-dense foods provide a consumer with more calories per dollar than lower calorie choices. Over the past few decades, retail price increases have been consistently lower for sugars/sweets and fats and oils than for nutrient-rich foods such as fruits and vegetables.

Food-insecure individuals often maximize the value of the dollar by purchasing foods that are cheaper in cost. As a result, this group tends to consume more calories per dollar than higher-income populations. For example, limited-resource shoppers on average pay 12 percent less per pound for ground beef,

yet they shop in stores where ground beef is costlier. (Ground beef costs are directly related to its quality and fat content.) This discrepancy can be explained in that limited-resource shoppers tend to buy low-quality, high-fat beef, because they cannot afford more expensive cuts. Foods richer in nutrients like lean cuts of meats, fish, and fresh fruits and vegetables are commonly more expensive. Correspondingly, individuals with the lowest household incomes (below 130 percent poverty) consume the fewest fruits and vegetables of any income group.

Economic constraints can induce an increase in dietary energy density in food-insecure individuals consuming a low-cost diet. This is so because individuals eat a constant weight of food. If individuals consume foods that contain a greater amount of energy per unit of weight, the result may be an overall increase in energy intake that can result in weight gain.

LIMITED ACCESS TO LARGE SUPERMARKETS

The built environment, broadly defined as the man-made surroundings in which individuals function, strongly influences the purchasing and consumption behaviors of individuals. Limited-resource, food-insecure populations tend to live disproportionately in areas where there are fewer large supermarkets than where higher-income groups reside. Consequently, this group is more reliant on smaller shops, convenience stores, and bodegas to acquire food. Not only do these smaller stores offer a limited selection of foods, but also their prices are typically higher than those of large supermarkets. Food availability in stores is linked to the diets of residents in the area of the stores. More healthful product offerings in stores are associated with increased consumption of more healthful foods by individuals living near the stores. As follows, those who live and shop in limited-resource neighborhoods tend to be more overweight than those who shop in wealthier areas.

PREVALENCE OF FAST FOOD RESTAURANTS

Limited-resource individuals are obtaining a greater percentage of their food outside the home and commonly from fast-food places where inexpensive, energy-dense foods dominate the menus, and "value" meals, such as oversized burgers, extra-large servings of fries, and buckets of soda are promoted. Food eaten away from home tends to contain higher amounts of total fat, saturated fat, and be more energy dense than foods eaten at home.

Food insecurity is associated with lower nutrient intake but an overall increase in energy intake. In food choice decision making, when financial resources are lacking, nutrition and health tend to function in a secondary role to economic considerations. Households reduce food spending by changing the quality or variety of foods consumed before they reduce the quantity of food eaten. As a result, while families may get enough food to avoid feeling hungry, they may be poorly nourished because they cannot afford a consistently adequate diet that promotes health; the need to satisfy hunger frequently overrides considerations for a healthy weight.

SEE ALSO: Access to Nutritious Foods; Hunger; Obesity and Socioeconomic Status.

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Food Intake Assessments in Children

A NUMBER OF methods exist for assessing dietary intake of children, including food frequency questionnaires, dietary recall, food records, doubly labeled water, and observation of intake. Assessing food intake in children is critical for both research and clinical purposes. To assess the connection between eating behaviors in childhood and physical and psychological health, researchers must have methods by which they can measure food intake. In clinical settings, assessment of food intake is necessary to appropriately formulate dietary interventions.

Different assessment methods have unique strengths and weaknesses and the decision of which method to use can depend on factors such as the reason for assessment, degree of accuracy required, and amount of time and resources available.

Food frequency questionnaires (FFQs) are used routinely to assess dietary intake in adults and less frequently utilized in children. These questionnaires query the parent or the child, if he or she is old enough to satisfactorily complete the form, regarding the frequency of the child's intake of various common foods and beverages over a given time. A few studies have suggested that such questionnaires may be useful in assessing the dietary intake of children, particularly in larger, epidemiological studies. The strengths of this method are that it is inexpensive, easily administered, and imposes little burden on the subject and the investigator. However, the accuracy of FFQs in estimating children's intake is questionable. Several studies have documented a tendency for FFQs to overestimate energy intake in children. Additionally, the FFQs used for adults may not be appropriate for use in children, because reference portion sizes used are generally larger than those recommended for children and the listed foods may not provide a representative sample of a child's diet. This may be remedied through the use of modified FFQs, which include more "kid-friendly" foods and portion sizes.

Dietary recall is another popular method used to assess intake. Using this method, the child and/or a parent is asked to retrospectively report all food and drink consumed by the child over a certain time (usually 24 hours). This method appears to have a reasonable level of accuracy when used on child samples. There are, however, some concerns about this method. Significant variability has been reported in the degree of accuracy, with some parents and children reporting intake with excellent and others with poor accuracy. Further, ethnicity and weight-status may affect the accuracy of recall assessments. For instance, overweight children are more likely to underestimate intake, while normal-weight peers are more likely to overreport intake.

Both FFQs and dietary recall rely on retrospective report. Studies in adults have long demonstrated a tendency to misreport caloric intake due to difficulties with recall, desirability effects, and lack of nutritional knowledge. This effect may be more pronounced when assessing a child's intake. Children may have more memory, time-conceptualization, and comprehension difficulties when reporting intake. They also tend to be less knowledgeable of the portion sizes and food preparation of foods consumed. Parent recall is similarly imperfect. Such methods rely not only on retrospective recall, but also on third-party observation. Parents can only report on the eating behavior that they directly observe and may therefore inaccurately report on meals and snacks eaten outside the home and any secretive eating. Accordingly, parents appear to be less accurate in reporting child's snack intake and disordered eating behavior.

To avoid the errors associated with recall, some studies have attempted to use weighed or estimated food records, doubly labeled water, or direct observation of food intake in order to assess children's eating. Using the food record method, children and/or their parents are asked to record the child's food and beverage intake over time, including information regarding specific types and portions of foods eaten. In the case of weighed food records, food is measured and weighed before and after eating in order to determine the quantity of any leftover food and drink. In the case of estimated records, these food quantities are estimated.

This method allows greater accuracy than recall methods by eliminating the problems associated with retrospective reporting. Although records are time-consuming to complete, fewer days of recording may be required to assess intake in children than in adults due to the lesser degree of variance in dietary intake in childhood. Food records are burdensome to complete; therefore, a high level of subject commitment is required. Accuracy of the data can be compromised by fatigue, forgetfulness, lack of compliance, effects of monitoring on intake, and desirability effects. This method also requires more of the investigator's time to explain to methods to the subject and to analyze. In the case of weighed records, special equipment is needed, which may be expensive to obtain.

The doubly labeled water technique involves tracking isotopes in an altered water solution through a child's system to measure metabolic rate. This is typically considered the most accurate measure of energy intake for children. However, there are limitations to the method. Growth must be taken into account when using this assessment in children and

proper gut function is required for accurate results. This method requires staff trained and knowledgeable in how to administer the test and analyze the results and it tends to be a more costly method. Additionally, while this method provides an accurate assessment of caloric intake, it cannot provide any information regarding the types of foods consumed or the eating behavior of the subject.

Another technique often utilized in the assessment of dietary intake in children is direct observation by investigatory staff. According to this method, a third-party observes children's eating habits directly in either a naturalistic (e.g., school cafeteria) or laboratory setting. This method helps to reduce the error associated with child- and parental-report methods, provides greater objectivity, and permits investigators to examine eating behaviors in addition to intake. Additionally, observation in the laboratory allows for controlled conditions and manipulation of target variables. There are specific limitations associated with this method. If aware that they are being observed, children may intentionally or unintentionally alter their eating behavior. This method is time-consuming, often costly, and can be burdensome for both the subject and the investigator. For this reason, it is difficult to use such methods to assess a child's eating habits over longer periods. If laboratory conditions are used, an artificial environment is created, and therefore results may not generalize to more typical eating situations.

When selecting a method for assessing children's eating habits, knowledge of the particular benefits and challenges associated with each method is critical. Such knowledge can help a researcher or clinician to select the most appropriate assessment method for the given circumstances and can reduce inaccuracies by anticipating difficulties associated with particular techniques.

SEE ALSO: Doubly Labeled Water.

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Food Intake Patterns

FOOD CONSUMPTION PATTERNS, including cooking practices and eating behaviors, are a set of norms, rules, and principles that population groups have established to provide nourishment, healing, and comfort to them. Examination of food consumption patterns, instead of intakes of single nutrients, gives a better idea of the relationships between diet, health, and disease patterns affecting population groups. In fact, people do not eat single, isolated nutrients nor do nutrients exert their effects in health status in a vacuum. On the contrary, people eat foods in the most complex and intriguing combinations, with several nutrients and other food compounds acting either synergistically or antagonistically. Therefore, to decipher the dietary effects on health, it makes sense to study foods as they are consumed instead of focusing in single nutrients.

Food patterns based on low-fat, nutrient-dense foods, particularly fruit and vegetables, have beneficial effects on cardiovascular health, protect against certain cancers, and are associated with healthier body weights. In the opposite direction, food patterns of poor diversity and those that include foods high in energy and poor nutrient density are associated with cardiovascular disease, Type 2 diabetes, certain cancers, and obesity.

Food patterns develop slowly, with the input of several factors associated with the physical environment and food availability. They are highly determined by cultural influences, as people construct their preferences, choices, perceptions, beliefs, and attitudes about foods on the basis of their ethnic and cultural values. Such cultural constructs are not always consistent with scientific knowledge regarding the safety or nutritional values of foods. Based upon the constructs of their specific cultures, different foods may be considered preferable and acceptable by some groups, but rejected and unacceptable by others. However, food

How people eat is complicated, making the picture of an individual's pattern of food intake difficult to decipher.

patterns are composed of dynamic constructs that are modulated and redefined as result of the interaction of people from different cultures and ethnicities.

Food choices affect health and well being, although these are not necessarily influential in such decisions. Social, cultural, and economic considerations are often decisive when selecting what to put on the table. It is notorious how the American population, since colonial times, has tested, improved, and adopted several foodstuffs, which are now integral elements of its eating patterns. A classic example is the food pattern defined to celebrate the Thanksgiving holiday—foods and meals incorporated into this celebration play important social, cultural, and emotional roles intrinsic in the American cultural identity. The food patterns followed by populations from the Mediterranean region, and known as the Mediterranean diet, developed slowly during centuries and became inserted into the lifestyles of these populations; such diet is associated with greater longevity and reduced mortality and morbidity for cardiovascular disease, certain cancers, and other nutrition-related diseases.

The easy and inexpensive access to food and the fluidity associated with the globalization of the food markets, along with several other factors influencing food choices, have brought profound changes in eating patterns of the population, including more diverse combination of foods, frequent snacking, and larger portion sizes. These changes in eating patterns, plus the perceived decreases in levels of physical activity, are associated with the epidemic of overweight and obesity that currently affect Americans. Efforts to promote better diets among the American population need to aim at the maintenance or adoption of healthful dietary patterns based on both cultural and modern foods that could satisfy the biological, emotional, and social needs of this population. In particular, efforts should be concentrated in guiding the population to adopt food patterns aimed at restoring or maintaining healthy body weights as more and better strategies are needed to halt or alleviate the current epidemic of obesity.

SEE ALSO: Food Guide Pyramid; Food Preferences; Fruits and Vegetables; Healthy Eating Index; High Carbohydrate Diets; Increasing Portion Sizes.

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Food Labeling

REGULATION OF FOOD labeling in the United States is largely within the purview of the U.S. Food and Drug Administration (FDA), an agency within the Department of Health and Human Services. The Center for Food Safety and Applied Nutrition within the FDA is responsible for regulating the safety and accurate labeling of most foods; the major exceptions are the products of domesticated animals (such as cattle and poultry), alcoholic beverages, and drinking water. Meat, poultry, and related products containing them are the responsibility of the United States Department of Agriculture Food Safety and Inspection Service. Beverages containing more than seven percent alcohol are regulated by the Bureau of Alcohol, Tobacco, and Firearms, part of the U.S. Department of the Treasury. Nonbottled drinking water is regulated by the Environmental Protection Agency, a cabinet department within the federal government. Bottled water is regulated by both the FDA and state governments, and dietary supplements are regulated by the FDA as foods (meaning they are not subject to the same safety and efficacy testing applied to drugs). The Federal Trade Commission, an independent agency within the federal government, governs the regulation of claims made for food products or supplements in advertising.

The United States government's role in regulating food labeling dates back to the late 19th century. The FDA's origins lie in this era also, as its beginnings can be traced back to the Division of Chemistry created within the Department of Agriculture. One of the activities of the Division of Chemistry was research into the adulteration and mislabeling of foods and drugs; results from these investigations were issued in a series of reports from 1887 to 1902. These reports, reinforced by a general interest in increasing government regulation of matters concerning public health, and the work of crusading journalists such as Upton Sinclair, led to passage of the Food and Drugs Act, which became law in 1906. Among other things, this Act banned interstate transport in adulterated or misbranded food and drugs (the legal structure of the United States generally does not allow the federal government to regulate commerce within individual states). Federal regulatory oversight increased with the founding of the Food and Drug Administration in 1927, and was further strengthened in 1938 with passage of the Food, Drug and Cosmetic Act. This Act remains the basis of most of the FDA's regulatory powers to the present day, although specific regulations are regularly added and revised.

The Office of Nutritional Products, Labeling and Dietary Supplements (ONPLDS) within the Center for Food Safety and Applied Nutrition is responsible for developing labeling policy, regulations, and standards; it was realigned in 2003 into units organized around product categories. Within the ONPLDS, the Division of Dietary Supplement Programs develops and implements policy, regulations, guidance documents and compliance activities related to dietary supplements, and reviews new dietary ingredients, labeling standards, and enforcement actions for adulterated or mislabeled dietary supplements. The Food Labeling and Standards Staff develops policy, regulations, guidance documents, and enforcement strategies concerning food labeling, and participates in international committees such as the Trilateral (United States, Canada, and Mexico) Technical Working Group on Food Labeling, Packaging, and Standards. The Nutrition Programs and Labeling Staff is concerned with scientific and regulatory review of nutrition labels, including health claims, nutrient content claims, and the information required to be provided by the Nutrition Facts panel; this staff also sets policy and creates guidance documents regarding nutritional labeling and promulgates and administers regulations. The Division of Research and Applied Technology provides expert advice to the Center for Food Safety and Applied Nutrition and evaluates the validity of dietary exposure esti-

mates. The Infant Formula and Medical Foods Staff deals with issues related to infant formula.

Basic laws regarding food labeling in the United States include the requirements that label information be conspicuously displayed and be expressed in common English, the amount of food in the package must appear, and if the food is imported, the name of the country of origin must appear in English. The common name of the food must appear, as well as the form (e.g., "whole" or "chopped"), and the individual food components must be listed in order by weight. With a few exceptions, food additives and colors must be listed; the exceptions include the use of most coloring agents in butter, cheese, and ice cream. Nutritional information must be provided for most packaged foods. Claims concerning nutrient content (such as "sodium free" or "low fat") must meet additional standards specified by the FDA.

Primarily the Nutritional Labeling and Education Act of 1990 governs the contents of nutrition labels, which are required on almost all packaged foods intended to be sold to the public. The Nutrition Facts Panel is required by law to contain information intended to help people choose foods that are nutritious and appropriate for their caloric needs. The first information provided is the serving size and the number of servings in the package; most of the information that follows is based on a single serving of the food, so it is important to understand what constitutes a serving in the context. Serving sizes must be given in standard units, such as cups or pieces, and also in metric weight (usually grams). Serving size information is followed by information about the amount of 15 nutrients, listed in standard order, contained in a serving of the food: calories, calories from fat, total fat, saturated fat, trans fat, cholesterol, sodium, total carbohydrate, dietary fiber, sugar, protein, vitamin A, vitamin C, calcium, and iron. This information is also presented as the percentage it constitutes of a recommended 2,000-calorie diet, and reference information about the amount of fat and saturated fat, cholesterol, sodium, carbohydrate, and dietary fiber recommended for a 2,000 and 2,500-calorie diet is also presented. If claims are made on the label about other vitamins and minerals for which a Reference Daily Intake (RDI) has been established, the amount of these ingredients must be also listed. Other information may be included on a voluntary basis, including calories from saturated fat and amounts of polyunsaturated fat, monounsaturated fat, potassium, soluble fiber, insoluble fiber, sugar alcohol, other carbohydrates, other vitamins and minerals, and beta-carotene. The addition of trans fat (trans fatty acids) to the labeling regulations (effective January 2006) is the first significant change since the Nutritional, Labeling, and Education Act regulations were finalized.

Strict definitions have been set for the following terms used in claims on food labels: free, reduced, lean, less, light, extra lean, low, fewer, high, more or extra, and good source. Definitions of the requirements for the use of each term are available from the Center for Food Safety and Applied Nutrition Web page. For instance, the word *free* must be used to signify that a product contains none or only a physiologically trivial amount of an ingredient. This is defined according to the context; for instance, in the case of "calorie free," it means five calories or less per serving, and in the case of "sugar free" or "fat free," it means less than 0.5 grams per serving. To take another example, "lean" and "extra lean" are used to describe meat, poultry, and similar products: lean means less than 10 grams of fat, less than 4.5 grams of saturated fat, and less than 95 mg of cholesterol per serving and per 100 grams, while extra lean means less than 5 grams of fat, 2 grams of saturated fat, and 95 milligrams of cholesterol per serving and per 100 grams.

The Food and Labeling Consumer Protection Act of 2004, which came into effect on January 1, 2006, requires that food labels clearly state if the food contains any protein derived from eight major allergenic foods. This regulation is a response to the fact that about 2 percent of U.S. adults and 5 percent of infants and young children suffer from food allergies, that about 150 Americans die each year from allergic reactions to food, and that the eight major allergens account for about 90 percent of all documented food allergic reactions. The eight major allergens are milk, eggs, fish, crustacean shellfish, tree nuts, peanuts, wheat, and soybeans. If a food contains any of these ingredients, they must appear in the list of ingredients or an extra statement including the food's common name beginning with "contains" must appear adjacent to the list of ingredients, e.g., "contains milk" for a product that include the milk-derived protein casein.

SEE ALSO: Carbohydrate and Protein Intake; Fat Intake; Food and Drug Administration; Government Agencies; Nutrition Education.

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Food Marketing to Children

ADVERTISEMENTS PERMEATE CHILDREN'S lives through all forms of media. Food advertising to children, in particular, has been the subject of great interest because of the volume experienced by children, the types of foods advertised to children, and the vulnerability of many children who cannot comprehend the nature of advertising. Further, an association has been made between food marketing to children and childhood obesity, although a causal relationship has not been established. Many studies document the types and effects of food marketing to children, and the results have only fueled debate on the topic. The food and beverage industry has attempted to allay concerns by introducing mechanisms for self-regulation. Internationally, many countries regulate marketing to children, but the United States does not. As such, a major unanswered question is whether the food industry can responsibly self-regulate food advertisements to children, or whether governmental oversight and policy action is required.

Obesity in children is now a well-known concern in many countries, including the United States. Recent decades have seen a doubling of the prevalence of obesity in children and a tripling in adolescents. Many causes have been identified as part of this problem, and diet is primary among them. Children are consuming more energy-dense and less nutrient-dense foods, and are thus failing to meet many nutrition recommendations. Children and adolescents consume calories, added sugar, fat, and saturated fat in excess. Conversely, they fall short in consuming whole grains, fruits, vegetables, and a variety of vitamins and minerals. Studies suggest children and adolescents are consuming a large percentage of their calories from snacks and sugar-sweetened beverages, and they are eating more and more foods away from the home. Many of these trends contribute to obesity rates in children, and there is question as to whether food marketing to children plays a role.

Food companies spend a large amount of money on advertising, especially on the numerous new food products introduced for children each year. Children and adolescents have become an important consumer population for food companies because they have spending power in the order of billions of dollars. Not only do children have money of their own to spend, they also influence the spending of others in their household. Because of the major role children and adolescents play in the marketplace, the food and beverage industry spends an estimated $10 billion on youth-focused marketing each year. Overall, the food industry is second in advertisement spending only to the automobile industry in the United States. The majority of food industry marketing dollars has gone to breakfast cereals, candy and gum, soft drinks, and snack foods. Advertising budgets for these food categories are large because many of the thousands of new children's food products introduced each decade fall into these categories. According to a report by the Institute of Medicine, from 1994–2004, 58 percent of all new children's foods were candies, snacks, cookies, and ice-cream products. Conversely, very few new fruit or vegetable products were introduced to the marketplace for children, and nearly all studies on the topic conclude that healthy food products are minimally advertised to children. As a result, more money is spent advertising energy-dense foods than nutrient-dense foods.

Food marketing to children comes in many forms because children interact with a wide variety of media, and for extensive periods of time. Children and youth spend many hours a day with media of all kinds. The majority of these hours are spent watching television,

but children and youth also spend significant amounts of time listening to the radio and using computers to browse the Internet. Food and beverage companies take advantage of this wide exposure to media to advertise their products, and they use every available media outlet. Most advertisements are conventional in nature, such as television or radio advertisements, billboard messaging, food packaging that is attractive to children, and product promotions. Other forms of advertisements constitute novel developments in marketing to children and include product placement in movies and video games, "advergaming" related to specific food products or brands, licensing of cartoon and other children's characters from movies or television shows, internet promotions, and food-product-based Web sites.

Children and adolescents are exposed to large amounts of advertising in a variety of venues. For instance, it is estimated that children are exposed to tens of thousands of television advertisements each year. Among these advertisements, foods are the most frequent products being promoted. However, the food industry has been reallocating marketing dollars toward other venues, such as the school setting and the Internet. Within schools, youth are exposed to a variety of advertisements in the form of brand names associated with vending machines, fast food vendors, and placements in school newspapers or on school televisions. The opportunities to advertise through these outlets are many, as the vast majority of middle and high schools in the United States maintain vending machines in their hallways. A large number of high schools also offer branded fast foods, and many middle and high schools contract with Channel One, a news program for children that includes advertisement time for foods and beverages.

Some advertising to children is meant specifically to increase brand recognition beyond the marketing of a single product. A variety of techniques exist to increase children's interest in particular brands of foods or beverages. Many children's breakfast cereals, for instance, include recognizable, brightly colored characters that are designed to exhibit fun and excitement. These friendly spokescharacters help increase loyalty toward a particular brand in children. Similarly, character licensing, in which movie characters or other recognizable children's characters are included on food packaging or promotions, also increase awareness of particular brands over others. Other strategies include use of pop-culture spokespersons, such as athletes or actors; inclusion of toys or other prizes within food products; and most recently, online activities and virtual environments in which children may play, all the while being exposed to brand names. These strategies are increasingly used across the entertainment and food industries.

The pervasive nature of food marketing, as well as the increasingly sophisticated methods used to advertise to children, might be related to children's dietary preferences. Researchers have hypothesized that exposure to advertisements might greatly affect children's attitudes toward, and interest in, particular foods. Concern has developed over food marketing to children because many studies have linked food marketing to food choices of children. The majority of studies conducted on the effects of advertising to children have shown that children choose advertised products significantly more often than do children who were not exposed to advertising. Also, advertising increases children's attempts to influence purchasing decisions of their parents, a concept referred to as "pester power." Further, the majority of foods and beverages advertised to children are high in sugar and fat, and low in nutrients. For example, the Institute of Medicine has concluded that the majority of television advertisements for food and beverage products for children are energy dense but not nutrient dense. The Institute of Medicine also has concluded that advertising, at least on television, can influence children and youth such that they both prefer these types of food products and request them more often.

A more important question might be how advertising is related to actual dietary intake among children and adolescents, as well as its relationship with overweight in children. Intriguing data exist for the youngest age groups, who might be most vulnerable to advertising in general. Consistent evidence supports the role of television advertising in influencing the short-term food choices and consumption patterns of children under 11 years of age. There is also evidence to suggest that television advertising might affect long-term intake of young children up to 5 years of age. This might be the case because young children, especially those under the age of 8 years, do not have the capacity to distinguish between regular television and other programming, and the advertisements interspersed

within the programming. Even older children might not be media-savvy enough to protect themselves in all cases against advertisements. Further, many studies have established an association between exposure to food advertisements and childhood adiposity. The evidence for this association, however, does not establish causality.

Given the extent of food marketing to children in the United States, and its potential to affect the diets and health of children, many questions have arisen regarding how best to regulate advertisements to children. In response, the food industry developed its own regulatory agency, the Children's Advertising Review Unit (CARU). The agency was founded by the National Advertising Review Council in 1974, is run by the Council of Better Business Bureaus, and is funded by all industries that advertise to children, including the food industry. CARU maintains and updates a list of guidelines meant to direct industry advertising in a responsible manner. Using these guidelines, CARU conducts reviews of advertising to children, can accept and review complaints about advertisements, and will make determinations as to whether advertisements violate its guidelines. When violations do occur, CARU requests voluntary action from the advertiser to resolve the problem. Because there is a high level of commitment by industries to CARU guidelines, some health organizations, such as the Institute of Medicine, have suggested working with CARU in support of this form of self-regulation. However, some public health advocates and organizations have argued that self-regulation is not sufficient to protect vulnerable children. Organizations such as the American Academy of Pediatrics and the American Psychological Association have called for stronger government regulation of food marketing to children. Worldwide, action has been taken on the issue. Many nations currently regulate advertisement to children, and in some cases, advertisement to children has been banned or severely limited. The World Health Organization also has weighed in on the issue, publishing a set of guidelines on advertising to children through the International Obesity Task Force. Referred to as the "Sydney Principles," the guidelines provide suggestions to reduce or entirely eliminate commercial promotions to children. In the United States, however, governmental regulation remains a controversial issue.

SEE ALSO: Advertising; American Academy of Pediatrics; Children's Television Programming; International Obesity Task Force; Television.

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Food Preferences

THE TERM *PREFERENCE* refers to the selection or choice of one item over the other. Thus, *food preferences* mean one's choice or selection of some foods but not others. In common usage, however, food preferences simply refer to the foods that one likes. Food preferences vary across individuals, particularly between people who are from different cultural backgrounds. For example, some children born in regions of India or Africa acquire preferences for chili peppers when they are young, while the typical American child tends to find these foods too hot and often dislikes them. A combination of genetic predispositions and environmental factors influence human food preferences, and for every individual, these factors might be different, thus complicating the study of human food preferences. Of importance to the study of human obesity, most humans tend to prefer foods that are sweet or high in fat, and these foods are often the most energy dense and overindulgence can result in obesity. This entry will give an overview of how food preferences are formed, and will review the salient factors that affect human food preference, both genetic and environmental. Where possible, direct parallels will be drawn between food preferences and obesity.

A widely accepted but incorrect viewpoint is that food preferences are innate or inborn responses to the

Common belief holds that a person's food preferences are at least partially determined at birth. In truth, such food preferences can be and frequently are learned throughout a lifetime of eating.

body's need for specific nutrients. This view stemmed largely from a misinterpretation of the work of pediatrician Clara Davis, who performed studies in the early part of the 20th century where toddlers were offered a variety of foods, and from these, they tended to choose "healthy" foods that were suitable for development. Because Davis used such a limited variety of foods in her study, all of which tended to be healthy, these findings cannot be translated to the current food environment, rich with energy-dense and palatable convenience foods. In this environment, it is apparent that children do not always choose healthy foods, and in fact, need much guidance to learn to prefer foods that will result in optimal health and avoid the development of obesity.

Researchers tend to agree that food preferences begin to develop early in life and are primarily learned as a result of interactions a child has between food and his or her environment. The terminology applied to this process has most commonly been Pavlovian or associative conditioning. The term *environment* in this case can mean any context, social cue, or postingestive (biological) consequence that is paired with recent ingestion of a food, or an eating experience. Thus, food preferences are not innate or inborn, as suggested by Davis's early work, but rather are learned throughout life by any number of these shaping experiences. As an example, high-fat foods like desserts and sweets are often used as rewards or are the centerpieces to many holidays. Some have argued that the positive experiences that surround these foods serve to increase preferences for them. In contrast, vegetables are often presented as contingencies to dessert foods, when parents use tactics such as "eat your vegetables, or you can't have any dessert." Research from Leann Birch's laboratory, a child psychologist from Penn State University, has determined that these strategies can decrease preferences for vegetables in the long run, possibly because the contextual cues to

consumption of these foods are predominantly negative for the child experiencing them.

Of additional importance to food preferences are postingestive consequences that can result either in food aversions (dislikes) or in increased preferences. Food aversions can result from ingesting a food that makes one sick or nauseous. The next time this food is ingested, humans will typically dislike the taste of it, and this is likely the organism's protection mechanism against consumption of potentially toxic food sources. In the same respect, positive postingestive consequences are often experienced from consumption of sweets or sugar-containing foods. These may partly be mediated by the positive postingestive consequences that come with the metabolism of sugar. In fact, studies have demonstrated with sugar and with fat that humans will tend to prefer flavors that are matched with a higher amount of calories (such as sugar and fat), as opposed to conditions where few or no calories are given (such as artificial sweetener or fat-reduced items). These preferences for the higher calorie version are likely associated with positive postingestive consequences to eating these foods and may serve to guide the organism toward foods that contain calories, and therefore will promote growth.

While food preferences are primarily learned, there are some well-researched genetic predispositions across humans that can affect this learning process. For example, humans and many other organisms tend to be born with a liking for sweet substances and a disliking of bitter and sour ones. These are likely protective and serve to drive the organism to ingest calories (sweet) and avoid toxic and bitter substances (bitter and sour). In addition to these inherent taste preferences, humans tend to be universally neophobic at birth, which is the fear of ingestion of novel edibles. Again, this serves to protect the organism from ingestion of potentially harmful food sources, and while most important in childhood, adults can exhibit neophobic reactions to food as well. Finally, the most well-known inherited difference in basic taste response is the inherited ability to taste bitter thiourea compounds, such as phenylthiocarbamide (PTC) and 6-n-propylthiouracil (PROP). Humans tend to either be sensitive to the taste of these compounds ("tasters") or nonsensitive ("non-tasters"). Researchers have demonstrated that food preferences can vary, depending on whether one is a taster or a nontaster. These genetic predispositions interact with both the food environment and cultural factors to influence and shape human food preferences.

SEE ALSO: Taste Reactivity.

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Food Reward

A FOOD REWARD aims to reinforce or strengthen a behavior through food. For example, a food reward is often used to reinforce a desirable behavior such as achievements or compliance (e.g., a pizza party for elementary students completing a book challenge). Food rewards are frequently used in shaping behavior because food is a primary reinforcer. As a primary reinforcer, food is biologically preestablished to be rewarding. Food satisfies the biological drive of hunger, although further investigation suggests that food rewards are commonly used in the absence of hunger. In many cases, food is eaten for its pleasurable reward of palatability rather than nutritional value. There are two components of eating behavior that explain how food acts as a reward: pleasure of eating, and biological need to eat.

Parenting techniques commonly utilize food rewards. Food can be used to encourage a child to perform a needed task or to maintain good behavior. This practice of using food as a reward is also referred to as instrumental feeding. Baughcum and colleagues conducted focus groups investigating maternal feeding practices inclusive of food to shape behavior. This study found that mothers used food to quiet a fussy baby or a toddler's tantrum rather than using food to satisfy the child's hunger. When a parent grants a food reward to a child who is acting out, the food reward reinforces the bad behavior.

Giving food as a reward for particular behavior can strengthen a person's view of that food item.

This form of rewarding is not effective in maintaining the desired behavior, and instead, reinforces the negative behavior.

Food rewards are problematic by interfering with an individual's ability to regulate physiological hunger over psychological food cravings. Regulating food consumption based on biological need versus the pleasure of eating can be shaped at a young age. Parental feeding studies have shown that food control through reward and punishment during childhood can influence eating behaviors in adulthood. Puhl and Schwartz found that parental food control through reward and punishment places children at higher rates for binge eating and dietary restraint later in adulthood. This finding is consistent with previous research which suggests food in a reward system alters a person's ability to self-regulate food intake based on hunger cues.

Food preferences for rewarding foods are selected primarily for taste. A food reward is commonly a favorite high-calorie food choice. The learned association between appropriate behavior and an appetizing snack can generate food cravings without appetite signals. This drive to seek out desired foods can lead to eating in the absence of hunger and excessive eating.

SEE ALSO: Appetite Signals; Food Intake Patterns; Food Preferences; Hunger.

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Food Stamp Nutrition Education Program

THE FOOD STAMP Program is a cornerstone of the U.S. Department of Agriculture nutrition assistance program. The purpose of the Food Stamp Program is to end hunger and improve the nutrition and health of low-income households. The Food Stamp Program is run at the state level, but is overseen by the federal government with current presence in all 50 states, the District of Columbia, Guam, and the U.S. Virgin Islands. Qualifying for the Food Stamp Program requires the household to meet certain eligibility standards that mainly center on income, looking for work, and citizenship status.

The Food Stamp Nutrition Education Program was created by the Food Stamp Program to provide education to assist households in making healthy food choices on a limited budget. The recommendations and educational initiative are based on the most recent dietary advice provided by the Dietary Guidelines of Americans and the Food Guide Pyramid. Some of the more specific goals of the Food Stamp Nutrition Education Program are to improve the self-sufficiency of Food Stamp recipients, increase skills in food budgeting and meal planning, increase knowledge of proper food choices, increase physical activity, and promote a healthy lifestyle. State governments who choose to participate in the Food Stamp Nutrition Education Program can be reimbursed up to one-half of the costs of the initiative by the United States Department of Agriculture. Currently, there are over 19 million people in the United States on the Food Stamp Program; more than half are children. The Food Stamp Nutrition Education Program will

assist these individuals in making the right choices, within a limited budget, about food and health as they transition from welfare to work.

SEE ALSO: Access to Nutritious Foods; Accessibility of Foods; Changing Children's Food Habits; Expanded Food and Nutrition Program; Food and Drug Administration; Nutrition Education.

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Fruits and Vegetables

ONE OF THE most important messages of modern nutrition is that a diet rich in fruits and vegetables promotes health while protecting against almost all diseases including cancer, cardiovascular disease (CVD), diabetes mellitus, and several other age-related degenerative disorders. Specifically, the intake of 400–600 grams per day of fruits and vegetables is beneficial. Every dietary plant contains numerous types of antioxidants with different properties. Because oxidative stress is common in chronic degenerative disease, dietary antioxidants may explain the protective effect of fruit and vegetable consumption. Many of these antioxidants cooperate in oxidative stress reduction in plants, so the many different antioxidants may also be needed for the proper protection of animal cells.

In addition to essential nutrients, such as protein, amino acids, vitamins, and minerals, plant foods also contain phytochemicals that have additional health benefits. The term "phytochemical" means any compound of plant origin. Phytochemicals may play a key role in reducing chronic disease risk and include phenolics, flavonoids, and carotenoids. Plant sterols, the minor lipid components of plants, may be beneficial due to their cholesterol-lowering effect. Polyphenols are substances with antioxidant activity, which give some flowers, fruits, and vegetables their color. Tea also contains polyphenols. The flavonoids are polyphenolic compounds that have antitumor properties as well as cardioprotective effects with respect to vascular function and platelet reactivity. Flavonoids are universally present as constituents of flowering plants, especially of food plants. Flavanol-rich, plant-derived foods and beverages include wine, tea, various fruits and berries, and cocoa and cocoa products.

Colored fruits are considered a trait that correlates with nutritional values and health benefits. For example, red foods contain lycopene, the pigment in tomatoes, which may be involved in maintaining prostate health, and has also been linked with a decreased risk of cardiovascular disease. Green foods, such as broccoli, Brussels sprouts and kale, contain glucosinolates, which have also been associated with a decreased risk of cancer. Garlic and other white-green foods in the onion family contain allyl sulfides, which may inhibit cancer cell growth. Bioactive substances in green tea and soybeans have additional health benefits. Individuals are recommended to ingest one serving of each of the seven color groups daily, as part of the five to nine servings of fruits and vegetables per day.

Research has shown that people who consume large amounts of fruits and vegetables have a lower incidence of CVD, stroke and tumors. CVD accounts for almost 50 percent of all deaths in industrialized nations and approximately 70 percent of CVD can be prevented or delayed with dietary choices and lifestyle modifications. Of the tree nuts, walnuts, pecans, and chestnuts have the highest antioxidants. Death attributed to cardiovascular and coronary heart diseases show strong and consistent reductions with increasing nut/peanut butter consumption. Because of their chemical structure, plant polyphenols are able to scavenge free radicals and inactivate other pro-oxidants involved in the etiology of many chronic diseases. Many nutrients and phytochemicals in fruits and vegetables, including fiber, potassium, and folate, could be independently or jointly responsible for the apparent reduction in CVD risk.

Currently, less than 25 percent of the American population meets the minimum recommendation of five servings a day. The Western diet contains large quantities of oxidized lipids, because a large proportion of the food in the diet is consumed in a fried,

Most fruits and vegetables are high in both carbohydrates and natural dietary fiber. Research has shown that people who consume large amounts of fruits and vegetables have a lower incidence of cardiovascular disease, stroke, and tumors.

heated, processed, or stored form. In contrast, the Mediterranean diet is high in fruits, vegetables, legumes, and whole grains and includes fish, nuts, and low-fat dairy products. Its high antioxidant content may contribute to the prevention of CVD and possibly several forms of cancer and other diseases. Antioxidants may reduce the risk of atherosclerosis through the inhibition of oxidative damage and the Mediterranean diet appears effective to decrease LDL particle oxidizability.

It has been estimated that 80 percent of cancer in the United States have a nutrition/diet component and 30 to 40 percent of all kinds of cancer can be prevented with a healthy lifestyle and dietary measures. Protective elements in a cancer-preventive diet include selenium, folic acid, vitamin B12, vitamin D, chlorophyll, and antioxidants such as carotenoids (alpha-carotene, beta-carotene, lycopene, lutein, cryptoxanthin). Diets rich in antioxidants and anti-inflammatory compounds may also lower the risk of developing age-related neurodegenerative diseases, such as Alzheimer's or Parkinson's diseases. Research suggests that the polyphenolic compounds found in fruits, such as blueberries, may exert their beneficial effects by altering stress signaling and neuronal communication, thereby protecting against age-related deficits in cognitive and motor function. In addition, a healthy diet that includes five servings a day of fruits and vegetables should optimize the intake of micronutrients required for bone health.

The optimal diet should emphasize fruits and vegetables, nuts, unsaturated oils, whole grains, and fish, while minimizing saturated fats (especially trans-saturated fats), sodium, and red meats. Also, the overall calorie content should be low enough to maintain a healthy weight. Fruits and vegetables have low energy density (i.e., few kilocalories relative to volume), and including them as a part of a reduced-kilocalorie diet

can be beneficial for weight management. However, the actions of the antioxidant nutrients alone do not explain the observed health benefits of diets rich in fruits and vegetables for chronic diseases. Therefore, it is the additive and synergistic effects of phytochemicals in fruits and vegetables that are responsible for these potent antioxidant and anticancer activities, and that the benefit of a diet rich in fruits and vegetables is attributed to the complex mixture of phytochemicals present in plants.

SEE ALSO: Nutrition and Nutritionists; Nutrition Education; Nutrition Fads.

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Functional foods are essentially foods that provide an additional benefit to the body beyond simply supplying energy.

Functional Foods

FUNCTIONAL FOODS ARE conventional foods conferring additional health benefits in addition to providing energy necessary for the body's function. Other terms associated with these foods include designer foods (containing cancer or disease preventive properties), phytochemicals (pharmacologically active food components), pharmafoods, and nutraceuticals (more often dietary supplements made from plant or animal products). The Food and Drug Administration (FDA) regulates the labeling of conventional foods and enriched or enhanced foods for health claims or structure function claims by evaluating the claims of potential health benefits with scientific evidence. This began in 1997 when the FDA ruled oatmeal could claim to be heart healthy.

Health claims relate food or food components to disease or health conditions and are limited to risk reduction and not cures, treatments, alleviation, or prevention. An example of a health claim is low-sodium diets may reduce the risk of high blood pressure.

Structure function claims relate food or food components to normal structure and function of the body and must be truthful and derived from nutritional value. An example of a structure function claim is calcium builds strong bones.

Nutrition professionals recommend a variety of foods in adequate quantities to provide for energy requirements and provide essential nutrients, those vitamins, minerals, and amino acids not produced in the body. Appropriate diet options in the 2005 Dietary Guidelines for Americans include recommendations to maintain a healthy body weight and reduce risks of chronic diseases (cardiovascular, cancer, diabetes). Functional foods may reduce health risks commonly associated with obesity—hypertension, diabetes, hyperlipidemia, coronary artery disease, and some cancers. Because of

the complex nature of nutrients in food and the factors of many components working together to promote health, health claims may be difficult to establish.

Current trends in scientific research of functional foods are aimed at determining the validity of common health claims and analyzing the components of foods for nutritional and health benefits. In general, for a study to be of use in substantiating health claims for functional foods, they should have a large enough sample size with a control group and sufficient duration. The findings should be consistent with or supported by other research.

When evaluating the research performed and used to substantiate a claim, additional factors are important including the sponsor of the research to ensure the information is credible, whether the researcher objective is to sell a specific product, and whether the information is current to keep pace with advances in medicine, science, and nutrition.

Some foods have become mainstays of functional foods for their component properties. Fiber has been shown to reduce feeling of hunger by providing bulk with low calories and may help to decrease blood sugar and cholesterol levels. Two kinds of fiber—soluble and insoluble— have different functions. The soluble fiber found in beans, oatmeal, and apple skin may retard nutrient absorption rates. The insoluble fiber found in bran and whole grains increases intestinal transit and has benefits against colon cancer.

Live active cultures or probiotics in yogurt may prevent gastrointestinal infections and colon cancer, boost the immune system, and reduce allergies. Phytoestrogens—for example, soy, whole wheat, seeds, grains, some vegetables, and fruits—may prevent hormone-related cancers of the breast and prostate, lower cholesterol and prevent memory loss, and possess antiinflammatory and antioxidation properties. Cranberries in foods or in juice for preventing and treating urinary tract infections have been studied and have been in common use for some time. Other research indicates possible reduction in dental plaque as well as anticancer and antioxidant activity. Antioxidants have received much press. As a group, antioxidants scavenge free-radicals created by oxygen-powered metabolism; the by-product is a reactive oxygen species capable of damaging cells. Antioxidants include flavonoids or polyphenols, carotenoids, and omega-3 fatty acids.

Flavonoids or polyphenols found in tea, nuts, and berries may produce antiinflammatory and antiallergenic actions. Carotenoids, a large family of vitamin A precursors including beta-carotene, lutein, and lycopene, are abundant in red and yellow fruits and vegetables with cancer and aging protective effects and offering reduced risks of heart attacks. Omega-3 fatty acids are found in fatty cold-water fish; these acids may reduce risks of heart disease, stroke, and memory loss, and prevent depression.

As part of a healthy diet, functional foods are best served in their natural state and not processed with potentially harmful additives; for example, oatmeal alone could be a better option than a high-fat, high-sugar granola bar. When a food carries a label with a health claim, read the whole label for complete nutrition information to make sure the food fits the diet. To achieve and maintain a healthy weight, read food labels, self-educate with information available from the National Institutes of Health and other health organizations, consult a dietitian or physician for scientifically based health claims regarding food, and follow an appropriate eating plan.

SEE ALSO: American Dietetic Association; Food and Drug Administration; Nutrition and Nutritionists.

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Functional Magnetic Resonance Imaging

OVER THE PAST three decades, magnetic resonance imaging (MRI) has developed into several more specialized imaging modalities including functional magnetic resonance imaging (fMRI). This relatively new technology allows for simultaneous visualization of both structure and physiological function of the brain. fMRI has a variety of applications that have earned it

a place in clinical medicine and have established it as a valuable tool for research. Among its many contributions, fMRI has added to the understanding of the brain as it relates to obesity.

fMRI provides a high resolution and noninvasive report of neuron activity in the brain by detecting blood oxygen levels. Regional increases in neural activity increase oxygen demand and subsequently causes localized increases in blood flow. As a result, oxygen concentration is altered. Detection of these changes, also known as the blood-oxygen-level dependent (BOLD) effect, is the basis of fMRI. Other related functional neuroimaging techniques include diffusion MRI, which measures diffusion of water molecules, and positron emission tomography (PET), which measures uptake of radiolabeled tracer molecules.

Because functional neuroimaging techniques are able to correlate physiological function with anatomy, they provide researchers, neuroradiologists, neurosurgeons, and radiation oncologists the ability to plan more precise treatments that will best preserve brain function. These imaging techniques are also important in the assessment of disease states such as stroke, dementia, seizure disorders, and multiple sclerosis (all disease states that affect the morbidly obese more than normal-weight individuals).

Understanding the brain's response to excessive food intake is a focus of obesity research as it is likely the major contributing factor for developing obesity. For this reason, obesity research with fMRI has targeted both the nonconscious (homeostatic) and conscious (perceptual, emotional, and cognitive) aspects of eating behavior.

The role of the hypothalamus in the nonconscious regulation of energy homeostasis is well established. fMRI has shown a profound and sustained decrease in neural activity of two distinct regions of the hypothalamus after glucose ingestion. This decrease in neural activity was significantly reduced in obese individuals when compared to lean individuals. In addition, several limbic and paralimbic structures such as the insula, hippocampus, and orbitofrontal cortex show exaggerated responses to hunger in obese individuals compared to lean individuals. Difficulty arises when trying to separate hunger under homeostatic control from hunger related to the pleasure of eating.

Functional neuroimaging helps to identify abnormal responses in various brain regions elicited by complex behaviors such as hunger and eating. More research, however, is needed to understand the relationships of these structures and their candidacy as neural risk factors for obesity. fMRI will continue to be an important imaging tool in identifying the role of the brain in obesity.

SEE ALSO: Autonomic Nervous System; Computerized Tomography; Hypothalamus.

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Future of Medical Treatments for Obesity

OBESITY IS A common medical problem worldwide with up to 300 million people falling within the accepted definition of obese (body mass index [BMI] above 30). Current estimates suggest that about a third of the population in the United States is obese, with rising obesity rates among children indicating that the problem will grow in coming years. Being overweight or obese is a risk factor for diabetes mellitus and cardiovascular disease and this combination is best termed *cardiometabolic risk* (CMR). While much interest has been focused in the past decade on the constellation of obesity, diabetes mellitus, cholesterol abnormalities, hypertension, and other factors called the metabolic syndrome, regulatory authorities in the United States and Europe do not recognize metabolic syndrome as a disease entity. Metabolic syndrome as a collection of disorders together does not appear to increase disease risk any more than the individual components themselves. In the future, it is

likely that terminology such as CMR will be favored. Weight reduction is medically desirable as even modest decreases (about 5 percent) in body weight in obese subjects results in a reduction in CMR. There is a great deal of interest in developing new therapies for the future because of the limited efficacy and tolerability of current therapies. Management of obesity includes behavioral, pharmacological, and surgical modalities which all have significant limitations.

DIETARY APPROACHES TO OBESITY

Behavior modification based on diet and exercise regimes is the standard initial approach to obesity management. U.S. government-backed dietary and exercise initiatives for healthy living and obesity containment over the past 20 years have focused on eating a balanced composition of foodstuffs and reducing fat intake have been unsuccessful in stemming the rise of obesity and overweight. A huge selection of commercial diet and exercise programs are available, many of which advocate a high-fat, low-carbohydrate approach, which is in contrast to national diet programs. Only recently have data become available comparing the utility of these approaches, and it is now acknowledged that future dietary recommendations may have to change. Subjects who follow low-carbohydrate diets can experience greater weight loss than other groups over a period of 12 months, thus a diet that is relatively low in carbohydrate and high in protein and fat may allow greater weight loss than diets containing a more traditional bias toward reduction of fat intake. Future studies will focus on whether such results can be applied to the initial medical treatment of the obese population in general.

PHARMACOTHERAPY OF OBESITY

Despite the high prevalence of obesity and the sizable health burden that accompanies obesity, few pharmacologic options are available. The development of successful drug therapies for the treatment of obesity is often stymied by the remarkable complexity of the physiological mechanisms that govern body weight. Indeed, many of these systems are overlapping and interlinked, providing a degree of redundancy, meaning that blockade of one system only can be overcome by continued activity of other systems. Feeding behavior is controlled by a tightly regulated network of central nervous system (CNS), gut, and fat cell (adipocyte) signaling that function to maintain energy storage and balance (known as energy homeostasis). This system has evolved to store energy in times of plenty in order to combat periods of relative scarcity. The continuous availability of plentiful sources of high-calorie foodstuffs in the developed world is a very recent occurrence, which constitutes a major challenge to energy homeostasis.

The approved medications that are available for weight loss include orlistat (a lipase inhibitor), sibutramine (a norepinephrine/serotonin reuptake inhibitor), phentermine, and diethylproprion (sympathomimetic compounds). When all clinical trials of U.S.-approved antiobesity medications are considered, mean weight loss in excess of placebo of 5.7–6.3 pounds was seen with orlistat, a 10 pound weight loss occurred with sibutramine, eight pounds with phentermine, and 6.5 pounds with diethylpropion. While these approved medications are associated with weight loss in excess of placebo during clinical trials, the benefits are modest. The sympathomimetic compounds are associated with cardiovascular side effects (hypertension, increased heart rate), while orlistat is associated with unpleasant digestive system adverse.

A new medical therapy for obesity that has recently completed clinical development is rimonabant, the first clinically useful endocannabinoid-1 receptor (CB-1) antagonist, which has been approved for use by the European Medicines Evaluation Agency in 2006 and is under consideration by the U.S. Food and Drug Administration. The CB-1 receptor is expressed widely throughout the body, including areas of the brain (hypothalamus) and on fat cells. CB-1 receptor blockers probably function in obesity by stimulating fullness, decreasing hunger signals and desire for food, although there is also good evidence that they can regulate fat storage in the liver and by adipose tissue.

After 12 months of therapy with rimonabant, mean placebo-corrected weight loss of 10 pounds was seen, which was accompanied by an improvement in abdominal girth and a variety of secondary metabolic measures. To maintain weight loss, however, continued treatment appears to be necessary. Psychiatric events associated with rimonabant, such as depression and anxiety, have been highlighted and require further evaluation. Given the apparent success of rimonabant as a treatment option for

obesity, many other CB-1 antagonists are in development at this time.

More information on drugs in present use is needed, for example, effects of prolonged treatment (more than one year) and on the comparative effects of concurrent and sequential administration of drugs with nonpharmacologic antiobesity procedures. Also, more data are required on effects of drugs on body fat variables, such as the amount, distribution, and type, which are important factors in the development of obesity and determine consequences for health. Very few data are available on the effects of chronic treatment with existing antiobesity drugs on subjective or objective measures of appetite in obese patients.

NEW TREATMENT APPROACHES IN OBESITY MANAGEMENT

A number of pharmacotherapies are currently under development for the treatment of obesity and related medical conditions. While the mechanisms of action of these developmental therapies are multiple and varied, they can be classified into a number of general categories. First, drugs that block (antagonize) the sensation of hunger and feeding behavior (orexigenic) can decrease food intake and promote weight loss. Second, drugs can be used to boost signals that suppress hunger and feeding behavior (anorexigenic). Finally, drugs can increase the body's consumption of fat or other energy sources. Ideal antiobesity pharmacotherapies might not only suppress appetite, especially for fat, but also oppose its deposition by decreasing its absorption or increasing its metabolism. Concurrent increase of thermogenesis or heat production through consumption of fat stores would also be advantageous. Because it is unlikely that a single drug would have all of these actions, treatments with combinations of drugs should be worth further investigation.

Novel agents for the treatment of obesity are legion, each with their own particular mechanism of action. A selection of newly identified therapeutic targets include oxyntomodulin, neuropeptide Y receptor antagonists, agouti-related peptide blockers, peptide YY, ciliary neurotrophic factor, uncoupling proteins, somatostatin analogs, melanocyte stimulating hormone analogs, coenzyme A carboxylase inhibitors, diacylglycerol acetyltransferase blockers, and adiponectin antagonists. The potential role of these programs for the future medical therapy of obesity will require extensive efficacy and safety assessments in preclinical and clinical trials.

Many therapies that are available for the treatment of other conditions are currently under investigation for their effects on weight loss; most came to light due to adverse events related to weight loss or loss of appetite. One benefit of studying established therapies is the availability of large amounts of data on safety and tolerability in the general population.

The availability of new therapies for obesity in the future depends on the ability to meet the criteria for relevant weight loss and weight-loss maintenance that are set by regulatory bodies. Draft FDA guidance for assessing developmental weight-loss therapies in late-stage clinical trials has recently been released. This guidance indicates that to be considered effective as a treatment to achieve weight loss and maintenance of weight loss, studies with future drugs should meet the following criteria:

- A representative demographic mix of patients at significant risk due to obesity or overweight plus concomitant CMR factors should be included; also a representative sample of patients with extreme obesity should be included.
- A study population of approximately 3,000 subjects, of whom at least 1,500 subjects are randomized to placebo for one year of treatment.
- The primary end point of studies should include mean weight loss and also the proportion of subjects that lost at least 5 percent of their body weight. These figures should be expressed as weight loss in excess of placebo.
- CMR factors are of interest as secondary end points and could include blood pressure, cholesterol, fasting glucose, and waist circumference. For diabetic subjects, the proportion of subjects with improvements in diabetic control as measured by reduction in concomitant diabetic medication use is suggested as an important outcome.

DEVELOPMENTAL PHARMACOLOGICAL TREATMENTS FOR OBESITY

Hormonal Modulation. Leptin is a hormone secreted by fat cells that has a variety of actions that govern energy homeostasis. Leptin decreases feeding behavior (anorexia) and increases baseline utilization of fat and other energy stores, and acts to counteract con-

tinuing food intake and storage in subjects with sufficient stores of fat. Human studies of administration of leptin in patients with obesity were, however, disappointing as it appears that many obese individuals are to some degree resistant to leptin's actions.

Ghrelin is a hormone secreted by cells in the gut in response to fasting and has sites of action in the brain and elsewhere. Ghrelin reduces the secretion of leptin and other hormones and neurotransmitters, and as such, ghrelin antagonists have been studied as obesity therapies

Oleoyl-estrone (O-E) is produced normally by adipose and other tissues and helps to regulate fat mass and body weight. Like the hormone leptin, O-E levels are correlated with percentage body fat in humans, while O-E synthesis by adipose tissue is stimulated by leptin. Experimental animal studies indicate that O-E significantly reduces both food intake and body weight. The effects of synthetic O-E in these studies were most pronounced in obese animal models and weight loss of up to 20 percent was due predominantly to a reduction in fat tissue. This reduction may be due to a central effect by decreasing food intake and peripheral effects on adipose tissue and skeletal muscle mediated through lipoprotein lipase. In early-stage clinical trials, O-E was well tolerated and was associated with weight loss and improvements in blood glucose and cholesterol. Midstage clinical trials of O-E are currently ongoing.

Stimulation of other hormones, such as growth hormone (GH) or blockade of others such as cortisol, have been suggested as potential treatments for obesity based on clinical scenarios of GH deficiency (increased fat mass) and cortisol excess (Cushing's disease, associated with obesity, hypertension, and increased fat mass). While GH therapy is associated with loss of fat mass, its widespread use as an antiobesity therapy is limited by an increased propensity for diabetes. Blockade of cortisol as a mechanism for treating patients with obesity, hypertension, and other CMR factors is currently being studied in early-stage human studies using inhibitors of the enzyme 11-beta-hydroxysteroid dehydrogenase-1.

Type 2 diabetes mellitus often accompanies obesity, and a series of approved antidiabetic drugs, particularly the incretins, have come under scrutiny as potential therapies for weight loss. These include the glucagon-like peptide-1 (GLP-1) agonist, exanatide, which is associated with modest weight loss of the order of 5.5–7.7 pounds during treatment periods of six–12 months duration in patients with diabetes. Large-scale trials in obesity per se are planned. The amylin analog, pramlintide, is approved for the treatment of diabetes mellitus, and has been shown to be associated with weight loss in patients with concomitant overweight/obesity. To address the complexity of the systems that govern energy balance and weight, combination therapies of hormones such as amylin, GLP-1 and leptin-based therapies are being investigated. The oral therapy for Type 2 diabetes mellitus, metformin, is one of the main therapeutic options in diabetic patients with insulin resistance. In multiple clinical trials, metformin has been shown to reduce body weight to a significant degree. As of 2007, weight-loss studies were ongoing with metformin in obese patients.

Neurotransmitter Modulation. Topiramate, an antiepilepsy medication, was known to be associated with weight loss as an occasional adverse event in the clinical-use setting. This led to large-scale clinical trials as a weight-loss therapy for obesity, which have shown some maintained efficacy over 12 months of treatment. Current developmental efforts are focused on improving the delivery of the drug with a preparation that has an enhanced duration of action and a decreased incidence of adverse events. A combination of the topiramate and the sympathomimetic weight-loss agent phentermine is also being studied as a future medical therapy for obesity. Current mid-stage clinical results indicate that this combination was associated with greater weight loss than placebo and than either topiramate or phentermine alone.

Zonisamide is an approved antiepileptic treatment that acts by modulating the activity of the neurotransmitters serotonin and dopamine in the brain. It was noted to lead to decreased appetite as a side effect of treatment and is being studied as a weight-loss therapy. In obese adult patients, 16 weeks of treatment with zonisamide led to significant weight loss as compared with placebo treatment and about half of the zonisamide group lost around 5-percent body weight during the study. Zonisamide is also being studied as an antiobesity therapy when given in combination with bupropion, with midstage clinical trials indicating that 16 weeks of therapy with this combination was associated with significant weight loss in excess of placebo; continued treatment was associated with maintenance of weight loss.

Atomoxetine, a treatment for attention deficit hyperactivity disorder, acts by modulating brain levels of the neurotransmitter, norepinephrine (noradrenaline). In a 12-week study of obese women, atomoxetine treatment led to significantly greater weight loss than placebo. It is currently being studied in patients with weight gain due to medical treatment of schizophrenia.

Bupropion is an antidepressant that modulates CNS levels of serotonin, dopamine, and norepinephrine. Bupropion is also used as a treatment to aid smoking cessation by reducing nicotine cravings, and its potential as a weight-loss therapy may be ascribed to parallel reduction in feeding behavior or hunger. When used as monotherapy, bupropion is associated with modest weight loss in obese individuals. A combination of bupropion and naltrexone (a CNS treatment for opiate and alcohol addiction) is also being studied in patients with obesity. Current results suggest that this combination results in greater weight loss than that achieved with placebo, although the degree of weight loss was moderate.

Lorcaserin is a stimulator of the serotonin 5-HT2C receptor in the brain, which acts to modulate the sensation of hunder and alter feeding behavior. It was designed to overcome adverse events (cardiac valve damage) associated with now-withdrawn weight-loss therapies that stimulated another serotonin receptor, 5-HT2B. In midstage clinical trials, lorcaserin treatment in obese patients led to significantly greater weight loss compared with patients treated with placebo. Larger-scale trials are ongoing to validate these initial findings in a larger population of obese individuals.

Histamine, which plays an important role in allergy, also acts as a neurotransmitter in the brain to modulate feeding behavior. Stimulation of histamine receptors (probably the H3 receptor) in the brain decreases appetite. Betahistine is a prescription medication for vertigo in many countries outside of the United States and acts to stimulate H1 and H3 histamine receptors in the brain. It is a well-tolerated therapy that has been used in more than 100 million people over the past decades. Initial short-term clinical trials have shown betahistine to reduce weight in obese subjects and in those taking medication for schizophrenia.

Amphetamine-like compounds such as phentermine and diethylproprion have a modest effect on body weight, but are associated with adverse events such as tachycardia, nervousness, and potential increase in blood pressure. Interest has focused on β-3 adrenergic receptor subtype activation, as these receptors are believed to play a role in fat mobilization and heat production in adipose tissue. Treatment of obese patients with experimental β-3 adrenergic receptor stimulating drugs has proven to be relatively well tolerated, although weight loss and energy expenditure results have been mixed and it remains to be seen if valid β-3 adrenergic receptor-based drug therapies will emerge.

Gut Absorption Inhibitors. Cetilistat inhibits gastrointestinal lipase, thus blocking the initial breakdown and subsequent absorption of fat from the gut. Early and midstage clinical studies of 12 weeks duration have shown cetilistat to reduce weight significantly more than placebo in patients with obesity and combined obesity and diabetes mellitus. The adverse-event profile of cetilistat in terms of unwanted gastrointestinal side effects may be more favorable than the currently available lipase inhibitor, orlistat.

SEE ALSO: Acomplia; Estrogen Levels; Hormones; Hypothalamus.

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Gallbladder Disease

THE PURPOSE OF the gallbladder is to aid in the efficiency of the fat digestion system. Gallbladder disease occurs when this function is obstructed or slowed down. There are different types of gallbladder disease, the most common of which are inflammation of the gallbladder and gallstones. Obese people are at an increased risk of contracting gallbladder disease.

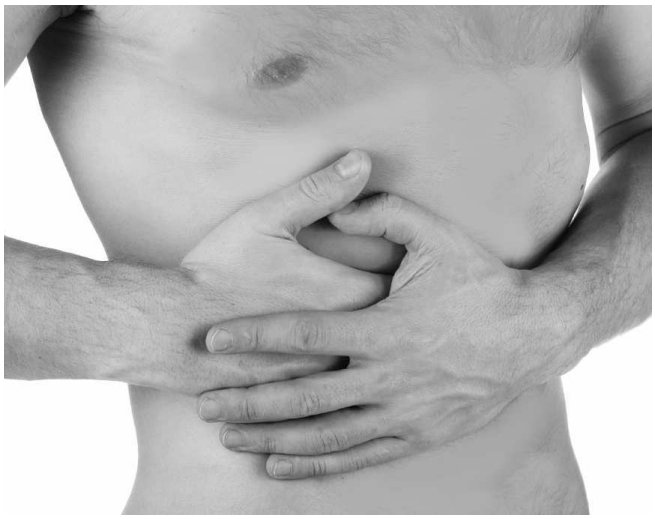
As the name suggests, the gallbladder is a hollow sac-like organ that contains gall (bile). It is the size and shape of a small pear and is located under the liver on the right side of the abdomen. Bile produced by the liver is stored and concentrated in the gallbladder. The gallbladder contracts after a person eats. When the gallbladder contracts, it releases bile into the upper part of the small intestine in order for the bile salts to assist in fat breakdown. Without the gallbladder, the bile would still be able to travel from the liver to the intestine but with less efficiency. Gallbladder disease is the obstruction or decrease in efficiency of the flow of bile through the gallbladder during fat digestion.

The two main types of gallbladder disease are cholecystitis (inflammation of the gallbladder) and cholelithiasis (gallstones). Gallstones are formed when the contents of the bile precipitate to form crystals, and they cause disease when they block the duct that leads to the intestine.

Obese people, especially women, have a higher chance of developing gallstones. At-risk for gallstones are middle-aged women, obese people who have more fat around their midsection than around their hips or thighs, people who undergo gastrointestinal surgery, and people on rapid weight-loss diets. Diets extremely low in fat or eating fewer meals could decrease a person's amount of bile salt. Because the gallbladder's function is to assist in the fat digestion system, without fat, the gallbladder will contract less. With fewer contractions, the bile is more static, which in turn increases both the chances of small stones forming and of those stones getting larger. Some types of diet and exercise may reduce the risk of developing gallstones.

Although gallstones are prominent in the developed world, only some cases require intervention. The majority of gallstones have no symptoms. When symptoms do occur, they commonly include abdominal pain on the upper right side or upper middle of the abdomen, especially following meals. Abdominal pain could be coupled with fever, nausea, vomiting, shaking, chills, and heartburn. Serious complications of gallstones requiring hospitalization can occur when an infection occurs behind the blocked gallstone. Gallstones can be detected using a blood test or computerized tomography (CT) scan, X-ray, or ultrasound of the abdomen.

It is possible to remove the gallbladder by a safe operation called laparoscopic cholecystectomy. This



Gallstone symptoms include abdominal pain on the upper right side or upper middle of the abdomen, especially following meals.

surgery has a high success rate and is easy for patients. Other possible interventions include dissolution therapy or certain low-fat diets.

SEE ALSO: Body Mass Index; Dieting: Good or Bad?; Elevated Cholesterol; Fat Intake.

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Gastroesophageal Reflux (GERD)

GASTROESOPHAGEAL REFLUX DISEASE (GERD) is defined as the reflux of acid-rich stomach contents into the esophagus, burning the inner layer of the esophagus and causing symptoms that are sufficient to interfere with quality of life. GERD is one of the most common causes for primary care physician visits and a leading cause of noncardiac chest pain. Symptoms of GERD include heartburn, a burning sensation felt in the chest, foul-smelling breath, and cough. Other symptoms include wheezing, hoarseness, and recurrent respiratory infections. Complicated GERD may cause esophagitis, permanent scarring of the esophagus, and even precancerous transformation of the esophageal lining.

A competent lower esophageal sphincter (LES) is needed to prevent GERD. The length of intra-abdominal esophagus, the strength of the circular muscle fibers at the LES, and the normal emptying function of the stomach dictate LES strength. Acid production is also an important factor in the symptoms of GERD.

Obesity is a strong independent risk factor for GERD, as 25 to 50 percent of the morbidly obese population will have GERD. Obesity is associated with GERD through several mechanisms. The increased intraabdominal pressure exerted by the abdomen promotes reflux. Fatty foods are also known to relax the LES, which, in combination with an increase in food volume, promotes reflux.

There are several effective treatment options for GERD, which range from lifestyle modification, medications, and surgical approaches. Lifestyle modifications include weight loss, decreasing nighttime eating, decreasing meal size, and avoiding foods that relax the LES. Medications include those that decrease acid production (H2-blockers and protein pump inhibitors) and those that increase the strength of the LES and improve the emptying of the stomach (prokinetics).

Surgical procedures are reserved for those whose symptoms are still severe despite other treatments. The standard surgical procedure in normal-weight and overweight individuals is called a Nissen fundoplication. However, in the morbidly obese, a gastric bypass or lap band may be more effective treatment. Both of these treat the underlying problem of elevated intra-abdominal pressure from too much intra-abdominal fat by allowing the patient to lose weight. If the morbidly obese patient has an incompetent LES or a hiatal hernia that are causing the reflux, then only a surgical approach will be effective. In these instances, a medical approach is contraindicated. Both procedures can be performed using minimally invasive surgical techniques.

SEE ALSO: Gastrointestinal Disorders.

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Gastrointestinal Disorders

THE GASTROINTESTINAL (GI) system extends from the mouth to the anus and is comprised of the esophagus, stomach, duodenum, jejunum, ileum, colon, rectum, liver, pancreas, gall bladder, and biliary tract. Its primary functions are absorption of nutrients and elimination of wastes, although the liver and pancreas in particular fulfill many additional roles. Common symptoms of GI dysfunction include nausea, vomiting, changes in bowel movements, pain, and changes in weight.

GI disorders account for about 10 percent of the total burden of illness in the United States.According to the Centers for Disease Control and Prevention, in 2004, GI disorders accounted for 44.9 million office-based and 15.1 million emergency department visits. Obesity increases the chances of developing many different types of GI disorders, including gastroesophageal reflux disease (GERD), irritable bowel syndrome (IBS), gall bladder disease, fatty liver disease, and GI cancers.

Because problems with many different organs within the GI system can present with similar symptoms, a careful history and examination is essential to identify the underlying cause. The timing of symptoms, for example, can help determine if diarrhea is related to food poisoning, irritable bowel syndrome, medication side effects, or other problems. Other important characteristics of symptoms, especially pain, include any associated factors that make the symptom better or worse, the quality of the pain, whether the pain radiates to other parts of the body, and the severity. On examination, the presence or absence of fever, abdominal tenderness or masses, changes in the overlying skin, pelvic examination, and color and consistency of the feces can all be helpful.

GI disorders can be further evaluated by laboratory studies. Special blood tests can evaluate the function of the pancreas or liver, check for proteins made by cancers, or for signs of infection or autoimmune disease. Stool tests for diarrhea can diagnose infections, bleeding, or malabsorption.

A range of radiology studies can also be helpful. Probably the most important test today is computed tomography (CT) scanning. This special technique reconstructs the appearance of the internal organs, so that many infections, cancers, and a range of other problems can be identified. CT is not perfect, though. Many people cannot receive the special intravenous contrast used for it because of an allergy or kidney problems. In other cases, the cause of the problem may not be seen at all on a CT scan because of its size, location, or underlying nature. In these instances, ultrasound, magnetic resonance imaging (MRI), special X-rays taken after swallowing barium, or directly visualizing the inside of the bowel with a special camera such as an endoscope may be helpful. Ultrasounds are especially good for looking at other structures in the abdomen such as the ovaries or bladder which can cause symptoms similar to that of GI problems. Barium X-rays allow radiologists to assess the contraction and movement of bowel which may cause swallowing problems, vomiting, constipation, or diarrhea.

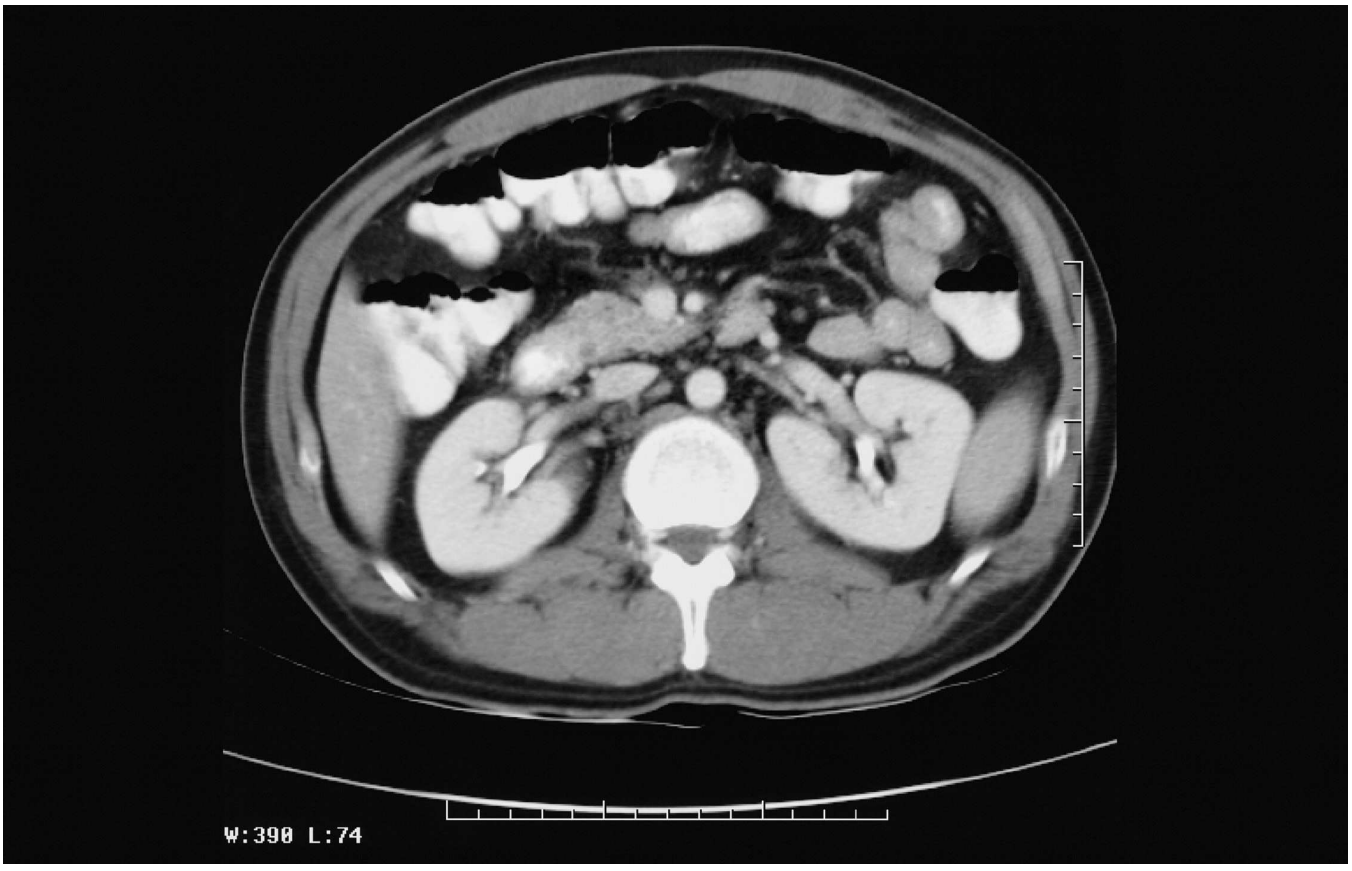
Endoscopes are special cameras within long tubes that can be inserted through the mouth or anus which may obviate the need for surgery. Upper endoscopy evaluates the esophagus, stomach, and duodenum, while colonoscopy assesses the rectum, colon, and end of the small intestine. Endoscopy can be used to screen for cancers, take biopsies of the bowel or a mass, stop bleeding, or obtain additional ultrasound images of the liver and pancreas.

Several GI disorders are more common with obesity. In GERD, stomach acid flows from the stomach back up into the esophagus, causing irritation of the esophagus's delicate lining. It is often associated with pain or pressure in the chest. It can lead to scarring and narrowing of the esophagus and increases the risk of developing one type of esophageal cancer. People with higher body mass indexes have more self-reported heartburn and regurgitation symptoms than their thinner counterparts.

IBS is a functional disorder—that is, scientists have been unable to identify a specific cause for it—characterized by chronic abdominal pain with diarrhea, constipation, or both. Obesity is associated with symptoms of bloating, upper abdominal pain, and diarrhea. People with IBS tend to have changes in GI motility, increased sensitivity to bloating and distension, coexisting psychological problems, and higher stress levels.

Gall bladder disease is the most common gastrointestinal disorder in obese individuals. Both obesity and rapid weight loss are associated with the development of gallstones. Gallstones form when liquid bile stored in the gallbladder hardens into pieces of stone-like material. Obesity increases the cholesterol content of bile and decreases the contraction of the gall bladder, predisposing to cholesterol-rich gallstones. These usually remain silent, but can cause right upper abdominal pain within 30 minutes of eating a fatty meal, pain between the shoulder blades or below the right shoulder, and nausea or vomiting. Stones that have become stuck in the neck of gall bladder commonly precipitate cholecystitis, or infection of the gall bladder. It produces symptoms such as right upper abdominal pain, fever, nausea, and vomiting.

Acute pancreatitis, or inflammation of the pancreas, is usually caused by gallstones or drinking alcohol. It can cause severe pain, nausea, and vomiting. Symptoms are typically worsened by eating, because this stimulates the pancreas to try to release more enzymes to digest food entering the



Because problems with many different organs within the GI system can present with similar symptoms, a careful history and examination is essential to identify the underlying cause. A range of radiology studies can also be helpful, such as this cross-section scan of the abdomen.

duodenum. The inflammation can lead to pooling of fluid around the pancreas and bowel, which in severe cases can lead to shock and even death. People who are obese are more likely to develop severe pancreatitis and its complications.

Cancers of the GI tract are closely associated with obesity, and may all present with unintentional weight loss. Adenocarcinoma of the esophagus, often related to long-standing GERD, may present with swallowing problems. Stomach cancer can present with pain or dark tarry stools, while colon cancer may be heralded by a change in the frequency or caliber of bowel movements, blood in the stool, or dark tarry stools. Pancreatic cancer may have no symptoms until it blocks the drainage of bile into the intestine, causing yellowing of the skin and eyes, or may present with abdominal pain.

Abdominal hernias occur when part of the bowel or fatty tissue protrudes through a weakened area of the abdominal wall such as around the umbilicus or at the site of a past surgery. The significance of abdominal hernias varies widely. Many cause no symptoms at all.

Hemorrhoids are engorged blood vessels in or around the anus, often caused by straining from constipation or increased pressure in the pelvis, as with pregnancy. Hemorrhoids may be associated with pain, bleeding, itching, or the sensation of a lump. Avoiding constipation by drinking fluids and eating lots of fiber can help prevent and treat hemorrhoids.

SEE ALSO: Colon Cancer; Fatty Liver; Gall Bladder Disease; Gastroesophageal Reflux (GERD).

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Gastroplasty

THE TERM *GASTROPLASTY* refers to the reshaping of the stomach to decrease its capacitance as a food reservoir. The early satiety induced by these restrictive procedures causes weight loss through decreasing caloric consumption. This is in distinction to malabsorptive operations such as the jejunoileal bypass and the biliopancreatic diversion, which decrease the number of calories that are being absorbed.

Advantages to gastroplasties compared to Roux-en-Y gastric bypass include the decreased risk for vitamin and mineral deficiencies, the lack of dumping syndrome, and the decreased immediate surgical complication rate, especially abdominal infections. Unfortunately, late-term problem such as poor weight loss and severe reflux have severely limited the application of these procedures.

Gastroplasty was the first purely restrictive operation performed for the treatment of obesity. Gastroplasties are typically divided into two types: the horizontal, and the vertical banded gastroplasty. The horizontal gastroplasty was first developed in the 1970s as an alternative to the Roux-en-Y gastric bypass and the jejunoileal bypass. The horizontal gastroplasty is performed by placing a set of staples transversely across the uppermost portion of the stomach, thereby separating the stomach into two parts.

A small channel is preserved between the upper pouch and lower stomach to maintain an outflow (stoma) for food to pass. The staples act as a seam that physically decreases the capacitance of the stomach. The lay term *stomach stapling* comes from this procedure. Unfortunately, late weight-loss failures were common, as the seam would invariably "pop" open, leaving the stomach in its native configuration. Due to weight regain, this procedure has been abandoned.

The vertical banded gastroplasty (VBG) is performed by orienting the staples in a vertical fashion against a bougie (sizing device) placed along the lesser curve of the stomach. The new stomach tube is made approximately one to two ounces in volume and just a few centimeters in diameter. To prevent dilation of this vertical channel, a prosthetic band is wrapped around the channel. Various materials are

used such as polypropylene, Silastic®, and even an adjustable gastric band.

The VBG is now rarely performed, as long-term studies have shown a significant percentage of long-term failures. Failures are either secondary to weight regain due to stomal dilation or severe reflux due to stomal stenosis (narrowing of the opening). Failures often respond to conversion to the Roux-en-Y gastric bypass. Erosion of the band into the stomach is another serious complication that is not uncommon. Randomized trials have found this operation inferior to the Roux-en-Y gastric bypass, especially in candy eaters. This procedure is waning in popularity as only a few surgeons still perform this operation.

Another reason that the VBG has become in disfavor is the decrease in complications with the Roux-en-Y gastric bypass procedure. The gastric bypass, when performed by experienced surgical centers has a mortality rate of 0.2 percent and can be performed laparoscopically in nearly all instances. Restrictive procedures have given significant insight in the causes of obesity. From these operations, it has become evident that the feelings of fullness and hunger are much more complex than the size of the stomach pouch. The weight loss with the VBG and the adjustable gastric band are significantly less than the gastric bypass possibly secondary to the more profound changes in neurohormones such as ghrelin, PYY, and leptin in the later procedure.

SEE ALSO: Roux-en-y Gastric Bypass.

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Genetic Influences on Eating Disorders

FOR DECADES, ANOREXIA nervosa, bulimia nervosa, and binge-eating disorder were considered to be disorders influenced primarily by family and sociocultural factors. However, over the past decade, it has become clear that these disorders run in families, that the observed familiality is due primarily to genetic and unique environmental factors, and specific areas of the genome and specific genes have been implicated in etiology. Eating disorders are relevant to obesity insofar as they represent forms of dysregulation of appetite and weight, and genetic mechanisms that are operative in eating disorders may be of direct relevance to understanding genetic factors that influence risk for obesity.

Anorexia nervosa is a serious psychiatric illness marked by an inability to maintain a healthy body weight, often dropping below 85 percent of expected weight. Despite increasing emaciation, individuals with anorexia nervosa continue to obsess about body weight and shape, remain dissatisfied with the perceived size and shape of their bodies, and engage in unhealthy behaviors to perpetuate weight loss (e.g., purging, dieting, excessive exercise, and fasting). Bulimia nervosa occurs in individuals of all body weights and is marked by binge eating (i.e., eating an unusually large amount of food and feeling out of control) coupled with inappropriate compensatory behaviors (e.g., self-induced vomiting, laxative abuse, diuretic abuse, excessive exercise, and fasting). Binge-eating disorder, often associated with obesity, is characterized by recurrent episodes of binge eating and associated distress, and the absence of regular inappropriate compensatory behaviors.

FAMILY STUDIES

Family studies have revealed consistent evidence for familial aggregation of eating disorders. Relatives of individuals with anorexia and bulimia nervosa are approximately 10 times more likely to have an eating disorder than relatives of unaffected individuals. Family members of an individual with binge-eating disorder are more than twice as likely to have the disorder themselves compared to those with an overweight or obese relative without binge-eating disorder. Intrigu-

ingly, anorexia and bulimia nervosa do not "breed true," meaning that risk is increased in relatives for an array of eating disorders, not just the disorder of the index case. Family studies are unable to determine the extent to which the familial pattern is due to genetic or environmental factors.

TWIN STUDIES

Although adoption studies are a valuable tool for unpacking the extent to which genes and environment contribute to risk for a disorder, no adoption studies of eating disorders exist. Therefore, attempts at determining the relative contributions of genes and environment to eating disorders have focused exclusively on twin studies. Twin studies utilize the fact that monozygotic (identical) twins share 100 percent of their genome, whereas dizygotic (fraternal) twins share only 50 percent. Thus, if both members of monozygotic twin pairs are more frequently affected (i.e., concordant) than members of dizygotic twin pairs, then a genetic contribution is suggested. Additional analyses enable more detailed decomposition of liability into genetic, shared environmental, and unique environmental influences.

The heritability of anorexia nervosa has been estimated to be between 33 and 84 percent, and the heritability of bulimia nervosa between 28 and 83 percent, with the remaining variance in both disorders attributable to individual specific environmental factors, and with negligible impact of shared environmental factors. The studies on which these estimates are based are primarily twin populations of European ancestry, and given the relative rarity of the traits under study, confidence intervals tend to be wide. One twin study has been conducted for a broad definition of binge-eating disorder, and yielded a heritability estimate of 41 percent. Individual environmental factors accounted for the remaining 59 percent of the variance. In summary, findings from twin studies have been replicated in several countries throughout the world—all indicating a substantial contribution of genetic factors to eating disorders. Additional analyses on more diverse populations will enable a more complete understanding of the role of genes and environment in risk for these disorders across racial and ethnic groups.

ASSOCIATION STUDIES

Further evidence supporting the role of genes in the pathogenesis of eating disorders has emerged from molecular genetics studies. Two primary approaches have been taken. Association studies compare cases that display a trait to controls that do not display the trait with respect to a candidate gene (or genes) hypothesized to influence the disorder under study. Genotype frequencies are compared in the case versus the control groups. Association studies are best when there is prior knowledge of the pathophysiology of a disorder, which can inform the choice of candidate genes. Modern genetic approaches (i.e., genomewide association [GWAS]) do not require prior knowledge, as specific candidate genes need not be selected. In the GWAS approach, hundreds of thousands to millions of single nucleotide polymorphisms (SNPs) are compared across cases and controls to identify significant differences across the genome. To date, there have been no GWAS of any eating disorder.

In terms of eating disorders, association studies have focused on genes that are known to affect appetite, weight regulation, and mood, with particular emphasis on the serotonin system. The serotonin pathway has been studied intensively in eating disorders due to its involvement in a broad range of biological, physiological, and behavioral functions, specifically weight regulation and eating behavior. Many of the association studies of eating disorders have been small and underpowered. Several serotonin 1D polymorphisms have been associated with anorexia nervosa or restrictive anorexia nervosa; however, only one SNP (rs674386) has been replicated in two adequately powered studies. No association has been observed in the rs6311 polymorphism of the serotonin 2A receptor gene in anorexia nervosa. A study examining four SNPs in HTR1D in 276 women with anorexia nervosa and 768 controls found evidence of association between two polymorphisms within HTR1D and the restricting subtype of anorexia nervosa.

Overall, for anorexia nervosa, the serotonin 1D gene looks promising, and is conveniently located under the linkage peak for restricting anorexia nervosa. However, "hard" replications in adequately powered samples are required.

Also studied have been factors associated with 5-HTTPLR within a sample of women with "binge-purge syndromes." In this sample, the S allele was not associated with eating disorder symptoms, including binge eating or vomiting frequency, body dissatisfaction, eating attitudes, or body mass index (BMI).

However, the S allele was associated with borderline personality disorder and related symptoms indexing impulsivity, affective instability, and insecure attachment. Intriguingly, individuals with the S allele had a significantly lower density of paroxetine binding sites, suggesting that they might not respond as well to traditional selective serotonin reuptake inhibitors.

Altered dopaminergic activity has also been hypothesized to be involved in many of the major symptoms related to eating disorders. Repulsion to food, food-based reward, weight loss, hyperactivity, menstrual abnormalities (amenorrhea), distortion of body image, and obsessive-compulsive behavior have all been related to dopamine activity.

The COMT gene encodes Catechol-*O*-methyltransferase which catabolizes brain catecholamine neurotransmitters such as dopamine and norepinephrine. No association was found between the rs4680 polymorphism located within this gene and anorexia nervosa in a combined transmission disequilibrium test and case-control analysis. Several polymorphisms within the dopamine D2 receptor gene were tested for association with anorexia nervosa. Association was reported with the purging type AN for the rs1800497 and rs6278 polymorphisms in a case-control design and the transmission disequilibrium test yielded preferential transmission for the rs6277 and the rs1799732 polymorphisms.

The dopamine receptor D2 gene remains of interest, although the findings require replication in a large independent sample. For catechol-*O*-methyltransferase, the existing data do not support a role for the rs4680 polymorphism in anorexia nervosa.

Three genes involved in neuropeptide and feeding regulation have been tested in methodologically adequate association studies: ghrelin, hypocretin receptor 1, and opioid receptor delta-1. No association was found between anorexia nervosa and the ghrelin or hypocretin receptor 1 genes. Despite the use of different polymorphisms, two studies reported associations between the opioid receptor delta-1 gene, anorexia nervosa, and the restrictive subtype of anorexia nervosa. Also genotyped were six SNPs in OPRD1 with three SNPs found to be associated with both the restricting subtype of anorexia nervosa and binge-purge anorexia nervosa.

The accumulated data do not support the involvement of ghrelin and hypocretin receptor 1 in the etiology of anorexia nervosa. The involvement of opioid receptor delta-1 should be replicated in an independent sample to confirm the reported association.

Brain-derived neurotrophic factor (BDNF) plays an important role in the growth and maintenance of several neuronal systems. Physiological and animal models indicate that BDNF induces appetite suppression and body weight reduction, and support the hypothesis that alterations in this system could influence abnormalities in eating behavior predisposing to eating disorders.

Two European collaboration studies have investigated the association between anorexia nervosa and two polymorphisms located in the gene encoding for BDNF. The rs6265 polymorphism was associated with anorexia in two studies, especially the restrictive subtype. Again, this gene looks promising although replication is required.

Fewer association studies for binge-eating disorder exist. Initial reports of melanocortin 4 receptor (MC4R) gene mutations being associated with a binge eating subset of obese individuals were promising, yet enthusiasm for these results were dampened after several nonreplications in adequately powered studies.

LINKAGE STUDIES

Another approach to molecular genetics that has been applied to eating disorders is linkage analysis. Linkage studies include large samples of multiplex families (i.e., families with more than one affected individual). Anonymous genetic markers scattered across the genome are used to identify chromosomal regions that may contain genes influencing the trait of interest. Linkage studies help to focus the search for genes within the human genome. Once linkage peaks are identified, specific genes that exist under those peaks can be further explored using association approaches. Theoretically, the function of those genes can then be determined and further study can reveal how they specifically influence risk for eating disorders.

A series of collaborative studies have yielded intriguing linkage results for both anorexia and bulimia nervosa. No linkage studies for binge-eating disorder have been performed. Linkage studies for anorexia nervosa have underscored the importance of detailed phenotyping. A linkage study of a heterogeneous sample of individuals with broadly defined eating

disorders yielded no statistically significant findings. However, restricting to relative pairs exhibiting the classic restricting anorexia nervosa yielded significant evidence for a susceptibility locus on chromosome 1. Additional approaches that incorporated the behavioral covariates of drive for thinness and obsessionality isolated several regions of interest on chromosomes 1, 2, and 13. The chromosome 1 region contained the serotonin 1D receptor (HTR1D) and the delta opioid receptor (OPRD1)—both plausibly related to risk for anorexia nervosa—and a subsequent association study found significant associations with anorexia nervosa.

Further work enriched methodology to select and incorporate behavioral covariates into linkage analyses. Obsessionality, age at menarche, and an anxiety measure displayed normal distributions and familial correlation and were selected for quantitative linkage analysis. Three variables emerged in which families showed highly concordant and extreme values—lifetime minimum BMI (lowest BMI attained during the course of illness), concern over mistakes, and food-related obsessions. These distributions are consistent with a mixture of populations, and applied to covariate linkage analysis. Subsequent analyses using these approaches identified a number of additional suggestive signals—obsessionality at 6q21, anxiety at 9p21.3, BMI at 4q13.1, concern over mistakes at 11p11.2 and 17q25.1, and food-related obsessions at 17q25.1 and 15q26.2.

One linkage study of bulimia nervosa has been conducted and reported significant linkage on chromosome 10p when using a broad sample of families with bulimia nervosa, with a second suggestive peak on chromosome 14.

From the perspective of identifying very strong candidate genes for eating disorders, however, these studies have not yet focused the genomic search space in a manner that has enabled the identification of functional variants that influence risk for these disorders.

We do not yet know whether these linkage findings contain one or more genes relevant to eating disorders. To date, there has yet to be a comprehensive fine-mapping study of these regions. Our current statistics of understanding must view these studies as preliminary knowledge awaiting both confirmation via replication and follow-up genotyping efforts.

SEE ALSO: Genetic Mapping of Obesity Related Genes; Genetic Taste Factors; Genetics.

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Genetic Mapping of Obesity-Related Genes

THE INCREASING PREVALENCE of obesity is a major public health concern. According to a World Health Organization (WHO) expert committee, there are about 1 billion overweight adults and one-third of a billion are labeled as clinically obese. Obesity is a multifactorial disease in which the environmental and genetic factors interact resulting in disorder of energy balance.

Excess food intake and lack of physical activity are the major lifestyle factors causing obesity, but until today, the role of genetic factors is still a challenge to scientists concerned. It has been known since the last century that both familial and nonfamilial factors play a major role in the development of obesity and the genetic component is fundamental to this

medical problem. From the mid-1980s, the evidence for genetic basis to human obesity came from a number of identical twins and family studies, which reported that 50 to 90 percent of obesity prevalence is genetic and 10 to 50 percent is heritable. The question of a major gene or potential chromosomal candidate gene in the development of human obesity is a particular challenge.

Published data have clearly demonstrated the complexity of genetics in obesity, which poses a great challenge to understanding the role of genetics associated with this medical condition. Although there is substantial evidence to show that genetics plays an important role in the body weight, knowledge about the genetic role is still in its first steps. Large numbers of different family studies has established an increase of obesity risk among obese families. This suggests the role of genetics in obesity, and at the same time explains the nature of polygenic obesity.

Obesity is a multifactorial disease that is likely affected by a multitude of genes. In rare instances, there are single dysfunctional genes that can result in a phenotype of severe obesity (eg., the ob/ob mouse that is deficient in the hormone leptin). But, most cases of obesity are likely polygenic, where numerous genes contribute to the resulting phenotype. To date, 250 genes have been found that relate to some form of human obesity, so the picture is quite complicated.

To date, about 200 medical conditions have been associated with single-gene mutation involving 11 genes. This clinical condition obeys Mendelian genetics and is characterized by severe phenotepics feature presenting early in life and associated with mental, developmental, and endocrine disorders. A large-scale linkage analysis in mice led to detection of disease loci and the identification of the candidate gene in the majority of mutations underlying monogenic murine obesity. The characterization of naturally occurring obese models such as the ob/ob, db/db, fat, and tubby mice led to the discovery of recessive mutations in the genes encoding leptin (lep or ob), leptin receptor (Lepr or bd), carboxypeptidase E (Cpe or fat), and tubby (Tub). Several additional monogenic obesity genes were discovered like single-minded homolog 1 (*Drosophila*) (*SIM1*) identified in a girl with early-onset obesity that expressed in the paraventricular nucleus of the hypothalamus, which appears to regulate feeding. Melanocortin 4 receptor MC4R-linked obesity is the most prevalent form of monogenic obesity identified to date, which represents two to three percent of childhood and adult obesity. Recent discovery of rare functional mutations in regions of proopiomelanocortin (POMC) encoding for alpha melanocyte stimulating hormone a-MSH provides support for the use of genetic screens to identify factors upstream and downstream of MC4R in early-onset and severe human obesity.

There are more than 25 medical syndromes that follow Mendelian inheritance associated with obesity. They are considered to be syndromic obesity which arises from discrete genetic effect that could either be autosomal or X-linked disorders. These groups of syndromes are known with their unique clinical features that present with dysmorphic features and mental retardation in addition to organ developmental abnormalities. The most frequent obesity syndromes known are Prader-Willi syndrome (PWS), Bardet-Biedl syndrome (BBS), and Alstrom syndrome in addition to other syndromes that are usually diagnosed in the early part of life. Each syndrome is characterized by chromosomal defect such as absence of paternal segment 15q11.2-q12 chromosome in case of PWS and sometimes associated with more than one chromosomal location as in BBS that have more than 11 different locations in six genes. Although syndromic obesity is presumed to be a single gene disorder, recent studies clearly defined the contribution of multiple genetic factors.

Polygenic obesity is by far the most acceptable genetic explanation by many scientific laboratories and trials. Although the environmental factors play a significant role in the etiology of obesity, yet more studies are needed to identify candidate genes unlike monogenic obesity where many genes and chromosomes regions contribute to defining the common obese phenotype. These genes have been linked to variable biological functions that are related to fat store in adiposities. Some have relation to food intake and others have to do with energy expenditure. There are certain genes that are linked to lipid and glucose metabolism and others have a role in adipose tissue development. To date, few candidate genes for obesity have been reported by at least five positive stud-

ies, for example, an association between three SNPs of GAD2, which codes for the 65-kDa subunit of the glutamic acid decarboxylase enzyme, and morbid obesity as identified in a French population following a genome-wide scan, but this was not achieved in a large, similar German survey.

The map reveals that putative loci affecting obesity-related phenotypes are found on all chromosomes except Y-chromosomes. The number of quantitative trait loci (QTL) reported for human obesity derived from genome scans continues to grow to more than 250 QTLs from 61 genome-wide scans. The number of studies reporting associations between deoxyribonucleic acid (DNA) sequence variation in specific genes and obesity phenotypes has also increased considerably with 426 findings of positive associations with 127 candidate genes. Among them, those showing replications in more than 10 studies include *PPARG* (30 studies), *ADRB3* (29 studies), *ADRB2* (20 studies), *LEPR* (16 studies), *GNB3* (14 studies), *UCP3* (12 studies), *ADIPOQ* (11 studies), *LEP* (11 studies), *UCP2* (11 studies), *HTR2C* (10 studies), *NR3C1* (10 studies), and *UCP1* (10 studies). Meta-analysis of genetic association studies conclude that although false-positive associations are abundant in the literature, 20 to 30 percent of genetic associations are real and have modest effect on risk of common diseases.

There is a long way to go for understanding the role of genetics in obesity, and the goal remains to identify the right combination of genes and mutations that are associated with increased risk of obesity and to understand the role of environmental factors to this medical health problem.

SEE ALSO: Adipocytes; Animal Models of Obesity; Db/Db Mouse; Ethnic Variations in Body Fat Storage; Genetic Influences on Eating Disorders; Genetic Taste Factors; Genetics; Genomics; Monogenic Effects that Result in Obesity; Ob/Ob Mouse; Prader-Willi Syndrome; Quantitative Trait Locus Mapping; SNP Technologies; Thrifty Gene Hypothesis; Tubby Candidate Gene; Twin Studies and Genetics of Obesity.

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Genetics

OBESITY AND OVERWEIGHT have been shown to have a clear genetic determinant, although it is often a misunderstood and underestimated one in the eyes of both the professional and lay communities. The belief that obesity is simply a function of lack of self-control and willpower persists in spite of the evidence to the contrary. The genetic underpinnings of human behavior is often a contentious issue, with some arguing that human choice determines the range of behaviors that are increasingly being linked to genes, such as alcoholism and addictions; overweight and obesity; and such things as impulsivity, angry outbursts, anxiety, depression, and personality traits such as optimism and pessimism. It has been argued that even reporting genetic research is irresponsible because it might cause obese people or alcoholics to use the findings as an excuse for their obesity or addictive behavior, rather than adopting healthier behaviors and lifestyles. Exactly how genes and the environment interact to cause obesity and various behaviors is often misunderstood and to a large degree still unknown. Free choice and individual control are issues raised repeatedly in the public discussion of genes and obesity and genes and behavior.

It is cited frequently in the literature that while one's genes cannot be changed, one's behaviors can. These beliefs and trends, coupled with the strongly documented societal bias against obesity often leads to the simplified conclusion that if fat people ate less and exercised more, the problem of obesity could be easily solved. Both professionals and the public use this information and reasoning to downplay the genetic contribution to the problem and to maintain a focus on individual behavior and responsibility when it comes to addressing and solving the widespread and growing problem of obesity.

THE EVIDENCE

Prior to the Human Genome Project of the early 1990s and the subsequent genetic research that has connected specific gene functions with body size and eating behaviors, it was known that genes played a role in obesity and overweight. By looking at the incidence of obesity in families, both those related biologically as well as those related through adoption, and studies comparing the incidence of obesity in identical twin pairs compared to the incidence of obesity in fraternal twin pairs and siblings in general, it has been demonstrated that there is a significant degree of heritability when it comes to body size and obesity. Studies have shown that adopted children, for example, have body sizes and weights that are closer to their biological parents than to those of their adopted parents. While this has been clearly and repeatedly demonstrated, it continues to be maintained that obesity may run in families because of similarities in eating behaviors and habits. While it is also known that culture and environment as well as genes play significant roles in the development of obesity, the genetic links show that there may not be as much individual choice and control over weight loss and weight gain as is generally believed. Yet, "lifestyle" is continually cited as the primary contributing factor in obesity, both in the mainstream and the professional literature.

Even when professional literature cites such findings as the correlation between the weight of adopted children with that of their biological parents, who often live in vastly different environments, cultural settings, geographical regions, and socioeconomic classes, the same literature will frequently conclude that obesity can best be eliminated by lifestyle change.

While the genetic evidence has solidly been gathered, demonstrated, and disseminated, it is often not integrated into the current beliefs, attitudes, and recommendations of both the health and the public health communities. In other examples, the professional literature downplays the importance of genetics at the same time it cites it, again recommending lifestyle modification and change. The treatment and prevention of obesity, and the recommendations of committees, experts and professionals often seem to not be informed by the research, but rather by persistent misconceptions and commonly held but unproven beliefs.

ANTI-FAT BIAS AND DISCRIMINATION

Literature in the fields of health, social science, and psychology continually show a great societal tendency to stigmatize obesity and obese people, even among health professionals, health educators, and healthcare practitioners. Discrimination in employment and college acceptance persist, and incorrect assumptions about why children and adults become and stay obese continue to be made even by those people who are charged with providing services and help for this very population. The American obsession with thinness has been widely documented, as has the distorted body image and preoccupation with weight and body size that exists among a significant number of normal-size women and girls. Professionals often decry the unhealthy attitudes toward thinness and food that exists among both girls and women, who have been repeatedly cited as wishing they were anorexic or sick with life-threatening illnesses, just so that the elusive slenderness they so desire might be achieved. There has been worldwide focus on fashion models and actresses who lose increasing amounts of weight to achieve the often unrealistic and underweight body size and weight goals set by these industries. In spite of all this, the health community continues to insist on the importance of food restriction among children, families, and adults to achieve lower and healthier weights.

THE ROLE GENES PLAY IN OBESITY

One misconception about genes and obesity is that there is an "obesity gene," or one gene that causes obesity. In fact, there are many genes that have been implicated in obesity, and these genes include those that play a role in how insulin is regulated; those that determine how various hormones are secreted and used; those that regulate how and where fat is stored; and those that govern how or whether fullness or satiation is felt or experienced. These and other genes play important roles in eating behaviors, energy use and metabolism, and in the experience of hunger and the desire for food. When a number of genes contribute to a particular behavior or condition, this is known as having a polygenetic cause. According to some, having a polygenetic cause, as opposed to having a single genetic mutation or difference causing a disease or medical condition means that the

genetic factors are less meaningful or even insignificant. Some view these genetic factors as merely having some influence over, rather than a causative relationship with, obesity. Another view is that because environmental, social, and psychological factors also have roles in determining various aspects of food intake and energy expenditure, biology and genetics appear to set the parameters within which these behaviors and conditions occur.

For example, during times of food shortages there will be a great deal less obesity than during times when food is available and abundant, demonstrating the environmental, behavioral, and societal factors that also govern food intake. However, during times of food availability and abundance, there are individual differences in feelings of hunger and satiation, in the ability to metabolize food and expend energy, and in the way nutrients are used and stored. As a result, not every individual will gain weight during a time of great food availability and abundance, but some will. Living continuously in times of food abundance, especially the abundance of processed, sugary, fatty, and salty foods has been called living in a "toxic environment," one that sets the stage for unhealthy food behaviors and weight gain to occur. Although not everyone in a toxic environment will become obese, those who are predisposed to do so, due to the presence of genetic variations and differences, will most likely put on excess weight and have great difficulty taking it off.

Additionally, it has been shown that the human body is built to respond and change according to environmental conditions and demands. During times of food shortage, human metabolism slows down, so as not to use up the energy it has taken in too quickly. Historically this has been played out during times of food shortages, seasonal changes in food availability, and during time of extreme environmental conditions such as famine. The ability for metabolism to slow down during these times would have provided an evolutionary advantage, enabling individuals with this ability to use much more slowly that which was in limited supply. Humans have been shown to react the same way to self-imposed food restriction, such as weight-reducing diets, and instead of losing weight due to limited food intake, the body adjusts to these limitations by slowing down metabolism and the need for food. As a result, the dieter, over time, needs less and less food as the body adjusts to reduced amounts and availability of food supplies.

While this does not demonstrate genetic changes due to dieting, it does demonstrate that over thousands of years, humans adjusted to periodic food shortages by changes in physiological functions. During times of food abundance and availability, it was common for people to desire excessive amounts of food. It is believed that these urges also played an evolutionary role in that it prepared people for a time when food shortages would occur. From an evolutionary and genetic standpoint, humans appear to tend to respond to abundance by eating more in preparation for future food scarcity. Now that we live with great food availability and abundance, these trends and tendencies do not serve us the same way as they did during times when scarcity and famine were more common. We may get the same internal signals our ancestors got, which direct us to eat somewhat excessively when there is overabundance, but this behavior no longer serves the same purpose it served historically. The ability to store and transport food, as well as to farm and produce it, means that in many societies food scarcity no longer exists.

This human tendency may have started as a genetic variation that in turn helped humankind survive during harsh conditions, enabling the species to not only survive but thrive. Now the tendency to react to excessive amount of available food by taking in more food may no longer aid in our long-term survival and health. This is an example of how genes can change and adapt to environments over a long time. This ability to both cope with scarcity with a slowdown in metabolism, and cope with abundance by taking in excess food no longer appears to be serving human health and longevity.

GENETIC DIFFERENCES

There is a great deal of individual variation as well when it comes to genes and genetic variants. Some variants are more common than others, and researchers are able to determine their existence in significant proportions in the population. There are also rare genetic differences that cause some forms of obesity. More commonly, among the obese and the overweight are numerous genetic variants that influence human behavior and physiology in a way that accumulates to result in the tendency to take in excess food energy

Genetic background appears to play a role in one's body size. Studies have shown that adopted children, for example, have body sizes and weights that are closer to their biological parents than to those of their adopted parents.

and to inadequately store, use, and expend it. In 2003, the Human Obesity Gene Map underwent its 10th update, resulting in the identification of more than 430 genes, markers, and chromosomal regions that have been associated or linked with obesity in humans.

The gene that codes for the a protein called leptin has been the focus of a great deal of research, initially in mice, to try to determine its role in the desire to eat and in the ability to self-regulate food intake. In mice, the hormone leptin was shown to dramatically control appetite and food regulation, and when it was clinically manipulated, the eating behaviors and subsequent weights of mice would either go way up or way down. Injections of leptin in mice showed dramatic changes in body weight. When studies were done into leptin and human physiology, these dramatic results did not cross over into the human population.

Much of the genetic research is done in rats or mice and, while sometimes promising, these findings often do not seem to cross over into human physiology and behavior. In the case of leptin, it was repeatedly demonstrated to drastically influence the food and eating behaviors in mice. Leptin appeared to control appetite and food regulation, and when it was clinically manipulated, the weights of the mice subjects would either go way up or way down as a result of mice eating in a regulated or disinhibited way. Although the research on leptin failed in its attempt to translate into an easy and swift intervention in human obesity, the research continues, as scientists try to discover why leptin works for some obese people, but not for others. The research on leptin has demonstrated that the ability to control appetite and body size is a complex process which in many ways hinges not just on human choice and behavior, but on genetically based biological processes that may regulate food intake and the drive to eat.

Another gene variant was recently discovered in Great Britain that was related to higher body weights in approximately half of the population studied. This gene, known as the FTO gene, produces a protein that appears to be related to the system of appetite

and satiety. Individuals who were shown to carry two variant FTO genes weighed more than those who had only one variant FTO gene; the one-only variant FTO gene carriers weighed more on average than did the general population.

More than one gene is involved in appetite control, and another gene with this function is called GAD2, which appears to play a role in stimulating appetite and hunger. Research has shown that people with a more active form of the GAD2 gene have a large quantity of gamma-amino butyric acid (GABA) in the hypothalamus, which appears to increase the drive to eat.

In other research, a gene known as PPAR has been identified and shown to control the amount and size of fat cells that develop in an individual. When there is a mutation, or variant, in the PPAR gene, the production and size of these fat cells are increased. If the gene is seen as a "switch" that turns on the production of these cells, the variant can be viewed as the condition whereby the switch is stuck in the on position, and overproduction of fat cells results.

Ghrelin is a hormone that is secreted by the stomach and the brain and it is believed to signal feelings of hunger. It goes up before meals and decreases after meals. It can be thought of as the opposite of the hormone leptin, which is related to the feeling of fullness. Some obese subjects have been shown to have an overabundance of ghrelin. It has also been shown that obese people who undergo gastric bypass surgery have a decreased amount of ghrelin in their stomachs, which is thought to result in their significant weight loss following surgery. While the surgery has not altered the genetic makeup of the individual, it appears to play a role in altering the production of ghrelin. The surgery seems to alter the biologic function that the genetic variant might have caused.

These are some of the significant genes that have been identified as having an influence in food intake, metabolism, and fat storage. While researchers are quick to point out that genes only predispose individuals to obesity, and that societal and environmental conditions must also exist and interact with genes and biology to produce the conditions of overweight and obesity, they also note that altering body size might not be something that is easily done through behavior alone. Research in this area continues to point out new and more effective ways of intervening in the treatment and prevention of obesity.

SEE ALSO: Appetite Control; Appetite Signals; Ghrelin; Leptin; Roux-en-Y Gastric Bypass.

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Genetic Taste Factors

THE TERM *"taste genetics"* refers to variations in basic taste responses that can be directly associated with variations in the human genome. While there is substantial variance across humans in response to all the basic tastes (sweet, salty, bitter, sour, and umami or savory taste), the genetic ability to taste bitter compounds like phenylthiocarbamide (PTC) and 6-*n*-propythiouracil (PROP), which share a thioruea chemical moiety (N-C=S), is the most widely studied. Genetic variations in the ability to taste these compounds were discovered in 1931 by a chemist named A. L. Fox. When some of the PTC he was synthesizing dispersed into the air, his colleague detected a strong bitter sensation while Fox detected nothing. In response to this accidental discovery, Fox collaborated with his colleague, A. F. Blakeslee, to test this finding on larger groups of related and unrelated individuals. They concluded that about 65 to 70 percent of participants find PTC crystals to taste extremely bitter ("tasters"), while between 30 to 35 percent detect no taste from the crystals ("nontasters").

It is now known that there are large variations in the breakdown of tasters and nontasters, depending on geographic location. In general, northern Europe has a higher percentage of nontasters (about 30 percent), while some parts of sub-Saharan Af-

rica have the lowest (less than 2 percent). From an evolutionary standpoint, the ability to taste bitter compounds is advantageous in that it helps individuals avoid the consumption of potentially toxic plants. The fact that the inability to taste the bitterness in PROP and PTC has remained as high as 30 percent in some populations is of particular interest, particularly because it does not appear to have obvious genetic advantages. Presently (2007), numerous internet publications and news media reports have publicized the phenomenon of tasters and nontasters, and health researchers continue to be intrigued as to why this variation in taste remains in the human population.

Inherited variations in response to PTC and PROP are associated with food preferences, diet, and possibly risk for chronic diseases such as obesity and cancer. The first studies to report this relationship did so in the 1960s, from the laboratory of Roland Fischer. Instead of using PTC, Fischer began using PROP, a related compound that has less of a sulfuric odor than PTC. Currently, most studies use PROP, although a few laboratories still use PTC because it has a more direct relationship to the genetic sequence that confers this trait. Fischer used questionnaires to determine that in general, PROP tasters have more food dislikes than nontasters. He also noted that tasters tended to be more "slender" and hypothesized that this was attributable to their having more food dislikes than nontasters.

From the time of these early discoveries, many advances have been made in the field of taste genetics. It is now evident that the ability to taste PTC and/or PROP is associated with the perception a variety of other basic tastes. For example, PTC/PROP tasters also perceive greater bitterness from caffeine and sweetness from the taste of sucrose. These general differences in taste perception between tasters and nontasters may be a result of differences in the number of taste buds (papillae) between the two groups. Tasters have more taste papillae, the anatomical structures that hold the taste cells that are responsible for detecting basic tastes. Because of this anatomical difference, tasters perceive greater taste sensations from many basic tastes compared to nontasters. Moreover, these differences in basic taste perception are also likely to affect food preferences between the two groups. Studies have noted that tasters tend to dislike foods that are bitter or hot, like green leafy vegetables and hot peppers. On the other hand, nontasters have demonstrated higher preferences for some high-fat foods. It is important to note that despite genetic differences, food preferences are complicated behaviors, influenced by a broad array of biological and environmental factors. Thus, influences due to PTC/PROP sensitivity are often masked by other more salient factors.

The relationship between inherited sensitivity to PTC/PROP and body weight is complex, and not fully clarified in the literature. Several studies have noted that PTC tasters tend to be taller and thinner, while nontasters tend to have a higher body mass index. Not all studies support this finding; therefore, further investigation is necessary. It is unclear why nontasters tend to have higher body weights in some studies, but increased fat preferences, and possibly intakes, is one potential contribution. Other studies have also associated PROP status with risk for certain cancers (i.e., breast and colon) and cardiovascular disease, but this literature is sparse and not yet widely recognized in the scientific community.

The inheritance of PTC and/or PROP tasting was originally thought to be transmitted to offspring as a dominant Mendelian trait. This model, however, has been revised due to family studies in which taster offspring are born to nontaster parents. The major gene underlying this trait has been identified as TAS2R38, a G-protein coupled bitter receptor located on chromosome 7q. Amino acid substitutions of this protein lead to two haplotypes which correlate to the taster and nontaster phenotypes. The knowledge of the gene underlying the ability to taste PROP provides a fundamental link between phenotypic observations on taste sensitivity and the PTC genotype.

In the mid-1990s, Dr. Linda Bartoshuk, a psychophysicist at Yale University, advanced the field of PROP classification with sophisticated scaling procedures. She noted that the group of PROP tasters can be subdivided into a group with extremely high sensitivity to PROP, "super tasters," and a group with moderate sensitivity to this compound, "medium tasters." This procedure of classifying PROP status is more sensitive than a simple two-group (taster/nontaster) classification, and thus allows researchers to study the relationship between taster status and diet across a broader range of PROP sensitivities. These recent

advances in both the genetics of PTC and the ability to measure this trait through scaling procedures have the potential to clarify why this unusual taste variance remains in the human population today.

SEE ALSO: Food Preferences; Taste Reactivity.

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Genomics

THE TERM *GENOMICS* refers to the study of an organism's entire genome. Each organism contains a genome that contains biological information that maintains the organism. Traditionally, individual genes have been investigated, but genomics differs because it encompasses the study of the entire genome. Genomics is a relatively new field with plenty of room for exploration. Newer technologies have made genomics and systems biology (the linking of molecular pathways to describe overall functioning of the organism) not only possible but a must for scientific advancement.

The human genome is made up of deoxyribonucleic acid (DNA) composed of subunits known as nucleotides. There are two main parts to the human genome: the nuclear genome and the mitochondrial genome. The nuclear genome contains roughly 35,000 genes split into 24 chromosomes. The mitochondrial genome consists of only 37 genes that are contained within the mitochondrial organelle. On its own, the genome is unable to release the biological information to the cell. Coordinated activity of enzymes and proteins in biochemical reactions is necessary for genome expression.

The initial product of genome expression is the transcriptome, which is maintained via a process known as transcription. Transcription copies individual genes into RNA molecules. Following transcription, a process known as translation produces the proteome (the cellular complement of proteins) that specifies the reactions that the cell is capable of. The RNA and proteins are important because, although the genes must be present to make a protein, if the gene does not make the protein, biochemical reactions of the gene will not be carried out.

One specific area of genomics that is extremely pertinent to obesity research is the field of nutritional genomics. Nutritional genomics refers to the study of the relationships between the nutrients that are consumed, the health consequences of these nutrients, and the genome of that particular organism. Nutritional genomics can be subcategorized into two main subfields: nutrigenomics and nutrigenetics. Nutrigenomics refers to the study of the effect of the nutrients on the genome, while nutrigenetics refers to the study of the effect of genetic variations on the interactions between nutrition and health. Practically, nutrigenetics, is used to study the impact of genetic variation on dietary requirements, while nutrigenomics is used to study the effect of diet on gene function. Nutrigenomics can be utilized to determine the effects that nutrients have not only on the genome, but also on the transcriptome and the proteome.

The primary technology used in nutrigenomics is the microarray. A microarray is an assay that is used to determine which genes are expressed in a sample. They can assess thousands of genes in a single experiment; therefore, the process is often known as gene expression profiling. Although not quantitative, a microarray allows for determination of a qualitative "yes or no" in terms of expression within the genome or transcriptome. Proteome investigations are primarily conducted via two-dimensional gel electrophoresis or liquid chromatography-mass spectrometry.

These technologies can be used with different interventions to determine what sets of genes are activated by the intervention. For example, one could examine the differences in gene expression in human

adipocytes when comparing groups of various calorie levels, dietary macronutrient percentages, or specific bioactive compounds to a placebo. Or it may be combined with a nutrigenetic approach. One example of this type of research was conducted by Burg and colleagues using a mouse model. A group of mice was treated with a drug designed to alter lipid metabolism. The goal was to determine the difference between normal individuals who do not absorb dietary sterols and those with sitosterolemia, a disorder in which dietary sterols are hyperabsorbed leading to hypercholesterolemia. A microarray analyzed differences in mRNA expression profiling a new gene was found. In fact, humans share the gene that produces two proteins and is responsible for removing dietary sterols from the tissues. In those with sitosterolemia, however, a mutation in this gene exists and sterols are therefore not properly transported.

Because of its relative infancy as a field, nutrigenomics still has many questions that are yet to be answered. One of these primary questions is how data obtained from nutritional genomic investigations will translate into consumer applications. For example, while DNA-specific nutritional advice and drug therapy programs seem highly beneficial, there has been little research showing that these techniques are more beneficial and/or resource efficient than traditional nutritional advice or drug prescription methods. Another difficulty at present is the relative fluidity of the term *nutrigenomics*. Experts disagree on whether the science is limited to studying the human genome and nutrient effects therein, or whether goals and applications are to be included as well. In any case, genomics is a burgeoning field of biomedical science that appears to hold great potential helping to investigate the growing obesity problem and may possibly lead to individualizing nutritional programs to help counter the issue more effectively than traditional nutrition alone.

SEE ALSO: Genetics; Microarray Analysis; Translational Research.

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Gestational Diabetes

GESTATIONAL DIABETES MELLITUS is a type of diabetes commonly seen only during pregnancy in overweight/obese women. Women older than age 25 with a family history of diabetes are more likely to develop gestational diabetes. Gestational diabetes affects about 4 percent of all pregnant women. It usually begins in the fifth or sixth month of pregnancy (between the 24th and 28th weeks). Most often, GDM will disappear once a woman gives birth, but the chance she will go on to develop Type 2 diabetes later in life increase by 5–10 percent. If the maternal blood has too much glucose, the pancreas of the fetus senses the high glucose levels and produces more insulin in an attempt to use the glucose. The fetus converts the extra glucose to fat. The predominant pathogenic factor in gestational diabetes could be the inadequate insulin secretion. As many as 90 percent of those identified with gestational diabetes have a deficiency of insulin receptors (prior to pregnancy) and a marked increase in abdominal obesity. The other 10 percent have deficient insulin production and will proceed to develop mature-onset insulin-dependent diabetes. Insulin release is enhanced in an attempt to maintain glucose homeostasis. Maternal pancreatic beta cells increase by threefold for insulin resistance. Increased hunger due to the excess insulin release as a result of elevated glucose levels.

An oral glucose tolerance test (OGTT) for gestational diabetes is carried out between the 24th and 28th week of pregnancy. A fasting plasma glucose level above 126 mg/dl (7.0 mmol/l) or a casual plasma glucose above 200 mg/dl (11.1 mmol/l) meets the threshold for the diagnosis of diabetes. The American Diabetes Association considers the following abnormal during the OGTT in gestational diabetes: Fasting Blood Glucose

Level = 95 mg/dl (5.33 mmol/L); 1-Hour Blood Glucose Level = 180 mg/dl (10 mmol/L); 2-Hour Blood Glucose Level = 155 mg/dl (8.6 mmol/L); and 3-Hour Blood Glucose Level = 140 mg/dl (7.8 mmol/L).

Stillbirths are uncommon in diabetic pregnancies; congenital malformations and complications of maternal hypertensive disorders account for most of the 1.5- to twofold higher perinatal mortality, compared with nondiabetic pregnancies. The perinatal mortality rate in diabetic pregnancies is approximately 30 to 50 per 1,000 births. Maternal risks in diabetic pregnancies are greatest in the presence of retinopathy and nephropathy. Diabetic retinopathy is present in 15 to 66 percent of women with insulin-dependent diabetes mellitus (IDDM) early in pregnancy, and the retinopathy frequently worsens during gestation. Overt diabetic nephropathy is present before pregnancy in approximately 5 to 10 percent of IDDM women, many of whom manifest hypertensive disorders during pregnancy. Nephropathy increases the prevalence of intrauterine growth retardation, prematurity, fetal morbidity, and fetal mortality. Maternal mortality during diabetic pregnancy is approximately 3 to 7 per 100,000, which is similar to the rate in nondiabetic pregnancies.

Nutrient deficiencies of chromium, magnesium, potassium, zinc, manganese, and pyridoxine may increase the hyperglycemia in gestational diabetic women. They also have imbalance of calcium and magnesium levels. Fasting hyperglycemia may be associated with an increase in the risk of intrauterine fetal death during the last four to eight weeks of gestation. Gestational diabetes is also associated with maternal hypertension and preeclampsia. Obesity/overweight and other associated risk factors of insulin resistance may enhance the risk of Type 2 diabetes after gestational diabetes. If gestational diabetes goes undetected, a baby has an increased risk of stillbirth or death as a newborn. Shoulder dystocia is a serious complication in gestational diabetes. Caesarean delivery is common in gestational diabetes. Babies born to gestational diabetes women are more prone to hypoglycemia, jaundice, respiratory distress syndrome, increased risk of obesity, glucose intolerance, and diabetes in late adolescence.

A healthy diet is important for every pregnant woman. Eating the right kind and amount of food is one of the best ways to control your blood sugar levels, and the more active you are, the lower your blood sugar. The key to prevention is careful control of blood sugar levels in the mother as soon as the diagnosis of gestational diabetes is made. By maintaining normal blood sugar levels, it is less likely that a fetus will develop macrosomia, hypoglycemia, or other chemical abnormalities. Nutritional management of gestational diabetes can be achieved with regular exercise, maintained body weight, and a healthy diet.

SEE ALSO: Pregnancy; Type 2 Diabetes.

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Ghrelin

GHRELIN IS A recently discovered hormone that is believed to play a role in the regulation of eating and body weight. Ghrelin is produced in the fundus of the stomach and is part of a class of compounds known as gut peptides. Other gut peptides include peptide YY and glucagon-like-peptide-1 (GLP-1). Ghrelin is the natural ligand (substrate) for the growth hormone secretagogue receptor.

Ghrelin levels fluctuate throughout the day and peak before a person begins to eat. After a meal is consumed, ghrelin levels decrease. This is part of the proposed mechanism for ghrelin's role in eating in which ghrelin travels from the stomach to the brain. Once in the brain, ghrelin sends its message through a series of other pathways to initiate eating. These include the activation of neuropeptide Y (NPY) and agouti-related protein (AgRP), compounds located within the brain that stimulate eating.

People who are underweight have elevated ghrelin levels (to stimulate food intake), while overweight and obese people have decreased ghrelin levels (to inhibit food intake). In one study, when ghrelin was infused into both animals and people, food intake increased,

and when ghrelin antagonists were infused, food intake decreased.

Weight loss by dietary restriction increases plasma ghrelin levels. Paradoxically, patients who underwent gastric bypass surgery, which is associated with large amounts of weight loss, had very low ghrelin levels and lacked the circadian pattern associated with the ghrelin. This may be the result of the gastric bypass surgery because the part of the stomach that makes ghrelin is no longer fully functioning. Taken altogether, this hormone may be one reason why it is difficult to maintain weight loss by dietary restriction.

Ghrelin may be related to leptin and insulin, two other hormones that affect eating and body weight. Leptin and ghrelin oppose each other in action and circadian rhythm (i.e., ghrelin is high before a meal and leptin is low before a meal and vice versa). However, while leptin is not the primary regulator of ghrelin levels, leptin was demonstrated to decrease ghrelin levels in the blood. Insulin also opposes ghrelin in function, but it was demonstrated that insulin only suppressed ghrelin levels at superphysiological levels. Given its role in eating and body weight, ghrelin may have significant potential as a pharmaceutical. Currently, ghrelin is being studied to be a pharmaceutical agent as either an agonist to increase patients' appetites (i.e., cancer patients) or as an antagonist to decrease patients' appetite (i.e., obesity).

SEE ALSO: Insulin; Leptin; Leptin Supplements.

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Glucagon Receptor

GLUCAGON RECEPTORS ARE members of the family of g-protein linked receptors (GPLR) that transduce an extracellular signal into an intracellular signal via ligand binding and G-protein activation. These receptors are found throughout the body, with primary sites being the liver and kidney, and secondary sites including the heart, adipose tissue, spleen, and gastrointestinal tract. Following stimulation of the receptor, the second messenger system via adenylate cyclase and cyclic AMP enhance the signal. The primary hormone agonist is glucagon, while a primary antagonist (which blocks the binding site of the receptor) is acylated aminothiophene nitril. The functions of glucagon and its receptors are as vital to the regulation of blood sugar as is insulin. Therefore, glucagon receptors are inherently important to the obesity and diabetes epidemics in society today.

The primary function of the receptor is to bind with glucagon to form a ligand and to carry out the functions of glucagon via the second messenger system. Glucagon itself is hormone secreted by the alpha cells of the islets of Langerhans in the pancreas. The primary functions of glucagon are to stimulate the breakdown of liver glycogen (glycogenolysis) and to increase gluconeogenesis in the liver to increase blood levels of glucose. Other effects of glucagon include activating adipose cell lipase (which makes fatty acids more available to the body) and inhibiting storage of triglycerides in the liver.

Glucagon's functions are regulated by several factors. If levels of glucagon are increased in the blood and antagonists remain stable; there is a greater chance of glucagon binding to a receptor and thereby imposing it functions. Glucagon levels are increased by increased blood amino acids (i.e., protein ingestion) as well as by exercise. The primary inhibitor of glucagon release is hyperglycemia, or high blood glucose.

While most people are currently concerned about diabetes mellitus in conjunction with insulin levels, tumors causing excess glucagon release from the pancreas can be a major obstacle as well. As described, the excess glucagon will continue to bind to multiple receptors, resulting in excessive hyperglycemia. The excess sugar that is not used will then be stored as fat.

A new drug that has shown potential in the scientific research for treating Type 2 diabetes is known as dual-acting peptide for diabetes (DAPD). It combines the promotion of insulin secretion via a com-

pound known as glucagon-like peptide-1 with the effects of antagonistic action on glucagon receptors. It is currently being developed as a novel dual-acting peptide to treat Type 2 diabetes without gastrointestinal side effects.

SEE ALSO: G-Protein Coupled Receptors; Hormones; Insulin.

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Glucocorticoids

GLUCOCORTICOIDS (GCS) ARE a group of steroid hormones that affect carbohydrate, lipid, and protein metabolism. The biological functions of GCs are maintenance of arterial blood pressure, alteration of the connective tissue response to injury, reduction in the number of circulating lymphocytes, inhibit corticotropin secretion, functioning of the central nervous system, and inhibiting the process of inflammation. They are powerful antiinflammatory agents. They may be administered to treat chronic inflammatory conditions such as arthritis, asthma, inflamed joints, and allergic reactions. Some examples of CG drugs are prednisone, prednisilone, methylprednisilone, dexamethasone, and hydrocortisone. The biophysiological effects of GCs are discussed below.

METABOLISM

GCs stimulate glucosneogenesis (glucose production from sources such as amino acids and lipids) in the liver. GCs enhance the expression of enzymes involved in gluconeogenesis. They serve as substrates for gluconeogenesis by mobilization of amino acids from extrahepatic tissues and help the inhibition of glucose uptake in muscle and adipose tissue. It stimulates fat breakdown by enhancing lipolysis in adipose tissue.

The secretion of GCs by the adrenal gland is regulated by the hypothalamic-pituitary-adrenal axis (HPA) axis via secretion of adrenocorticotropic hormone (ACTH). The main plasma cortisol is protein bound with 4 to 5 percent free fraction. The plasma cortisone is in the free unbound form. The equilibrium of cortisol and cortisone between the plasma and tissues are illustrated with the dotted bidirectional arrows. Tissue-specific GC metabolisms are also depicted. GCs are metabolized primarily in liver and the metabolites are excreted in the urine. Increased tissue concentrations of GCs in obesity are related to increased fat mass.

OBESITY

GCs seem to be important in the development of obesity, as adrenalectomy or GC receptor antagonists attenuate weight gain and associated metabolic abnormalities. The role of GCs in obesity is unclear. The enhanced inactivation of cortisol by 5α-reductase and altered reactivation of cortisone to cortisol by 11ß-hydroxysteroid dehydrogenase type 1 (11ßHSD1) was reported in obese men. The changes in GC metabolism may influence corticosteroid receptor activation and feedback regulation of the HPA. Increased secretion of GCs (e.g., in Cushing syndrome) is associated with obesity, and increased cortisol secretion is seen in subjects with idiopathic obesity, especially of central distribution.

Insulin resistance is viewed as an insufficiency in insulin action, with GCs being recognized to play a key role in its pathogenesis. In addition, clinical administration of GCs to treat acute and chronic inflammatory diseases has been associated with metabolic adverse effects such as hypertension, obesity, hyperlipidemia, and insulin resistance as seen in the metabolic syndrome. GC action is mediated by glucocorticoid receptors, a nuclear receptor that regulates physiological events through activation or repression of target genes involved in inflammation, gluconeogenesis and adipocyte differentiation.

INFLAMMATION AND IMMUNE FUNCTION

GCs have potent antiinflammatory and immunosuppressive properties. GCs are widely used as drugs to

treat inflammatory conditions such as arthritis or dermatitis, and as adjunction therapy for conditions such as autoimmune diseases.

CARDIOMETABOLIC SYNDROME

GC metabolism is also abnormal in insulin-resistant states and may promote including insulin resistance, diabetes, and hypertension. Use of GCs may enhance the risk of heart failure. In a large population-based study, the use of GCs was associated with an increased risk for cardiovascular events (more than 2.5 times higher than nonusers), with a clear dose-response relationship.

BONE HEALTH

The predominant effect of the GCs is a reduction in bone formation due to direct detrimental effects on osteoblasts, although there is also evidence that they increase the activity of osteoclasts. They can also interfere with calcium metabolism, and adversely affect the levels of sex hormones. In addition to the GC treatment, the primary disease itself might also predispose to bone loss through factors such as nutritional deficiencies (e.g., in gastrointestinal diseases) or the increased production of inflammatory cytokines, which enhance bone loss (e.g., in rheumatoid arthritis).

PULMONARY HEALTH

GCs are the most effective therapy available for patients with asthma. GCs bind to GC receptors in the cytoplasm and translocate to the nucleus, where they interact as dimers with GC response elements in the promoter region of steroid-sensitive genes to switch on transcription of antiinflammatory genes, (such as MKP-1). This involves binding of GR to coactivator proteins and acetylation of core histones, particularly histone-4, through the intrinsic histone acetyltransferase (HAT) activity of these coactivators.

CATARACTS

Glucose level in the lens increased markedly with lens opacidication and decreased with recovery from cataract after hydrocortisone administration. GC-induced cataract formation is through a step of oxidation or peroxidation. The use of inhaled corticosteroids and the development of posterior subcapsular and nuclear cataracts association were reported.

COGNITIVE FUNCTION

The secretion of GCs influences cognitive function. These hormones are important in the maintenance of normal brain health and functions. Few studies indicate that endogenous GCs are essential for maintaining prefrontal cortex (PFC) cognitive function and suggest that HPA disruption contributes to PFC cognitive deficits.

GCS DEFICIENCY

Familial glucocorticoid deficiency (FGD) is an autosomal recessive syndrome of adrenal unresponsiveness to ACTH characterized by GC deficiency, high plasma ACTH levels, and a normal renin-aldosterone axis. Defects of the ACTH receptor have been suggested as a possible cause. Insufficient production of cortisol, often accompanied by an aldosterone deficiency, is called hypoadrenocorticism or Addison's disease.

SIDE EFFECTS OF GCS

Long-term use of GCs drugs may cause many adverse effects. These include immunosuppression, hyperglycemia due to increased gluconeogenesis, insulin resistance, and impaired glucose tolerance; reduced bone density (osteoperosis, higher fracture risk, slower fracture repair), increased skin fragility, easy bruising, weight gain due to increased visceral and truncal fat deposition (central obesity) and appetite stimulation, adrenal insufficiency, muscle breakdown (proteolysis), weakness; reduced muscle mass and repair, expansion of malar fat pads and dilation of small blood vessels in skin, anovulation, irregularity of menstrual periods, growth failure, increased plasma amino acids, increased urea formation; negative nitrogen balance and excitatory effect on central nervous system. Dexamethasone can cause loss of appetite, weight loss, stomach upset, vomiting, drowsiness, headache, confusion, fever, joint pain, and peeling skin.

SEE ALSO: Asthma; Osteoporosis.

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Glucokinase

GLUCOKINASE IS AN enzyme that traps glucose in the liver. Glucokinase is part of the hexokinase enzyme family and is sometimes referred to as hexokinase IV. This enzyme functions by attaching phosphorus to glucose inside the liver and forming glucose-6-phosphate (G-6-P). Glucokinase differs from the other hexokinases, which are found in muscle and other organs. Specifically, glucokinase is highly effective at converting glucose into G-6-P in the liver, while muscle hexokinase can only convert a limited amount of glucose into G-6-P. In muscle, elevated levels of G-6-P inhibit hexokinase activity (referred to as negative feedback). While in the liver, elevated levels of G-6-P do not inhibit glucokinase activity. Through the actions of glucokinase, the liver plays a more critical role in the storage of excess glucose than does the muscle. Once glucokinase has trapped glucose in the liver, the liver will decide which metabolic pathway for glucose to enter. This is one reason why the liver is referred to as the gatekeeper of metabolism.

G-6-P is the enzyme that opposes glucokinase. G-6-P works by removing the attached phosphorus (from glucokinase) and creating free glucose. This permits the stored glucose in the liver to be released into the plasma. Glucokinase is regulated by other by-products of glucose metabolism such as fructose-1-phosphate (activates) and fructose-6-phosphate (inhibits). Glucokinase regulatory protein (GKRP) also affects the enzymatic functioning of glucokinase enzyme. GKRP will inhibit glucokinase when glucose levels are low in the blood and activate it when glucose levels are high in the blood, subsequently allowing for either the release or removal of glucose in the body.

Glucokinase is also present in the β-cells of the pancreas. The β-cells are responsible for producing and releasing insulin, the hormone that regulates blood sugar levels. Glucokinase is present in the β-cells in order to sense the presence of glucose, then signal the production of insulin, and release insulin into the body. Glucokinase is regulated by insulin only on a long-term basis such that chronically high levels of insulin may alter glucokinase expression.

Maturity onset of diabetes in youth (MODY) is a rare, genetic form of diabetes mellitus. Patients who suffer from MODY are primarily children who are hyperglycemic (high blood sugar) but have normal β-cell function. Several genetic mutations have been found to be responsible for MODY including a mutation in the gene that makes glucokinase. In this genetic disorder, there is decreased glucokinase level.

SEE ALSO: Metabolic Disorders and Childhood Obesity.

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Gout

GOUT IS A broad term used for the problems with uric acid metabolism in the body. Gout is also referred to as metabolic arthritis due to similar symptoms. As such, gout causes severe pain in joints and associated tissues from the buildup of monosodium urate crystals that lay stagnate in the articular cartilage of joints. This articular cartilage tissue covers the

ends of bones and allows the distribution of weight and compression for normal body movement. It also provides a wear-resistant surface for joint movement and when inflamed is very painful. The inflammatory response from this buildup of urate crystals causes pain to the joints and swelling at the site. If not treated, the swelling can grow and increase in size until it bursts through the skin. Obesity is a major risk factor for developing gout and early diagnosis and treatment are essential to reduce disease morbidity.

HISTORY

Gout is one of the oldest human ailments seen throughout time and obesity has played a major role in how it has been perceived throughout cultures. Some accounts identify gout first with the Egyptians circa 2640 B.C.E. Later, Hippocrates in the 5th century B.C.E., identified gout as separate from other joint disorders and referred to it as the "unwalkable disease." Nonetheless, healers have consistently associated gout with the consumption of rich foods and excessive alcohol. This led those who suffer from gout to be seen as decadent and corpulent, as the foods that seem to contribute to the disease were only available to the wealthy. This negative stereotype is most evident by seeing that gout was also referred to as the "disease of kings" by some historical accounts. Now, with the disease process better understood, these negative stereotypes are no longer tolerated.

In the United States today, gout is twice as prevalent in African-American males as it is in Caucasians. Internationally, the prevalence of gout varies widely from country to country. For example, in England, gout affects about 16 out of every 1,000 men and three of every 1,000 women. There is an increased prevalence of gout among obese individuals across populations. Gout can also develop as comorbidity of other diseases, including diabetes, hypertension, and obesity.

SIGNS AND SYMPTOMS

The clinical presentation is severe and sudden pain, swelling, redness, warmness, and stiffness to the affected joint. The urate crystals inside the joint cause intense pain whenever the affected area is moved and the inflammation of the tissues around the joint also cause skin to be swollen and tender to touch. Hyperuricemia, a high level of uric acid in the blood, is a common sign of gout and is considered an aspect of the metabolic syndrome. However, a high uric acid blood level does not exclusively mean a person will develop gout.

TREATMENT

Treatment of proven crystal-induced arthritis is directed at relief of the pain and inflammation. Narcotic pain relievers, nonsteroidal antiinflammatory drugs (NSAIDs), and steroids are standard treatment. Last, for extreme cases of gout, surgery may be necessary to remove large areas of affected tissues and correct joint deformity. The best prevention measure for gout is a low purine diet and a reduction in alcohol consumption.

SEE ALSO: Alcohol; High Protein Diets.

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Government Agencies

ALTHOUGH GOVERNMENTAL AGENCIES at the federal, state, and local levels play an important role in issues related to obesity in the United States, not everyone approves of the current level of involvement. Some feel that governmental involvement in public health should be restricted to matters such as providing municipal water treatment and monitoring communicable diseases, and that the government should not be involved in diseases that have a strong volitional or lifestyle component, while others feel that the governmental role should be greatly increased, in recognition of the societal costs of obesity and because they do not see the purpose in drawing a distinction between infectious and chronic diseases, both of which are harmful to human health.

Of course, opinions about the proper level of governmental involvement are informed by differing conceptions of who is responsible for obesity; some place the blame on the obese individual as a person

who makes poor food or lifestyle choices, while others point to an environment that encourages overconsumption of unhealthy foods and discourages exercise. These are differences in philosophy rather than questions of fact, and fruitful lines of investigation have been instigated from both points of view.

HISTORY

Governments have been involved in public health at least since the Roman Empire, where the government constructed aqueducts, sewer systems, and public baths and appointed officials to oversee the public water supply, drainage system, and food supply. Charity medical care was also offered in both classical Greece and Rome, and the Roman Empire established charity hospitals by the 4th century B.C.E. Unfortunately, these health measures were not maintained in Europe during the Middle Ages, during which time cities often lacked secure water supplies, drainage systems, and garbage collection systems. These deficiencies in infrastructure are among the factors implicated in the epidemics of communicable diseases, which were a regular feature of European life during this period.

The Renaissance brought a greater interest in public health matters, boosted in part by increased interest in collecting and analyzing data. Notable in this effort was John Graunt (1620–74), who produced the first calculations of life expectancy and noted the association of demographic variables with mortality. The first widespread public health campaigns that involved individual treatment rather than creation of public works such as sewers, were the variolation campaigns that began in the late 18th century. These campaigns offered vaccination against smallpox to large numbers of people within a geographic area, and in some cases, made the vaccination mandatory in recognition of the public benefit of reducing the susceptible population.

The development of the modern public health approach is usually dated from the mid-19th century, when the concentration of population in cities lacking adequate housing and sanitation led to frequent outbreaks of disease. The sanitarian movement in Europe and the United States was founded on the belief that provision of basic public health facilities such as a municipal water supply and sewage system were a worthwhile investment in securing the population's health, and were best provided by governments rather than leaving such matters to the choice of individuals. Public health was often organized the city level in this period, and the Metropolitan Board of Health, created in New York City in 1866, was one of the earliest municipal public health authorities.

The Capitol in Washington, D.C.: Some feel that government should not be involved in diseases that have a strong volitional component.

Public health efforts were expanded in the late 19th and early 20th centuries to deal with many more threats to public health, including quarantine of persons suffering from infectious diseases, mass immunization campaigns, and elimination of mosquito breeding grounds.

As antibiotics and other advances in medicine brought most infectious diseases under control in the United States, chronic diseases such as cancer and coronary heart disease became responsible for a comparatively higher proportion of morbidity and mortality in the population, and occupied more of the attention of medical practitioners and public health officials as well.

Many chronic diseases are influenced by modifiable behaviors such as smoking, alcohol consumption, diet, and physical activity, often called lifestyle factors, and for this reason, many public health campaigns aimed at improving health through modifying these factors have a large educational component. For instance, the Food Pyramid popularized by the U.S. Food and Drug Administration is intended to teach people about the types and quantities of food which make up a healthy diet for an average person. In its current incarnation, the Food Pyramid includes information about the benefits of physical activity as well, and uses information about physical activity, age, and gender to create personalized nutritional recommendations for individuals.

Although few would deny that health behaviors such as smoking and overeating extract a cost both to health of individuals and to society in terms of increased costs for healthcare and loss of productivity, some feel that public health campaigns aimed at modifying such risk behaviors is intrusive and exceeds the proper role of government. If adults choose to smoke, the argument runs, they are aware of the risks, have decided that the pleasure of smoking outweighs the potential harm to their health, and it is not the government's place to interfere with this decision. Similarly, if a person enjoys eating the wrong foods, some people would argue that it should not be the concern of the government to exhort them to do otherwise.

Recent efforts to encourage healthier lifestyles have also included legal restrictions such as banning the sale of soda and junk foods within schools and increasing taxes on products such as tobacco and beer to discourage their consumption. These measures have met with more controversy, particularly when proposals are made to extend such regulations into less clear-cut areas, for instance, to tax fast food at higher rates than other restaurant food. Among the reasons this type of legislation is less popular is because they would then affect adults as well as children, would involve regulation of food rather than tobacco or beer, both of which are traditionally considered "vices" which has been socially acceptable to tax at a high rate, and would require defining what constitutes fast food. For instance, would it include a salad purchased at McDonald's, a hamburger and french fries purchased in a traditional restaurant? Additionally, because fast-food restaurants are more prevalent in poor neighborhoods and offer food at lower prices than many traditional restaurants, this type of tax has been questioned as a tax, which would disproportionately penalize people who have fewer resources to begin with.

Beyond the many well-known public health campaigns, one of the most important functions the government performs in the study of obesity is the collection and analysis of information about the health and health behaviors of the public. It is largely due to such governmental efforts that we have reasonable estimates of how many Americans smoke, how many are overweight or obese, and what the relationship is in the general population between, for instance, body mass index (BMI) and Type 2 diabetes.

AMERICAN GOVERNMENTAL STRUCTURE AND PUBLIC HEALTH

The governance structure of the United States is composed of many layers, and often the federal, state, and local levels of government are all involved in a single public health concern, such as promoting maternal and child health. This multiplicity of governments can be confusing, particularly to observers used to a more centralized form of public health organization, because each state and at least 3,000 local governments as well as numerous federal agencies are involved in regulating and delivering services related to public health. This decentralization is largely due to the fact that the U.S. Constitution limits the authority of the federal government to the specific powers (none of which directly involve public health) and reserves all powers neither allocated to the federal government nor prohibited to be the concern of state governments. State governments, in turn, often grant responsibility for many health functions to local governments. Although this complex structure of governance has advantages in terms of allowing local control and adaptability to local circumstances, it has disadvantages including fragmentation of responsibility and unequal distribution of resources, so that people with the greatest needs may have the least access to services.

The federal role in public health in the United States today principally involves leadership and expertise, provision of funding, and regulation and intervention in matters concerning more than one state (for instance, interstate commerce). The Department of Health and Human Services (DHHS) is the primary federal agency concerned with public health; agen-

cies within the DHHS concerned with public health include the United States Public Health Service, the Centers for Disease Control and Prevention (CDC), the National Institutes of Health, the Indian Health Service, and the Centers for Medicare and Medicaid Services. In addition to the programs carried out within these and related agencies, DHHS is a major funding source for state and local health agencies. Some public health efforts such as the Behavioral Risk Factor Surveillance System (BRFSS), an ongoing survey that collects data about individual risk factors and health behaviors, are conducted as a partnership between the federal government and the states. In the case of the BRFSS, the CDC provides technical expertise and some of the funding, while the states do the actual data collection; the data are then returned to the CDC for processing, then returned to the states and (with some exceptions) made publicly available through the CDC Web site.

Each of the 50 states in the United States has an agency responsible for health, although the organization differs from one state to another. In some states, there is a heath department devoted exclusively to health, while in others, health is included within a department that is also responsible for other social and welfare programs. Functions performed by most or all state health agencies include collection of vital statistics, disease surveillance and control, regulation of health facilities, and regulation of food safety and environmental health. Federal grants and state tax revenues provide most of the funding for state health functions.

Organization of health agencies at the local level also takes many forms. In some states, local health agencies are part of the local government; in others, they are an agency of the state health department; and in yet others, they are free-standing entities independent of both state and municipal governments. Many public health services are actually carried out at the local level, and the services provided vary widely from one area to another. Local health efforts are usually funded by local taxes (primarily property taxes), state subsidies, and federal grants.

EXAMPLES OF GOVERNMENTAL INVOLVEMENT IN OBESITY ISSUES

Vending Machines in Schools. Many elementary and secondary schools have vending machines that may sell sodas and other beverages, candy, and other types of food. Often revenues from these machines are used to fund school or extracurricular activities, such as music programs, class trips, or library purchases, and schools often enter into a contract with a single supplier (such as Coca-Cola) and agree not to sell products from other suppliers. The rising rate of obesity among children and adolescents has increased scrutiny of children's dietary habits, and within the last 10 years or so, people have begun questioning why food and beverages of little nutritional value should be available to students during schools hours. A related question also raised is whether students should be subjected (during school hours) to the advertising typically displayed on the vending machines and on the packaging of the foods and beverages sold.

According to a survey conducted by the CDC in 2000, 43 percent of elementary schools, 89 percent of middle and junior high schools, and 98 percent of high schools in the United States had vending machines or snack bars within the school where students could purchase foods other than those provided through the United States Department of Agriculture (USDA) meal programs. As of 2005, six states have passed laws regulating foods and beverages sold in schools outside of USDA meal programs, and 27 states have introduced legislation regarding this issue. Arkansas has banned elementary schools from allowing student access to vending machines selling soda and food, but most legislation has taken a more moderate approach. Typical regulations include limiting the types of food that may be sold within schools or during school hours, requiring healthy alternatives to be offered, limiting the hours during which vending machines are accessible to students, and requiring information about nutrient and caloric composition of foods to be provided.

Physical Activity. Because many surveys have demonstrated that many American schoolchildren are insufficiently active, there have been calls for establishing physical education (PE) as a mandatory activity within schools. However, like public health, education in the United States is primarily the responsibility of state and local governments, because regulation of education is not one of the powers granted to the federal government in the U.S. Constitution. Due to the lack of a central authority for education in the

United States, there is no way to mandate PE in all U.S. schools. In addition, funding for education is primarily provided through local taxation, supplemented by state revenues and federal grants, so the resources available to schools differ by their location. Current state laws vary widely. In Arkansas and California, the state mandates that PE is required in every grade level, and the amount of time allocated varies by grade level. In contrast, in Colorado only local school districts may impose curricular requirements. In Florida, there are no requirements for elementary and secondary schools, but two semesters of PE are required in high school, while in Idaho PE is required through the eighth grade but not in high school.

Another way governmental agencies have tried to encourage increased physical activity in children is through programs that encourage children to walk or bike to school. Examples of these programs include the Kids Walk-to-School program of the CDC and the International Walk to School in the USA program funded by the CDC and U.S. Department of Transportation. These and similar programs take a community-based approach which encourages parents, local police departments, civic associations, and business to create an environment in which children can safely walk to school, and educate parents and children about the importance of regular physical activity as part of a healthy lifestyle.

Trans Fats. Most trans fats (also called trans fatty acids) are created through partial hydrogenation of plant oils, which reduces rancidity and raises the melting point of the fat. Trans fats are most commonly found in processed foods such as margarine, baked goods, and prefried taco shells, and in the fat used in deep fryers. Consumption of trans fats has also been linked with increased risk of heart disease and has been implicated in a number of other health conditions, including obesity and diabetes. In the United States and many other countries, labels on processed foods are required to list the amount of trans fats they contain.

However, many nutritionists recommend that consumption of trans fats be limited to trace amounts. The National Academy of Sciences determined in 2002 that there is no benefit to the consumption of trans fats, and that there is no known safe level of consumption of trans fats, but has not recommended they be eliminated entirely from the diet because they occur naturally in many animal foods. The World Health Organization recommended in 2003 that consumption of trans fats constitute no more than 1 percent of an individual's total caloric intake.

New York City began a public education campaign against trans fats in 2005, which included a request to restaurant chefs to stop using trans fats in their kitchens. The voluntary effort was not successful, and in December 2006, the city passed legislation banning the use of trans fats in restaurants. New York City is the largest American city to ban trans fats in restaurants, although similar legislation has been proposed in Chicago and in the state of Massachusetts. Partly because of legal actions, some major food manufacturers, including Kraft, have begun exploring alternatives to trans fats for their products, as have some fast-food chains include McDonald's, Kentucky Fried Chicken, and Wendy's. Predictably, the regulation of food consumed by adults, as opposed to those consumed by children in schools, has generated more controversy. Some see such regulation as an imposition by the nanny-state who is overly protective and deprives adults of their right to make free choices, and some restaurant chefs protested the interference with their right to run their businesses, while others point out that many regulations already apply to restaurants (such as health inspections) and that there is no benefit consuming trans fats and no need to include them in any dish.

SEE ALSO: Eating Out in the United States; Obesity in Schools; Physical Activity and Obesity; Physical Activity in Children; Physical Activity Patterns in the Obese; Soda and Soft Drinks.

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Governmental Policy and Obesity

GOVERNMENTS PERFORM MANY tasks. One of the tasks is to deliver services to the people they govern. Healthcare promotion and regulation is a government service that promotes the good of the population of the state. Policies to meet the challenge of a globally spreading epidemic of obesity are being developed and implements by all those countries experiencing an obesity epidemic in contrast to those countries where poverty and malnourishment still prevail.

All governments perform three general functions. They make laws, enforce laws, and adjudicate cases arising under the law. The institutions of government that perform these functions vary from country to country. In the United States, they are performed by the constitutionally defined three branches of government—the legislative, the executive, and the judiciary. In the states, a similar division of legal labor exists, but in many states the executive branch may be a plural one in which the state governor shares power with other elected executive officials.

Policy-making in the United States occurs within the separation of power system between the three branches of the government of the United States and also in the federal system that joins it into a union with the several states. The effect is usually a policy-making process that is democratic without a single person or group in charge, but one that is also at times inefficient.

The politics of the policy process requires the identification of a problem. In the case of obesity, it was noted by a number of health specialists, including public health officials who measure demographic features of schoolchildren or scan them for health issues. It was also noted by many observers that many people in the adult population were gaining considerable weight. These observations were interpreted as a significant health issue that was not simply private or personal but a public issue that needed to be addressed by government. Consequently, the problem was pushed onto the agenda of the government, where in due time, it would be considered for some kind of solution.

Once the government identifies a problem, the next step on the agenda is to define that problem. Otherwise the "problem" that will be "solved" may be something entirely different so that the problem does not actually get addressed. In the case of obesity, the problem was obvious, or so it seemed, until challenged. Critics claimed that despite casual observations that Americans had gained enormous amounts of weight, there really was not an obesity epidemic. The accusation asserted that government bureaucrats and health researchers were colluding with drug and weight-loss industries to mislabel 60 million Americans as overweight to attract funding. Instead of a health problem, it was a political corruption problem, according to critics.

Despite opposition, Congress held hearings and other government agencies in cooperation with health officials moved toward defining the causes of obesity. Again, the politics of the policy process came into play as a number of food growers, processors, and distributors were accused of being responsible for the epidemic of obesity. These include the manufacturers and distributors of corn syrup, the fast-food industry, and even vending machine companies that placed "junk food" in schools for children to purchase. In the struggle to define the problem of obesity and a solution(s) as legal policy all of these participants—healthcare providers, food producers, food distributor, advocacy groups, government agencies with an interest in the development of policy, and others—engaged in a struggle to hammer out a policy that deals with the issues surrounding the problem. This stage of policy adoption is crucial, for without a success at this stage, bills in Congress die and do not become policy. Once adopted, the policy process moves to the fifth stage, which is implementation.

Agencies responsible for putting the policy into play develop programs for the distribution of benefits

It is hoped that the 5-a-day program school program will encourage healthy eating in children, both at school and in the home.

(e.g., money for training those who will counsel the obese, or research money to study obesity), rules for administering educational programs, or other activities connected with meeting the challenge of obesity. In the case of the Department of Education, it would be expected to develop rules affecting school programs education against obesity. In the case of the Department of Agriculture, the school lunch program would be modified to meet health standards. Vending machines may be prohibited from selling junk food. Much of current obesity policy is at the implementation stage, but many developments are being made on evaluating the success of the newly implemented policies.

Significant amounts of money are being spent by government agencies on what people are eating to discover what is and what is not healthy. The focus on what is and is not healthy not only includes the foods people eat, but also the exercise in which people engage. It has been observed by numerous state politicians and educational officials that physical education and nutrition classes have been abolished in a great many schools to put more resources into mathematics and science courses.

Policy makers and those who implement policy-affecting obesity also engage in educational campaigns to raise public awareness of the problem of obesity and ways to deal with it. Bureaucrats may do such simple things as walk to work wearing a pedometer to stress the need for exercise. Because most people today live in a "built environment," which is essentially urban, it means that people are inclined by the environment to be sedentary. To promote antiobesity behaviors, methods of developing ways to burn calories when going about daily activities, such as taking the stairs instead of the elevator, need to be accomplished.

Another illustration of the governmental policies working at cross-purposes and their connection to obesity is to be seen in the fact that there is an almost direct correlation between obesity and income. The wealthier, the healthier, is in part due to the fact that carbohydrates and the calories they provide have never been cheaper in American economic history. As a result, it is possible for people with limited income to purchase large quantities of carbohydrates but not comparatively of vegetables such carrots. Processed foods are much more affordable than are foods that need to be prepared. The very success of American farm policies is also a defeat of its health policies because the cheapest foods are not those with the most health benefits. The politics of the farm subsidy programs are such that Twinkies cost less than a bunch of carrots although the latter are healthier.

The policy trends in the war against obesity is moving beyond simple solutions such as taking vending machines out of schools to engaging the Department of Housing and Urban Development and the Department of Transportation. These departments can development policies that promote changes in the "build environment" that make sidewalks, bicycle lanes, and other exercise-friendly opportunities commonplace in the modern urban world of America.

An important change in the policy of the federal government came in 2004 when the Centers for Medicare and Medicaid Services changed its policy on obesity. Previously, it had denied the claim that obesity was a disease. Since then, it has defined obesity as a disease. Because insurance companies tend to follow government polices in the way they provide coverage of medical services, this allowed for future coverage of treatments for obesity. The policy change was applauded by the American Obesity Association, which is an obesity advocacy group. Healthcare providers also supported the change.

Governmental action usually includes enlisting scientific or social science studies of some policy problem. The Food and Drug Administration requested

the Keystone Center to study the issue of away-from-home foods and their connection to the obesity epidemic. Its report urged the adoption of corporate and public programs to encourage the availability of healthy foods in appropriate proportions rather than the sale of oversized portions of foods heavily laden with fats, sugars, and carbohydrates.

The recent movie *Supersize Me* (2003) starring John Banzhaf and Bridget Bennet, not only used fast food to illustrate the growth of obesity, but it also devoted a portion of the film to the failure of a developer to build exercise opportunities as well as houses. The attention to this in the film points to the dynamic nature of the policy process as one of problem development and redefinition as well as one of implementation and policy reimplementation.

In the American system of politics, the judiciary also makes public policies through its decisions in cases. Most of the suits today have been focused on food and not on other possible causes of obesity. Obesity lawsuits have been filed against fast-food purveyors such as McDonald's. If successful, such suits would establish as government policy that it is a judicial action if food sales can be identified as connected with the development of obesity.

The tactic of using lawsuits to gain policy goals is one that has been successfully used in America against tobacco and others. It will take years to see if the courts decide in favor of the claims of plaintiffs that their obesity is not due to nature or to their personal choices, but was wrongfully caused by the sale of fatty foods. The obesity epidemic is not just an American problem it has been recognized as a major health problem by government in other countries such as Mexico, Australia, Great Britain, and Germany. In those countries, critics also demand changes, legislators resist spending, and interest groups struggle to end excess weight.

SEE ALSO: Federal Initiatives to Prevent Obesity; Governmental Subsidizing of Energy Dense Foods; Healthy People 2010.

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Governmental Subsidizing of Energy Dense Foods

SUBSIDIES ARE PAYMENTS from the government to food producers intended to keep the price of various foods constant. In 2005, the U.S. government spent $23 billion on farm subsidies. Most of this money was spent on three grains: wheat, soybeans, and corn. Because the government is supporting the price of these goods, it tends to keep them (and products made from them) inexpensive. Many of the foods made from these products have been linked to obesity. A large supply of inexpensive obesity-promoting foods could contribute to the current obesity epidemic.

Subsidy programs were originally designed to protect farmers and ensure a steady food supply to the

U.S. population. These policies also tend to decrease the price of wheat, corn, and soy, leading to decreased prices of corn syrup, hydrogenated fats, and meats produced from corn-fed animals. Corn syrup is used for soft drinks and other sweetened foods that have been linked to obesity. The meats produced from corn-fed animals (such as red meat) have also been linked to chronic diseases such as cancer and heart disease.

The controversial question is whether the less expensive price of these materials has actually changed consumption, and thus supports the current obesity epidemic. If food prices affect consumption, they are most likely to have a maximal effect on low-income populations. Research supports the theory that limited economic resources may shift dietary choices toward an energy-dense, highly palatable diet that provides maximum calories per the least volume and the least cost. One study demonstrated that lower prices for vegetables and fruits were found to predict a significantly lower gain in BMI between kindergarten and third grade. Lower meat prices had the opposite effect, although this effect was generally smaller in magnitude and was also insignificant for body mass index gain over three years. If the subsidy programs for the above commodities were removed, there would likely be a small change in the price of energy-dense meat and foods with added sugar, resulting in a small change consumption of these foods. Because small changes multiply rapidly in public health, this small change would likely have an effect on the health of Americans.

This is evident in Poland where government policies have rapidly affected the public health status of the country. The elimination of subsidies for animal fats and the alteration of other agricultural programs in Poland increased consumption of unsaturated fats and made fruits and vegetables more widely available, resulting in a decrease in cardiovascular disease in 12 years. The summary of evidence on subsidies points to a conclusion that removing subsidies of energy-dense foods will have to be combined with programs to decrease fruit and vegetable prices in order to have an effect on public health.

SEE ALSO: Economics of Obesity; Governmental Policy and Obesity; Policy to Prevent Obesity.

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G-Protein Coupled Receptor

G-PROTEIN COUPLED RECEPTORS (GPCRs) are a class of membrane receptors that transduce extracellular signal into intracellular events. Many hormones and neurotransmitters act on GPCRs to produce their biological effects. Upon binding its agonist, GPCRs will activate specific G proteins, which in turn, modulate other intracellular effectors such as enzymes or ion channels.

Besides sharing a common signal transduction mechanism via G proteins, all GPCRs share one common structural feature, possessing a transmembrane domain with seven helixes. The transmembrane domain is crucial in converting the binding energy of GPCR agonists into intracellular second messenger signaling. A particular GPCR may interact with one of several major classes of G-proteins, leading to a change in intracellular cAMP level, calcium level, potassium level, or the level of other second messengers.

GPCRs are important in treating obesity, because many medicinal compounds act on GPCRs directly or indirectly to exert their therapeutic effects. For example, sibutramine, known by the trade name Meridia, inhibits serotonin transporter and norepinephrine transporter, leading to increase of serotonin and norepinephrine which in turn activate G-protein coupled serotonin receptors and adrenergic receptors. Ephedrine acts indirectly on adrenergic receptors by releasing norepinephrine from nerve terminals. These biological events ultimately serve to suppress appetite. New experimental anti-obesity agents are being developed to target GPCRs directly, including cannabinoid receptor inverse agonists/blockers and serotonin 5HT2c agonists.

SEE ALSO: Cannabinoid System; Serotonergic Medications; Sibutramine (Meridia).

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Growth Hormone

GROWTH HORMONE IS a hormone that is produced in the pituitary glands and has anabolic (promoting growth) effects on the body. Growth hormone is critical for normal growth and development of cells. Growth hormone's primary functions include stimulating protein synthesis, increasing blood sugar, maintaining calcium levels, promoting muscle growth, helping fat utilization, and stimulating the immune system.

Growth hormone has long-term effects from promoting the regulation of growth. Levels of this hormone are elevated in children and progressively decrease as a person gets older. One pathway that growth hormone works through uses two compounds known as insulin-like growth factors I and II (IGF-1 and IGF-II). These compounds play a role in enhancing the message of growth hormone in the pathway for protein synthesis. When fasting blood glucose (sugar) levels are low, growth hormone will play a role in helping to increase the glucose levels, but is not responsible for the immediately rise. Growth hormone can also stimulate the levels of a certain enzyme, 1-alpha-hydroxylase, in the kidney, which leads to an increase in vitamin D levels. This in turn will increase calcium absorption and lead to growth of cartilage and bone in the body.

Elevated levels of growth hormone are associated with two conditions: acromegaly, and pituitary gigantism. Several of the clinical symptoms associated with acromegaly include thickening of bones (noticeable in fingers, toes, and the jaw), visual problems and disorders within the cardiovascular (high blood pressure and cardiac myopathy), pulmonary (sleep apnea or narcolepsy), renal (elevated sodium retention), endocrine, and metabolic (low thyroid activity, lower sex hormones, diabetes mellitus, high triglycerides) systems. Pituitary gland tumors are the cause of acromegaly. This disease develops in adulthood. When a pituitary gland tumor is present in children, it will lead to excessive growth, known as pituitary gigantism. Treatment of these disorders involved removing the pituitary gland. Children who have their pituitary gland removed are given synthetic growth hormone at normal levels. Adults who have their pituitary gland removed have traditionally not been given growth hormone. Recent research has shown that supplementation with growth hormone in adults is associated with increased muscle growth, decreased body fat, and an improved general overall feeling.

Growth hormone deficiency is a lack of growth hormone. In children, this condition is associated with decreased growth. Adults may also suffer from a growth hormone deficiency, although it is not completely understood what the consequences from a lack of growth hormone are. It is believed that it may be associated with problems associated with the heart, bone strength, and general energy. To treat growth hormone deficiency, patients are given injections of a synthetic growth hormone. Prader-Willi syndrome is a genetic disorder in which children eat excessively and are short. Growth hormone is the recommended therapy for patients with Prader-Willi syndrome. Some children who used growth hormone therapy to treat their Prader-Willi syndrome have died while on the therapy. This might have been related to growth hormone's effect on respiratory functions. Some researchers recommend testing for respiratory function prior to beginning growth hormone therapy.

SEE ALSO: Metabolic Disorders and Childhood Obesity; Prader-Willi Syndrome.

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Gustatory System

THE GUSTATORY SYSTEM utilizes taste receptors in the oral cavity to differentiate various stimuli through several neurological pathways. The gustatory system is also intricately linked to many of the physiological responses that control gastric secretions, electrolyte balance and the avoidance of toxins.

The gustatory system is comprised of anatomical features of the oral cavity, taste receptors, facial nerves and regions in the brain. The anatomical features of the oral cavity include the tongue, salivary glands and structures involved with swallowing.

Taste receptors are found throughout the oral cavity and most notably on the tongue. Taste receptors are harbored within small, visible protuberances or bumps on top of the tongue called papillae or taste buds. More specifically, papillae found on the anterior portion of the tongue are called fungiform papillae for their broad, flat structures resembling a fungus, and those found on the posterior portion of the tongue are called circumvallate papillae, which are large, dome-shaped structures forming two rows of eight to twelve papillae across the tongue.

The facial (VII), glossopharyngeal (IX) and vagal (X) cranial nerves innervate the taste receptors of the oral cavity. Additionally, each cranial nerve innervates a specific region of the oral cavity. The facial (VII) nerve and its branches are responsible for innervating the anterior and lateral surfaces of the tongue as well as the roof of the oral cavity. The glossopharanygeal (IX) nerve and its branches innervate taste receptors on the anterior, lateral and posterior regions of the tongue. Finally, the vagus (X) nerve is responsible for innervating taste receptors on or in the regions of the larynx and epiglottis. When taste receptors are stimulated, the message is sent from the oral cavity via cranial nerves to an anatomical structure in the brain called the nucleus of the solitary tract (NST) located within the medulla and a portion of the pons. The NST acts as a relay center in the brain, transmitting sensory and motor information to and from the brain in response to gustatory sensations.

The neurophysiologic role of the gustatory system is expansive. Some of the functions of the gustatory system include, distinct taste sensations, maintaining homeostatic and energy levels, recognizing varying degrees of temperature, and palpable stimuli. These perceived stimuli are related to a complex milieu that also encompasses many visceral (autonomic nervous system) and motor reflexes.

There are four taste sensations, which include sweet, salty, sour and bitter. These four taste sensations enable the body, physiologically, to control intake and monitor nutrient levels. Monitoring salt and sugar concentrations is necessary for the body to maintain proper electrolyte and energy levels respectively. Additionally, bitter and sour taste sensations provide the body with a defense mechanism, as these sensations are often associated with poisons and toxins.

Taste sensations are perceived in various locations on the tongue, but some taste receptors exhibit more specificity than others. Taste sensations are detected using ion-channels, which are pore forming proteins that allow for the passage of ions, and G-protein coupled receptors—a system that utilizes ligand receptors on the cell's membrane to activate mechanisms within the cell.

The region of the brain that receives information from the cranial nerves innervating taste receptors in the oral cavity is positioned in close proximity to the region that controls visceral and motor reflexes. These reflexes include chewing, swallowing and gastric secretions, salivary secretions and motility.

It has been proposed that the gustatory system is related to obesity through hedonic or palatability, texture or the energy content of food. However, many of the studies published up to this point have found little conclusive evidence suggesting a hedonic or texture related link to obesity. One fact is certain, obese individuals have a tendency to consume more energy-dense foods than lean individuals, regardless of the hedonic experience or texture of the food.

SEE ALSO: Autonomic Nervous System; G-Protein Coupled Receptors; Palatability; Peripheral Nervous System; Taste Reactivity.

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Habituation

HABITUATION IS A process that has been observed in humans and in all other animals which results in the lessening of stimulus over time. An example is pain, which may be very sharp initially, but which becomes more manageable in the course of time. The same phenomenon results in people apparently ignoring the sound of trains regularly passing outside a house or the powerful odors that might be found in the workplace. Habituation is important because otherwise it would be difficult for people to separate the arrival of new stimuli in the environment, which clearly has an impact on security and efficiency. Changes in the level of a stimulus seem to be more important and urgent than their absolute level. This appears to be a learned ability.

Habituation has a particular meaning with respect to drug use. Drugs have a tendency to promote the body's ability to tolerate their effects and, hence, reduce effectiveness. The use of opiates, for example, presents a significant challenge to the body, which in response, can change its chemical configuration to a significant degree and this leads to problems when the drug is withdrawn or otherwise no longer available because the body has converted itself into a system in which the drug is a necessary presence. This is different from the process of addiction, which contains elements of psychological craving quite separate from the physical changes. The use of substances such as caffeine, amphetamines, and sedatives does not customarily involve such a high level of ingestion that physical changes are produced. Other substances, including heroin and alcohol, to a lesser extent, can be used in large enough doses to produce the physical changes and, consequently, may subsequently represent a greater physical risk to the body. However, it is a very complex undertaking to determine to what extent different types of drug affect different types of people and when individuals are at risk of habituation or of addiction. The World Health Organization has since 1964 used the term *drug dependence* to encompass any condition in which an individual has a strong requirement or need for a drug. In the management of obesity through using pharmaceuticals, it has become apparent that long-term reliance on drugs is generally ineffective without the patient successfully negotiating a variety of changes relating to lifestyle, eating habits, and similar issues.

The same or at least similar combination of factors exists with respect to habituation to tastes of extreme sweetness, saltiness, or other tastes associated with unhealthy food choices. Food technicians expend considerable efforts in creating tastes and food ingredients to attract consumers. It has become clear over recent years in particular that, at least in Western countries, but also other developed and developing countries, consumer tastes have changed to

Over time, the response to any given stimulus such as candy is deadened. This phenomenon is known as habituation.

accommodate more extremely pleasing tastes. In the course of time, the process of habituation ensures that what was once a very sweet, very salty, or otherwise stimulating taste becomes less extreme and, hence, less pleasing. Consumers therefore request additional quantities of the desired taste stimulant. When this results in the creation of extra-hot chili dishes, the problem is rarely severe, although desensitization of the taste buds because of excessive spiciness can lead to an intensifying vicious cycle. However, when the process leads to the intensifying desire for sweetness, in particular, this can lead to obesity as more and more sugary products are added to a wider range of products. In combination with intensive marketing of snack foods, of instantly available gratification from fast foods, and the breakdown of the nuclear family as the defining influence upon eating patterns, it has become ever more the case that habituation is promoting obesity in large numbers of people.

One additional issue with respect to habituation is that of increasing disregard for public information notices with health-related information or exhortations included. Just as cigarette advertising on the packet has been forced to become ever more extreme in terms of the graphic illustrations of the possible outcomes of smoking, with approximately the same results as were once achieved by much milder approaches, the same will be true of food-related health messages.

SEE ALSO: Fat Taste; Gustatory System; Sweet Taste.

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HDL Receptors

ATHEROSCLEROSIS IS A leading cause of cardiovascular disease, contributing to deaths worldwide. However, high-density lipoprotein (HDL) has a protective effect. Indeed, atherosclerosis is inversely related to plasma levels of high-density lipoprotein (HDL) cholesterol.

Specifically, HDL performs an important role in reverse cholesterol transport by removing accumulated cholesterol from extrahepatic tissues. Receptors enhance the flux of unesterified or free cholesterol. Scavenger receptors are cell-surface transmembrane proteins that can bind modified lipoproteins. Cell surface receptors for HDL on liver cells (hepatocytes) are major partners in the regulation of cholesterol homeostasis. The scavenger receptor class B type I (SR-BI) has been identified as a functional HDL binding protein. Nonetheless, the molecular mechanism by which the receptor mediates selective cholesteryl ester uptake still needs to be explored

SR-BI is abundantly expressed in several tissues, including the liver, where its expression is regulated by various mechanisms. SR-BI mediates both the selective uptake of lipids, mainly cholesterol esters, from HDL to cells and the efflux of cholesterol from cells to lipoproteins in liver and steroidogenic tissues. In addition to mediating selective lipid uptake from lipoproteins to cells, SR-BI can mediate the bidirectional movement of unesterified cholesterol between lipoproteins and cells. The binding of HDL to SR-BI mediates cholesterol movement bi-directionally, down a concentration gradient, the extent of which seems to depend on the cholesterol concentration gradient between HDL particles and the plasma membrane. The constitutive expression of SR-BI alters the steady state level of cellular cholesterol and phospholipid. However, the effects are proportional to the level of receptor on the cell surface. The level of SR-BI expression determines both the rate of free cholesterol flux and the steady state level of cellular cholesterol.

HDLs consist of a variety of particles with different sizes, densities, and lipid and protein compositions. The most abundant protein component of HDL is apolipoprotein A-I (apoA-I). Differences in apoA-I conformation in different-sized particles notably influence apoA-I recognition by SR-BI. Indeed, the preferential binding of larger HDL particles to SR-BI promotes selective cholesteryl ester uptake from larger cholesteryl ester-rich HDL over lipid-poor HDL. Particularly, the larger, cholesteryl ester–rich, lower density, spherical -HDL particles bind more tightly to SR-BI than higher density HDLs, lipid-poor pre-ß-HDL or lipid-free apoA-I. The ability of SR-BI to bind a wide variety of lipoprotein classes suggests that the conformation of apoA-I in HDL particles is important for the formation of a productive HDL/SR-BI interaction. The relative contents of apoA-I and apoA-II in spherical HDL particles may also affect their interactions with SR-BI.

The scavenger receptor class B type I (SR-BI) was the first molecularly well-defined cell-surface HDL receptor to be described. SR-BI mediates selective HDL cholesterol uptake by formation of a lipoprotein/receptor complex, which requires specific structural domains and conformation states of apolipoprotein A-I which is present in HDL particles. HDL signaling requires cholesterol binding and efflux and SR-BI serves as a cholesterol sensor on the plasma membrane. Hepatic SR-BI expression can be regulated by a variety of dietary, hormonal, metabolic, and pharmacological manipulations.

SEE ALSO: Cardiovascular Disease; Cholesterol; Low Density Lipoproteins.

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Head Start

LAUNCHED AS A summer project in 1965 by the United States Office of Economic Opportunity, Head Start is federal program that was designed to help break the cycle of poverty by providing educational, social, and other services to preschool-aged children from low-income families. Since 1980, the Department of Health and Human Services has overseen Head Start as a program under the Administration on Children, Youth, and Families. Over 40 years since its creation, the basic tenets of Head Start have been maintained, although refinements and expansions have been inevitable with congressional reauthorizations occurring every five years. For example, in 1995, Head Start services were extended downward to children under three via Early Head Start, aimed at promoting healthy prenatal outcomes for pregnant women, enhancing the development of very young children, and working toward healthier family functioning. In 2007, nearly 900,000 three- to five-year-old children were served, with approximately another 9,000 children under age three.

At the core of the Head Start model since its inception has been its emphasis on addressing the development of the whole child, not only in the realms of social behavior and cognition, but their emotional, mental, and physical health, and nutritional needs as

well. With respect to the nutritional needs, frequent medical screening, immunizations, and dental services are provided, as well as hot meals and nutritional guidance. Unfortunately, children of low-income and minority status—the very demographic groups that are overrepresented in Head Start enrollments—are most likely to develop childhood obesity. For example, two recent studies have reported alarming rates of overweight (above the 95th percentile for age and sex) for black and Hispanic preschoolers attending Head Start centers in Texas (17 percent) and New Jersey (28 percent). On the positive side, the opportunity to serve nourishing meals and establish good eating practices in Head Start centers represents an unmatched opportunity for nutrition education and the promotion of physical activity in at-risk children. To this end, the 2007 U.S. Senate reauthorization bill includes important language regarding childhood obesity prevention efforts as part of Head Start.

SEE ALSO: Department of Health and Human Services; Ethnic Disparities in the Prevalence of Childhood Obesity; School Lunch Programs.

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Healthcare Delivery

THE HEALTHCARE SYSTEM in the United States has traditionally focused on treatment rather than prevention. Obesity is a major risk factor for many diseases and as rates of obesity have increased in recent decades, insurance coverage has typically provided reimbursement for its comorbidities, but not for the treatment or prevention of obesity itself. Although the World Health Organization, National Institutes of Health, Food and Drug Administration, Centers for Disease Control and Prevention, and Social Security Administration all define obesity as a disease,

As the problem of obesity increases, so will the problems of providing adequate health care for patients.

health insurance companies do not consider it as such for reimbursement purposes. There is a lack of understanding that obesity is a disease and a perception that treatments lack effectiveness. Obstacles to healthcare delivery for obese patients include inadequacies in healthcare facilities and insensitivity to obese patients' needs. In order to adequately provide healthcare to patients who are obese all patients need to be treated with respect, facilities should have appropriate equipment and supplies available for obese patients, and practitioners need to be willing to talk to patients about weight-related goals and have a working knowledge of the available obesity treatments.

INSURANCE COVERAGE OF OBESITY

In 2000, the total cost of obesity in the US was an estimated $117 billion: $61 billion for direct medical costs and $56 billion for indirect costs. Direct medical costs include hospitalization, medication, physicians' services, and the services of allied health professionals. Indirect medical costs consist of the value of lost output due to morbidity and mortality. These medical

costs are largely due to comorbidities associated with obesity, including: Type 2 diabetes mellitus, heart disease and stroke, gallbladder disease, osteoarthritis, obstructive sleep disorder and other respiratory problems, and depression. Obesity may also increase the risk of developing some forms of cancer and is associated with complications during pregnancy.

Type 2 diabetes is a disease in which blood sugar levels are above normal. High blood sugar is a major cause of early death, heart disease, kidney disease, stroke, and blindness. Heart disease is the leading cause of death in the US and stroke is the third leading cause. People who are overweight are more likely to suffer from high blood pressure, high levels of triglycerides, high LDL cholesterol, and low levels of HDL, all of which are risk factors for heart disease and stroke. Being overweight may also increase a person's risk of developing cancers of the colon, esophagus, and kidney, and is also linked with uterine and postmenopausal breast cancer in women.

The increase in the prevalence of overweight in children in the United States has been associated with a dramatic increase in the number of children diagnosed with Type 2 diabetes. Sixty percent of overweight children already have at least one risk factor for heart disease. Among children overweight-associated annual hospital costs have more than tripled over two decades, and as these children become obese adults the healthcare costs associated with obesity will continue to rise. The life expectancy for the average American could decline by as much as five years unless efforts are made to change this trend.

Prior to 2004 Medicare coverage stated that obesity was not a disease. This wording has since been removed from the Medical Coverage Issues Manual, however the wording change did not authorize any new Medicare coverage for obesity treatments. Under Medicare, hospital and physician services to treat obesity are excluded unless the treatment is for certain comorbid conditions. Surgery for the treatment of obesity is covered only if it is medically appropriate for the individual and the surgery is to correct an illness, which cased the obesity or was aggravated by it. In 2007 the federal government did not require states to reimburse for the treatment of obesity under Medicaid programs.

Whether or not and to what extend insurance companies and Health Maintenance Organizations (HMO) cover obesity care varies. In 2006 Georgia, Indiana, and Virginia were the only states that mandated health insurers offer the coverage for medical treatment of morbid obesity, and Maryland was the only state that mandated health insurers cover the treatment. Historically, insurance companies have viewed obesity as an individual responsibility not considered a disease for reimbursement purposes. Many insurance companies and HMO's use "condition coverage" for reimbursement of obesity treatments in which coverage is based on the presence of other diseases. They will pay for treating the comorbidities of obesity, but not for preventive treatments that address obesity directly. The American Obesity Association (AOA) is an advocacy group that has committed itself to expanding insurance coverage for obesity treatment. The OPERATE project, "obese people entitled to receive appropriate treatment equitably" was developed to promote access to treatment by persons with morbid obesity.

Many aspects of managing weight are considered discretionary and not reimbursed by insurers. However, there are some insurance providers that are starting to develop wellness programs to help enrollees live healthier lifestyles. For example, Blue Cross Blue Shield of North Carolina offers eligible members a program at no additional charge in which enrollees are given enhanced insurance benefits including doctor's visits to assess and monitor their weight, visits with a dietician for nutrition counseling, and weight loss medications for long-term weight management with prior authorization. Another example of a comprehensive approach to obesity prevention is Kaiser Permanent's "Healthy eating, active living" program. Eligibility for some of these programs, however, does require the diagnosis of obesity and a comorbid condition.

HEALTHCARE FACILITIES

There are challenges in treating obese patients resulting from both the patient and the healthcare facility. Patients who are obese may delay seeking medical care, and they also may be less likely to receive certain preventive services such as Pap smears, breast examinations, and pelvic examinations. Obese patients may be self-conscious about their weight, not want to go for an appointment because of weight gain or failure to lose weight since their last visit, fear negative

comments from the physician or medical staff, or not want to go back because of past disrespectful treatment from physicians or medical staff.

The healthcare facility should demonstrate sensitivity to an obese patient's needs. Care needs to be taken to assure that facilities are adequate to safely and comfortably accommodate obese patients. There should be appropriate medical equipment to accurately assess and treat obese patients. The intake process should include the patient's Body Mass Index (BMI) and severity classification which requires the availability of appropriate equipment such as scales that weigh to at least 500 pounds, spring loaded tape measures for waist circumference, a stadiometer for accurate height measurements, and large-size blood pressure cuffs. Due to the lack of sufficient equipment, patients' frequently have to self-report their height and weight measurements. These measurements should be conducted in the privacy of an exam room, and XXL or larger size gowns should be available size for a patient's comfort and modesty. Additional equipment needed to provide safe healthcare includes adequately sized stretchers and wheelchairs for the transport of patients, and hospital beds and exam tables.

HEALTHCARE PRACTITIONERS

Healthcare practitioners are very important to quality healthcare delivery for obese patients. However, there is evidence of uncertainty among healthcare practitioners when it comes to their role in the prevention and treatment of obesity. Among reasons cited by primary care practitioners for not treating overweight and obesity is the lack of authoritative information to guide treatment: medical school curricula have avoided the topic of obesity and physicians have generally lacked specific skills in the diagnosis and treatment of obesity and the effective utilization of multidisciplinary teams.

Improving the healthcare system to better address the needs of obese patients also requires a change in attitude and belief about obesity by some healthcare professionals. The healthcare field is not free of the social bias and discrimination obese patients often face. Using the Implicit Associations Test (IAT), a method used to examine automatic associations a person has toward a social group, researchers have illustrated that there are unconscious negative attitudes toward obesity even among health professionals that specifically treat and research obesity. These beliefs involve the perception that a patient's obesity is mainly due to lack of willpower. Negative attitudes and false beliefs can lead to impaired clinical judgment by health professionals, disrespectful treatment of obese patients, and reluctance by health professionals to treat patients who are obese. The physician who is successful in treating obesity has a belief in the potential of patients to change, and also has a working knowledge of behavioral therapy, an understanding of the physiology of obesity, and the pharmacological and surgical treatment options available.

Quality healthcare delivery to obese patients also includes access to treatments for obesity. Treatment for obesity may include a combination of diet, exercise, behavior modification, and sometimes weight-loss drugs and in cases of extreme obesity surgery may be recommended. Psychologists, nutritionists, and physicians can all provide dietary and behavioral therapy, which focuses on changing eating habits and physical activity. Intentional weight loss, as little as 5 to 10 percent of the patient's initial body weight, can reduce health risks and improve symptoms of obesity-related conditions.

Losing weight and increasing physical activity can lower an individual's risk for developing Type 2 diabetes and help control blood sugar levels. It can improve blood pressure, triglyceride, and cholesterol levels and lower the risk for developing heart disease or having a stroke. .

Drug therapy is recommended as a treatment option for persons with a BMI greater than or equal to 30 with no obesity-related conditions, or a BMI greater than or equal to 27 with two or more obesity-related conditions. Drug treatment may be used for weight loss and weight maintenance. Most available weight-loss medications approved by the Food and Drug Administration (FDA) are appetite-suppressant medications which make the patient feel less hungry by increasing one or more brain chemicals that affect mood and appetite: Phetermine and Meridia (sibutramine) are the most commonly prescribed appetite-suppressants. Xenical (orlistat) is another FDA-approved obesity treatment drug and it works by reducing the body's ability to absorb dietary fat by about one third. Weight-loss medications on average lead to a weight loss of 5 to 22 pounds more than a

person would lose with non-drug obesity treatments and weight loss with some medications improves blood pressure, blood cholesterol, triglycerides, and insulin resistance. Weight-loss medications may also help individuals keep off weight they have lost.

Surgery is recommended as a treatment option for persons that have a BMI greater than or equal to 40, or a BMI between 35 and 39.9 with serious medical conditions. Bariatric surgery is used to modify the stomach and or intestines to reduce the amount of food that can be eaten, and it can cure and improve many of the comorbidities associated with obesity. In response to the prevalence of obesity hospitals are increasing the availability of these surgical procedures. According to the American Society for Bariatric Surgery, the number of procedures increased from approximately 16,000 in the early 1990s to more than 103,000 in 2003. Most patients lose weight quickly and continue to lost for 18 to 24 months after; patients can have a total weight loss of 60 to 80 percent of their excess weight.

SEE ALSO: Cost of Medical Obesity Treatments; Health Coverage of Gastric Surgeries

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Health Coverage of Gastric Surgeries

GASTRIC BYPASS (BARIATRIC) surgery is one treatment option for individuals with morbid (clinically severe) obesity. The number of bariatric surgeries performed in the United States each year has risen steadily from just under 13,000 in 1998 to over 177,000 in 2006, and demand for the procedure continues to increase. The availability of health coverage for gastric bypass, however, is highly variable and dependent upon a number of factors including insurance company criteria, employer benefit plans, type of gastric bypass procedure, and state laws governing insurance coverage. In fact, as the number of bariatric procedures has increased over the past few years, it has become increasingly challenging for individuals to obtain insurance coverage or approval for the surgery. Insurers cite escalating costs, surgery risks, inexperienced surgeons, and unknown long-term outcomes as reasons to exclude, deny, or limit coverage for gastric bypass procedures.

Generally accepted criteria for coverage of gastric bypass procedures are derived from the National Institutes of Health (NIH) consensus statement on gastrointestinal surgery for severe obesity published in 1991. Gastric bypass surgery can be considered as a treatment option for individuals who have a body mass index (BMI) of at least 40, or for individuals with a BMI of at least 35 who also have a serious obesity-related condition such as cardiovascular disease or diabetes. BMI is calculated by dividing a person's weight in kilograms by his or her height in meters squared [(kg)/height (m2)]. The criteria represent the minimum starting point, however, for gastric bypass surgery as insurance companies and employers may put in place more restrictive criteria for obtaining surgery. (Gastric bypass providers may also apply more stringent criteria, but those will not be covered here.)

Insurance coverage for gastric bypass procedures is not guaranteed; in fact, it is highly variable depending upon a number of factors. Employers can choose whether to offer gastric bypass coverage as part of the benefits package. Employers who are self-insured, that is, they assume the costs of coverage, often elect not to offer coverage for bariatric surgery because of the initial cost for the procedure. In addition, cost savings from bariatric surgery, such as reduced need for medications and greater employee productivity may not be realized until two to three years or more after surgery. Some employers choose to offer bariatric surgery coverage but at an extra cost to employees.

Insurance companies vary widely in coverage of gastric bypass. Some insurers provide coverage with very few out-of-pocket costs for the insured; others limit, or cap, the amount of reimbursement paid for the procedures and related costs, while still other insurance

companies exclude coverage for gastric bypass altogether. Very few states require insurance companies to specifically cover the cost of gastric bypass surgery; rather, the requirements are to provide coverage for medically necessary treatments or procedures. This requirement leaves room for interpretation about what constitutes as medical necessity, and individuals may be denied approval for bariatric surgery on the grounds that the procedure does not constitute a medical necessity, despite satisfying the general criteria set forth by the NIH. Denials can be appealed; however, this is a time-consuming process and there is no guarantee that a denial can be overturned.

In addition, insurance companies have increasingly added more complex and difficult-to-satisfy criteria for obtaining surgery approval. These criteria often include, for example, detailed documentation of physician supervised medical weight-loss programs of varied durations (from six to 18 months to even longer) within a recent time period (generally the last two to five years). The criteria are often quite difficult to satisfy because although many individuals with morbid obesity have tried multiple dieting strategies over their lifetimes, commercially marketed diet plans such as Weight Watchers and NutriSystem® do not satisfy insurance company requirements. Few individuals can provide documented evidence of having participated in a physician-supervised weight-loss program and will need to do so before approval can be considered.

In a strategy to reduce risk, insurance companies also tend to limit gastric bypass coverage to facilities that have achieved a Center of Excellence (COE) designation from special accrediting organizations such as the Surgical Review Corporation (SRC) or the American College of Surgeons (ACS). Facilities with a COE designation have met certain criteria for safety and other short- and long-term outcomes of gastric bypass. Research has demonstrated that outcomes differ between facilities depending upon surgeon experience, type of procedure, and number of procedures performed per year, among other factors. Some insurance companies have established their own application process for bariatric surgeons and centers and will not provide reimbursement unless additional criteria are met.

Finally, some patients decide to pay for bariatric surgery because they either have no insurance coverage at all or have not been able to obtain an approval for the surgery despite having coverage. Financing plans for gastric bypass surgery have become increasingly common over the past few years.

SEE ALSO: American Society for Bariatric Surgery; Cost of Medical Obesity Treatments; Future of Medical Treatments for Obesity; Lap Band; Roux-en-Y Gastric Bypass.

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Health Disparities—NIH Strategic Plan

ALTHOUGH MORE AMERICANS overall are enjoying better health and living longer than ever before, racial and ethnic minorities, the medically underserved, and urban and rural poor populations in the United States continue to experience a disproportionate burden of mortality and disease due to higher rates of cancer, cardiovascular disease, diabetes, asthma, obesity, stroke, and infant mortality. This inequality in health outcomes results from complex interactions between biological, environmental, sociocultural, political, and behavioral factors. The National Institutes of Health (NIH) implemented the NIH Strategic Research Plan and Budget to Reduce and Ultimately Eliminate Health Disparities to address these disparities via a comprehensive program focused on

research, research infrastructure, and public information and community outreach. To complement the overarching plan, each institute within the NIH was required to develop a strategic plan addressing health disparities within its individual area of expertise. The plans are aligned with other national health disparities initiatives and continue to evolve based on public comment, funding, and public health needs, among other factors.

HISTORY AND BACKGROUND—REDUCING AND ULTIMATELY ELIMINATING HEALTH DISPARITIES

Because the NIH is part of the Department of Health and Human Services (HHS), the trans-NIH Working Group on Health Disparities sought to align its objectives with other existing HHS national health disparities initiatives when it began the Program of Action to Address Health Disparities in 1999. The resulting NIH strategic plan is based on elements from both Healthy People 2010 (released in 2000) and the HHS Initiative to Eliminate Racial and Ethnic Disparities in Health. Healthy People 2010 (a set of national health objectives updated every 10 years) includes the elimination of health disparities as one of its primary goals. The 1998 HHS Initiative to Eliminate Racial and Ethnic Disparities in Health identified infant mortality, immunizations, cancer screening and management, cardiovascular disease and stroke, diabetes, and human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS) as the health disparities of greatest concern.

These disparities were chosen in part based on data from a 1985 report issued by the HHS Task Force on Black and Minority Health. The task force conducted a comprehensive study on health outcomes inequalities experienced by African Americans, Hispanics, Asian/Pacific Islanders, and Native Americans and found that six causes of death combined accounted for more than 80 percent of excess mortality in minority groups. The report recommended that the NIH become more engaged in determining the underlying causes of health disparities and to reduce and ultimately eliminate them.

Congress also directed the NIH to take action on health disparities. The Minority Health and Health Disparities Research and Education Act of 2000 (Public Law 106-525) established the NIH National Center on Minority Health and Health Disparities (NCMHD) to collaborate with all NIH institutes, centers, and program offices. The NCMHD provides leadership, coordination, support, and progress assessment for all NIH-sponsored research and activities related to eliminating health disparities.

GOALS AND OBJECTIVES OF THE NIH STRATEGIC PLAN

The objectives of the strategic plan are grouped into three major areas: research, research infrastructure, and public information/community outreach. Through research, the NIH intends to improve diagnosis, treatment, and prevention of diseases and disabilities contributing to health disparities.

Examples include the Multi-Ethnic Study of Atherosclerosis (detection of heart disease before appearance of clinical symptoms in African Americans, Hispanic Americans, Asian Americans, and Caucasians), the Strong Heart Study (American Indians), and the African Americans Diabetes Mellitus Study (AADM). In addition, the NIH Strategic Plan for Obesity Research, developed in partnership by the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) and the National Heart, Lung, and Blood Institute (NHLBI), focuses on research that will reduce obesity and related health conditions that are also associate with health disparities.

The research infrastructure objective encompasses NIH plans to increase participation by health disparity populations in clinical trials, increase funding and research opportunities for individuals from these populations, and increase health disparity population representation in peer review. In addition, the NIH intends to enhance the ability of institutions across the country to conduct health disparities research by providing funding for construction, renovation, equipment, and other resources. Examples of programs within the research infrastructure objective include Community-Based Participatory Research awards (partnering scientists and communities in health disparities research), Research Supplements for Underrepresented Minorities (RSUM), and Biomedical Research Infrastructure Networks (BRIN).

Public information and community outreach is the third and complementary objective. The NIH dissem-

inates research information to healthcare providers serving populations experiencing health disparities, incorporates science-based information into medical curricula and continuing education materials, and provides information in publicly accessible databases and other Internet resources.

In addition, the strategic plan calls for ongoing dialogue with members of health disparity populations and the development of targeted public health education programs. Education campaigns include the HIV Communications Campaign (HIV treatment awareness for African Americans and Hispanic Americans) and Real Men about Depression (depression awareness in Native American, Hispanic, Asian, and African-American populations).

The first draft of the NIH Strategic Research Plan and Budget to Reduce and Ultimately Eliminate Health Disparities was made available for review in 2000 and has undergone a series of revisions, reviews, and updates.

The plan is continually evolving to incorporate new data on additional groups affected by health outcomes inequalities while contextualizing these disparities within the biological, environmental, sociocultural, political, and behavioral factors contributing to them.

SEE ALSO: Healthy People 2010; National Heart, Lung, and Blood Institute; National Institutes of Health; NIDDK; Office of Minority Health.

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Healthy Eating Index

THERE ARE MANY ways that researchers measure people's diets to better understand the obesity epidemic and prevent it the trend from increasing. Two common methods include statistical analysis and scoring an individual's diet. Scoring an individual's diet is based on preset criteria and looks at food intake quality.

The Healthy Eating Index (HEI) was originally created in 1995, and recently in 2005, a new HEI was created to complement the release of the 2005 Dietary Guidelines for Americans. The HEI is a measure of an individual's overall diet quality. The U.S. Department of Agriculture (USDA) Center for Nutrition Policy and Promotion developed the HEI to assess how well American diets comply with the 2000 Dietary Guidelines for Americans and the Food Guide Pyramid.

There are 10 dietary components that the HEI measures to obtain a score out of 100 points. The score breakdown consists of "good," "fair," and "poor." Any score greater than 80 is considered "good," a score between 51 and 80 is considered "fair," and any score below 51 is considered "poor."

In general, the HEI-2005 is a standardized tool that can be used to monitor nutrition, conduct research studies, and educate consumers. Components 1 to 5 assess how well an individual follows the food guide pyramid. Components 1 to 5 will give a score of 10 points for each of the five food groups included in an individual's diet, for a maximum of 50 points. Component 6 assesses total fat consumption. If fat consumed is less than or equal to 30 percent of total calories, 10 points are awarded.

Component 7 assesses saturated fat. If saturated fat is 10 percent or less of total calories, 10 points are awarded. Component 8 assesses the intake of cholesterol. If an individual ingests less than or equal to 300 milligrams of cholesterol, 10 points are awarded. Component 9 takes a look at sodium intake. If an individual's diet contains 2,400 milligrams of sodium or less, 10 points are awarded. Component 10 looks at variety in a diet. If at least half of a serving of eight or more items from different food groups are eaten during the day, 10 points are awarded.

The USDA will sometimes use the HEI on national food consumption surveys, such as the National Health and Nutritional Examination Survey

The Healthy Eating Index is a standardized tool used to measure how closely a person conforms to eating recommendations.

(NHANES) and Continuing Survey of Food Intakes by Individuals (CSFII). The HEI is used for individuals 2 years of age and older. Some of the past HEI findings are that most Americans need to improve their diets, specifically within the fruit and dairy groups. It also determined that; the HEI improves with education, HEI is only modestly affected by income, and women tend to have higher scores than men. Reports from 1999–2000 give a mean HEI score for the U.S. population of 63.8. Ten percent of the population had what was considered a good diet, and 16 percent had a poor diet. The most current information for a summary comes from 1999–2000, which suggests that American diets have slightly improved from 1989 to 1999–2000. In 1989, the HEI score for all people 2 years old and over was 61.5, compared with 63.8 in 1996 and 1999–2000.

SEE ALSO: Food Guide Pyramid.

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Healthy People 2010

PUBLISHED IN 2000, Healthy People 2010 is a set of 10-year health objectives for the United States developed by the U.S. Department of Health and Human Services (HHS). The statement marks the third time HHS has outlined a comprehensive, nationwide health-promotion agenda since its first attempt in 1979. It has two overarching goals: to increase quality and years of healthy life, and to eliminate health disparities. The plan is divided into 28 focus areas containing 467 specific objectives. The objectives set out measurable goals for improvements in health status, risk reduction, public and professional awareness of prevention, delivery of health services, protective measures, surveillance, and evaluation to be achieved by 2010. Three of the objectives directly address obesity: increase the proportion of adults who are at a healthy weight; reduce the proportion of adults who are obese; and reduce the proportion of adolescents who are overweight or obese. Other objectives address obesity-related issues such as physical fitness and nutrition. Many government initiatives affiliated with Healthy People 2010 are charged with achieving the goals or evaluating progress toward the objectives. Despite these governmental programs and other prevention efforts, data published in the Healthy People Midcourse Review indicate that all three obesity-specific objectives have moved away from their target values.

The initiatives most closely related to Healthy People 2010 are DATA 2010, HealthierUS, Steps to a HealthierUS, the Guide to Clinical Preventive Services, and the Guide to Community Preventive Services. DATA 2010, developed by the National Center for Health Statistics, is an interactive database that compiles data for all Healthy People 2010 objectives. HealthierUS is a health-promotion program that shares common goals with Healthy People 2010; it is based on four pillars: "physical fitness," "nutrition," "prevention," and "make healthy choices." The Steps to a HealthierUS program funds community-based projects that embody the HealthierUS goals. The STEPS program uses information from DATA 2010 and progress toward Healthy People 2010 objectives to assess the efficacy of its programs. Finally, the Guide to Clinical Preventive Services and the Guide to Community Preventive Services both attempt to

Healthy People 2010 is a national health-promoting agenda with the goal of improving the general health of Americans.

provide and disseminate a systematic review of public health information.

Data from the National Health and Nutrition Examination Survey (NHANES) have been used to establish a baseline for and assess the progress of all three obesity-specific Healthy People 2010 objectives. In the Healthy People 2010 Midcourse Review, all three objectives showed significant movement away from their targets. From 1988–94 to 1999–2002, the proportion of adults aged 20 years and over at a healthy weight (objective 19-1), defined as a body mass index (BMI) between 18.5 and 25, decreased from 42 to 33 percent. During the same period, the proportion of adults who were obese, defined as a BMI greater than 30 (objective 19-2), increased from 23 to 30 percent. And the proportion of children who were overweight (BMI greater than 25) or obese (objective 19-3) increased from 11 to 16 percent, away from the goal of 5 percent.

SEE ALSO: Body Mass Index; Centers for Disease Control; Food and Drug Administration; Health Disparities—NIH Strategic Plan; Prevalence of Childhood Obesity in the United States; Prevalence of Obesity in U.S. Women.

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High-Carbohydrate Diets

DIETS OF HIGH carbohydrate intake generally consists of 50 to 60 percent of total daily energy intake in carbohydrates, less than 30 percent fat, and less than 20 percent protein. Some common high-carbohydrate diets include the National Cholesterol Education Program (NCEP) Step I and Step II Diet with energy intake restriction; the Dietary Approaches to Stop Hypertension (DASH) diet, which is based on the U.S. Department of Agriculture Food Guide Pyramid; and some commercial programs such as Weight Watchers. The National Weight Loss Registry is one of the best places to go to find information on long-term successful weight-loss maintenance strategies. Over 5,000 people are registered because they have lost over 30 pounds and maintained for over a year. Recent results of the large prospective study reveal that the majority of people who are keeping it off are on high-carbohydrate, low-fat diets. Therefore, despite the Atkins craze of recent years, high-carbohydrate, low fat, and high-fiber diets are proven to be effective in terms of successful weight loss and maintenance, as well as improvements in cholesterol, thereby potentially reducing the risk of many major chronic illnesses, such as diabetes and certain types of cancer.

The general consensus for weight-loss recommendations would promote diets that are high in fiber, while some may say that these diets should also have a low glycemic index. The glycemic index, a concept introduced by D. J. Jenkins to classify foods containing carbohydrates, represents the blood glucose response (incremental area under the curve) to a food portion containing 50 grams of available carbohydrate compared with the response to an equivalent amount of either glucose or white bread.

A recent debate has arisen regarding the use of glycemic index/load for the control of diabetes, blood

lipids, and weight control. In a review of clinical trials that compared the effects of high- and low-GI foods or diets on appetite, food intake, energy expenditure, and body weight, 15 out of 31 short-term studies found that low-GI foods were associated with greater satiety. On the contrary, reduced satiety or no differences were seen in 16 other studies. Further, reduced ad libitum food intake was associated with low-GI foods in seven studies, with opposite results in eight other studies. Whether reduced food intake translated to weight loss was explored in 20 studies of less than sux months in duration. However, 14 studies found no difference, whereas four studies showed weight loss achieved with a low-GI diet versus two studies on a high-GI diet. As reported by the comprehensive review, the average weight loss was 3.3 pounds on a low-GI diet and 3.5 pounds on a high-GI diet. With similar results, the evidence that low-GI diets are superior to high-GI diets with regards to weight loss is skim. Nonetheless, because each study varies slightly by design and execution, there have yet been long-term studies of low-GI versus high-GI diets where ad libitum intake and fluctuations in body weight are permitted.

High-carbohydrate diets have previously been criticized because unprocessed sugars cause hyperinsulinemia, which further leads to insulin resistance, obesity, and cardiovascular disease. Particularly, postprandial hyperglycemia has been found to increase the risk of cardiovascular disease and diabetes. However, if the unprocessed sugars are replaced with whole grains, then there would be many health benefits. High-fiber and low-glycemic index (GI) diets are associated with increased satiety, lower postprandial glycemic response, and lower insulin levels. A review of epidemiological studies looked at dietary glycemic load, whole grains, and systemic inflammation in patients with diabetes. Despite the current controversy surrounding the glycemic index, there is some evidence that suggest diets low in glycemic index/load or high in whole-grain products have been associated with decreased concentrations of inflammatory markers and increased adiponectin levels among diabetic patients. Such effects may be explained by the reduction in hyperglycemia-induced overproduction of oxidative stress and by the amelioration in insulin resistance, adiposity, dyslipidemia, and hypertension. Therefore, diets high in whole grains may have a protective effect against systemic inflammation in diabetic patients and may therefore be recommended to diabetic patients for the prevention of cardiovascular complications.

High-fiber diets may increase satiety to control hunger and aid in the adherence to low-calorie diets, especially because they are lower in energy density than high-fat diets, as humans respond mostly to volume of food eaten rather than calories. Additionally, diets that are high in fiber may ensure a favorable serum lipid profile. High-carbohydrate diets may reduce the risk of diabetes in adults who are prediabetic. It was emphasized, however, that the best action a prediabetic patient could take is to reduce her/his body weight, no matter what type of diet she/he would go on, to reduce future health risks. Moreover, in a review by Lara-Castro and Garvey, it was suggested that dietary fiber, rather than dietary GI per se, may be involved in the effects of carbohydrates on insulin sensitivity. Therefore, high-fiber, high-carbohydrate diets comprised of foods with low caloric density may be used for effective weight reduction and to ameliorate insulin resistance.

In addition to its beneficial effects on insulin, high-carbohydrate, low-fat diets also appear to have a favorable impact on levels of cholesterol. Studies done with the NCEP Step I and II diets have been shown to reduce circulating low-density lipoprotein (LDL) cholesterol levels by 12 and 16 percent, respectively. Specifically, evidence points to the beta-glucan in oats as a major player in the improvement of cholesterol levels, especially in individuals with mild hypercholesterolemia. Over-

Dieters using a high-carbohydrate diet have reported significant weight loss that has been maintained for more than one year.

weight normotensive subjects with mild to moderate hypercholesterolemia on the American Heart Association (AHA) Step II diet were divided into two groups: One group ate whole-wheat bread, whereas the second group ate bread formulated with 6 grams of beta-glucan, a type of soluble fiber derived from oats. After only one week, the high-density lipoprotein (HDL) cholesterol of the group receiving oat bran bread increased by 27.8 percent, whereas there was no change in the group receiving whole-wheat bread. Both groups decreased their LDL cholesterol, but the beta-glucan–fortified diet was significantly more effective. Additionally, the group on the beta-glucan–fortified diet decreased fasting plasma glucose, lost more weight, enjoyed a higher reduction of total cholesterol/HDL cholesterol ratio, and LDL cholesterol/HDL cholesterol ratio. Therefore, the inclusion of oats in a low-fat, high-carbohydrate, and high-fiber diet may improve cholesterol.

Besides the beneficial effect of fiber on cholesterol levels, there are inconsistent data regarding the association of high-carbohydrate diets in general with an increase in triglyceride levels, which may be due to high levels of postprandial glucose and insulin caused by the relatively high amount of carbohydrates. High triglycerides are usually associated with lower HDL and small LDL particles that are more susceptible to oxidation, which makes up an atherogenic metabolic profile. However, these negative effects may be attenuated if monounsaturated fats are replaced with carbohydrates, or if these diets are high in fiber and have a low glycemic index, although there is still controversy surrounding the glycemic index. However, conflicting evidence comes from a study that analyzed data from the Women's Health Initiative, one of the largest and longest prospective studies addressing the most common causes of death. In the study, it was found that over a mean of 8.1 years, a dietary intervention that reduced total fat intake and increased intakes of vegetables, fruits, and grains did not significantly reduce the risk of coronary heart disease (CHD), stroke, or cardiovascular disease (CVD) in postmenopausal women and achieved only modest effects on CVD risk factors, suggesting that more focused diet and lifestyle interventions may be needed to improve risk factors and reduce CVD risk.

While a high-carbohydrate diet has been shown to have beneficial effects on health, some athletes are interested in its ergogenic properties. As glycogen is the primary fuel source utilized during moderate-intensity exercise, many endurance athletes consume high-carbohydrate diets to enhance training and performance. A study that looks at the efficacy of carbohydrate supplementation and chronic high-carbohydrate diets suggests that a large chronic intake of carbohydrates may be necessary for optimal adaptations to training. Another study that looked at the macronutrients necessary for optimal endurance performance reaffirmed that a relatively high daily carbohydrate intake of above 6 kilograms per day and carbohydrate ingestion of 30–60 grams per hour during exercise appears to delay the onset of fatigue.

However, the mechanisms of this effect are governed in part by attenuation of effort perception, rather than solely as a consequence of delaying an impending energy crisis. Besides its effects on delaying time to fatigue, carbohydrate loading and ingestion also impart some neuroprotection from fatigue during prolonged exercise, as well as high-intensity, intermittent exercise. Furthermore, carbohydrate and protein intake in the postexercise period aid in protein synthesis and restoration of muscle glycogen stores. Hence, high-carbohydrate diets may be recommended for endurance athletes and other athletes who participate in exercises of moderate to strenuous intensity.

SEE ALSO: Exercise; High-protein diets.

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High-Density Lipoproteins

HIGH-DENSITY LIPOPROTEINS (HDLS) are often referred to as the "good" cholesterol. HDL is cholesterol packaged in a coat of protein and phospholipid. HDL carries approximately one-third of blood cholesterol. HDL protects arteries by harvesting cholesterol from the arterial walls and the blood. It brings the scavenged low density lipoproteins (LDLs) back to the liver where it is converted to bile and excreted. HDL counteracts the accumulation and growth of plaque from Low Density Lipoproteins (LDL), also known as the "bad or lousy cholesterol. A higher HDL level is equated with the body operating at optimal capacity. Low levels of HDL may be linked to a higher risk of coronary heart disease (CHD). Opposite trends are seen between LDL and risk for cardiovascular problems, including coronary heart disease.

Dietary change appears to influence cholesterol levels. Monounsaturated fats and polyunsaturated fats are healthier alternatives to saturated fats and transfats. Monounsaturated fats include olive oil, canola oil, avocadoes, and most nuts. Polyunsaturated fats include the oils of seeds and grains including safflower oil, corn oil, sunflower oil, walnut oil, and soybean. In determining cholesterol levels, the type of fats included in the diet are critical. Saturated and trans fats tend to raise LDL cholesterol levels, while at the same time, these fats do not show beneficial effects on HDL levels. In contrast, fatty fish provides a source of omega-3 fatty acids which may help to raise HDL. Examples of good sources of omega-3 fatty acids include mackerel, herring, sardines, anchovies, and salmon.

It is desirable for HDL to be 60 milligrams of cholesterol per deciliter of blood (mg/dl) or higher. HDL less than 40 mg/dl is considered to be low and raises the risk of CHD.

Lifestyle factors appear to have the most significant influence on HDL levels. Obesity, smoking, and inactivity may lower HDL. Genetics may play a role in determining total cholesterol levels as well. Further, the presence of type 2 Diabetes Mellitus also tends to be related to lower levels of HDL cholesterol (although factors such as the existence of obesity and related conditions may confound this relationship). Exercise and weight loss key lifestyle factors that can contribute to more favorable blood cholesterol levels (higher HDL, and lower LDL). Furthermore, quitting smoking may raise HDL and decrease the tendency of the blood to form clots. There is no medication specifically designed to raise HDL. Some medications designed to lower LDL may raise HDL. Lifestyle changes appear to exhibit the most significant positive impact on raising HDL.

SEE ALSO: Atherosclerosis; Blood Lipids; HDL Receptors; LDL Receptors; Low Density Lipoproteins.

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High-Protein Diets

MANY PEOPLE, FROM exercise enthusiasts to people who want to lose weight, have used high-protein diets. Some of the popular weight-loss diets with a central theme around high protein intake include the ZONE diet, Protein Power diet, CSIRO diet, and Sugar Busters. The theory behind these diets emphasizes the increased satiating effect of protein, with only modest reductions in carbohydrate intake, as opposed to the Atkins diet, which allows for only a small amount of carbohydrates. In general, these diets consist of approximately 30 percent protein as a percentage of energy, 40 percent carbohydrates, and 30 percent fat. Importantly, these diets need to contain protein of high biologic value, or high in essential amino acids, to preserve lean body mass. Moreover, these diets must contain an adequate amount of essential fatty acids, and vitamin/mineral supplements to prevent the development of a malnourished state.

There are many health benefits to a high-protein diet. In general, doubling the amount of protein as a percentage of energy occurs by reducing the amount of fat and carbohydrate energy with only a 10 to 15 percent increase in the actual amount of protein. Moreover, high-protein diets are associated with better fat loss and relatively less lean mass lost. A study that used a diet similar to that of CSIRO, protein intakes of greater than 1.05 g/kg of actual, rather than desirable, body weight were associated with 1.3–2.6 pound better retention of lean body mass.

The high-protein diet was popularized most recently by the Atkins Diet. While many people have reported losing weight on such programs, there are potential health risks involved with such a diet over longer periods of time.

Therefore, a high-protein diet may ameliorate the loss of lean body mass accompanied by weight loss.

Many studies have examined the effects of protein on satiety. Typically, studies use a crossover design and presented subjects with one of several preloads of different protein content. Subjective satiety ratings were measured repeatedly for several hours after consumption. In a review of 14 studies, 11 found that the protein preload significantly increased subjective ratings of satiety. Therefore, protein is thought to be the most satiating of the macronutrients, although this does not mean that the other macronutrients should not be consumed in a diet weight loss, because glucose is essential for brain function, and fat plays a role in signaling meal termination.

In the same review, of the 15 studies that measured absolute weight loss, seven studies found a significantly greater weight loss with a higher protein diet. Five out of these seven studies had durations of greater than six months as opposed to the studies with null findings. On the whole, diets high in protein may enhance weight loss compared to diets with lower protein in the short term.

A possible reason for the success of high-protein diets may be due to their greater thermic effect. The thermic effect of a food is the increase in energy expenditure above baseline following consumption, or the energy required for digestion, absorption, and disposal of ingested nutrients. Generally, the typical thermic effect of protein is 20 to 35 percent of energy consumed and for carbohydrate, between 5 to 15 percent. It is hypothesized that one main reason for the differential thermic effects of food may be due to the lack of storage capacity for protein. Thus, there is a need for its immediate metabolic processing. Additionally, other reasons include the high ATP cost of peptide bond synthesis, the high cost of urea production, and the gluconeogenesis involved in the synthesis of protein.

High-protein diets have been associated with an improvement in blood lipid levels. In a study conducted by Gardner et al., higher protein diets led to improvements in weight loss, triglyceride levels, and high-density lipoprotein (HDL) cholesterol levels, with increased satiety. Another study found that when protein, as a percent of energy, is doubled from 15 to 30 percent, weight loss is increased at 12 months by about 6.5 pounds compared with a high carbohydrate diets, with benefits on triglyceride levels. High-protein diets have been shown to benefit triglyceride levels and sometimes HDL cholesterol levels as well. People with elevated triglyceride levels gain specific

benefits, at least at three months into the weight-loss study, such as greater weight, fat, and central fat loss. Because of these improvements in triglycerides, patients with metabolic syndrome, especially elevated triglycerides and high blood glucose, may benefit more from carbohydrate restriction. Hence, the effects of high intakes of proteins may aid in the improvement of levels of blood lipids such as triglycerides, HDL cholesterol, low-density lipoprotein (LDL) cholesterol, and total cholesterol.

In addition to improvements in blood lipids, high-protein diets have also been associated with improvements in blood pressure. Epidemiological studies have linked high-protein diets with lower blood pressure and decreased risk of cardiovascular disease. OmniHeart was a study that compared the effects of three diets on blood pressure, cholesterol levels, and heart disease risk. The diets used in the study were variations of the Dietary Approaches to Stop Hypertension (DASH) diet, which emphasizes low sodium intake. One diet was the control, with high amount of carbohydrates. Another diet partially replaced carbohydrates with protein with about half from plant sources, whereas another diet partially replaced carbohydrates with unsaturated fat, predominately in the form of monounsaturated fat. It was found that in the setting of a healthy diet, the diet with more protein intake showed better improvements on blood pressure, lipids, and estimated risk of cardiovascular disease compared to the other diets.

On the other hand, several studies have found that the levels of triglycerides, HDL cholesterol, blood pressure, and measures of insulin resistance were not significantly different or were more favorable for very-low-carbohydrate groups. Furthermore, other studies have expressed concerns about the adverse effects of blood lipid levels and risk of cardiovascular disease.

Not just for people aiming to lose weight, many exercise enthusiasts have long been supporters of high-protein diets. Athletes such as bodybuilders may consume as much as 3 grams of protein per kilogram body weight. In the late 1970s, branched chain amino acids (BCAAs) were suggested to be the third fuel for skeletal muscle after carbohydrate and fat. Moreover, it was thought that carbohydrate ingestion during exercise can prevent the increase in BCAA oxidation. However, later studies that used exercise and treatment designs and different forms of administration of BCAAs have found no effects on performance, anticatabolic effects during and after exercise, or that BCAA supplements may accelerate muscle repair after exercise. According to a review by Gleeson, activities of enzymes in BCAA oxidation are too low to allow a major contribution to energy expenditure during exercise. In fact, excess protein intake of greater than 3g/kg body mass per day, as achieved by protein supplements, may cause kidney damage, increased blood lipoprotein levels, which are associated with an increased risk of atherosclerosis, and dehydration. Increased protein intake leads to an increase in nitrogen excretion in the urine, which results in increased urinary volume and dehydration. Therefore, athletes consuming a high-protein diet must increase their fluid intake to prevent dehydration. Last, in addition to the health risks of a diet with daily protein intake above 3g/kg body mass, there are normal food alternatives that are not as harmful to the wallet as protein supplements.

Even though there are benefits to a high-protein diet, there are health risks as well. Common complaints include constipation, headaches, muscle cramps, and halitosis. Dietary protein is known to modulate renal hemodynamics by increasing glomerular filtration rate and renal blood flow. High intakes of protein increase the urinary excretion of calcium through the decrease in renal absorption, possibly resulting in negative calcium balance and metabolic bone disease. Therefore, the calciuretic effect of protein may lead to an increased risk of fractures. Moreover, the association of animal protein consumption with hyperuricosuria, hypercalcuria, and a reduction in urinary pH are all risk factors for stone formation.

Long-term high protein intake may accelerate renal function decline in individuals with mild renal insufficiency. One study found that women with impaired renal function have a greater decline in renal function with a greater protein intake, whereas those with normal renal function had no such decline. Therefore, individuals with kidney problems may want to steer away from consuming large intakes of protein.

In addition to the effects of protein on kidneys, a study found that there was a 30 to 40 percent increase of rectosigmoid cancers in the quintile with the highest intake of red meat. However, the European Prospective Investigation into Cancer and Nutrition study found that this was only true for processed meats. On a similar note, a study by Clifton

claims that eating more than 28 grams of fiber per day removes the effect of meat altogether. In general, animal sources of protein are characteristically high in saturated fat. Therefore, wiser and leaner choices would include lean red meat, low-fat dairy, chicken, fish, as well as plant sources of protein such as soy, nuts, and whole grains.

SEE ALSO: Atkins Diet; High Carbohydrate Diet.

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Hispanic Americans

HISPANICS COMPRISE THE largest minority ethnic group in the United States and their proportion is expected to double by the year 2050, becoming nearly 25 percent of the population. Therefore, attention to obesity in this population group is crucial to assure the health of all Americans.

WHO ARE HISPANIC AMERICANS?

Sometimes a distinction is made between the terms *Latino* and *Hispanic*, because the first term includes people from Latin America who are not of Hispanic origin. However, Hispanic is the broader term used in the U.S. Census and most research. Therefore, for simplicity's sake, the term *Hispanic Americans* is used to denote both Hispanics and Latinos/Latinas who reside in the United States.

The U.S. Hispanic population is quite heterogeneous, with great diversity in country of origin, culture, history, and race, as well as immigrant experience, duration of residence, and socioeconomic status. Over 60 percent (25 million) are of Mexican origin; 15 million are of Cuban and Puerto Rican origins, as well as Central and South America, the Dominican Republic, and Spain. Many Hispanic Americans are of mixed ancestry that includes Spaniards and other Europeans (including Italians, Russians, French, Germans, Dutch, Irish, and Scandinavians),

It can be difficult to draw firm conclusions about Hispanic-American health due to the diversity of the population.

Africans, Amerindians, and East Asians. Nearly half of those who identified themselves as "Hispanic" on the federal 2000 Census also identified themselves as "white," and the remainder were of other races.

Hispanics may be first-generation immigrants (born outside the United States), second generation, or third generation and beyond. Some Hispanic communities in the Southwest trace their origins as far back as the 15th through 17th centuries. Patterns of social incorporation and acculturation among different generations of Hispanic Americans are related to the acquisition through time of beliefs and behaviors more in tune with recipient societies, which may affect their obesity trajectories. The ongoing process of incorporation and acculturation, along with other variations among Hispanic Americans, makes it difficult to compare and generalize about obesity in this population, although there are discernable trends.

OBESITY RATES IN HISPANIC AMERICANS

Most Census and research data relate to Mexican Americans, whose obesity and overweight prevalence has been increasing steadily for three decades and is estimated to be 34 percent and 73 percent, respectively. Approximately the same percentage of women and men are overweight (72 percent and 74 percent, respectively), but many more women are obese than are men (40 percent versus 30 percent) and twice as many women as men are severely obese.

As is the case with immigrants in general, newly arrived Hispanic immigrants generally have had lower obesity rates than the overall U.S. population. However, over time, much of this advantage disappears; as years go by, Hispanic immigrants and those of subsequent generations tend to have increased rates of obesity and obesity-related diseases that surpass those of non-Hispanic Caucasians. Here, too, disparities have been found within this population—longer residence in the United States is associated with increased overweight among Puerto Rican and Cuban Americans, but not among Mexican Americans.

Although obesity is a risk factor for many chronic diseases for both genders, Hispanic women may be of particular interest because of the impact of obesity on maternal, child, and adolescent weight and well-being. Hispanic children are twice as likely to be overweight as non-Hispanic Caucasian children, with rates among 2- to 5-year-olds as high as 35 percent, while 30 percent of Hispanic adolescents are obese.

FACTORS INFLUENCING OBESITY IN HISPANIC AMERICANS

It is thought that culture, acculturation, and changing socioeconomic status (SES) interact to influence obesity trajectories over time. Hispanics Americans face many unique interrelated life stressors including lack of social support, language and cultural barriers, employment issues, caregiver status, conflicting sex-role expectations, and discrimination.

Among certain Hispanic cultures, overweight and obesity may be more tolerated and even preferred than among whites. Excess weight may be equated with health in children and curves may be equated with desirability in women. As a result, some Hispanics may be less preoccupied with weight control, less likely to consider themselves as overweight, and thus less motivated to control their weight. However, data are contradictory and may be evolving with time as the mainstream ideal of thinness becomes more widespread.

Acculturation and socioeconomic status may influence Hispanics' diet and physical activity by affecting the availability, accessibility, and appeal of healthy food and physical exercise opportunities in their environment. Although acculturation has generally been associated with obesity, presumably via obesigenic health behaviors such as high caloric intake and sedentary behavior, at this point in time, the data are not clear or consistent. For example, acculturation has been found to have opposite effects on healthy diet and amount of physical activity; some studies show a strong acculturation effect, while others show little or none; and low income may be a proxy for other factors such as accessibility.

Nevertheless, it should be noted that Mexican and many other Hispanic cuisines traditionally depend on plant foods—and emphasize fruits, vegetables, whole grains, and legumes—and with a few exceptions are not rich in the desserts and added fats that typify the American diet. These differences may be protective—if they are maintained. With increased acculturation, however, the relatively healthy Hispanic immigrant diet appears to deteriorate. Poor urban neighborhoods tend to favor convenience-type bodegas and fast-food

restaurants that sell energy-dense, high-fat, and high-sugar foods. Other related factors found to affect food access are a lack of familiarity with the community, language barriers, and lack of transportation to food markets where fresh or familiar foods are sold.

Studies on physical activity indicate that Hispanic women have the highest rate of inactivity—40 percent compared with Hispanic men (33 percent) and Americans overall (24 percent). However, these studies measure only leisure time physical activity and may underestimate total physical activity, especially in people with physically demanding occupations, such as blue-collar Hispanics who may be more active when nonleisure time is considered. Indeed, Hispanic women report more housework, dependent care giving, dancing, and work activity than non-Hispanic women. In contrast to the case with diet, acculturation (according to language acculturation, but not length of residence) appears to improve the level of physical activity in both men and women. Hispanics have named many barriers to physical activity including gender roles, language difficulties, peer pressure, lack of transportation, lack of facilities or programs, personal or neighborhood safety concerns, and cost. People living in low-income and high-minority neighborhoods have reduced access to physical activity facilities.

Finally, acculturation and socioeconomic status influences access to healthcare, especially preventive care, which in turn can influence health behaviors related to obesity through lower use of healthcare services, lower quality and less intensive healthcare, and lower likelihood of dietary counseling than U.S.-born whites.

Current thinking focuses on developing culturally specific interventions and encouraging immigrants to maintain the healthy aspects of their cultural traditions while adopting healthy aspects of Western life. Further research may additionally shed light on possible strategies to facilitate retention of other protective factors such as strong family and cultural ties and other social behaviors while promoting English-language skills, education, upward mobility, access to healthy food and physical activity opportunities, and greater ability to navigate the U.S. healthcare system.

SEE ALSO: Body Fat Distribution in Hispanic Americans; Central America and Caribbean; Dominican Americans; Immigration and Obesity; Mexican Americans; Puerto Rican Americans.

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Histamines

HISTAMINES ARE BIOGENIC amine compounds that play roles in local immune responses and regulation of physiological function in the gastrointestinal system. Histamines are neurotransmitters. There is evidence that histamines are factors affecting body-weight regulation and these compounds are known as mediators with critical role in homeostatic regulatory processes (e.g., control of food intake and maintenance of body weight). Histamine H3-receptor antagonists have been suggested as new drugs in control of obesity.

It has been shown that central infusion of histamine diminishes the accumulation of fat, upregulates uncoupling protein, decreases leptin, and increases the insulin sensitivity in a leptin-resistant animal model of obesity. It is believed that weight gain associated with the use of typical and atypical antipsychotic drugs is related to H1 receptor blockade.

Histamine receptors have been used as obesity drug targets, and "antihistamines" are common medications. Drugs targeting histamine receptors include classic antihistamines (such as diphenhydramine) targeting histamine H1 receptors and used in allergic reactions and histamine H2 receptor antagonists (such as cimetidine, ranitidine, and famotidine), which block histamine H2 receptors and are used in gastric ulcers. The next generation of antihistamine drugs has been suggested to block histamine H3 receptor. Histamine H3 receptor is a G protein–cou-

pled receptor. This receptor has a role in regulation of neurotransmission in the brain and can control some cognitive and homeostatic functions. This receptor has been proposed as a drug target for treatment of attention-deficit hyperactivity disorder, dementias, schizophrenia, obesity, and sleep disorders.

The preclinical studies have confirmed histamine H3 receptor antagonists to be effective in controlling obesity, but no clinical trial has been conducted to evaluate it. The biology of histamine H3 receptor is very complex and further understanding of this drug target will lead to designation of new drugs for obesity. Despite this fact, many selective H3 receptor antagonists have been produced, but they have not been used in any clinical trials.

SEE ALSO: C-Reactive Protein; Cytokines; Interleukins.

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Hormone Disorders

HORMONES PLAY A crucial role in the etiology and consequences of obesity. Insulin, glucocorticoids, glucagon, androgens, thyroid hormones, and growth hormone affect the energy homeostasis in the body, and in some cases, impairment in their normal functions and activities can cause obesity. Several neuroendocrine diseases are believed to have a possible association with the development of obesity. Because hormones play such a crucial role in obesity, accurate measurement of these proteins with hormone assays is essential in obesity treatment. Hormone assays can help establish possible endocrine pathogenesis of obesity, and can reveal hormonal impairment due to obesity itself.

The hormones function in different ways as modulators of energy balance in human body. For example, insulin can affect the volume of fat cells by reducing food intake; glucocorticoids and glucagon can control fat storage through their effects on energy intakes; and androgens, thyroid, and growth hormones modulate the fat storage in the body via their effects on energy expenditure. All these show that in evaluation of a patient with obesity, hormone disturbances must be evaluated by hormone assays.

In such an evaluation, some disorders such as hypothalamic obesity, Cushing's syndrome, hypothyroidism, and polycystic ovary syndrome should be considered. In other words, in pathogenesis of obesity, hormonal factors should be noted and therapeutic strategies must include treatment of endocrine disorders as well. For example, studies suggest that growth hormone therapy can decrease the amount of stored body fat.

The rare syndrome of hypothalamic obesity is due to damage to some regions in human hypothalamus or the amygdala. In these parts of the brain, some regions process the information related to metabolic status of the body. Obesity is a presentation of damage to these regions caused by impairment in hypothalamus hormonal activity.

Obesity is an important clinical presentation in Cushing's syndrome too and in hypothyroidism, the decreased metabolic activity can lead to obesity and fat storage in the body.

In polycystic ovary syndrome (PCOS), obesity is a clinical feature in more than 50 percent of the patients. Some other neuroendocrine diseases related to obesity are hypogonadism, growth hormone deficiency, and pseudohypoparathyroidism.

All the described disorders are due to disturbances in hormone production and activities and the evaluation of endocrine system by hormone assays is crucial in finding the pathogenesis of obesity in any patient. Obesity itself can cause endocrine disorders such as insulin resistance with hyperinsulinemia. In this disorder, disturbance of glucose removal due to

obesity and elevated insulin resistance cause hyperinsulinemia. In a patient with obesity, hormone assays can reveal the harmful effects of obesity on the hormonal activities as well.

SEE ALSO: Cortisol; Cushing Syndrome; Glucocorticoids; Growth Hormone; Hormone Sensitive Lipase; Hormones; Insulin; Insulin-Like Growth Factors; Menopause; Pituitary Gland.

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Hormones

HORMONE COMES FROM the Greek word *hormao*, which means "a substance which starts, urges on, initiates, irritates, stimulates or excites." It was in 1656 that Thomas Wharton gave the first thorough account of the glands of the human body. It was only in 1905, however, that the word *hormone*, on the suggestion of Sir William Bate Hardy, was first used by Ernest Henry Starling, in one of his Croonian lectures, to name the internal secretions. This established the basis for the subsequent development of the discipline of endocrinology.

Virtually all multicellular organisms (plants and animals) produce hormones. Generally, they are described as chemical messengers that control metabolism, growth, development, and even reproduction. Through these chemical messengers, the cells are able to communicate and interact, maintaining a steady state which we call homeostasis. In this entry, we are going to concentrate on the hormones secreted by the human body, their functions and regulation.

Many aspects of the human body, including appetite, are controlled by a number of hormones.

In principle, hormones function gradually. More often than not, their effects are not immediate. They also do not act directly on behavior; rather, they change its probability and intensity. They are influenced by factors that are both endogenous (to do with development and maturation) and exogenous (to do with the environment). Hormones often have multiple target sites and are produced in pulsatile

secretions; others follow a circadian rhythm regulated by other hormones or exogenous triggers. In action, they are target specific; they can only affect cells with receptor sites for a particular hormone. Last, the effects of hormones are interactive and long term, because they influence the buildup and breakdown of carbohydrates, lipids, and proteins, together with other metabolic changes.

There are three kinds of chemical signaling:

- Autocrine—the cells signal themselves through a chemical that synthesizes and then responds. Autocrine signaling can occur
  -solely within the cytoplasm of the cell, or
  -by a secreted chemical interaction with receptors of the same cell.
- Paracrine—chemical signals can diffuse into an area and interact with receptors on nearby cells. Examples are
  -the release of cytokines which cause an inflammatory response in the area,
  -the release of neurotransmitters at synapses in the nervous system.
- Endocrine—the chemicals can be secreted into the blood and carried by blood and tissue fluids to the cells upon which they act.

There are five known chemical classifications of hormones found in the human body. The first group is amine-derived hormones. Amine-derived hormones originate from the amino acids tyrosine (e.g., dopamine) and tryptophan (e.g., melatonin). The second is peptide hormones. Peptide hormones are made up of chains of amino acids (e.g., hormones of the stomach). The third is steroid hormones. Steroid hormones are derived from cholesterol (e.g., the androgens). The fourth is sterol hormones; they are derived from vitamin D (e.g., calciferol, calcitriol). The fifth group contains lipid and phospholipid hormones. Lipid hormones (e.g., prostaglandin) are derived from linoleic acid; phospholipid hormones are derived from arachidonic acid (e.g., leukotrienes).

In terms of cellular action, protein and amine hormones bind to the extracellular portion of a receptor site; it releases a second messenger within the cell; and its effects are relatively fast (seconds to minutes). In contrast, steroid hormones pass through cells; they bind to receptor genes within cells (receptor-gene complexes), which in turn bind to deoxyribonucleic acid (DNA). The action of the DNA may interact with other factors, which determine the cell's receptivity to the hormone.

The human body has a hormone system (endocrine system) for detecting and evaluating hormone levels. It has four known feedback mechanisms:

1. Autocrine feedback—hormones inhibit endocrine cell release
2. Target cell feedback—target cells produce effects which are detected by endocrine cells
3. Brain regulation—the hypothalamus drives endocrine cells and is sensitive to the effects produced by cells
4. Brain/pituitary regulation—the hypothalamus and pituitary drive endocrine glands and are sensitive to the effects produced by target cells

In contrast to the neural communication of the body, hormonal communication is broadcast, while neural is channel specific. It is slower than neural communication, with a graded action, as opposed to the all-or-nothing principle of neural communication. There are, however, several similarities between them—glands and cells both produce products as a result of stimulation; both systems rely on binding mechanisms; and both utilize second messenger systems.

SEE ALSO: Adipocytes; Hormone Assays.

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Hormone Sensitive Lipase

HORMONE SENSITIVE LIPASE (HSL) is the major enzyme that regulates the release of stored fatty acids from the fat tissue, a process known as lipolysis. This enzyme is important because it plays a role in providing energy to tissues and organs when food intake is low. HSL works by cleaving off the three

fatty acids that are attached to a triglyceride molecule. Once these fatty acids are cleaved, they can then be attached to albumin (a protein in the blood) and travel to tissues and organs that need fatty acids for energy. Several different hormones regulate HSL; insulin inhibits HSL while glucagon, ACTH, epinephrine, and norepinephrine activate HSL. Traditionally, HSL was believed to be the only enzyme that could cleave triglycerides. However, several experiments were performed in mice that removed the gene that made HSL. Even though there was no more HSL activity in these mice, they were still able to cleave the fatty acids off triglycerides. As a result, other hormones such as adipose triglyceride lipase have been identified to play a role in lipolysis.

HSL has been found in organs other than fat cells. It is present in skeletal muscle and in the heart where it functions to cleave the stored fatty acids that are subsequently used for energy within these organs. It is also present in the beta cells of the pancreas, adrenal glands, testes, ovaries, placenta, and mammary glands. In these organs, HSL is hypothesized to regulate cholesterol and steroid metabolism.

Changes in HSL activity may play a role in the development of heart disease, diabetes, and obesity. Mice that are deficient in HSL have decreased amount of lipolysis and a favorable lipid profile with low triglycerides and LDL cholesterol and elevated HDL cholesterol. However, humans who have decreased levels of HSL are more likely to be overweight or obese. Some people who have lower HSL levels could be the result of an alteration in their genetic code, known as a polymorphism. People with the polymorphism for this gene may be at an increased risk of developing obesity. Altered HSL activity may also play a role in the development of Type 2 diabetes mellitus (T2DM). Patients who are insulin resistant (also known as prediabetic) have decreased lipolysis in response to HSL. This subsequently affects fatty acid levels in the blood and fat storage in tissues. These perturbations in fatty acid metabolism can lead to the development of T2DM.

SEE ALSO: Hormones.

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Human QTLs

HUMAN QUANTITATIVE TRAIT loci (QTL) are pieces of deoxyribonucleic acid (DNA) closely linked to genes that underlie a particular phenotypic trait. Many important human traits (such as blood pressure and body mass index) are of a quantitative nature. It is well established that obesity is under strong genetic control. Body size is an archetypal quantitative trait with variation due to the segregation of many gene loci. Knowing details of QTLs helps us understand the genetics of that trait and identify specific genes associated with that trait. One of the major approaches to detecting obesity genes are whole-genome scans which have been extensively performed, resulting in identification of many human QTLs. As of October 2005, the number of human obesity QTLs is reported to be 253 of obesity-related phenotypes and continues to grow. The obesity gene map shows putative loci on all chromosomes except Y.

The genomic localization of human QTL involves a linkage concept using family-based sampling designs. Localization of human QTLs requires large samples to be rendered successful. The identification of the genes underlying QTLs, however, remains difficult and thus several approaches are used such as genetic and genomic methods that help discover genes that are involved in health conditions such as obesity. One linkage strategy for detection of human QTL involves the use of genetics studies of family members such as the use of sib pairs. Sib pairs with one member sampled from each extreme decile are called ED sib pairs and are used to denote their trait values. If both

members of sib pairs are from the same extreme decile, they are called EC sib pairs.

Several studies performing quantitative linkage analysis studies have found associations between obesity and various genes. Studies suggest that the beta-3 adrenergic receptor is a strong candidate gene for obesity. Other studies using pedigree-based analysis demonstrated a QTL on chromosome 3 (3q27), which is strongly linked to the trait of the metabolic syndrome, comprised of conditions such as insulin resistance and obesity. Other studies have suggested that one or more genes affecting obesity are located in chromosome region 20q13 and chromosome region 10p12. Other genomic regions (e.g., 1p36, 1p31–p21, 2p21, 11q23–24) have been replicated across different studies.

SEE ALSO: Genetic Mapping of Obesity Related Genes; Genetics.

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Humoral Factors and Satiety

THERE ARE A number of cerebral and gastrointestinal humoral (hormone) factors which regulate satiety. These hormones often work together and signal to each other to produce satiation, defined as a feeling of fullness. Studies which examine satiety utilize rodent models, associational data, and human trials. Rodent and human studies often examine the effects of intravenous infusion of these satiety factors, while correlative data often examines differences in circulating levels of satiety hormones in lean versus obese subjects. The following is a review of the most well-established humoral factors involved in satiety.

Alpha melanocortin stimulating hormone (α-MSH): Strong evidence indicates that this neuropeptide is involved in feeding behavior and energy expenditure. α-MSH and drugs able to mimic its action (such as MTII) are able to reduce adiposity and food intake in rodent models. Moreover, humans with mutations which lead to aberrant α-MSH production are obese, suggesting that this peptide is critical in body weight regulation.

Amylin: After food intake, amylin is secreted in tandem with insulin from the beta cells of the pancreas. Studies demonstrate that levels of amylin are increased in obese individuals.

Apolipoprotein A-IV: This hormone is synthesized and released in the small intestine; stimulus for release is fat ingestion. Animal data indicates ApoA-IV infusion reduces food intake.

Bombesin/bombesin-related peptides (ie-gastrin-releasing peptide, neuromedin B, neuromedin C): These peptides are released from the gut in response to food intake. Bombesin injection has been shown to decrease food intake in lean, but not obese subjects. Similar results were observed for gastrin-releasing peptide.

CCK: CCK is a hormone found in the duodenum and jejunum of the small intestine, as well as several different sections of the brain. In the gut, CCK is responsible for gallbladder contraction, secretion of pancreatic enzymes, and inhibition gastric emptying. Dietary fat and protein in the small intestine stimulate CCK release, and levels of this hormone increase 10-30 minutes after meal initiation, and taper off after 3-5 hours. Numerous studies have examined the effect of CCK administration on satiety. When administered alone, CCK has been shown to decrease meal size and duration; however a compensatory increase in meal frequency also occurs. When CCK is administered with other hormones such as ghrelin, leptin, or insulin, the results are more positive. For example, CCK is able to blunt ghrelin's appetite-stimulating effects. Additionally, CCK has been suggested to work synergistically with the hormones leptin and insulin. Associational data indicates that fasting CCK levels are increased in obese women, and decreased in anorexia nervosa, however this data is still preliminary. Potential therapeutic targets utilizing the ability of CCK to act as a satiety factor are underway.

Cocaine and Amphetamine Regulated Transcript (CART): CART is a neuropeptide involved in satiety. Evidence suggests that CART modulates the activity of other metabolic hormones such as neuropeptide Y

and leptin. Furthermore, administration of CART has been demonstrated to transiently inhibit normal and starvation-induced feeding.

Enterostatin: Enterostatin is a bi-product of pancreatic enzyme cleavage, which occurs after a fat-containing meal. Administration via infusion decreases food intake.

Glucagon-like peptide 1 (GLP 1): GLP1 is secreted 5-30 minutes after meal initiation. Like many other humoral factors, it is released based on the caloric content of the meal. GLP1 suppresses the gastric acid and pancreatic juice secretion that usually occurs after meal initiation. Additionally, this hormone reduces gastric emptying time. Levels of GLP1 are reduced in obese subjects, and increase after weight loss. Additionally, intravenous injection of GLP1 in humans has been demonstrated to decrease energy intake and promote weight loss. There is currently a GLP1 agonist (an agonist is able to mimic the action of the endogenous hormone) available commercially (exendin-4) which can be used to increase levels of GLP1 and promote its weight-reducing effects. However, its current intended use is for promoting glycemic control.

Oxyntomodulin (OXM): OXM is released from the stomach in proportion to the energy content of the meal. Particularly, the fatty acids in the meal stimulate OVM's release. Blood levels of this hormone increase after 30 minutes of meal initiation, and remain elevated for several hours after a meal. OXM inhibits gastric emptying and gastric acid secretion, which leads to stomach distention and satiation. OXM administration is also able to inhibit feeding, as well as suppress fasting levels of the appetite-stimulating hormone ghrelin.

Peptide YY (PYY): PYY release is stimulated by food intake, and its release is dependent upon the caloric content and composition (ie-fat, protein, carbohydrate) of the meal. PYY administration inhibits pancreatic and gastric secretion, gallbladder contraction, and gastric emptying. Consequently, it is suggested to act as a satiety signal that terminates feeding and stimulates digestion and absorption. Human data indicates that PYY administration can suppress appetite and food intake in both obese and lean subjects.

Pancreatic polypeptide (PP): PP is a hormone predominantly produced in islet cells of the pancreas. Release of PP is stimulated by food intake (based on the caloric load of meal), hypoglycemia (low blood sugar) and exercise. Other hormones can affect PP release; for example, ghrelin (appetite-stimulating), motilin (stimulates gastric motility), and secretin (controls gastric acid secretion) stimulate PP release, while somatostatin inhibits its release (somatostatin inhibits the release of a number of peptides involved in digestion). PP infusion has been shown to reduce food intake in normal-weight individuals. Research indicates that obese individuals may have suppressed PP levels, however this data has is still preliminary.

Serotonin: Serotonin is a neurotransmitter that also acts as a short-term satiety factor. Serotonin and/or serotonin precursors (tryptophan, an amino acid) have been demonstrated to reduce food intake in rodent models. Additionally, administration of a serotonin agonist was able to block the appetite-stimulating effects of another hormone, neuropeptide Y. A potential downstream target for serotonin is the anorexigenic peptide α-MSH, which reduces feeding. Human studies have demonstrated that fasting and/or "dieting" decreases levels of both serotonin and the serotonin precursor tryptophan. Additionally, obese individuals are found to possess low tryptophan levels; weight loss does not increase these levels. There are a number of drugs currently available/in progress which act to capitalize on the effects of serotonin on satiety. These include: Dexfenfluramine, Fluoxetine, Sertraline, Paroxetine, Femoxitine, DOI, MK212, mCPP, TFMPP, RU-24969, CP-93129, CP-94253, Ro 60-0175, Org 12962, VERT-3323, BTV-933, YM348, Lorcaserin, Sibutramine.

Urocortins/Corticotropin-releasing factor (CRF): Urocortins work together with CRF receptors to contribute to satiation. CRF2, found in the gut and brain, works with urocortins 2 and 3 to promote satiety. Injection of these specific urocortins suppresses feeding in several rodent models; their purported mechanism of action is similar to that of other appetite suppressants.

SEE ALSO: Agouti and Agouti-Related Protein; CART Peptides; Dexatrim; Ghrelin; Hypothalamus; Insulin; Leptin; Melanocortins; Neuropeptides; Neurotransmitters; POMC Proopiomelanocortin; Tryptophan.

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Hunger

HUNGER HAS MULTIPLE definitions; the ones most pertinent to obesity are the physiological definition and hunger within the social context. Physiological hunger is characterized by a sensation of individual discomfort.

The range of hunger experienced may be as mild as a lack of satiety or so severe that one may experience a reduction in energy so limiting that one's ability to perform activities is impaired. The most commonly experienced symptom is stomach pains. In more severe instances, particularly in cases where repeat instances occur, hunger may result in physiological changes brought about by insufficient nutrient intake and lead to anthropomorphic changes, such as decreased body mass indices.

Although the symptoms of physiological hunger may be experienced in response to the anticipation of a desirable meal or the approach of a time during which food is routinely consumed, it is universally experienced 10–12 hours after the last ingestion of food when the stomach is empty and the liver is no longer able to maintain blood glucose levels by its glycogen stores.

In this situation, gluconeogesis signals the hypothalamus, the brain's hunger center, to stimulate the release of the hormone ghrelin into the stomach, which increases the desire to eat and results in the sensation of hunger.

Within its social context, hunger is a lack of food due to limited economic or resource availability. This lack causes individuals to skip meals or eat less than desired. Over time, these conditions can result in malnutrition, poor nutrition, or overconsumption of calories for various reasons, for example, due to the consumption of less expensive, less nutrient-dense food sources or overconsumption on multiple occasions due to the fear associated with hunger experiences on other occasions.

According to the Food Research and Action Center (FRAC), a leading organization in the hunger advocacy and related policy development arena, "Hunger and food insecurity have been growing [in the United States]. Nearly 35 million people—including 13 million children—in the United States were hungry or living on the edge of hunger in 2002 (the most recent year for which data were available). Since 1999, food insecurity has increased three years in a row, in total by 3.9 million individuals—2.8 million adults and more than one million children. Overall, 11.1 percent of U.S. households (12.1 million households) experienced food insecurity or hunger in 2002, and black (22 percent) and Hispanic (21.7 percent) households suffered from food insecurity or hunger at double the national average."

FOOD INSECURITY

National statistics suggested that U.S. food insecurity rates dropped somewhat in 2005, yet, more than 35 million people resided in households considered food insecure. Each year the most recent U.S. hunger data and related statistics are made available in the following reports: FRAC, State of the States; America's Second Harvest, Hunger in America; U.S. Conference of Mayors' Annual Hunger and Homelessness Survey; and, reports from the U.S. Department of Agriculture (USDA) regarding Household Food Security in the United States.

In developing countries, it is not uncommon for hunger to be experienced by individuals to a level where they experience severe malnutrition, multiple clinical symptoms associated with undernutrition or malnutrition, and/or starvation.

Although severe malnutrition and starvation are less frequently associated with hunger in the United States, milder forms of chronic malnutrition are common. Lower rates of more extreme hunger in

Physiologically, hunger is a sensation of discomfort, generally in the form of stomach pains. While the presence of food can activate such a response, in general, the body becomes hungry 10 to 12 hours after the last ingestion of food.

the United States are credited to established federal nutrition programs, for example, the federal Food Stamp Program, that help to provide a safety net for many low-income families. In addition to the physical issues associated with the milder forms of chronic malnutrition in the United States, overweight and obesity, are common among those experiencing hunger in the United States. Overweight and obesity seem counterintuitive in terms of their association with hunger; however, some experts suggest that it is caused by excessive caloric initiating from a variety of factors, that is, individuals overeating when food is available due to fear of impending hunger, the lower cost of high caloric, less nutrient-dense foods, and similar factors.

Whether malnutrition or overnutrition results, even mild forms of hunger have detrimental effects on physical health, psychological welfare, learning, development, productivity, and personal well being. For example, these may include stunted or abnormal growth, poor mental health, poor academic productivity and performance among children, and reduced concentration and ability to sleep.

Within its social context, two terms are closely identified with hunger: food insufficiency and food insecurity. In 1990, Anderson defined food security as "access by all people at all times to enough food for an active healthy life [and] includes, last a minimum, the ready availability of nutritionally adequate and safe foods, and an assured ability to acquire acceptable foods in socially acceptable ways, e.g., without resorting to emergency food supplies, scavenging, stealing, or other coping strategies." When this phenomenon does not hold true for individuals or households, they are considered to be food insecure. Food sufficiency is similar in definition to food security; however, it fails to account for constructs, such as the nutritional value of the food or how it was

obtained. Thus, those who are considered to suffer from food insufficiency are likely to have minimal or no access to food of any kind.

When hunger is defined within the social context, it is measurable at the household, community, national, and global levels. At the household level, hunger is typically evaluated in terms of food insecurity, and households are further classified according to the severity of their circumstances, for example, low food security and very low food security or food insecurity with or without hunger.

There are many measures used to assess household food insecurity and hunger in the United States, but by far the most commonly cited is the U.S. Food Security Survey Module. Two terms currently used to describe food security when this measure is employed are high food security, that is, households that did not answer "yes" to any of the food insecurity questions; and marginal food security, in cases when families answered "yes" to one or two of the food security questions, meaning they have had some difficulties securing ample food. Originally, these families would have still been considered to be food secure.

The terms currently used to define food insecure households are low food security, previously classified as food insecurity without hunger, and very low food security, which was originally referred to as food insecurity with hunger. Those who are categorized as having low food security indicate they have had to make changes in the quality or the quantity of their food, while those defined as experiencing very low food security are those who have struggled with having enough food for the household, and reside in households where adults and/or children have frequently experienced hunger and have frequently engaged in behaviors such as cutting back on the food they eat or skipping meals.

Community assessment of hunger and associated factors is done in a variety of ways, for example, survey assessment, needs or asset mapping, and so forth. Tools available for community assessment are available in the USDA Community Food Security Assessment Toolkit. In the United States, community-level data are collected and published by the Second Harvest National Food Bank Network Agency Survey and from the Task Force on Hunger and Homelessness of the U.S. Conference of Mayors.

SEE ALSO: Food Insecurity and Obesity; Sensory-Specific Satiety.

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Hydrodensitometry

HYDRODENSITOMETRY IS A method for determining percentage body fat. The Archimedes' principle is applied by comparing the mass of a subject in air and underwater. It usually involves the use of a specially constructed tank in which the subject is seated on a suspended chair or placed on a cot attached to a weighing device. Once the body volume (V) of the subject has been determined, body density can be calculated using the mass in air (Ma, kg) and the weight while submerged (Ws, kg) with allowances being made for residual gas in the lungs (Vr) and GI gas (100 mL) and the density of water (Dw) at the temperature of submersion. Density is calculated using the following equation:

$$Db=Ma/\{[Wa-Ws/Dw]-Vr+0.1\}V$$

The value body density is of little use. Instead, it is used to determine percentage body fat. Entering body density into either Brozek or Siri's equations does this. These equations are based on the principle that the average densities of fat mass and lean mass are

0.90 g cm–3 and 1.01 g cm–3, respectively. Brozek's equation states that

$$\% \text{ body fat} = (4.57/Db - 4.142) \times 100$$

and Siri's equation states that

$$\% \text{ body fat} = (4.95/Db - 4.5) \times 100.$$

The equation developed by Brozek for the conversion of body density to percentage body fat was based on the chemical composition of a reference male. It is based on a young, nonathletic adult male.

While hydrodensitometry is considered a reliable technique for determining percentage body fat, there are some problems to consider with the measure. The procedure is not for everyone, because the subject must be submerged; therefore, confidence in water is important. In addition, most tanks are raised above ground level, which makes it difficult for morbidly obese, pregnant, elderly, and disabled subjects to access them.

The measurement is also sensitive to residual air in the lungs and gut; thus, total expiration is necessary. Most measuring devices are sensitive to movement and temperature. Often, multiple measurements are taken. The volume of gas in the intestine is usually included in the calculation as being 100 milliliters, but this volume may need to be increased for large adults and decreased for children. Measurements are most reliable if taken during the fasting state. Dehydration and hyperhydration also cause changes in the body density calculation.

The calculations are based upon the principle that the body consists of two homogeneous components. There are several limitations of this model, however; the main limitation is individual variation in the components of bone and muscle components. Athletes tend to have denser bones and muscles, while the elderly have lesser bone density especially those with osteoporosis. Furthermore, preadolescents have different bone mineral content than adults.

SEE ALSO: Bod Pod and Pea Pod.

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Hypertension

IN THE PAST decade, the prevalence of obesity has dramatically increased. It should be no surprise that the prevalence of hypertension has also increased given the well-recognized association between obesity and hypertension. Approximately 30 percent of the population is hypertensive (defined as a blood pressure [BP] >140/90 mmHg or using hypertensive therapy). Unfortunately, the nature of the relationship between obesity and hypertension is not entirely clear. Therefore, an understanding the development and pathophysiology of obesity hypertension is important for the treatment and prevention of this disorder.

NOT ALL OBESE INDIVIDUALS ARE HYPERTENSIVE BY CLINICAL STANDARDS

Weight gain is almost invariably associated with an increased BP. However, there is substantial variability in the BP response to weight gain among individuals and not all will become hypertensive, at least by the standard of 140/90 mmHg. In addition, weight loss is associated with a reduction in BP in many normotensive obese individuals. Therefore, BP is higher in obese humans than would be achieved at a lower level of adiposity. It is important to emphasize that any elevation in BP above optimal levels (i.e., below 120/80 mmHg) will increase an individual's risk of developing cardiovascular diseases. In turn, reductions in BP from above optimal levels should confer a health benefit.

MECHANISMS LINKING OBESITY WITH HYPERTENSION

Obesity-related hypertension is similar to other forms of hypertension in terms of abnormal kidney function due to increased tubular sodium reabsorp-

Hypertension, or high blood pressure, is one of the most common medical problems linked to obesity.

tion, which causes sodium retention and expansion of extracellular and blood volumes. The increase in sodium reabsorption results in a rightward shift in the renal pressure-natriuresis relation and BP elevation. Thus, higher levels of BP are required maintain sodium and fluid homeostasis in obese individuals. The results of animal and human studies suggest that activation of the SNS, sympathetic nervous system, and renin-angiotensin system play an important role in the development of obesity hypertension.

CONSEQUENCES OF OBESITY HYPERTENSION

Target organ damage is a common consequence of obesity hypertension. For example, cardiac dysfunction and left ventricular hypertrophy are common in obese individuals. In addition, obesity and hypertension worsen the degree of left ventricular hypertrophy in a synergistic manner, which translates into a greater risk of congestive heart failure. There is also evidence that obesity is associated with peripheral and coronary endothelial dysfunction. The increasing prevalence of chronic renal disease has also been linked to obesity. This is not surprising as two of the most common causes of renal failure, hypertension and diabetes, are closely associated with obesity. In fact, even mild to moderate forms of obesity are associated with renal disease and weight loss is associated with improvements in renal function.

TREATMENT OF OBESITY HYPERTENSION

Weight loss is considered the most effective nonpharmacological therapy for lowering BP in obese hypertensives; however, future long-term studies are required to fully understand the impact on long-term weight loss on BP. Regular physical activity and sodium restriction are also effective in lowering BP and should be included as part of the comprehensive treatment of hypertension.

There are currently no specific recommendations for the pharmacological treatment of obesity hypertension. However, therapy should be based on etiology of the disorder. As such, pharmacological blockade of the sympathetic nervous system and the renin-angiotensin-aldosterone system are logical choices for intervention. Unfortunately, there is little direct clinical evidence to justify their specific use in this patient population. As emphasized in the Seventh Joint National Committee on Prevention, however, good clinical judgment is paramount in the selection of hypertension therapy.

PREVENTION OF OBESITY HYPERTENSION

There is no sign that the rising prevalence of obesity seen over the past two decades is dwindling. The average weight gain of the population in the United States is estimated to be about two pounds per year. Weight gain is almost invariably associated with an increase in BP. Thus, prevention of weight gain should be a primary therapeutic target for reducing the problem of hypertension.

CONCLUSIONS

There is a continued problem of weight gain and obesity in the United States and most industrialized countries with no clear dwindling of this trend in sight. Because obesity is a major cause of essential hypertension, rising BP and its associated comorbidities will continue to impart their health and economic consequences. The prevention of weight gain and its metabolic and cardiovascular sequelae should be a focus of future efforts to combat the growing epidemic of obesity hypertension.

SEE ALSO: American Medical Association; American Obesity Association; Autonomic Nervous System.

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Hypertension in African Americans

HYPERTENSION (HTN) IS high blood pressure (BP). African Americans (AAs) have the highest prevalence of HTN in the world. HTN is seen in 41 percent of adult AAs compared with 27 percent of Caucasians. It occurs at an earlier age, is more severe, and has more complications than in Caucasians. Obesity increases the BP in these patients, and weight loss significantly decreases their BP. The prevalence of obesity is 28 percent in AA men, 50 percent in AA women, 27 percent in Caucasian men, and 30 percent in Caucasian women.

Systolic blood pressure (SBP) is the pressure in arteries while the heart is beating; diastolic blood pressure (DBP) is the pressure while the heart is resting between beats. It is measured in millimeters of mercury (mmHg). HTN is defined as SBP above 140 and/or DBP above 90 (above 140/90 mmHg). No one knows what causes elevated BP in 95 percent of the patients with HTN. It is one of the biggest mysteries in medicine. Hypertension is probably caused by many mutations (abnormalities) in several genes. In addition, environmental factors, such as diet and exercise, affect BP.

HTN is a major reason why heart disease mortality is 50 percent higher, stroke is twice as common, and hypertensive kidney disease is four times as common in AAs. Scientists do not know why HTN in AAs is different from HTN in Caucasians. There are some gene mutations seen in hypertensive AAs that could explain part of this difference. Salt raises BP and causes fluid retention. The BP in AAs is more sensitive to salt in the diet than in Caucasians. The kidneys of many hypertensive AAs do not excrete salt in the urine as efficiently as the kidneys of many Caucasians. The reason for this is unclear. Furthermore, obesity is thought to exacerbate the effects of salt sensitivity in all populations. In 2000, there was no significant difference between the prevalence of overweight (body mass index [BMI] >25 kg/m2) or obesity (BMI above 30) in AA and Caucasian men. But AA women had a much higher prevalence of overweight (77.3 vs. 57 percent) and obesity (49.7 vs. 30.1 percent) than Caucasian women. There is some evidence that the arteries in many AA hypertensives are more sensitive to hormones and other chemicals in the body that cause constriction of the arteries and less sensitive to those that cause dilation of arteries.

Social factors contribute significantly to the prevalence of HTN in AAs. One in four AAs lives in poverty, compared with one in 12 Caucasian Americans. Poverty is a strong risk factor for HTN in many populations. Poverty is associated with poor nutrition, obesity, increased alcohol and tobacco use, and increased stress, all of which increase the risk of developing HTN. The diet of poor persons is often low in fruits, vegetables, and milk, which are rich in potassium and calcium. Diets high in potassium and calcium lower BP. Moderation of alcohol intake also lowers BP. Long-standing social stress may also be a significant factor in the development of HTN.

The HTN in AAs can almost always be brought under good control. That is, 140/90 mmHg, except if diabetes or kidney disease is present; then the goal BP is 130/80. Lifestyle modifications can significantly lower BP, including weight reduction, a diet rich in fruits and vegetables, reducing salt intake, exercising at least 30 minutes most days of the week, and limiting alcohol consumption to two drinks for men and one drink for women per day. If lifestyle modifications are not enough, medications can almost always bring the blood pressure down to goal with few side effects.

SEE ALSO: Hypertension; Hypertension in Asian Americans; Hypertension in Hispanic Americans

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Hypertension in Animal Models

OBESITY AND HYPERTENSION are closely related in humans. In most cross-sectional studies, body mass index (BMI) and systolic blood pressure are highly correlated. In fact, BMI is more closely correlated with blood pressure than any factor other than age and family history.

In animal models, the relationship between obesity and hypertension is not as clear. Species that become spontaneously obese, such as pigs, do not show increases in blood pressure with increasing weight. Genetically obese mice and rats do not have elevations in blood pressure except in association with renal disease. Part of this may be because leptin increases blood pressure by activating the sympathetic nervous system. In genetic models of obesity, the capacity to make leptin or respond to it is usually lost, in contrast to obese humans who have high levels of leptin and are capable of responding to the hormone.

Dietary obesity in animals is often associated with increased blood pressure. Feeding dogs two pounds of lard in addition to their regular diet induces hypertension as well as weight gain. Diets high in sucrose or fructose elevate blood pressure in rats and mice, which is sometimes associated with moderate weight gain. For dietary obese models, it is not clear that the weight gain causes the hypertension. Instead, the increased intake of saturated fat and sugars may raise blood pressure along with body weight. A similar conundrum arises in human studies, where it is difficult to separate the influence of obesigenic diets from the effects of obesity itself.

Another approach to experimental models of obese hypertension is to induce hypertension and obesity independently, because increased body weight by itself is not sufficient to evoke hypertension in species other than humans and possibly nonhuman primates. A genetic model that arose spontaneously is the spontaneously hypertensive obese (SHROB) or Koletsky rat in which an obesity-causing mutation arose spontaneously in a colony of spontaneously hypertensive rats (SHR). The combination of obesity and hypertension leads to severe organ damage and an early death from renal failure. Genetically hypertensive animals are usually resistant to dietary obesity, so data on the synergy between dietary obesity and genetic or experimental hypertension are limited. The induction of experimental hypertension by renal surgery or mineralocorticoid excess causes weight loss in animals, limiting the availability of models. Thus, the leading models of obese hypertension are the lard-fed dogs as a model of hypertension induced by dietary obesity and the SHROB model of genetic hypertension and obesity.

SEE ALSO: Hypertension; Hypertension in African Americans; Hypertension in Children; Hypertension in Hispanic Americans; Hypertension Pharmacotherapy.

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Hypertension in Asian Americans

HYPERTENSION (HTN) IN Asian Americans is especially important, because they are the fastest growing racial population in the United States in terms of percentage. Asian Americans are defined as "any of the original peoples of the Far East, Southeast Asia, or Indian subcontinent." From 1990 to 2000, the

population of Asian Americans increased by 48 percent (overall U.S. rate was 13 percent). In 2003, Asian Americans made up 5 percent of the U.S. population. Between 2003 and 2050, the population of Asian Americans will increase by 213 percent (overall U.S. rate 49 percent). That will be 33.4 million, 8 percent of the U.S. population. Central obesity is an important risk factor for coronary heart disease in South Asians. Obesity increases the blood pressure (BP) in Asian Americans, and weight loss decreases their BP. Hypertension occurs at lower levels of obesity in South Asians than in Western populations.

HTN is one of the strongest risk factors for coronary heart disease and stroke, two of the leading causes of death in all U.S. populations. Higher salt intake is associated with higher BP in China. In China, salt intake is higher than in the West. Stroke is more common in Asian Americans than in Caucasians. Higher salt intake is one of the most important risk factors for their higher stroke mortality rate than seen in the West. HTN is a greater risk factor for stroke in Asian Americans than in Caucasians, with diastolic BP (DBP) being particularly implicated.

The prevalence of HTN (BP above 140/90 or taking HTN medication) varies among the Asian American populations. A study of Chinese living in San Francisco found a prevalence of HTN of 69 percent. Another study of Asian Americans in northern California found the prevalence of HTN to be 22 percent in Chinese, 26.9 percent in Filipinos, 21.7 percent in Japanese, and 18.6 percent in "other Asians." Among Koreans in Maryland, 32 percent had HTN. In comparison, in 2004, the prevalence of HTN in the United States was 29.3 percent. A study found that Filipino men and women had higher BPs than other Asian American groups.

In San Francisco in 2005, of the Chinese Americans with HTN, 41 percent were taking antihypertensive medication, and only 6 percent of them had their HTN under control (BP below 140/90). In Maryland in 2001, of the Korean Americans (aged 60–89) with HTN, 33.6 percent were taking antihypertensive medication, and 7.5 percent were under control. In 2004, the overall U.S. treatment rate for hypertensives was 53.7 percent, and control rate was 33.1 percent.

There are little data comparing the response to antihypertensive medications of Asian Americans and Caucasians. One study found that Chinese were more responsive to one BP medication than Caucasians. Another study found that the response of persons from the Indian subcontinent to HTN medications was similar to that of Caucasians.

These data demonstrate the need for more education of Asian Americans regarding the importance of HTN and its control. It also shows the need for more effective treatment of HTN by the physicians caring for this population.

SEE ALSO: Hypertension.

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While the Asian-American population is diverse, hypertension is, in general, an increasing problem across the ethnic group.

Hypertension in Children

HYPERTENSION, OR ABNORMALLY high blood pressure, is a serious medical problem in children. Children who are overweight are at increased risk for hypertension. Hypertension must be treated to avoid permanent health problems. Weight reduction, physical activity,

and dietary changes are often used as first-line treatments that can be supplemented with antihypertensive pharmacological agents as needed.

Hypertension can be classified as primary (also known as essential) or secondary. Essential hypertension is a diagnosis of exclusion (i.e., there is no clear underlying medical cause) that is associated with several risk factors, including obesity. Secondary hypertension is the direct result of another condition, most often renal, endocrine, or cardiovascular disease. The subcategories of pediatric hypertension include prehypertension, stage 1 hypertension, and stage 2 hypertension; the percentile cutoff of blood pressure for each category is based on the child's sex, age, and height. The prevalence of pediatric hypertension is approximately 1 to 5 percent. On average, blood pressure appears to be rising; from approximately 1990 to 2000, blood pressure in children increased 1.4 mmHg in systolic measurement and 3.3 mmHg in diastolic measurement.

In children under age 10, the majority of cases of hypertension are secondary to an underlying condition such as renal parenchymal disease or renal vascular disease. Other causes include endocrine abnormalities, coarctation of the aorta, and the use of certain medications. In adolescents, most cases of hypertension are primary, rather than secondary. Risk factors in adolescents for primary hypertension include family history of cardiovascular disease and high body mass index.

Hypertension often presents with no visible symptoms. If present, symptoms can be vague and may include irritability, headaches, and sleeping difficulties. The American Heart Association recommends that all children aged 3 and older have yearly blood pressure measurements. For a proper diagnosis, it is important that the child avoid intake of stimulant (such as caffeine) before the measurement. When the measurement is taken, the child should be calm, with a steady pulse in the normal range. A blood pressure cuff of appropriate size must be used to obtain an accurate measurement. If hypertension is suspected, the measurement should be repeated twice during that visit, and on at least one other visit for confirmation.

Childhood hypertension contributes to early development of cardiovascular disease. Long-term complications from hypertension include stroke, heart failure, renal failure, and retinopathy. Maintaining a healthy weight, eating a low-fat and low-sodium diet, and engaging in regular physical activity may help to prevent pediatric hypertension. The first line of treatment for primary hypertension is lifestyle change, which can include weight reduction, dietary modification, and physical activity. Antihypertensive medications are suggested for patients who do not respond to lifestyle changes, have evidence of end-organ damage, or have stage 2 hypertension. There are a variety of medications approved for use in children, including thiazide diuretics, angiotensin-converting enzyme (ACE) inhibitors, beta-blockers, calcium channel blockers, and angiotensin-receptor blockers. Secondary hypertension may resolve simultaneously if the disease causing it is treated.

As the rate of childhood obesity increases, so too does the incidence of obesity-related illnesses like hypertension.

SEE ALSO: Child Obesity Programs; Hypertension.

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Hypertension in Hispanic Americans

HYPERTENSION (HTN) IS high blood pressure (BP), above 140/90mmHg. Hispanics are the largest and fastest growing ethnic minority in the United States, increasing from 14.5 million in 1980 to 37.4 million in 2002. Hispanic is not a racial term, but it refers to a diverse ethnic group, comprised of people from different cultures and ancestries. The two major subgroups of U.S. Hispanics are those from Mexico and Latin America, living mainly in southwest states, and those from the Caribbean, living mainly in Florida and the northeastern states. Most available data involve Mexican Americans, the largest group. HTN is seen in 25.1 percent of Mexican Americans, 27.4 percent of Caucasians, and 40.5 percent of African Americans. Obesity increases the BP in these patients, and weight loss significantly decreases their BP. The prevalence of obesity in Mexican Americans is 29 percent in men and 40 percent in women. In Caucasians, it is 27 percent in men and 30 percent in women.

In 2000, 16 percent of Caucasians and 19 percent of Mexican Americans in San Antonio, Texas, had HTN. A study in Mexico City found that 19 percent of Mexicans there had HTN. In a study in San Antonio, Mexican Americans had higher all-cause, cardiovascular, and coronary heart disease mortality than Caucasians. HTN is a major risk factor for cardiovascular disease.

There are little data on the prevalence of HTN in Caribbean Hispanics. A study in northern Manhattan found that the prevalence of HTN was 58 percent in Caribbean Hispanics compared with 43 percent in Caucasians. They were 62 percent Puerto Rican, 12 percent Cuban, and 12 percent from other Caribbean islands and South America.

The causes of the differences in prevalence of HTN between Hispanics and Caucasians is unknown. Most of the differences are due to socioeconomic factors, and the importance of genetic factors is unknown.

In 2002, 49.8 percent of Mexican Americans who had HTN were aware of it. Of those with HTN, 34.9 percent were being treated for HTN, and only 17.3 percent had their BP under control (below 140/90).

The BPs of Hispanic Americans with HTN can be brought under control with a combination of lifestyle changes and medication. Decreasing salt intake, restriction of alcohol, losing weight if overweight, and regular exercise can significantly lower BP. BP medications are effective in Hispanic Americans with HTN. Some barriers to treatment have been identified, including language problems, poverty, and lack of access to primary care physicians. Many Hispanic men seek a machismo image in which admission of an illness and a need for treatment is a sign of weakness and is discouraged. Also, many Hispanic families have a strong sense of fatalism, feeling that "It is God's will and no treatment will make much difference." Initiatives must be designed to reduce these barriers and increase education about the importance of HTN in our Hispanic communities.

SEE ALSO: Hypertension.

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Hypertension Pharmacotherapy

OBESITY IS STRONGLY associated with hypertension. American Heart Association guidelines call for prompt antihypertensive treatment in patients with blood pressures over 140/90, regardless of body weight. Weight-loss programs are not a substitute for pharmacotherapy of hypertension. Following success in weight loss and maintenance, blood pressure medications may be tapered or discontinued with close monitoring.

Diuretics, especially thiazides, are the most commonly recommended first-line therapy for hypertension. Reasons for favoring thiazides include proven reductions in mortality and cardiovascular events, low cost, and the ability of diuretics to be given with almost any other agent as part of combination therapy. For obese patients, diuretics may be favorable because expanded plasma and body water compartments are common in the obese and may contribute to weight-related elevations in blood pressure. Thiazides are not effective against obesity-related edema, but can act synergistically with other diuretics to reduce excess fluid accumulation. Unfortunately, diuretics also have unfavorable metabolic effects such as elevating glucose and lipids. Diuretic use is associated with an increased risk of diabetes. For this reason, the dose of thiazide may be kept low (25 mg or less) and a second potassium-sparing diuretic may be added to maintain electrolyte balance.

Beta-blockers are also commonly recommended by expert panels on similar grounds to the diuretics. Beta-blockers are usually indicated after a myocardial infarction, because they reduce mortality. Unfortunately, beta-blockers have unfavorable effects on glucose and lipid metabolism and may favor weight gain through slight reductions in metabolic rate and lipolysis in adipocytes. In addition, beta-blockers can exacerbate asthma. For these reasons, beta-blockers are less commonly used in obese patients.

Angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers are increasingly favored as first-line treatment for hypertension, commonly in combination with a low dose of thiazide. In contrast to diuretics and beta-blockers, these agents have favorable effects on metabolic profile. Patients receiving ACE inhibitors and angiotensin receptor blockers are at reduced risk of developing diabetes. Furthermore, they reduce the risk of diabetic and hypertensive kidney disease. These agents may be particularly appropriate for use in obese hypertensives.

Calcium channel blockers are possibly the most effective agents for lowering blood pressure, and are widely used as antihypertensives. The metabolic profiles of these agents are mainly neutral, with neither negative nor favorable effects. Unfortunately, edema is a significant side effect to which obese patients may be particularly susceptible.

SEE ALSO: Hypertension; Hypertension in Animal Models; Hypertension in Asian Americans; Hypertension in Children; Hypertension in Hispanic Americans.

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Hypothalamus

THE HYPOTHALAMUS IS at the base of the brain, above the pituitary gland. The hypothalamus governs higher autonomic control, including mechanisms that maintain constancy of the internal environment. Included are controls of food intake and energy expenditure, making the hypothalamus important for regulating body weight and adiposity.

The hypothalamus is part of the diencephalon. It lies beneath the thalamus and borders the third cerebral ventricle. The hypothalamus is divided into nuclear areas distinguishable by cell appearance and neuropeptide and neurotransmitter contents. Hypothalamic nuclei and other brain areas communicate via complex neural circuits. There are also neural connections between the hypothalamus and the posterior lobe of the pituitary gland, and vascular connections with the anterior lobe of the pituitary gland. Thus, distinct populations of hypothalamic neurons are positioned to influence neural activity in other brain regions, as well as the release of pituitary hormones, and govern body temperature, "appetitive" processes such as food and water intake and sexual behavior, defensive reactions, and various endocrine and activity rhythms.

The hypothalamus receives nutrient, neural, and hormonal inputs indexing whole-body energy status, integrates these signals, and coordinates central nervous system and peripheral mechanisms that defend energy balance. In a simplified model, for example, the arcuate nucleus is a main receiver

of energy signals. The arcuate nucleus contains two neuron types with receptors for both leptin and insulin, hormones that indicate, respectively, fat mass and levels of blood glucose. One neural population makes proopiomelanocortin (POMC), precursor of alpha-melanocyte-stimulating hormone, which stimulates melanocortin-3 and -4 receptors, resulting in decreased food intake and increased energy expenditure. Food deprivation or underweight decreases, and overconsumption or overweight increases POMC. Other arcuate neurons make neuropeptide Y (NPY) and the melanocortin-3 and -4 receptor inhibitor agouti-related protein (AgRP), which increase food intake and decrease energy expenditure. Food deprivation and underweight increase NPY and AgRP, and overconsumption and overweight decrease them.

The NPY/AgRP and POMC neurons project to the paraventricular nucleus (PVN), lateral hypothalamic area (LH), and other hypothalamic nuclei with receptors for these neuropeptides. PVN is important in anorexigenic/catabolic signaling, whereas the LH is involved in orexigenic/anabolic signaling. The PVN contains multiple neuronal subtypes that make oxytocin, corticotrophin-releasing hormone, or other neuropeptides that lead to reduced food intake or increased energy expenditure. The LH contains neurons that produce either orexins or melanin concentrating hormone, which increase feeding. PVN and LH projections to the pituitary gland and other brain sites mediate these effects on energy balance.

SEE ALSO: Agouti and Agouti Related Protein; Appetite Control; Central Nervous System; CNS/Hypothalamic Energy Sensing; Leptin; Melanocortins; Neuropeptides; NPY (Neuropeptide Y); Obesity and the Brain; Pituitary Gland; POMC (Proopiomelanocortin).

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Hypothyroidism

THYROID HORMONE, MADE in the thyroid gland located in the neck, is an important regulator of the body's metabolism. Hypothyroidism refers to a lack of thyroid hormone, usually from underproduction. It can result in myriad symptoms, including weight gain or obesity, loss of muscle mass, cold intolerance, constipation, changes in menstruation, depression or slowed thinking, fatigue, congestive heart failure, dry skin, and more. Hypothyroidism is more common in women and in the elderly. Estimates of the prevalence of hypothyroidism vary throughout the world, but it may affect as much as 10 percent of older women in the United States.

Normally, the production of thyroid hormone is very tightly regulated by complex feedback loops. The pituitary gland regulates thyroid gland hormone production by altering levels of thyroid-stimulating hormone (TSH) in response to stress and levels of thyroid hormone in the blood. In turn, the thyroid gland uses iodine to make two different forms of thyroid hormone—T4, a storage form, and T3, the active one—which is released into the bloodstream. An increase in TSH should make the thyroid gland produce and release more of the hormone. However, in most cases of hypothyroidism, the thyroid gland is unable to respond. In developing countries, this is often a result of iodine deficiency; in the developed world, destruction of the gland by autoimmune disease, radiation, trauma, or infiltrative processes is more commonly the culprit. With any of these causes, the TSH level will continue to climb unless thyroid hormone is replaced.

Less commonly, hypothyroidism can be caused by pituitary problems and underproduction of TSH. In these cases, the thyroid gland is normal but simply has not received the signal to make hormone. The TSH may be normal or even low in the face of low T4 and T3 blood levels.

On examination, people with untreated hypothyroidism may appear normal or have coarsened facial features, dry hair and skin, rashes, swelling of the feet, and obesity. The thyroid gland may also be enlarged, resulting in a goiter. The most sensitive test for diagnosing hypothyroidism, except in cases of pituitary disease, is measurement of TSH. Low levels of free T4 confirm the diagnosis. Hypothyroidism,

whether from thyroid or pituitary disease, is readily treated by replacement of thyroid hormone in pill form.

SEE ALSO: Thyroid Medications.

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Immigration and Obesity

IMMIGRANTS COMPRISE A large and growing portion of the U.S. population. The term *immigrants* usually refers to first-generation immigrants or people who were born outside the United States. However, there are also second-generation immigrants (those born in the United States with at least one foreign-born parent) and third-generation immigrants (born in the United States of one or more second-generation immigrant parents).

Historically, many immigrants have been born in countries where the prevalence of obesity is lower than it is in the United States. Thus, upon arrival in the United States, the majority of immigrants have had a lower body weight than native-born Americans. However, with time, immigrants and their subsequent generations of children have tended to have increased rates of obesity that meet and even surpass those of their native-born counterparts.

Although this entry focuses on U.S. immigrants, developed countries all over the world, including Canada, Britain, Austria, Germany, Sweden, Australia, and Israel, have experienced a similar pattern of healthy-weight immigrants growing overweight and obese at a rate beyond that of the native born. In addition, developing countries have been undergoing a massive internal migration of people moving from the poor rural areas to urban areas with similar results. Because the immigrant population is the fastest-growing population segment in the United States and other developed countries, and is significant in developing countries as well, addressing the issue of obesity in immigrants is of increasing consequence worldwide.

WHO ARE IMMIGRANTS?

According to the U.S. Census Bureau, in 2003, the U.S. population consisted of 33.5 million foreign born, representing 11.7 percent of the population. (In comparison, according to the United Nations, there were a total of 91 million immigrants in high-income developed countries, and 51 million in low- and middle-income countries in 2005.)

U.S. immigrants are a heterogeneous population. The largest group (53.3 percent) were born in Latin America (comprised of Central America, the Caribbean, and South America). The second largest group (25 percent) were born in Asia. The remainder hail from Europe and other regions of the world. Mexico has been the top-sending country in contemporary times, and has been the wellspring for approximately one-third of all immigrants arriving in the United States each year since 2000. Historically, most immigrants have settled in large metropolitan areas and in California and New York. However, the immigrant population is becoming more dispersed, with small towns and suburbs and states such as Texas, Florida,

By definition, immigrant populations are incredibly diverse. Similarly, rates of obesity in immigrants have a wide range.

New Jersey, Illinois, Massachusetts, and North Carolina absorbing an increasing share.

Most of the existing U.S. data on obesity represent immigrants of Hispanic origin because they comprise the largest and fastest-growing proportion of immigrants in the United States. However, data on Asian Americans are increasing.

OBESITY RATES IN IMMIGRANTS

There is considerable variation in overweight and obesity rates among immigrant groups according to country of origin, race, length of time in the United States, generation, age, and gender. For example, over 73 percent of Mexican-American adults are overweight and 37 percent of Mexican Americans are obese. On the other hand, the percentage of Asian Americans classified as obese is very low, with East Asian (Chinese, Japanese, Korean) and Southeast Asian female immigrants having the lowest rates of obesity or overweight: only 9 percent of Vietnamese or Chinese women are overweight, but 25 percent of Asian Indian women are overweight. Vietnamese men have a low prevalence of overweight (17 percent) compared with Japanese (42 percent).

However, weight is not always an accurate reflection of body fat composition or distribution, both of which influence obesity-related risk of disease. This is important in relation to Asian immigrants because they tend to have a higher percentage of body fat per body weight, and for Asian and Hispanic immigrants because they tend toward upper-body obesity. Indeed, at comparable body weights to Caucasians, Asians have higher rates of obesity-related diseases; they are twice as likely as Caucasians to have Type 2 diabetes despite their lower rates of overweight and obesity. Therefore, the known obesity rates of Asians may not be meaningful. The World Health Organization has suggested different criteria for measuring overweight and obesity in Asians. However, research to date has not been conclusive in suggesting different criteria for Asians.

Very little is known about obesity among black immigrants, who may include people from a variety of African, Caribbean, and South American cultures, but are often not distinguished from American-born blacks known as African Americans. The overall obesity rate for non-Hispanic blacks is thought to be approximately 30 percent for men and 50 percent for women. There is some evidence that black immigrants tend to follow the immigrant pattern of lower rates of obesity upon arrival and increasing rates with duration of residence.

OBESITY IN CHILDREN AND ADOLESCENT IMMIGRANTS

Obesity and overweight have been increasing among people of all ages, races, and ethnicities; however, the rate of increase among immigrant children in the United States is particularly alarming. Overweight children are at higher risk for Type 2 diabetes and other conditions that raise their risk for cardiovascular disease, and are more likely to become overweight as adults. Hispanic children are twice as likely to be overweight as non-Hispanic Caucasian children, with the rate among 2- to 5-year-olds as high as 35 percent.

On the other hand, Chinese, Vietnamese, Hmong, and Pacific Islander immigrants have the fastest-growing rate of overweight and obese children. Adolescents fare just as badly; according to a national survey, 30 percent of Hispanic adolescents are obese, compared to 21 percent of Asian Americans and 24 percent of Caucasian non-Hispanic adolescents. Among this age group, variations also occur; for example, the obesity rates among Chinese adolescents is 15 percent and 19 percent among Filipinos.

FACTORS AFFECTING IMMIGRANT OBESITY

As is the case with the overall population, many factors have been associated with overweight and obesity in immigrants. These include age, sex, marital status, socioeconomic status (education, income, employment status), health behaviors (diet, physical activity), and access to healthcare. For immigrants, the latter three are the most salient and are intertwined with each other and with acculturation.

Acculturation—changes in immigrants' behaviors, beliefs, habits, and values toward the host society's cultural norms—generally increases over time and with each subsequent generation. It has become associated with obesity rates in immigrants, because the prevalence of overweight and obesity also increases the longer immigrants live in the United States.

The overall obesity rate for immigrants who have been living in the United States less than one year averages about 8 percent. This rate rises slowly for the first 10 years after immigration, but by 15 years, it jumps to 19 percent, except in foreign-born Blacks. Other data demonstrate a remarkable fourfold increase in the risk of obesity in longer-term immigrants (15 years or more) compared with immigrants of 5 years or less. The pattern intensifies with each generation, with a striking increase in overweight between first- and second-generation immigrants. For example, Asian-American and Hispanic adolescents born in the United States (second generation) are more than twice as likely to be obese than are first-generation adolescent immigrants.

Acculturation implies that immigrants are choosing to trade their active lifestyles and traditional foods—which are generally low in fat and sugar and high in whole foods such as grains, fruits, and vegetables—for a sedentary lifestyle and high-fat, high-sugar, calorie-dense foods. However, one must also consider the impact of changing living conditions inherent in immigration, and interactions with socioeconomic status (SES).

SES may exert its influence on health behaviors related to obesity in many ways. SES may create unique conditions relating to access issues, as a large proportion of immigrants are of low SES. Nearly half of immigrants live in central cities and most of the remaining live in metropolitan areas, which tend to have a high proportion of people living in poverty. This has implications for both eating and activity patterns because many immigrants may lack access to healthy foods and places to be physically active.

Researchers have found that acculturation generally worsens immigrant diets. Healthy foods such as fresh vegetables are more difficult to find in poor urban neighborhoods, compared with affluent areas. People living in low-income neighborhoods generally have poor access to stores that sell healthy foods such as fresh vegetables and easy access to fast-food restaurants, and the cheaper, more accessible foods tend to be energy-dense, high-fat, and high-sugar goods. Other factors affecting immigrants' diet include lack of familiar foods in their new country; lack of familiarity with the community; language barriers; memories of hunger, deprivation, and malnutrition before migration; high-caloric foods equated with status; and traditional plant-based diet equated with poverty.

Acculturation appears to have a rather mixed effect on physical activity. In one study, Hispanic women who were less acculturated had a 58-percent rate of inactivity compared with 28 percent for those who were more acculturated, and other studies show that Asian females become more active with acculturation. Yet, the physical environment in the United States, which depends on cars for transportation, does not encourage the walking and bicycling that is common in immigrants' countries of origin. For example, rural immigrants routinely walked to an open market every day in their birthplace, but in the United States, they make weekly trips to a supermarket. In addition, people living in low-income and high-minority neighborhoods have reduced access to physical activity facilities due to lack of transportation, lack of facilities or programs, personal or neighborhood safety concerns,

and cost. Rural minority women are more likely to be completely sedentary than their urban counterparts, and this may be due to a lack of access to exercise programs and facilities.

Immigrants face more barriers to quality healthcare and are less likely to receive preventive care than are native-born Americans, making obesity prevention even more urgent. Although acculturation appears to play a strong role in obesity, there is controversy over the way acculturation is defined, the methods used to measure it, and the interplay between acculturation and SES. Interventions will need to be culturally specific if they are to be effective and encourage immigrants to maintain the healthy aspects of their cultural traditions while adopting healthy aspects of Western life.

SEE ALSO: Ethnic Disparities among Obesity in Women; Hispanic Americans; Obesity and Socioeconomic Status; World Patterns.

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Implications of Gestational Development

CONCEPTION IS THE result of intricate biology, but the intricacies of biology do not end at that point as the growth and development of the fetus continues within the woman's body for nine months. As the fetus is not a parasite, these required nutrients come directly from the mother's nutrient intake; therefore, proper gestational development of the fetus is directly dependent upon the intrauterine environment that is provided by the mother, and risk of heart disease, diabetes, hypertension, and other health problems during adulthood have roots linked to maternal nutrition during pregnancy.

When maternal intake is unable to facilitate its own survival, thus falling below optimal levels, fetal growth and development are compromised more than maternal health. In general, nutrients will first be utilized by the maternal biology to facilitate her health and physiological changes and then for the placental development and finally to the fetus. To adequately provide the developing fetus with its nutritional requirements, changes occur in the maternal body composition and functions in a very specific sequence as the successful completion of each change depends on the one before it. Critical periods, which are preprogrammed time periods during development when specific cells, organs, and tissues are formed and integrated and functions established can be drastically influenced by the nutritional status of the mother. For optimal growth during these times, it is essential that certain nutrients be available in specific amounts. These critical periods are one-way routes; any errors or lack of development during these periods are irreversible and so cannot be corrected.

Both deficits and excesses in nutritional status during the critical periods can lead to lifelong defects in organ and tissue structure and function. Deficits and adverse prenatal environmental conditions induce adaptive changes that are permanent, promoting survival in the short term, but increasing vulnerability to later environmental stimuli, leading to obesity. Data have shown that maternal undernutrition during pregnancy leads to intrauterine growth restriction in the offspring, who then plays catch-up after weaning and develops metabolic abnormalities later in life including obesity, hyperinsulinemia, and hypertension.

The purpose of developing the ability to store fat was biologically and historically one of adaptation. During times of food shortage, it would be adaptive to be able to store fat for times when food was unavailable. However, in the present day and environment where food shortage is rare, this adaptive feature has turned maladaptive. Many animal models have been widely used as a model to decipher how intrauterine

restriction affects obesity and one such study concluded that growth-restricted rat pups demonstrated significant hypoglycemia and hypotriglyceridemia at birth and due to their catch-up growth period, they developed adulthood obesity with evidence of hyperglycemia and insulin resistance. However, if the catch-up period was delayed, those animals resulted in normal adult body weight, hyperglycemia, and postnatal insulin insufficiency. Protein restriction during gestation results in growth retardation which persists even if a normal diet is introduced after birth but if normal diet is introduced during the period of lactation, the pup regains weight, although the accelerated body weight gain continues and leads to obesity. This information can be a useful tool as it points to the lactation period during which interventional means can be implemented to gain proper weight.

Epidemiological studies have further shown that fetuses exposed to famine during pregnancy became adults with higher rates of intraabdominal adiposity than those exposed to undernutrition later in pregnancy. Furthermore, the catch-up period, if followed right after this deficiency, produced an increased likelihood of developing insulin resistance and central obesity. This is of escalated concern as intraabdominal obesity is linked to an increased risk of cardiovascular disease and Type 2 diabetes. Both the type of dietary restriction during pregnancy as well as the period during which malnutrition occurred are critical for programming adult obesity.

Gestational diabetes affects women who previously did not have diabetes but rather develop it during pregnancy and its prevalence is steadily increasing along with obesity. It is the second leading complication of pregnancy. Although controversial, the cause of this specific diabetes is considered to be a form of noninsulin-dependent diabetes, known as Type 2 diabetes. During pregnancy, maternal metabolism adjusts to provide nutrition to both the fetus and the mother by the increased production of hormones such as human chorionic somatomammotropin (HCS), bound to a free maternal serum cortisol, estrogen, and progesterone which all help to increase production and secretion of insulin as well as enhance insulin sensitivity.

Fetal glucose levels are directly proportional to maternal glucose concentrations, but insulin is not because it does not cross the placenta. Since blood glucose crosses the placenta but insulin does not, the fetus's pancreas must compensate by producing more insulin to prevent metabolic abnormality. The fetal liver responds to rising insulin levels by increasing glucose production and uptake and the conversion of glucose into triglycerides. The mechanism by which fetal insulin secretion affects growth is anabolic accretion of fat, muscle, and bone and may program metabolic adaptations, which may increase the likelihood that issues such as diabetes and obesity will develop later in life. Maternal hyperglycemia leads to excess fetal insulin, itself a growth hormone for the fetus and so offspring of mothers with gestational diabetes have higher birth weights.

For the first nine months of development, the fetus undergoes incredible growth and this is solely influenced by the environment the mother provides the fetus via the intrauterine environment.

SEE ALSO: Infant Weight Gain and Childhood Overweight; Low Birth Weight.

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Implications of Restriction of Foods on Child Feeding Habits

AS MORE CHILDREN are becoming overweight at school age and even preschool age, researchers have focused their attention on the feeding strategies of parents as one possible factor affecting weight problems in children. One theory holds that parents, by restricting their children's access to unhealthy foods, impair the children's ability to regulate their own intake, leading paradoxically to overweight children. Indeed, for some children, parental restriction seems to be associated with disordered self-regulation and even increased weight. However,

this finding is inconsistent and varies depending on age, gender, and ethnicity.

Left to their own devices, infants and young children are able to follow internal cues of hunger and satiety in order to regulate their intake appropriately as young as 6 weeks of age. As they become older, external factors in the environment can influence or even replace this internal feedback mechanism. These factors may include the availability and accessibility of foods, modeling of dietary patterns by parents or others in their vicinity, and conditioning, either intentional or accidental, by caretakers.

The tendency for parents to try to influence the amount that infants eat is one explanation for the well-known fact that breastfeeding infants are less likely to become overweight; if parents cannot see how much milk an infant takes, they are less likely to push the infant to take a little more. A recent study demonstrated that this tendency explains some, but not all, of the protective effect of breastfeeding.

In some settings, a parent's tendency to control what a child eats clearly affects both the kinds of food a child prefers and the amount of calories a child consumes. Increased pressure to eat fruits and vegetables, for example, may make these foods less desirable and decrease intake, while restriction of sweets may make them seem more desirable and increase intake in the long term. Repeated exhortations to "clean your plate" or limiting access to food when a child is hungry can teach a child to ignore his or her own internal signals and instead eat food based on availability. In today's environment of cheap, easily available, energy-dense food, such behavior can easily lead to overweight.

The connection between restriction of food by parents and increased energy intake in children, and in some cases between increased energy intake and overweight, has been shown for mothers and daughters in several settings. Of note, the majority of these mother and daughter pairs are middle class and white. The findings have not held true for boys nor have they held true for other ethnic or racial groups. Some large studies of mixed ethnic populations have also not found any association.

Complicating this picture is the question of causation. While it may be true that a controlling parental style can lead to an overweight child, it is also certainly true that a parent's concern about a child who is overweight (or underweight, for that matter) can lead to a controlling style. Because these relationships can influence each other, separating out which is the initial cause can often be difficult.

It is not surprising that children may lose their ability to regulate their food intake based on internal hunger cues as they age. Most adults are poor regulators of food intake based on hunger cues alone as well. Indeed, in most cultures, food serves a social function much greater than simply that of providing sustenance. The importance of culture in mediating behaviors surrounding food may explain at least in part the wide variation in effects of food restriction based on gender and ethnicity.

The complicated relationship between restriction, food preferences, and energy intake creates some difficulty in terms of treating the overweight child. While it is important that a child learns to adjust his or her own intake, there is no available evidence that teaching these skills alone will lead to weight reduction or even weight maintenance in an overweight child. Many current nutrition experts advocate the "division of responsibility" in feeding—that is, the parents decide what kinds of food are available and the child decides how much of each food he or she wants to eat. This approach is predicated on the notion that the parent will provide healthy foods from which the child may choose. Again, evidence is lacking regarding the effectiveness of this approach. Nor does it address the question of the "unhealthy" foods that are widely available outside the home—if they are not available, will that be perceived as restriction by a child, and if so, will that create even more desire for the unavailable food? Further research is needed to sort out these issues as well as the overall impact of parental feeding restriction over time.

SEE ALSO: Accessibility of Foods; Changing Children's Food Habits; Dietary Restraint; Energy Density; Food Intake Patterns; Food Preferences; Food Reward.

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Impotence

OBESITY CAN ADVERSELY affect sexual function and reproductive success, in both men and women. A study of women with an abnormal female sexual function index (FSFI) compared to women with a normal FSFI when matched for age and menopausal status found that obesity adversely affected sexual function. All women were free of diseases known to alter sexual function. Body mass index (BMI) correlated with sexual function but not waist-to-hip ratio. Pain during intercourse and desire did not correlate to BMI, but arousal, lubrication, orgasm, and satisfaction did correlate. FSFI was lower in overweight women and cholesterol and triglycerides were higher. Thus, being overweight can affect female sexual function and this is generally correlated with an increased BMI.

Erectile dysfunction (ED) is self-reported by one in five older males. It is especially common in Hispanic males and African-American males and those with a history of diabetes, obesity, smoking, and hypertension. Not all men seek treatment, but if they did, the cost is high for treatment pharmacologically.

Studies suggest that higher LDL cholesterol and triglyceride levels, regardless of the presence of metabolic syndrome, are associated with lower testosterone levels. BMI is also inversely correlated with testosterone levels across men. When the data were examined as a whole, diabetes, fasting glucose greater than 110 mg/dl, BMI greater than 30 kg/m, and triglycerides of 150 mg/dl or greater correlated with lower testosterone. These men are likely to have elevated estrogen-to-testosterone ratios as well.

A study of men 40 years and older who completed the International Index of Erectile Function revealed that 40- to 60-year-old men's central obesity did not correlate with ED. In men older than 60 years, ED correlated with waist–hip index, waist–thigh index, sagittal abdominal diameter, sagittal abdominal diameter–height index, and maximal abdominal circumference. Therefore, central obesity, also known as the "apple shaped" body type, is a significant factor in ED for men over 60 years old.

Looking at the topic from the other side, ED is a predictor of metabolic syndrome. Using data from the Massachusetts Male Aging Study, a population sampled 1987–89, 1995–97, and 2002–04, and metabolic syndrome as defined by the Adult Treatment Panel Guidelines III, ED predicted metabolic syndrome in men with a BMI of 25 or lower. The clinical significance is that the presence of this chief complaint should lead to investigations regarding early signs, symptoms, and physiological changes suggestive of obesity, metabolic syndrome, cardiovascular disease, and diabetes so often seen with this condition.

Another cause of ED is Peyronie's disease (PD). In 206 male patients with diabetes, 20 percent had PD. The risk factors for PD include age, obesity, and smoking. Diabetes should be added to the list of known risk factors. The pathogenesis is likely due to plaque deposition in the blood vessels of the penis. Such plaque is most commonly seen in the obese or men at risk for diabetes, central obesity, and metabolic syndrome.

Obesity affects sexual function in both women and men. In women, it is correlated with an increased BMI. In men, ED is correlated with decreased testosterone, serum lipid and glucose values, increased central obesity with increased age, metabolic syndrome, and Peyronie's disease. The effect of obesity on declining sexual function occurs by several pathways.

SEE ALSO: Body Mass Index; Central Obesity; Estrogen Levels; Hormones.

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Impulsivity

IMPULSIVITY IS A psychological condition that manifests itself in destructive behavior that is repeated uncontrollably. Binge-eating disorder is a recent clinical finding related to impulsive behavior that has shown increasing prevalence as the incidence of obesity has continued to rise. Treatments include selective serotonin reuptake inhibitors and cognitive behavioral therapy.

Impulsive behavior occurs quickly and spontaneously with impaired inhibition of an act that is harmful to the individual or to others. This impulsive act is performed repetitively, with little thought involved. The behavior begins with impulsivity (arousal or tension) that precedes the committing act. The compulsive drive releases this tension during the act itself. This experience perpetuates itself as a short-term gain of gratification followed by a larger, long-term negative consequence. These behaviors can interfere with personal relationships, family ties, and career performance.

The disorder has been classified under a spectrum of impulse control disorders (ICDs) since the early 1990s, which in itself is a large component of obsessive-compulsive spectrum disorders (OCSD). This category of OCSD is distinct from obsessive-compulsive disorder (OCD), which manifests itself as an anxiety disorder, whereas OCSD (at least initially) ensues as a pleasurable experience.

Binge-eating disorder is similar to bulimia nervosa in that a binge generally occurs in a short amount of time, occurs discreetly away from others, and the food consumed has a far higher caloric content than a typical meal. Those with either disorder eat when they are not physically hungry and generally consume food until they are overly satiated. The main difference is that those with bulimia nervosa will attempt to forcefully remove the ingested food from their bodies by vomiting, laxatives, or intense physical exercise, and those with binge-eating disorder will not.

Binge-eating disorder has gained recent notoriety due to the obesity epidemic and is subsequently being more frequently evaluated by mental healthcare providers. Obesity is not necessarily a feature of this disorder; however, the obese population may have the highest prevalence of binge-eating disorder with rates found to be as high as 30 percent in weight-loss clinics compared to 2 to 3 percent in the general population.

While tests do not test for impulsivity alone, they can determine basic identifying characteristics that may suggest issues problematic for the patient. The SCOFF (SCOFF is a mnemonic for the key word in the five questions: Sick, Control, One, Fat, and Food) questionnaire and the Eating Disorder Screen for Primary Care (ESP) are two short surveys that can screen those in need of further evaluation. The gold-standard assessment is the Eating Attitudes Test (EAT), which has 90 percent accuracy in determining eating behaviors and attitudes.

Pharmacological treatments vary considerably in different ICDs. Selective serotonin reuptake inhibitors (SSRIs), primarily fluoxetine and fluvoxamine, are generally used across the board with varying effectiveness in most ICDs. Mood stabilizers, primarily lithium and carbamazepine, are often also used in disorders such as pathological gambling, intermittent explosive disorder, and compulsive-impulsive shopping. Other drugs commonly used in ICDs include opioid antagonists, such as naltrexone.

Another suggested genetic physiological component between obesity and impulsivity is a melanocortin-4 receptor gene mutation. It was found that of the population study (24 patients), all of the carriers of the gene had episodes of binge eating. Less than 15 percent did not have the mutation, but still had binge eating.

Nonpharmacological treatment for impulsive behavior includes cognitive behavior therapy (CBT). This focuses on three main components: determining the initial behavior trigger, changing the behavior, and evaluating consequences. The patient can determine the causes by writing down events as they transpire and the surrounding circumstances, which is essential for behavior modification. Examples of behavior modification for the obese can be as simple as limiting locations of eating to drinking water between each bite. Finally, to reinforce the remodeled behavior, positive rewards (other than food) are utilized.

Combining CBT with a medication can increase the success rate of the treatment. Serotonin reuptake inhibitors are the most commonly prescribed drugs and the most effective pharmacotherapy for impulsive behavior. However, when this drug is

stopped, the condition will relapse without other treatments, so a multidisciplinary approach will be most effective.

SEE ALSO: Neurotransmitters; Obsessive-Compulsive Disorder; Psychiatric Medicine and Obesity.

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Inaccessibility of Exercise

IT IS WIDELY accepted that widespread physical inactivity plays a role in the current high prevalence of excess weight. Physical activity, or exercise, may both prevent and reverse obesity. In addition, even when little or no weight loss occurs, regular physical activity can mitigate many of the detrimental health conditions associated with obesity. Unfortunately, most people do not get enough exercise even when they understand its benefits and would like to be more active. Studies have found that inaccessibility of exercise programs and places to exercise is a major barrier for many people. Exercise may be inaccessible for many reasons including lack of convenience, awareness, affordability, and appropriateness for age, culture, or ability.

National and international organizations such as the Centers for Disease Control and Prevention and the World Health Organization recognize the issue of exercise accessibility because improving access has the potential to influence entire populations, not just individuals. Public health is undergoing a paradigm shift toward approaches that promote physical activity by addressing structural and design factors in the environment, as well as personal and social factors. Along these lines, places to exercise include not just athletic facilities, health clubs, and sports fields. Current thinking encompasses a myriad of ordinary public places—parks, streets, trails, schools, waterfronts, and even indoor staircases—as places where people can be physically active regardless of income or ability. It also broadens the scope of exercise beyond push-ups and tennis to include such everyday activities as walking and bike riding for transportation as well as recreation, gardening, playing catch, and taking the stairs instead of the elevator. Such activities can be integrated into daily life more easily and less expensively than trips to the gym. Evidence is building that improving the actual and perceived availability, awareness, and appeal of public spaces would enable many more people to engage in "active living."

Some segments of the population such as children, minorities, immigrants, people of low socioeconomic status, seniors, and people with activity limitations such as mobility disabilities and visual impairments are at particular risk for obesity as well as insufficient access to exercise. Therefore, increasing the accessibility of places to be physically active would benefit everyone, especially those who are currently at a disadvantage. For example, schoolchildren would benefit from more and better sidewalks, shorter distances to schools, and less trafficked streets, enabling a greater number to walk or bike to school. Communities with low incomes and ethnic disparities would benefit from free or low-cost exercise and recreation facilities including trails and parks, especially if culturally appropriate activities were featured.

As research continues to fine-tune knowledge about the role and characteristics of accessibility, efforts are already under way to act on the knowledge that already exists. Such efforts encompass a range of actions, from simply posting reminder signs to employees to take the stairs, to fundamental changes in policies and practices that govern the places where people can be more active. Some communities are implementing existing policies such as enforcing the mandated amount of physical activity required in school by hiring or training more staff and providing safe attractive school yards. Some are improving existing policies such as incorporating more bicycle lanes of a design that is more parent/child/senior friendly. Still others are enacting new policies such as guaranteed funding for parks or legislation giving tax breaks to employers who provide workplace fitness programs and facilities. These and other efforts, it is hoped, will combine to create activity-friendly communities that provide for more accessible exercise and thus increased physical activity.

SEE ALSO: Safety of Urban Environments.

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Income Level

INCOME LEVEL IS a term generally derived from personal income, or assessed via income proxies, to provide demographic information about individuals and/or groups. High, medium, and low income levels reflect varied levels of affluence and educational attainment, and therefore are contributing factors to socioeconomic status. Lower income levels have been found to be associated with increases in obesity and comorbidities such as diabetes, hypertension, and high serum cholesterol.

Income level has been negatively correlated with obesity; lower income people have higher rates of obesity.

In the least complex accounting terms, income from which income level is derived is all money earned during a specified period of time. The Bureau of Economic Analysis defines sources of personal income as wages, salaries, employee compensations, and supplements fewer contributions for government social insurance, adjusted proprietors' income, rental income, interest and dividend income, and government social benefits. However, the U.S. Census Bureau measures money income, defined as earnings before taxes that do not include the value of noncash government social benefits such as food stamps, Medicare, Medicaid, public housing, and employer-provided fringe benefits, and uses money income to calculate per capita, real median income and household incomes. Nonregular income, from bartering to illegal activities, is not definable for the purposes of data collection and statistics, even though it may contribute to income levels.

Researchers working with various groups sometimes choose to assess "resources" as income, and thus choose to include government benefits, for example, social security income, food stamp benefits, and so forth, in income determinations. Median and household incomes that take into account annual changes in the consumer price index are available to discern the general economic well being as well as the number of people in varied income levels per region.

The minimum income level below which a person is officially considered to be lacking in adequate sub-

sistence and to be living in poverty is annually calculated and published in the *Federal Register* by the Department of Health and Human Services. This calculated value is referred to as the poverty level, poverty threshold, or poverty line and is used for qualifying individuals within families with an income deficit for various government social benefits. The U.S. Census Bureau publishes the absolute poverty threshold and other income levels, for example, 125 percent poverty level, by sex, age, and race by state, county, and so forth. It is important to note that these calculations do not vary geographically and are therefore insensitive to cost-of-living differentials.

To compare households' incomes to the federal poverty threshold, family unit size and annual income must be discerned. Researchers should take note that in lower income households, family unit size varies radically and its discernment can be a difficult due to the inherent complexity of defining "household member" in populations with a high prevalence of extremely varied intergenerational households and shared living arrangements, for example, residence in group housing situations or shelters. Furthermore, self-reported income figures can be difficult to collect and/or discern due to inherent complications, for example, weekly variations in income, the embarrassment some individuals experience in revealing their income, and so forth.

Because income level can be difficult to collect, various other proxies have been used in its stead. These include variables such as education, educational degree type, job type and industry, dwelling size, landholdings/landlessness, ownership of various resources such as cars or refrigerators, or in developing countries, possession of livestock or plumbing, and so forth. Because income levels are used for qualifying individuals within families with an income deficit for various government social benefits, participation in such programs is a popular income level proxy. For example, incomes at or below 130 percent of the poverty level qualify individuals/households for participation in the Food Stamp Program or for free-meal eligibility in Child Nutrition Programs; incomes at or below 185 percent of the poverty level qualify individuals/households for participation in Women, Infants, and Children (WIC) or for reduced-priced meal eligibility in Child Nutrition Programs.

Alternatively, resources are often used as income-level proxies. Thus, the term *limited-resource audiences* is often used synonymously to describe low-income communities. Limited-resource audiences have been defined as "those whose income and other combined available assets, such as savings, credit availability, and real and personal property (e.g., real estate, furnishings, vehicles, etc.), are sufficiently low that, if other requirements unrelated to their assets are not taken into account (e.g., residency and/or citizenship) they and/or their family members would be eligible for one or more public assistance programs, such as the Food Stamp Program, the Special Supplemental Program for Women, Infants, and Children (WIC), Head Start, and Child Nutrition Programs. Individuals and/or families whose income and/or assets are slightly above this qualifying level may still be considered to be limited-resource individuals if their assets remain scarce and they continue to reside in an area where limited-resource individuals are prevalent."

Regardless of the income level assessed, this socioeconomic variable has been repeatedly identified as a significant variable in relation to overweight and obesity, both in terms of obesity's epidemiology as well as many pertinent related variables, that is, the foods individuals and group eat and the leisure time physical activities in which they engage.

SEE ALSO: Food Stamp Nutrition Education Program; Head Start.

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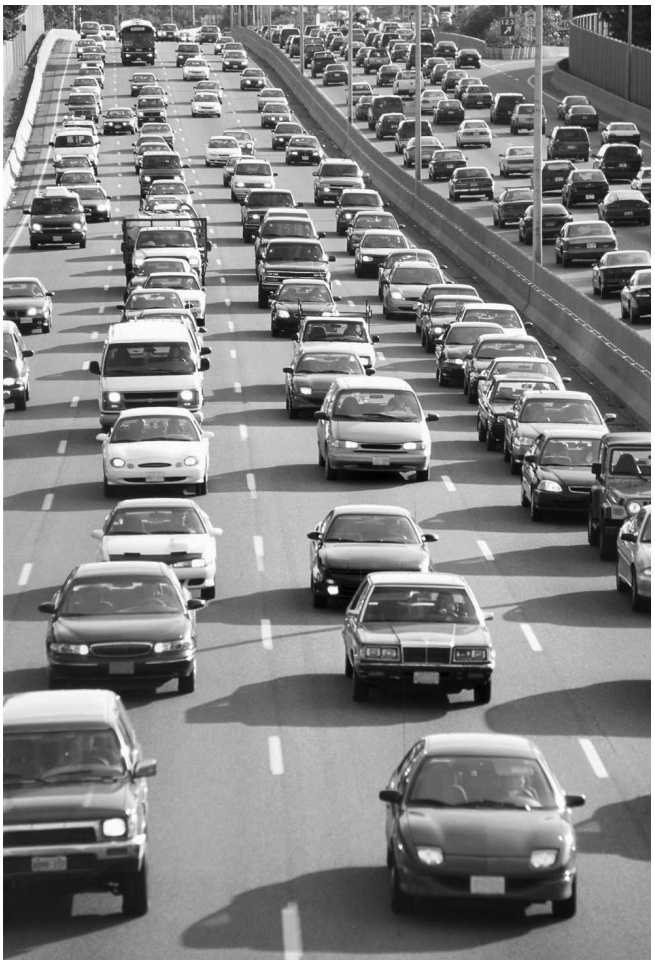
Increased Reliance on Automobiles



Automobile reliance has grown and has decreased the amount of exercise the typical American gets.

THERE ARE MULTIPLE, interrelated reasons for the obesity epidemic. One causal factor is believed to be the increase of dependence upon the car for mobility. With ever-increasing rates of vehicle ownership, expanding cities, and expanding waistlines, healthcare researchers and urban planners are extensively examining the link between the physical and social environments and obesity.

There is great debate about the true scope of the role of automobiles in causing America's weight gain. Some claim that people living in disadvantaged urban areas (which have the lowest rate of vehicle ownership) have the highest risk of obesity; others claim that lifestyles attached to suburban sprawl are key factors. These claims are not necessarily in conflict with each other; they are merely different viewpoints within the obesity discourse. Regardless of demographics and geographies, the automobile impacts lifestyle choices, which in turn affects daily decisions and options for mobility.

To understand how the automobile became an agent of obesity, one must know a little about how cities and society have changed in the past several decades. Before World War II, most Americans did not live in urban areas. Those who did were concentrated primarily in city centers. In general, these centers were traversed by foot, bicycle, and bus to access commercial needs and employment opportunity. Because of economics as well as the point in history, vehicle ownership was low as America emerged from the Great Depression. The onset of World War II created an economic boom. Many women went to work for the first time to fill the employment gap left by men serving in the military. As GIs returned from the war, the savings, incomes, and financial incentives to purchase new suburban single-family homes created major population shifts. Families left the city centers and countryside to populate the new communities. These changes encouraged and required the purchase of a family car, the construction of the highway system, and reduced the need for public transportation among the emerging middle class. Many suburban communities did not provide bus lines that linked directly to the employment centers. This forced workers to become car dependent and reduced the exercise associated self-propelled forms of mobility. At the same time, lifestyle changes also increased the demand for vehicle ownership. For example, as women increasingly entered the workforce, households required an additional vehicle for commuting to work. With the increase in disposable income, travel for shopping and recreation also increased, and dining out became a form of entertainment. All the while, bedroom suburban communities continued to grow.

Since the 1960s, the number of people per household has decreased, while the number of cars per household has increased. The number of American drivers has also increased along with the length of vehicle trip and number of miles driven per year. Overall, Americans spend more personal time driving, and they travel farther each year. Likewise, American work hours and commuting time are increasing. In 2005,

the U.S. Census Bureau reported that Americans spent more than 100 hours commuting to work each year. While 76 percent of to/from work trips were by driving, only 15 percent of car travel was commuting to and from work. Automobiles cannot be blamed as the only, or even primary, cause of obesity; however, the reliance can easily translate to decreased physical activity, less time for physical activity, and increase of carryout meal consumption.

The automobile reliance is a choice for many, whereas it is a requirement for others. Factors that impede other mobility options as well as encourage obesity include physical environment design issues (such as sidewalks, bicycle lanes, and play spaces), area safety, access to fast-food drive-through lanes, and lifestyle choices that demand more time spent driving because of distance between home, work, retail, and so forth. Even in suburban communities where physical distance is not a major factor, Americans still largely rely on the automobile to travel for ease, comfort, and time saving. Today's schoolchildren are more likely to be driven to school by parents rather than walk or ride bicycles. Without enough physical playtime, this decrease in activity is also causing a rise in childhood obesity.

While researchers continue to examine the factors surrounding car ownership and obesity, it is safe to make certain logical assumptions. Automobile ownership or access decreases the physical activity associated with walking and bicycling as travel modalities. Although many individuals will replace this lost activity with other forms of exercise, many others will not by choice or lack of time. Even as cities continue to expand outward, more communities are being designed to promote healthy lifestyles by reducing the need to depend upon an automobile for all trips.

SEE ALSO: Eating Out in the United States; Exercise; Fast Food; Income Level; Safety of Urban Environments.

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Increasing Portion Sizes

OVER THE LAST 30 years, portion sizes of many food items available in restaurants, fast-food establishments, and supermarkets in the United States have increased. This increase has paralleled the rise in the rate of obesity over the same time. Increased intake of calories from larger portions may be one cause of the obesity epidemic.

At its most basic level, obesity is caused by an energy imbalance. When energy intake (the number of calories ingested as food) exceeds energy expended (the number of calories used during exercise or other metabolic processes), the excess energy will be stored as fat. Large portion sizes can increase energy intake directly because they contain more calories, but also indirectly because they encourage people to eat more. When individuals monitor their food intake, many focus on the content of the food (i.e., how much of the food comes from protein, carbohydrates, or fat), but fewer pay attention to the portion size.

Although they may not be aware of it at the time, people given larger portion sizes generally increase their caloric intake. In both laboratory and real settings, adults given varying portion sizes of food will consume more calories when given the larger portion and will not adjust their intake at the following meal. This effect is seen regardless of age, gender, or current body weight.

Evidence for the increase in portion sizes comes from many locations. Portion sizes from restaurants and food manufacturers have been found to be almost uniformly higher than recommended Food and Drug Administration (FDA) and United States Department of Agriculture (USDA) standards, sometimes by as much as seven times. This increase in availability of larger portions began in the 1970s and accelerated in the 1980s. Consumer survey data also shows increasing portion sizes for items such as soft drinks, beer, wine, and cereal. Companies have increased product to the point where the smallest size currently available is often larger than the only size offered 30 years ago.

Several approaches may be helpful in slowing or reversing these trends. Individuals may be taught how to estimate appropriate portion sizes themselves, or be taught to pay closer attention to internal cues of satiety. Governments or businesses may make serving

size information more available, or discount or reward reasonable portion sizes. Another approach might be to encourage intake of large portions of foods such as fruits and vegetables. Because of high water content, these foods have lower energy density (the number of calories per given weight of food) and may create feelings of fullness while decreasing the total energy intake during a meal.

SEE ALSO: Accessibility of Foods; Dietary Restraint; Economics of Food; Energy Density; Food Intake Patterns.

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Indirect Calorimetry

INDIRECT CALORIMETRY IS a method used to determine how many calories a person is using during different physical conditions. This is a useful technique because the amount of calories a person uses will vary from one person to the next based upon physical characteristics such as body weight and percent body fat. Indirect calorimetry works by measuring the amount of oxygen a person consumes and carbon dioxide the person produces. Additionally, the nitrogen content in the urine is calculated to determine the total number of calories utilized. Indirect calorimetry differs from direct calorimetry, which measures the amount of heat released from the body. Scientists convert information about the heat that is released from the body to calories based upon several principles from physics. Direct calorimetry is difficult to measure; it is both expensive and laborious and thus indirect calorimetry is the preferred method to measure how many calories are used by certain activities. Other measurements to assess the caloric needs of an individual are based upon equations that estimate an individual's age, height, weight, and gender. These assumptions are less accurate than indirect calorimetry.

For research purposes, indirect calorimetry can be used to measure how many calories are used during sleeping, resting, response to exercise, or a 24-hour period. These different terms apply to different types of calories utilized. Sleeping indirect calorimetry measurements can estimate how many calories are utilized while a person is sleeping. Resting (sometimes referred to as basal, fasting, or postabsorptive) indirect calorimetry measurements can estimate that amount of calories used by the body to maintain all essential life processes (i.e., breathing and heart rate). Response to exercise indirect calorimetry measurements can estimate the amount of calories utilized during physical activity (i.e., running or swimming). Finally, the 24-hour indirect calorimetry measurements can estimate the total amount of calories a person uses during the course of a day (all activities).

Indirect calorimetry collects breath samples to determine the oxygen consumption and carbon dioxide production. The breath samples may be collected through several different methods including a facemask for calories used during exercise, a large plastic hood for calories used during resting conditions, and a human respiratory chamber (sometimes called a metabolic chamber) to measure sleeping or 24-hour energy expenditure. Each of these methods has its advantages and disadvantages. The use of a facemask will most accurately assess how many calories a person is using during exercise. However, a person must wear this mask while performing his or her exercise and needs to be near an analyzer to determine oxygen and carbon dioxide levels. This can limit the testing to riding a stationary bike or running on a treadmill, and cannot be used for hiking or swimming.

Additionally, some people find the mask uncomfortable. The large hood for measuring calories during resting conditions is much more comfortable than the mask used during exercise, but this test is limited to a person lying in bed. Therefore, it cannot be used in many experimental settings. A human respiratory chamber is an effective way to measure the total amount of calories utilized over a certain time period, usually 24 hours. These chambers are sealed off from the outside environment and a person re-

mains inside for an entire test period. The chamber is normally equipped with all of the items a person needs for a 24-hour period including a bed, toilet and sink, exercise equipment, telephone, and television. Patients are normally instructed to follow certain protocols while inside a chamber. They spend a certain amount exercising, resting, eating, and doing light activities (i.e., reading or watching television). Analyzers in the chamber measure the amount of oxygen and carbon dioxide in the room. This test reflects more free living than the other methods described and may better calculate the total number of calories used over the course of a day. These chambers are very expensive and laborious to maintain and can be found only in research institutions.

Indirect calorimetry may also be used in a clinical setting to assess the caloric needs of patients in hospitals. Different disease states will alter a patient's caloric requirements and it is important to accurately assess their requirement from a normal recovery. For example, a burn victim would need to increase the amount of calories he or she consumes. However, it might be difficult for the clinician to determine the exact amount of calories the patient needs because of the severity of the burn. Bedside indirect calorimetry machines are utilized in hospitals to accurately determine the caloric requirements of an individual.

SEE ALSO: Caloric Restriction; Metabolic Rate.

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Infant Growth Rate

GROWTH IN INFANCY follows a pattern which is influenced by multiple factors including genetic such as mid-parental height, low birth weight, calorie intake, and psychosocial factors such as availability of food. Infants' energy and successful development is dependent on good nutrition in the first year of life. Infancy is a period marked by rapid physical growth and brain development. The energy required to support these processes is extraordinary. Undernutrition in infancy can lead to later cognitive delays. Infancy is divided into several stages, with physical growth, developmental achievements, nutrition needs, and feeding patterns varying significantly in each. The most rapid changes occur in early infancy, between birth and 6 months of age. In middle infancy, from 6–9 months, and late infancy, from 9–12 months, growth slows but is still rapid. Healthy full-term infants who are fed on demand usually consume the amount they need to grow well. Feeding infants is much more than simply offering food when they are hungry, and it serves purposes far beyond supporting their physical growth. Feeding provides opportunities for emotional bonding between the parent and infant. When feeding their infant, parents strengthen their sense of what it means to be a parent.

PHYSICAL GROWTH

Infants lose approximately 6 percent of their body weight after birth because of fluid loss and some breakdown of tissue. They regain their birth weight by 10–14 days after birth. Typically, infants double their birth weight by 4–6 months and triple it by one year. On average, infants gain 5–7 ounces per week, or 26–30 grams per day in the first 4–6 months and 3–5 ounces per week, or 17–18 grams per day. Infants gain 3–4 ounces per week and 12–13 grams per day and from 9–12 months 2–3 ounces per week or 9 grams per day. Infants usually increase their length by 50 percent in the first year, but the rate of increase slows down during the second half of the year. From birth to 6 months, infants gain approximately an inch a month, and from 6–12 months of age, they gain about 1/2 inch each month.

Growth rates of breastfed and formula-fed infants differ. Breast-fed infants grow more rapidly in the first 2–3 months but less rapidly from 3–12 months of age. Infants who are genetically determined to be tall but

who are born short may experience catch-up growth during the first 3–6 months. There has been some suggestion in the literature that rapid periods of catch-up growth may not be a good thing, as during these times, infants may be more likely to add fat, as opposed to muscle. Rapid early growth rate has been associated with increased risk of obesity in adulthood. However, infants who are genetically determined to be short but who are long at birth tend to maintain the same rate of growth for several months and then experience a lag-down ingrowth. To meet growth demands, infants require a high intake of calories as well as adequate intake of fat, protein, vitamins, and minerals. From birth to 6 months of age, infants require about 108 kcal/kg of body weight per day. By 12 months, this need decreases to 100 kcal/kg of body weight per day. Thirty percent of calories should be obtained from fat to meet the demands of growth and development. Vitamin and mineral needs are usually met if the full-term infant is breastfed by a well-nourished mother or receives correctly prepared infant formula.

A variety of factors can influence the rate of growth of an infant, including genetics, caloric intake, and availability of food.

NUTRITION

The World Health Organization supports breastfeeding infant as the best feeding method for achieving optimal growth. Feeding is crucial for developing a healthy relationship between parents and infants. At 4–6 months old, infants are developmentally ready to eat supplemental foods. As infants grow, their ability to digest a greater volume and variety of foods increases. By 6 months old, an infant's digestive system has matured enough to allow the absorption of more complex foods.

For infants who are not breastfed, formula is recommended for the first year and then all infants should be on whole milk until two years because of the need of extra fat for brain growth. Juice should not be given to infants before 6 months old. Juice should be given in a cup and limited to 4–6 oz a day. Infants over 6 months should receive or drink fluoridated water; or for those who drink bottled water, a brand in which fluoride is added at a concentration of approximately 0.8 to 1.0 mg/L (ppm) is recommended. Infants 6 months and older who receive breast milk or infant formula prepared with water that is severely deficient in fluoride (less than 0.3 ppm) require fluoride supplementation. Introduce a cup for drinking at 6 months and wean infants from the bottle by 12–14months.

CONCLUSION

Assessing growth alone is not enough to adequately evaluate an individual's health status. Growth references are among the most commonly used and most valuable tools for assessing the general well-being of individuals, groups of children, and the communities in which they live, and for tracking progress in reaching a range of health. The value of growth references resides in the fact that numerous physiological processes must proceed normally and many needs must be met in fetal life and childhood if growth is to proceed normally. Normal physical development is a necessary aim of any strategy that includes aspects of well being. The marked vulnerability of the health of infants and young children also makes assessments of child growth a "sentinel" indicator in evaluations of the health and socioeconomic development of communities in which they live.

Children who are not meeting physical or developmental milestones should be screened early so that the appropriate interventions are initiated. The Centers for Disease and Prevention growth charts are important in evaluation of infants in relation to patterns of growth.

Infants who cross two lines in any direction for weight, height, or head circumference should have a complete assessment including nutritional intake, psychosocial, and behavior including sleep history and temperament. Calculating weight to height ratios is imperative in all children, not just those who are at the extreme ends of the growth chart. Problems with energy balance should be suspected in any child with a weight-to-age ratio below the 3rd percentile, and above the 97th percentile on standardized growth charts.

SEE ALSO: Infant Weight Gain and Childhood Overweight.

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Infant Weight Gain and Childhood Overweight

IN LIGHT OF the increasing prevalence of overweight children and a lack of effective interventions, it has become necessary to consider early prevention strategies. Understanding the implications of infant weight gain on childhood overweight (and the development of later disease as well) is one area studied for its applicability to early prevention and making evidence-based public health recommendations surrounding infant feeding practices.

To understand the relationship of weight gain in infancy and overweight in childhood, normal developmental growth patterns must first be considered. Peak growth rates vary for different organ systems. Neural tissue, including the brain, primarily grows during the prenatal period and extends to early childhood, while linear bone growth is fastest in childhood. The peak growth rate for reproductive organs is during puberty. Adiposity increases rapidly during the first year of life, declines during early childhood, and increases again at puberty. Interruption of normal growth during any stage of infancy and childhood may adversely affect organ system development, especially the organ system that is expected to grow rapidly when an interruption occurs.

Researchers have noticed that there are predictable patterns for infant growth over time. Nearly half of all infants will grow along the same centile (i.e., percentile, which indicates the baby's weight relative to all other babies of the same age and gender) as their weight at birth. These infants will follow their genetic growth trajectory and their adult height will be most closely associated with the height of their parents. About 25 percent of infants will grow faster than the average child, so that if they start out at the 10th percentile, they might reach the 50th percentile by 12 months to 2 years of age (also known as upward centile crossing or accelerated growth). Many infants exhibiting accelerated growth were born small for gestational age (SGA); thus, some researchers have referred to this phenomenon as "catch-up growth." The remaining 25 percent of infants, often those with increased adiposity at birth, will grow slower than the average child. This is referred to as downward centile crossing or decelerated growth.

Multiple studies have demonstrated that rapid weight gain in the first few years of life is positively related to childhood and adulthood obesity. The risk of childhood and adult obesity was found to increase two- to threefold in these infants. In fact, it is now well established that the trajectory and pace of growth from conception through adolescence is associated with the development of obesity and related chronic illnesses in later in life. This evidence has given rise to the theory of developmental plasticity, which posits that during critical periods (e.g., in utero) the environment affects gene expression. Accordingly, a developmental model for human disease suggests that undernutrition in a developing fetus has lifelong consequences to the individual by altering hormones, metabolic processes, and the functional capacity of various organ systems. In support of this hypothesis, using a cohort of 8,760 people born 1934–44 in Helsinki, Finland, David J. P. Barker found that lower birth weight, thinness at 2 years of age, and overweight at 11 years (indicated by upward centile crossing between ages of 2 and 11) were all associated with increased risk of coronary events and insulin resistance in adulthood. These data, drawn from a series of studies, suggest that rapid weight gain after the age of 2, rather than during infancy (as noted above) is associated with obesity-related outcomes later in life. Reconciling the contradictory evidence is difficult when methods of analysis and the definitions of predictors and outcomes vary widely.

Because many of the children who exhibit upward centile crossing were born SGA, it is important to note that rapid weight gain in infancy may offer survival advantage to the child. Larger infants are less likely to suffer morbidity and mortality in infancy and childhood, particularly in a nutrient-poor environment. SGA is a marker of less-than-optimal fetal nutrition. The commonly observed growth acceleration among these children represents developmental plasticity, which is explained by the "thrifty phenotype hypothesis." This hypothesis suggests that the fetal nutritional environment is critical for metabolic "programming." The programming may allow the newborn infant to differentially utilize nutrients that were not available in utero to improve the development of the nervous system, particularly the brain. Indeed, there is a growing body of evidence showing that accelerated growth for infants born SGA, while maintaining normal growth velocity for infants born appropriate for gestational age (AGA), results in optimal neurodevelopmental outcomes when compared to SGA newborns who fail to exhibit accelerated growth, and AGA newborns who exhibit decelerated growth.

The mechanisms for fetal programming of childhood weight gain have not been elucidated. Investigators have focused on endocrinological variations in SGA infants to help understand the genetics and biochemistry involved in fetal programming. SGA infants undergoing catch-up growth have been found to have increased insulin resistance and hyperinsulinemia, but it is not clear whether it is the catch-up growth or being born SGA that is responsible for this association. In a nutrient-starved fetal environment, insulin resistance and hyperinsulinemia in utero may be advantageous to supply glucose to the fetal brain. However, persistence of this effect in childhood and adulthood may be detrimental to later health, particularly in a nutritionally rich postnatal environment.

In summary, infant weight gain appears to be associated to the development of obesity and related diseases later in life. While the importance of accelerated weight gain during the early postnatal period remains unclear, there is consistent evidence that accelerated growth after 2 years of age is related to later obesity and adverse health outcomes. Further research is warranted to clarify the effect of rapid weight gain during the early postnatal period.

SEE ALSO: Barker Hypothesis; Infant Growth Rate; In Utero Programming; Intrauterine Growth Restriction; Low Birth Weight.

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Inflammation

OBESE ORGANISMS HAVE higher levels of inflammation-associated messengers. As researchers investigated where these signals originated and their relation to increasing body weight, they discovered that the accumulation of excess fat tissue leads to the recruitment of immune cells into the tissue. These cells manufacture and recruit molecules, which perpetuates more inflammation. The ongoing nature of the inflammation and its role in instigating diabetes, heart disease, and other chronic obesity-associated illness are well known, but the stimulus for continued inflammation is under some debate.

Inflammation is our body's response to infection, foreign substances, or injury. The white blood cells in the blood stream release chemicals and set up responses to deal with disruptions to the body. Fat tissue is an organ just as the heart or the skin are organs and, as such, has to maintain a balanced environment to operate optimally. As more fat tissue accumulates, the organ starts to malfunction, acting injured. Fat cells make chemicals of inflammation just as white blood cells do, recruiting more cells and manufacturing more substances characteristic of an inflammatory response.

Inflammation in obesity differs from other injuries, although because it is a foreign invader, substance or infection is not present to be cleared. In fact, usually more fat is being stored as the person continues to overeat, leading to an increased inflammatory response. Consequently, chemicals that were meant to repair a short-lived disturbance in the system are continually released and ultimately lead to more disease. These chemicals are produced by the fat cells and lead to the proliferation of inflammatory substances made by cells recruited into the fat tissue.

The hormone leptin is the classic example of a proinflammatory substance made by the fat cells that increases proportionately with the increase in fat tissue. Moreover, it stands at the crossroads linking appetite regulation in the brain (via the hypothalamus), the nervous system, and the endocrine system (hormonal messaging system that becomes deregulated in obesity). Research in the 1980s looking at fat tissue led to the hypothesis that an unknown signaling mechanism linked energy expenditure and food intake. Over the following two decades, an interconnected regulatory system involving the hypothalamus—a master gland linking the nervous system to the endocrine system through the pituitary gland—the fat cell, and food intake were pieced together. Researchers found many important players that were central to the inflammatory process through this research including leptin, macrophages (cells that recruit more immune cells, plus engulf and digest cell debris, defending the organism as part of the immune system), cytokines (cell messengers which are particularly important in the immune response), and chemokines (small cytokines which send messages to cells nearby).

While leptin's primary function appears to be the regulation of appetite and metabolism acting centrally on the hypothalamus, it acts peripherally to increase inflammation. Leptin stimulates the differentiation of precursor blood cells, helping to determine their fate. One such blood cell involved in the immune system is the macrophage. As the fat tissue increases, more leptin is produced and more macrophages are recruited to invade the fat tissue. These macrophages then make highly proinflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-alpha). Leptin also influences T-cells, a group of white blood cells called lymphocytes that are central to cell-mediated immunity. Leptin changes the profile of cytokines that T-cells produce and prevents programmed cell death (a complex process called apoptosis that is partly meant to clear dead, dying, or incorrectly programmed cells from the system). Leptin also affects the endothelium (the cell-lining of all blood vessels), increasing the amount of adhesive molecules. For example, platelet activating factor-1 (PAF-1) activates platelets and consequently the clotting cascade—linking leptin to the initiation of cardiovascular disease by increasing the propensity to form blood clots in the coronary arteries. Leptin's proinflammatory nature is underscored by research demonstrating that leptin-deficient animals have reduced inflammation.

While fat cells manufacture leptin, which then acts locally and centrally, the toll-like receptors (TLRs) are positioned within the adipocytes and send signals in response to increasing fatty acids, leading to increased inflammation. TLRs are primitive cell receptors that trigger the immune system in response to a number of activators. Nutrient excess has been implicated as one of these activators through an intersection of metabolic and pathogenic pathways and the proliferation of common inflammatory molecules. TLR activity acts

to stifle the message of a receptor that alters gene expression called liver X receptor (LXR). This receptor's messenger is to decrease inflammation. The result is increased inflammatory mediators. There are numerous other downstream effects of this interaction including the enhanced formation of cholesterol plaques in coronary arteries. Truly, TLR activity represents another intersection of the immune and metabolic systems that is in part responsible for the inflammatory cascade activated by nutrient excess.

Chronic inflammation, as described above, results from the interplay of nutrient excess and the overproduction of molecules whose purpose is to keep an organism's system in balance. Leptin, meant to decrease appetite, gets overproduced and its dual function peripherally in generating inflammatory mediators and altering immunity causes untoward effects. TLRs, implicated in infectious disease to handle infectious insults to the body, responds to nutrient excess by trying to combat a perceived diseased state in the face of fatty acid excess. The downstream effect of these metabolic alterations is numerous diseases that are increasing at alarming rates in the face of our increasingly obese population—cancer, asthma, sleep apnea, osteoarthritis, nerve degeneration, gall bladder disease, fatty liver disease, atherosclerosis (the hardening of arteries due to cholesterol plaque), hypertension, and stroke, to name a few. Finally, insulin resistance results from inflammation, and is one of the first metabolic conditions associated with nutrient excess and obesity. While the exact nature of insulin resistance generation is somewhat complex, the overarching idea is simple. Proinflammatory cytokines in addition to excess fatty acids intersect with the insulin signaling pathway through the disruption of insulin receptors insulin-receptor signal 1 and 2 (IRS-1 and IRS-2, respectively). Thus inflammation has been directly linked with the initiation of disease.

The stimulus for the ongoing inflammation is not entirely known, but among competing hypotheses is that as the fat tissue expands, it outgrows its blood supply leading to cell death in the periphery. The dead tissue then acts as the injured stimulus for the inflammatory response. In addition, as noted above, leptin inhibits apoptosis (the clearance of dead and dying cells through a noninflammatory, noninjurious process), again instigating a more inflammatory pathway to clear dead tissue debris.

Obesity is, indeed, an inflammatory state, resulting from fat tissue's proliferation of inflammatory molecules, the recruitment of more cells, which manufacture more substances, which perpetuate inflammation and result in many obesity-associated diseases. The commencement of chronic inflammation occurs by an as yet not fully elucidated mechanism. What is known, however, is that where inflammation usually heals the sick, in the case of obesity, it is making people sicker.

SEE ALSO: Obesity and the Immune System.

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Inherited Taste Preferences

THE TERM *INHERITED* implies that certain preferences for the basic tastes, sweet, salty, sour, bitter, and umami, have a component that is genetic or innate. While both the environment, and biology influence preferences for both foods, and the basic tastes, there are some preferences which are thought to be primarily innate. For example, all humans, and many mammals, exhibit an inherent liking of sweet taste from birth, and when presented with a sweet tasting solution, infants will increase their heart rate and show facial signs of pleasure. In contrast, humans tend to universally reject bitter and sour tastes at birth. This rejection of bitter and sour is thought to be a protective evolutionary advantage, since many poisonous substances have bitter and/or sour tastes.

One genetically influenced taste preference that has been well studied is the inherited ability to taste bitter thiourea compounds, such as 6-n-propylthiouracil (PROP), and phenylthiocarbamide (PTC). These compounds are both extremely bitter to 70 percent of U.S. adults and children (tasters), but are tasteless to the re-

mainder of the population (non-tasters). This bimodal distribution of taste response is extremely rare, and therefore, has been of interest to scientists since it's discovery by a chemist name Arthur Fox in 1931. The distribution of tasters and non-taters varies widely around the world, such that some regions in Sub-saharan Africa have virtually no non-tasters, while India and the U.S. have some of the highest prevalences of non-tasters. The hypothesis that has been suggested, and supported by several studies, is that tasters and non-tasters have different food preferences. Tasters tend to dislike foods that are too bitter or hot (eg., black coffee, broccoli, hot peppers), while interestingly, non-tasters tend to have higher fat preferences and risks for obesity.

Several studies have been completed on the "heritability" of food preferences. Heritability is a term that means the amount of a particular trait or behavior that can be explained by genes. For example, heritability studies on obesity suggest that anywhere from 25-70 percent of obesity can be explained by heritable influences, or genes. Heritability studies are typically done using monozygotic (identical) and dizygotic (fraternal) twins, where one assumes that the monozygotic twins share 100 percent of their genetic material, while dizygotic twins share about 50 percent. Behaviors that are more similar in monozygotic twins would have a higher heritable component that behaviors that are more similar in dizygotic twins. Heritability studies on human food preferences suggest that for most specific food preferences, the contribution made by genes is low. However, some research has suggested that preferences for food groups, like high protein foods, fruits, and vegetables, may show higher heritability, at least in children.

Interestingly, a recent study (2007) in the American Journal of Clinical Nutrition found that liking for sweets and use of sweetened foods might have a significant heritable component. These studies are intriguing, as they suggest that some dietary inclinations might be genetically motivated.

SEE ALSO: Childhood Obesity as a Risk Factor for Adult Overweight; Genetic Taste Factors.

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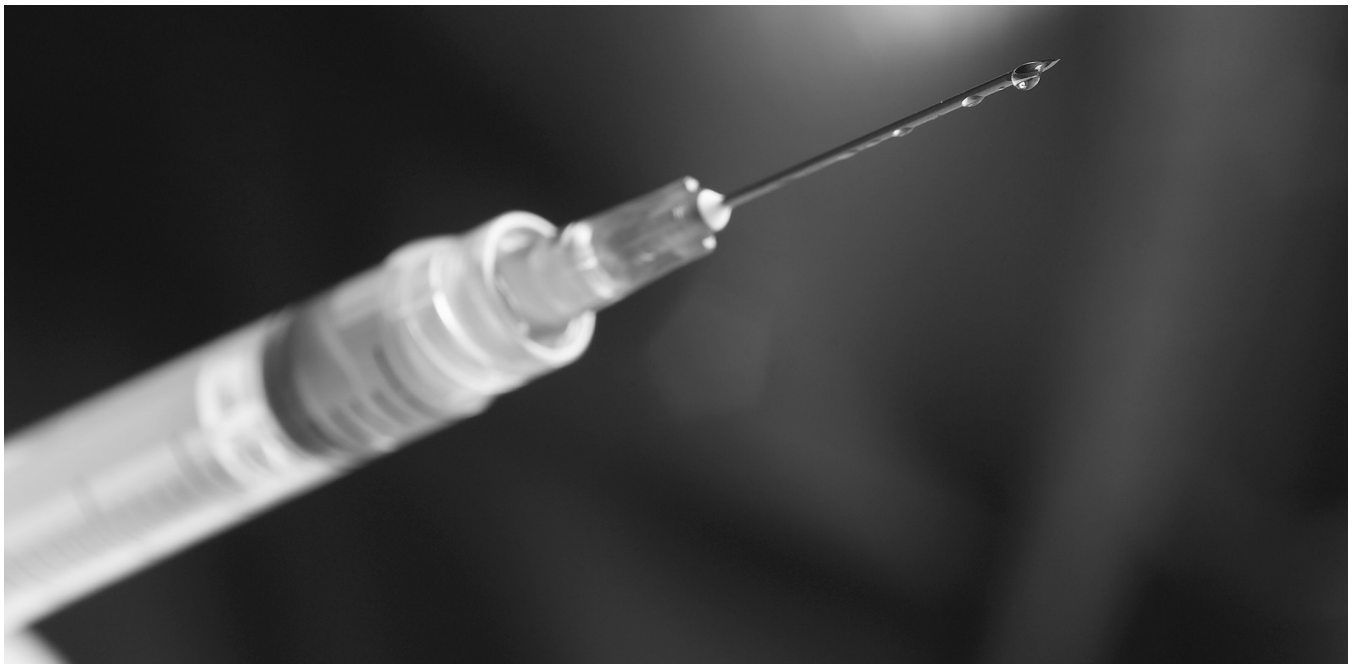
Insulin

INSULIN IS A hormone that is made in the beta cells of the pancreas and plays a critical role in the use of glucose (sugar) in the body. In the pancreas, insulin is made in an inactive form known as proinsulin. Once proinsulin is released, a portion of the compound known as c-peptide is cleaved off and the active form of insulin becomes available for the body to use. Insulin levels rise after a person eats and are low when a person has been fasting.

Insulin has many functions in the body. The primary function is to help bring sugar from the blood into muscle and fat tissue, two principal users of glucose. Insulin works by binding to a receptor on the outside of a cell and subsequently initiating a series of reactions inside the cell. The final step of these reactions is to activate glucose transporters, which are proteins that bring sugar inside the cell. Once inside the cell, the sugar can either be used to create energy by a process known as glycolysis or stored by a process known as glycogenesis. Insulin also promotes fatty acid synthesis, which is known as de novo synthesis. Insulin's role in this pathway is to activate enzymes that favor the creation of fatty acids. Additionally with fatty acids, insulin may also promote the storage of excess fatty acids as triglycerides. Insulin also regulates protein metabolism by increasing the uptake of amino acids, the building blocks of dietary protein, and by increasing protein synthesis. All of these metabolic pathways are classified as anabolic pathways because they promote growth in the body and thus insulin is classified an anabolic hormone.

Insulin also activates the enzyme lipoprotein lipase and inhibits the enzyme hormone sensitive lipase, which work to cleave and store fat in the body, respectively. More recently, it has been discovered that insulin may regulate gene expression. Some of the



Insulin is most commonly thought of in regard to diabetes (and shots). In nondiabetic individuals, insulin is produced in the pancreas, and is used to transport sugar from the bloodstream to muscle and fat tissue.

genes whose expression increases in the presence of insulin include SREBP 1-C, GLUT 1, GLUT 2, GLUT 3, GLUT 4, hexokinase II, glucokinase, glucose-3-phosphate dehydrogenase, and pyruvate kinase. Some of the genes whose expression is decreased in the presence of insulin are glucose-6-phosphotase, fructose-1, 6-bisphophotase, and PEP carboxykinase. These proteins that are regulated by insulin all play a role in glucose metabolism. Insulin has also been demonstrated to increase food intake. This is because insulin's main role is to bring glucose into the cells. If glucose levels are low but insulin levels are high, then insulin will send a signal to the brain to begin eating. Once a person eats, his or her blood glucose will rise and subsequently the extra insulin will be used.

Glucagon is a catabolic hormone whose actions oppose insulin. To assess which metabolic pathways are currently occurring in the body, researchers examine the insulin-to-glucagon ratio. A low insulin-to-glucagon ratio means that there is more glucagon than insulin and thus catabolic pathways are favored. Conversely, a high insulin-to-glucagon ratio means that there is more insulin than glucagon and anabolic pathways. Other hormones that oppose insulin include epinephrine, norepinephrine, and ACTH. During stressful situations, insulin levels are decreased and these other hormones become elevated. This allows for our body to favor a catabolic response and provide a large amount of energy during a short period of time.

Insulin sensitivity is a measure of how well a person's body utilizes insulin. People who utilize insulin well are known as insulin sensitive, while people who do not use insulin well are known as insulin resistant. It is also possible to measure the insulin sensitivity of a specific organ such as the liver (hepatic insulin sensitivity) or the muscle (peripheral insulin sensitivity). There are several different ways to assess insulin sensitivity with some being very easy to measure and thus more favorable for clinical use, while others are more labor intensive and subsequently are favored in research settings. Two of the more common ways to assess insulin sensitivity in a clinical environment are using the homeostasis model of assessment (HOMA) and fasting and two-hour insulin levels. HOMA is a clinical term that measures fasting glucose and insulin levels and through a conversion a value is obtained. Elevated HOMA may be a sign of insulin resistance, but may also be a sign of sugar intolerance if insulin levels are normal but glucose levels are el-

evated. Some clinicians may assess insulin sensitivity by measuring a patient's insulin levels at fasting and two hours after they consume a standard amount of sugar. Three common ways that insulin sensitivity is assessed in a research environment is by an oral glucose tolerance test, intravenous glucose tolerance test, or a hyperinsulinemic-euglycemic clamp. These tests are much more accurate than HOMA or fasting measurements. They can also be used to measure regional insulin sensitivity.

There are several conditions associated with abnormal insulin metabolism. Diabetes mellitus is the major disorder in which the body is deficient or resistant to the actions of insulin. Patients who suffer with type 1 diabetes are deficient in insulin. Their beta cells do not produce any insulin and levels of this hormone are not detected in the blood. This was formerly known as child-onset diabetes. Patients with Type 2 diabetes actually have very high levels of this hormone in their plasma, but the insulin does not function properly and thus glucose does not enter cells for normal metabolism. This was formerly known as adult-onset diabetes. However, along with the rise of the obesity epidemic in children, there has been a rise in Type 2 diabetes in children. Ninety percent of new cases of diabetes in children are for Type 2 diabetes. Patients with untreated DM (both type 1 and type 2) are often said to be metabolically starving because they are unable to use sugar in their cells despite elevated sugar levels in the blood. Insulin resistance is a prediabetic condition in which the body is beginning to become resistant to the actions of insulin. Some people may be insulin resistant for 10 to 20 years before Type 2 diabetes develops. Risk factors for insulin resistance include a family history of diabetes, being overweight, age (older individuals), and lack of exercise. Polycystic ovary syndrome (PCOS) is also a disorder related to impaired insulin metabolism. Women who suffer from PCOS have altered testosterone and estrogen levels and subsequently will become insulin resistant.

There are several medications that people can take to improve their insulin sensitivity. These include sulphonylureas which act to increase insulin release from the pancreatic beta cells. Biguanide (also known as metformin) mechanism of action is not completely understood, but it has been demonstrated to improve insulin sensitivity. Another class of medications is the thiazolidinediones (TZDs). TZDs work by activating peroxisome proliferator-activated receptors (PPAR). This in turn leads to the activation of certain genes that enhance a person's insulin sensitivity. Chromium is a mineral that has been suggested to be related to insulin function. Chromium may be consumed in supplement form as chromium picolinate or chromium GTF. Chromium has been hypothesized to play a role in insulin secretion or insulin receptor function.

In summary, insulin is a critical hormone for glucose utilization as well as many other anabolic metabolic pathways.

SEE ALSO: Estrogen Levels; Metabolic Disorders and Childhood Obesity; Polycystic Ovary Disease; Type 2 Diabetes.

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Insulin-Like Growth Factors

INSULIN-LIKE GROWTH FACTORS (IGFs) are multifunctional hormones that regulate cellular proliferation, differentiation, and metabolism. These proteins resemble insulin in structure and function; hence, the metabolic actions of IGFs mimic those of insulin. IGFs are transported in the circulation bound to insulin-like growth factor-binding proteins (IGFBPs), which regulate the bioavailability and function of IGFs.

There are at least two distinct IGFs—IGF-I and IGF-II, the former being the major form produced in many adult tissues and the latter being the major form produced in the fetus. The liver is the predominant source of IGFs as well as the predominant site

of IGFs degradation. There are six types of IGFBPs. The most important of these chemicals in clinical practice are IGF-I and IGFBP-3. The major portion of IGF-I (75 percent) is bound to IGFBP-3, while 20 percent is bound to other high-affinity IGFBPs (mainly IGFBP-1 and IGFBP-2), and 5 percent is free-floating in the circulation.

IGF-I is a potent mitogen (stimulus for cell growth) and a strong stimulus for cell differentiation (functional maturation). It has a particularly profound effect on bone and cartilage, stimulating osteoblasts (bone cells) replication, and collagen and bone matrix synthesis. IGF-I is the mediator for growth hormone (GH) action, and its level in the circulation is regulated by GH, which is one of the pituitary hormones.

Nutritional status and growth situations influence IGF-I levels. When ample supplies of diverse nutrients are available, the high serum amino acid levels stimulate GH and insulin secretion and the high serum glucose levels stimulate insulin secretion. The high levels of GH and insulin stimulate IGF-I production, and these conditions are appropriate for growth. If the diet is high in calories but low in amino acids, this will result in high insulin but low GH and IGF-I levels. Insulin also stimulates IGF-I production, and per se, GH cannot stimulate IGF-I production in the absence of insulin. Starvation, on the other hand, effectively inhibits IGF-I secretion, even when GH levels are high.

There is evidence suggesting a potential role for IGF-I in the development of obesity. Obesity is associated with growth hormone undersecretion; therefore, it may be expected that total IGF-I concentrations would be lower in obese subjects compared with nonobese subjects. Obesity is also associated with hyperinsulinemia (elevated insulin levels), which may increase IGF-I. Insulin also regulates IGFBP-1 and IGFBP-2 levels through inhibition of their hepatic (liver) synthesis. Finally, it has been recently suggested that IGFBP-2, the principal binding protein for IGF-I in preadipocytes (fat cell precursors), may play a role in the development of obesity.

SEE ALSO: Growth Hormone; Insulin.

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Interleukins

THE REGULATION OF vital physiological processes, for example, replenishment of mature blood cells from bone marrow stem cells, termed *hematopoiesis*, and the activation of defense mechanisms against pathological microbes and injury has been shown to depend on the production and action of a variety of biologically active proteins, collectively known as cytokines. Central among cytokines is a class of mediators, largely involved in the regulation of immune, inflammatory, and hematopoietic functions, called the interleukins. They are produced mainly by leukocytes and act locally on other leukocytes in surrounding tissues. Each interleukin, of which there are now 29 designated ones, has a spectrum of biological activities via specific cell surface receptors. The 29 known interleukins can be clustered into three groups: *noxious* (the *"bad,"* eight members), comprising IL-1, IL-2, IL-6, IL-7, IL-8, IL-15, IL-17, and IL-18; *protective* (the *"good,"* five members), comprising IL-4, IL-10, IL-11, IL-12, and IL-13; and *"aloof,"* comprising IL-5, IL-9, IL-14, IL-16, and IL-19 through IL-29 (15 members).

Obesity is known to give rise to a number of inflammatory conditions in the body that are known to cause other disease conditions such as Type 2 diabetes, hypertension, arthritis, and so forth. In obesity, there is a marked rise in the amount of adipose tissue in the body. Historically, it was believed that adipose tissue consisted only of adipocytes or fat cells that were the sole sites of synthesis and storage of lipid. However, it has now been established that the adipocyte cells also contain a large number of other cells such as the fibroblasts, mast cells, macrophages, leukocytes, and other cells involved with inflammation. A marked increase of various cytokines such as tumor necrosis factor-α (TNF-α), interleukin (IL)-6, IL-8,

and so forth, are seen among the obese individuals. The five interleukins that have an increased production in obesity are briefly mentioned below:

1. IL-1β: Along with TNF-α, IL-1β is considered a proinflammatory (causing inflammation) cytokine. The release of IL-1β is 50 percent higher in tissues of obese individuals. Both TNF-α and IL1-β modulate the release of other cytokines such as IL-6 and IL-8 in the adipocytes.

2. IL-6: This cytokine has been known to play a key role in the development of coronary heart disease. It is associated with the stimulation and release of some specialized proteins in the liver called the acute phase proteins. These proteins increase production of other markers of inflammation such as the C-reactive protein (CRP) by 10- to 100-folds. Enormous amounts of IL-6 are released by the human fat cells which then pass into the bloodstream and reach the liver. Higher amounts of circulating IL-6 in the blood stream and the liver are associated with insulin resistance, a cause of Type 2 diabetes. However, IL-6 is not associated with insulin action; it only stimulates the formation of anti-insulin factors.

3. IL-8: It is an interleukin that belongs to an ever-expanding family of proteins that exert chemoattractant activity to leukocytes and fibroblasts. This family of proteins is termed the *chemokines*. IL-8 is produced by monocytes, neutrophils, and NK cells and is chemoattractant for neutrophils, basophils, and T-cells. IL-8 activates neutrophils in the adipose tissue. Elevated levels are associated with increased risk of coronary artery disease and types I and II diabetes.

4. IL-10: This interleukin inhibits cytokine production, promotes B cell proliferation and antibody production, and suppresses cellular immunity and mast cell growth. The serum levels of IL-10 are enhanced in obesity primarily from the nonfat cells of the monocytes and it functions as a feedback inhibitor, that is, it prevents the secretion of the inflammatory cytokines. This is the only good cytokine secreted in obesity.

5. IL-18: Increased levels are associated with increased risks of both diabetes and cardiovascular disease. This cytokine is released more by the nonfat cells of the adipose tissue.

The visceral as well as the subcutaneous fat depots of the body produce interleukins and other inflammatory cytokines. However, it has been seen that elevated visceral fat accumulation is a risk factor for cardiovascular diseases, hypertension, and Type 2 diabetes mellitus than accumulation of fat in the subcutaneous regions of the hips and legs. The reason is unclear, but the most likely cause is that of the portal circulation. It results in a greater delivery of cytokines, free fatty acids, and other substances from the liver from the visceral adipose tissue depots compared to the subcutaneous depots.

SEE ALSO: Adipocytes; Inflammation.

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International Obesity Task Force

THE INTERNATIONAL OBESITY Task Force (IOTF) is the advocacy arm of the International Association for the Study of Obesity (IASO), an umbrella organization for national obesity associations whose aim is to promote understanding of obesity and related diseases through scientific research, and development policies for their prevention and management. The IOTF also serves as a global network of experts on obesity and health and as an incubator of research and ideas related to obesity prevention and management. The mission of the IOTF is to alert the governments and general populations throughout the world of the impending health crisis produced by increasing rates of obesity, and to persuade governments to take action immediately to counter this threat. The IOTF works with the World Health Organization (WHO) and other nongovernmental organizations and stakeholders, including partners in the Global Prevention Alliance, toward this goal.

The IOTF has declared obesity to be "the millennium disease" in recognition of the fact that it was first introduced into the international classification

of diseases over 50 years ago, and is reaching epidemic proportions in early years of the 21st century. As stated on the IOTF Web site, over 300 million people around the world are obese (defined as a body mass index [BMI] over 30) and their numbers are rising; because serious health consequences are associated with obesity, this situation demands recognition and immediate action by governments throughout the world.

Childhood obesity is a particular focus of the IOTF. A working group consisting of eight scientists from Australia, the United Kingdom, the United States, and the Netherlands is consulting on guidelines to reduce food marketing to children (under age 18), currently known as "The Sydney Principles." These guidelines, in draft form as of May 2007, are based on the following principles: the right of children to adequate, safe, and nutritious food; the particular vulnerability of children to commercial exploitation, and responsibility of parents, governments, civil society, and the private sector to protect them; the necessity of statutory regulations (rather than merely industry self-regulation) to reduce marketing aimed at children and the deleterious effects this has on their diets; a broad definition of commercial promotions, including conventional advertising and media, product placement, sponsorships, and internet-based promotions; assurance that schools and child care settings remain free of commercial promotions; regulation of cross-border media such as satellite and cable television and the internet; and the need for enforcement and evaluation of the effectiveness of regulation.

A number of informational and promotional materials are available from the IOTF Web site. Press releases and other media information are presented, including links to current news and research reports, and reports from international conferences. Separate Web pages are provided for cancer, cardiovascular diseases, and diabetes, which include a summary of current knowledge of the relationship between obesity and the disease in question, statistical tables, and links to further information and to other organizations working in each field. A large general links section of the Web site includes links to many other Web sites, including academic organizations, campaigns (such as the BBC Education Health Site "Fighting Fat, Fighting Fit"), online journals, relevant articles, governmental sites, pharmaceutical companies, professional societies, conferences, and online articles and studies.

The IOTF has compiled a number of statistical reports that are available through the organization's Web site. These include *Global Obesity Prevalence in Adults* (which includes the percent obese and overweight by gender by country), a map of global obesity prevalence for men and women, *Childhood Overweight* (by country, arranged by WHO region, for boys and girls), *Childhood and Adolescent Overweight and Obesity* (for European boys and girls, in age categories 7–11 and 14–17 years), *Adult Overweight and Obesity in the European Union (EU25)* (by country, males and females separately), and *Trends in Obesity Prevalence* (maps for males and females, and adults and children, showing rates of obesity in Europe and globally).

SEE ALSO: Child Obesity Programs; Government Agencies; Governmental Policy and Obesity; Nutrition Education; Prevention; Type 2 Diabetes.

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Intestinal Microflora Concentrations

MICROINTESTINAL FLORA IS the group of microorganisms that live in the digestive tract of the human body. This microbiota has a mutualistic and symbiotic relationship with the gut and is involved in many functions such as fermenting unused energy substrates, preventing harmful pathogen growth, and vitamin and hormone production. Both the fermentation and hormone production roles can contribute to body weight regulation. Certain hormones aid in fat deposition, and microintestinal flora digests sub-

strates that the human body is normally unable to, which means that more energy can be extracted from certain foods. Evidence in both mice and humans support that varying microbiota compositions lead to differences in caloric extraction from food. This may, in turn, contribute to differential body weights.

Genetics play a large role in determining an individual's body weight. However, the human genome is not the only genome of interest. The normal healthy adult has around 1023 gut microbes, about 10 times the total number of cells in the body, all of which bring their own genetic makeup into the mix. There are between 300 and 1,000 different species in the gut (30 to 40 species account for 99 percent of all gut microbes).

It was shown that obese humans have a higher proportion of a certain strain of bacteria than lean individuals. And when those obese individuals lost weight, their flora proportions shifted to resemble that of the lean individuals. In addition, mice with microbiota had almost 42 percent more total body fat when compared with mice lacking microbiota, even though they had consumed 29 percent less in their diets. Also, mice born lacking microbiota with an introduction of microbiota 8–10 weeks after birth showed a 57-percent increase in their total body fat. Similarly, when microbiota of obese mice was transferred to lean microbe-free recipient mice, they began extracting more calories from their food and had a modest fat gain.

It is not clear, however, if these small changes in caloric extraction will always result in significant and meaningful differences in body weight. Also, the question still remains on how gut flora is regulated. Why is it that obese humans, who already have a great deal of stored energy, still have a microflora environment shifted to be even more efficient at calorie uptake? Future exploration may lead to a better understanding of how to manipulate the microbiotic environment of the human intestinal tract to help treat or prevent obesity.

SEE ALSO: Fiber and Obesity; Functional Foods.

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Intrauterine Growth Restriction

CURRENTLY, OVER 200 million children are considered to be undernourished; many of them first experience nutrient deprivation in utero and were born small compared to healthy children. Maternal diet, along with a host of other physical and environmental factors, is the primary source of energy that promotes fetal growth. Energy restriction of the mother, and ultimately the fetus, results in impaired fetal growth, manifested in intrauterine growth retardation, or restriction (IUGR). Two classifications of IUGR are currently used by healthcare professionals: symmetric is defined as less than the 10th percentile for birth weight and asymmetric defined as less than the 10th percentile for birth weight and normal head but small abdominal and muscle development. It is estimated that between 17 and 27.6 million babies born each year experience IUGR, the vast majority of whom are born in less developed or developing countries. For example, Asia, specifically India, has the highest prevalence of IUGR followed by Latin America and Africa. Still, regardless of the geographic location, causes of IUGR are universal and generally associated with inadequate delivery of nutrients to the fetus, caused by either inadequate intake by the mother or poor placental circulation.

Inadequate maternal caloric intake immediately before and during pregnancy is the primary factor predisposing a fetus to IGUR. However, in developed countries, the causes of IUGR are cigarette smoking, low weight gain during pregnancy, and low prepregnancy body mass index (BMI). In developing countries, the primary causes of IUGR include low weight gain during pregnancy and low BMI prior to pregnancy. Regardless of the level of development, a less prevalent dietary risk factor for IUGR extends to

micronutrient deficiencies, such as low foliate, zinc, and magnesium, lending support to aid programs that promote the intake of foods rich in protein and micronutrients.

Among children born with IUGR, a percentage are born small for gestational age (SGA), defined as a birth weight for gestational age less than the 10th percentile of a healthy population, such as the United States. While maternal age, smoking, altitude, and maternal height are all factors that contribute to babies being born SGA, the main cause of SGA births is maternal undernutrition, especially in developing countries.

The timing of maternal undernutrition has important implications for future risk of chronic diseases in offspring. The relationship between when a child is exposed to IUGR and later disease risk varies by particular disease. Again, maternal undernutrition resulting in IUGR and babies born SGA occur primarily in the second and third trimester of pregnancy. Undernutrition throughout pregnancy and prior to pregnancy also increases the likelihood of IUGR occurring, a phenotype that is now believed to be a risk factor for chronic diseases in adulthood.

Maternal nutrition is essential for normal and proper growth of the fetus and placenta. It is clear that undernutrition results in reduced fetal and placental growth and that fetal growth retardation that is often permanent. Perhaps more importantly, and central to this entry, is that maternal diet during pregnancy not only has immediate effects on fetal growth, size, and development, but also that energy and/or protein deprivation in utero increases the risk for many chronic diseases in adulthood, such as Type 2 diabetes, hypertension, and other forms of cardiovascular disease.

SEE ALSO: Low Birth Weight; Pregnancy.

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In Utero Programming

LARGE EPIDEMIOLOGICAL STUDIES have reported an association between being born SGA, or small for gestational age with a birth weight less than 2,500 grams, and chronic disease during adulthood, a concept known as "fetal programming." Of particular interest has been the association between maternal caloric restriction (MCR) and cardiovascular disease (CVD), hypertension, and Type 2 diabetes. One of the most noted studies is that of the Dutch famine in which a cohort of men born before, during, and after the famine in Holland of 1945 were studied for body weight in relation to exposure to famine during gestation. Men who had been exposed to the famine during late gestation and early infancy were more likely to be underweight as adults compared to men who were exposed to the famine during the first two trimesters of gestation, suggesting that early, prenatal exposure to undernutrition is related to weight gain later in adulthood.

In one cohort of over 15,000 men and women born in the Hertfordshire region of England, men and

women with lower birth weight were more likely to suffer from CVD than men and women with normal birth weight. Barker et al reported that a correlation between birth weight and CVD mortality in a cohort of 1,586 men born between 1907 and 1924. It has also been reported that for every 1,000g increase in birth weight, the rate ratio for mortality from cardiovascular disease decreased by one-third, even when adjusted for adult sociodemographic characteristics. Finally, results of a retrospective study of US nurses found that those who were born small were more likely to suffer from CVD compared to those with normal birth weight.

Mechanisms to explain these observations are lacking, but several potential hypotheses exists, such as abnormal fat distribution. Several studies have reported that persons with low birth weight are fatter and tend to deposit fat centrally during adulthood. Results from the Dutch famine study suggest that men who had been exposed to the famine during late gestation and early infancy were more likely to be underweight as adults compared to men who were exposed to the famine during the first two trimesters of gestation. This would imply that early, prenatal exposure to undernutrition is related to weight gain later in adulthood. However, a high body mass index (BMI) is not the most important body phenotype to increase disease risk, how body fat is distributed, whether centrally or peripherally, is often reported to be a major risk factor for chronic disease.

In terms of fat distribution, some have reported that waist-to-hip ratio (WHR) was negatively associated with birth weight, but it should be stressed that WHR is an indirect and inaccurate assessment of central adiposity. Low birth weight girls are more likely to deposit fat in the upper body, estimated using skinfold measurements. Women with short stature, an indication of previous undernutrition, are more likely to be obese and had higher WHR than women of normal stature. A study of 229 male twin pairs reports that low birth weight is associated with greater abdominal fat and less lean body mass, independent of maternal and genetic influences. Similarly, birth weight is negatively correlated with central adiposity, a known risk factor for diabetes, in American children, and birth weight is associated with higher lean tissue mass, but not higher adipose tissue mass. The fact that lean, but not adipose, tissue is directly associated with birth weight suggests that persons born with low birth weight either develop less lean tissue or deposit more adipose tissue during growth.

Recently, it has been suggested that a potential factor that unites many of the observations relating low birth weight and chronic diseases is an abnormality in the developing of the hypothalamic-pituitary axis (HPA). Central to this work is an abnormal stress response, measured by circulating cortisol, a stress hormone released in acute bursts in times of stress, given that cortisol is associated with central adiposity and increased risk of chronic diseases.

Evidence from well-designed clinical studies of adults who were born SGA suggests that programming of the HPA may occur inutero. For example, persons born SGA have increased basal cortisol values and increased cortisol released during cortisol stimulation tests. In addition, increased cortisol is released in response to stress; this is evident even in childhood. While cortisol is increased in persons born SGA, others have not universally reported this observation. Still, previous studies have shown that there is a relationship between birth weight and cortisol and cortisol and insulin resistance. First, in older adults who were born SGA, the results of an oral glucose tolerance test showed that of the 370 participants, 66 had impaired glucose tolerance and 27 were diabetic. Additionally, it was also found that they had increased plasma fasting cortisol levels that were associated with increased glucose levels, insulin resistance and decreased HDL. Finally, it was reported that as the birth weight of the subjects increased, cortisol levels decreased.

In terms of a potential mechanism explaining the association between MCR and programming of the HPA-axis, it has been hypothesized that in utero stress may program the control of cortisol secretion. In one study, a stress challenge using dexamethasone (designed to induce cortisol secretion) followed by administration of adrenocorticotropic hormone (ACTH), a test designed to stimulates secretion of glucocorticoids such as cortisol. As birth weight decreased, plasma cortisol increased, suggesting a hypersensitivity to ACTH in lower birth weight adults. Thus, fetal exposure to stress may alter stress responses and influence the daily secretion of cortisol, a powerful hormone that can alter not only fat oxidation, but body fat distribution as well.

While human clinical studies provide significant physiological evidence of fetal programming of key tissues and metabolic processes that promote chronic diseases, there are only so many protocols that can be conducted ethically in humans. Such limitations are overcome through the use of well-controlled animal studies. Thus, studies conducted in animals have provided a wealth of information that illustrate the effect of low protein intake during pregnancy on the physiology and health of the offspring. For example, rats exposed to maternal protein restriction during gestation had significantly reduced the lifespan and both male and female offspring. Explanations for the reduced lifespan include gestational exposure to oxidative stress and poor development of key tissues.

In terms of development, it was reported that the offspring of rats fed an 8 percent protein diet, compared to 20 percent protein, had reduced pancreatic cell mass at birth and a lower insulin secretion in adulthood. It was also found that the offspring from protein-restricted mothers had significantly greater islet cell apoptosis from birth through adulthood. Others have reported that protein-restriction in utero results in poor growth of hepatic cells such that the DNA synthesis of cultured hepatocytes was 30 percent less than that from control offspring. It was also reported that the LP hepatocytes produced less IGF-1 and more IFBGP-3 compared to the control hepatocytes, suggesting a more complex response to LP other than abnormal tissue development. The fact that maternal energy restriction, in general, and protein-restriction, in particular, influences the powerful IGF axis has generated substantial research on the cellular and molecular changes that result from gestational exposure to such restriction.

These cellular effects appear to persist into adulthood and alter the physiology of animals exposed to protein-restriction in utero. The effect of protein-restriction appears to continue following pregnancy as offspring of either protein-restricted or control mothers had a lower insulin to glucose ratio when breastfed from a mother protein-restricted during lactation. However the effect appeared to be more profound for female, but not male, offspring. Thus, it was concluded that the timing of protein restriction is important for long-term developmental changes, but also that these effects appear to be gender-specific.

As far as specific mechanisms linking maternal protein-deficiency to risk for chronic diseases in the offspring, several studies have reported that changes in pancreatic development, enzyme function, or cellular morphology are altered following protein restriction in utero. When fed less than 30 percent of the protein as control mothers, offspring of protein-restricted mothers had decreased glucokinase levels. Specifically, both prenatal and postnatal exposure to maternal protein-restriction appeared to increase the affinity of glucokinase to glucose, an observation that was not reversed when the offspring were fed a normal protein diet until adulthood. Desai et al found that following exposure to protein-restriction during either gestation or lactation, the activity of both glucokinase and phosphoenolpyruvate carboxykinase decreased in the pups exposed to gestational protein-restriction only. More important is the fact that insulin and glucagon levels were similar in control and restricted groups, but the mRNA levels of both enzymes paralleled their activity, suggesting an influence on gene expression as well as enzyme activity.

Most chronic metabolic diseases are related to abnormal or insufficient glucose metabolism and/or hypertension, diseases that stem from abnormal functioning of key tissues, such as endocrine or renal development. In terms of endocrine development, several animal studies have reported a clear association between insufficient maternal protein intake and abnormal development of endocrine tissues. The developing pancreas is sensitive to amino acid availability and requires a specific concentration of essential amino acids to propagate normal beta cell multiplication. Maternal protein restriction has been reported to promote not only a decreased islet cell proliferation, pancreatic insulin concentration, and reduced islet vascularity. Moreover, offspring of protein-restricted mothers also had increased apoptosis and decreased IGF-1 following birth and persisting even when fed a normal protein diet. The metabolic outcome of this profile was a 50 percent reduction in insulin secretion compared to control pups. Perhaps most striking from the research on maternal diet and pancreas development, is that most of the observations found in the first generation of offspring, persisted into subsequent generations, even when protein was restored to the diet. Thus, pups born small and with abnormal pancreas development gave birth to pups that were growth-retarded insulin-resistant.

For humans, these studies support epidemiological and clinical studies in humans that suggest that growth retardation in utero predisposes a person to insulin resistance in adulthood. Insulin resistance is commonly associated with LBW in adults and young adults, and is a known precursor for type 2 Diabetes Mellitus. LBW has been associated with elevated plasma glucose and insulin levels, even when controlling for sex, BMI, and maternal BMI. Yajnik et al reported that 4-year old children in India with LBW had higher plasma glucose and insulin concentrations following an oral glucose load (8.1 mmol vs. 7.4mmol, p=0.01), independent of their current body weight. Initial studies suggested the mechanism behind the relationship between birth weight and glucose intolerance/Type 2 diabetes was based on abnormal fetal endocrine pancreatic development. However, recent research has targeted peripheral insulin resistance as a key factor in etiology of diabetes.

Recently, novel research into the epigenetic effects of MCR is providing some evidence that fetal experience may result in long-term regulation of key metabolic enzymes through DNA modifications. In terms of uterine environment, reduced uterine blood flow, a factor that would retard fetal growth through nutrient and oxygen restriction, decreased methylation of the renal p53 promoter, affected mRNA levels of key apoptosis-related proteins, and increased renal apoptosis, factors that promote hypertension in adulthood. In addition, the expression of genes associated with hypertension and Type 2 diabetes increased in the pups and, more interestingly, the methylation of these same genes decreased.

There is an abundance of evidence, from both human and animal studies, that support the concept of fetal programming. Additional research on epigenetic relationships and work with human studies are warranted to develop more precise mechanism to explain why caloric restriction in utero, or even during childhood, may predispose a person to chronic diseases in adulthood.

SEE ALSO: Pregnancy; Tubby Candidate Gene.

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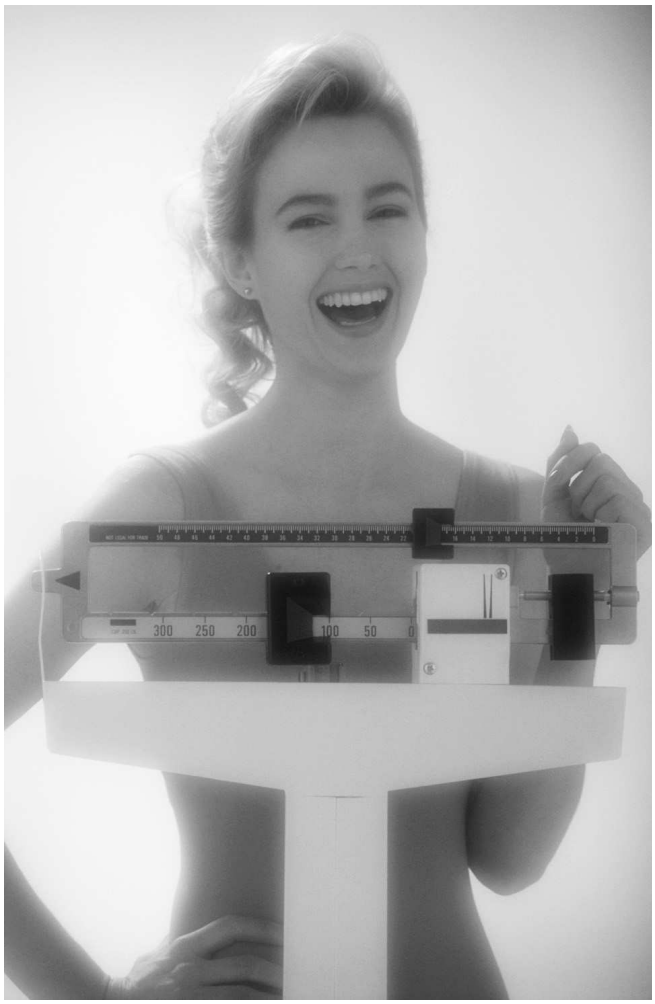
Jenny Craig

JENNY CRAIG, INC. is a corporation based in Carlsbad, California, which operates a chain of weight-loss centers in the United States and several other countries. It was founded in Australia in 1983 by Jenny Craig (née Genevieve Guidroz) and her husband Sidney Craig. At the time of founding the company, Jenny Craig had no training or expertise in nutrition or medicine, but based the Jenny Craig system on Jenny's successful personal weight loss, coupled with her husband's experience as National Director of Operations for Body Contour, Inc., a chain of fitness salons. Jenny Craig, Inc. was one of the first commercially successful weight-loss programs, and today is one of the largest: there are Jenny Craig Centers in the United States, Canada, Puerto Rico, Guam, Australia, and New Zealand. Jenny Craig, Inc. was sold to Nestlé for approximately $600 million in 2006.

The Jenny Craig program emphasizes a balanced approach to diet and exercise supported by individual counseling; counselors are trained within the Jenny Craig program and do not necessarily have formal medical or nutritional training. A number of different programs are offered, which are based either out of a local Jenny Craig Center or are based out of the individual's home. Both types of program involve purchase of packaged Jenny Craig foods based on an individual menu plan designed for a weight loss of one to two pounds, or 1 percent of body weight, per week. Center-based programs include weekly individual counseling sessions, weight take at each consultation, online message boards, and a live chat site for peer support. The home-based program (known as the Jenny Craig Direct At-Home Program) includes weekly phone counseling, home-delivered support materials, and home-delivered Jenny Craig foods. Several pricing options are available. In April 2007 the Jenny Craig Web site listed six types of plans, with variation in pricing within each based on geographic region and other factors.

The Jenny Craig program has been praised for its comprehensive nature, as it incorporates diet and exercise, and provides individual counseling and coaching intended to teach the dieter new cooking and eating habits. On the other hand, the program initially requires purchase of Jenny Craig packaged food, which is relatively expensive but is intended to remove the guesswork from portion size and food choice while the individual is learning new dietary habits. Dieters are gradually encouraged to eat more ordinary food as they near their target weight, although some believe the use of packaged foods (in single-portion sizes) only delays the day when the individual must learn to choose appropriate foods and portion sizes. In addition, use of the prepackaged foods are so central to the Jenny Craig concept that some participants have referred to Jenny Craig as a food marketing program supplemented by counseling.



The Jenny Craig program emphasizes a balanced approach to diet and exercise supported by individual counseling.

A 2005 *Forbes* magazine article found Jenny Craig to be the most expensive of 10 systems of dieting, including NutriSystem, Weight Watchers, Sugar Busters!, the Atkins Diet, Slim Fast, and the Zone Diet. They estimated the total price of one week's worth of Jenny Craig meals to be $137.65, or 152.8 percent of the amount the average single American spends on food in one week.

The Jenny Craig program is based on sound weight-loss principles including learning new eating and lifestyle habits, making incremental changes, and finding social support during the learning process. The Web site also contains much useful information for anyone trying to lower or maintain their weight, including recipes, shopping tips, information about nutrition and exercise, and general information such as advice about maintaining balance in one's life. However, the commercial nature of the program is also obvious. Individual Jenny Craig Centers are franchises and information for prospective investors, including the amount of investment capital required and anticipated returns are available from the Jenny Craig Web site.

The program has achieved high name recognition due largely to the number of individual Jenny Craig Centers (there were approximately 660 as of 2007), television and print advertising of individual success stories (ironically often followed by the disclaimer "results not typical"), blogs of successful individual dieters, and use of celebrity spokeswomen including Monica Lewinsky, Kirstie Alley, and Valerie Bertinelli.

As of 2002, Womble and colleagues found no peer-reviewed studies of the efficacy or safety of Jenny Craig programs for weight loss or health improvement, and a PubMed search conducted in 2007 also revealed no articles examining the success of the program or comparing it to other weight-loss and maintenance strategies.

SEE ALSO: Advertising; Nutrition and Nutritionists; Women and Dieting.

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Kidney Failure

THE KIDNEYS ARE complex organs that perform many functions, including maintenance of fluid and acid–base balance, removal of waste products, stimulation of red blood cell production, and regulation of calcium stores. Failure of the kidney can affect one or all of these functions, and may occur suddenly or gradually. Physicians commonly use creatinine, a blood test, to estimate the kidney's function. The glomerular filtration rate (GFR) reflects how much fluid one glomeruli (the smallest functioning unit in the kidney) can filter per minute.

Sudden or acute kidney failure can be caused by poor blood flow to the kidney as with dehydration or heart failure, very high blood pressure, toxins, inflammation, or kidney stones, to name a few possibilities. Patients can present with confusion, reduced appetite, vomiting, leg swelling, or reduced urine output. Acute kidney failure is managed by treating the underlying cause and allowing the kidneys to heal themselves. If needed, physicians can remove excess fluid or waste products directly from the blood by using hemodialysis for a short period.

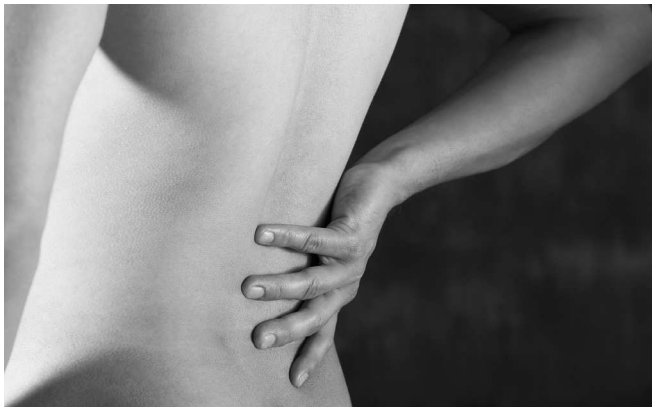
Chronic kidney failure, on the other hand, is a gradual and largely silent disease caused most often by uncontrolled diabetes and hypertension. One in nine adults in the United States has chronic kidney disease (CKD). Racial and ethnic minorities and people with obesity and associated conditions like hypertension, diabetes, and hypercholesterolemia are at higher risk for kidney failure.

Simple tests like creatinine and urine protein can identify people with early stages of CKD. Early intervention with medications to treat hypertension, such as angiotensin converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), and lower blood sugar can slow or even halt disease progression.

People with advanced CKD commonly develop poor appetite, loss of sleep, lack of energy, poor concentration, muscle cramps at night, and swollen feet. CKD itself can cause high blood pressure and increase cholesterol deposition in arteries, thereby predisposing to heart attack and stroke.

People with CKD should avoid taking over-the-counter pain medications, and take new medications only after proper consultation with a physician. Because damaged kidneys cannot process high levels of potassium, sodium, or protein, a modified diet is recommended. In addition to monitoring the GFR, physicians also monitor calcium, vitamin D, and red blood cell levels. Treatment with vitamin D to prevent osteoporosis or erythropoietin to treat anemia may be necessary. In end-stage CKD, patients may require hemodialysis, peritoneal dialysis, or kidney transplant to survive.

SEE ALSO: Hypertension; Hypertension in African Americans; Hypertension in Asian Americans; Hypertension Pharmacotherapy; Type 2 Diabetes.



Acute kidney failure is managed by treating the underlying cause and allowing the kidneys to heal themselves.

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Kidney Stones

KIDNEY STONES ARE formed by a complex process of crystal formation and deposition within the kidney, ureter, or bladder. People with kidney stones most often present because of severe pain or blood in the urine. About 5 percent of women and 12 percent of men will have at least one episode of kidney stones by the age of 70. Kidney stones are more prevalent in obese patients, particularly men and those who have abdominal obesity (higher waist circumferences). The mechanism linking obesity to a greater prevalence of kidney stones is not yet known."

There are three types of stones: calcium, struvite, and cystine. The most common type is calcium with oxalate or phosphate. These can be caused by high calcium levels in the blood, genetic factors, intestinal bypass surgery, or diets rich in oxalate. Struvite stones are caused by chronic urinary tract infections with certain bacteria. Uric acid stones tend to develop in people with high blood levels from gout or chemotherapy, or people eating high-protein diets causing acidic urine. Cystine stones occur rarely in association with hereditary disorders.

In some people, kidney stones pass through the urinary tract without causing symptoms. However, stones can also cause obstruction of the kidney or ureter. This causes severe episodic pain that usually begins in the back and moves to the groin and is associated with nausea, vomiting, and blood in the urine. Physicians can diagnose kidney stones based on urine tests, X-rays, computerized tomography (CT) scans, or ultrasounds.

Most kidney stones eventually pass on their own with two to three quarts of fluid intake a day. Treatment with a calcium channel blocker, a type of blood pressure medicine that relaxes smooth muscle like that in the ureter, may speed up the process. Stones that need to be removed urgently are very large or fail to pass on their own may require additional intervention, such as uretroscopy or extracorporeal shock wave lithotripsy.

Kidney stones frequently recur but can be prevented. The single most important lifestyle change is to drink up to a gallon of water a day. People with calcium stones should restrict foods rich in oxalate such as beets, chocolate, coffee, cola, nuts, rhubarb, spinach, strawberries, tea, and wheat bran. Reduction of dietary salt and animal protein also helps. As a rule, restricting calcium is not recommended because calcium in normal amounts combines with oxalate in the gastrointestinal tract and prevents its absorption. When calcium is low, oxalate is absorbed, which leads to more stone formation. Less commonly, specific medications can correct the underlying problem resulting in stones.

SEE ALSO: Calcium and Dairy Products; Kidney Failure.

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Laparoscopy

MINIMALLY INVASIVE SURGERY has truly revolutionized how abdominal procedures are performed. Minimally invasive surgery in the abdomen is performed using a laparoscope. Laparoscopy is derived from two Greek words, *laparos* = "abdomen" and *skopein* = "to look or see." Laparoscopy involves the principle of minimal access in which operations in the abdomen are performed through small incisions compared to larger incisions needed in traditional "open" surgical procedures.

The benefits of minimally invasive surgery include less pain, improved cosmetic results, and quicker return to baseline functionality. Other benefits include lower wound-related complication rates such as infections and hernias. The morbidly obese patient arguably benefits from laparoscopic surgery to an even greater degree than the normal-weight patient. The elements in laparoscopic surgery are the laparoscope, a telescopic lens system that is connected to a video camera and attached to a lighted fiber-optic cable system. Laparoscopes are currently available in several sizes from 2 millimeters in diameter to 10 millimeters. The abdomen is usually insufflated with carbon dioxide gas to create a working space. Carbon dioxide is almost always chosen because it does not support combustion and it is inexpensive, easily available, and rapidly absorbed.

Any operative procedure performed in the morbidly obese patient is inherently more risky than in normal-weight individuals. Specific to abdominal surgery, not only are there technical challenges to operating on the obese individual due to the thickness of the abdominal wall and amount of visceral adiposity, but also obese individuals are prone to a number of severe physiologic derangements that substantially increases their perioperative risk.

The obese patient suffers from a decreased cardiac reserve. Systemic hypertension, so common in the obese patient, can result in left-sided ventricular hypertrophy, cardiomyopathies, and valvular dysfunction. Obesity is strongly associated with the metabolic syndrome, which promotes arteriosclerosis and coronary artery disease. Arrhythmias are also more common in the morbidly obese and may be further aggravated by obstructive sleep apnea.

In the obese, respiratory physiology is altered. Oxygen consumption and carbon dioxide production is increased and the chest wall compliance is reduced. This, in turn, decreases pulmonary functional residual capacity (FRC) and result in the premature airway closure and subsequent ventilation/perfusion mismatch in the lungs. Chronic intraabdominal hypertension secondary to visceral obesity also may lead to decreased FRC. Severe sleep apnea occurs frequently in the morbidly obese patient and can be associated with

a number of complications including arrhythmias, hypoxia, pulmonary hypertension, right-sided heart failure, and cor pulmonale (a failure of the right ventricle of the heart). Gastric physiology is also altered; obese patients are more likely to have large gastric volumes, lower gastric pH, and delayed gastric emptying, which increases their risk for gastric aspiration during surgery. Gastroesophageal reflux is very common in the morbidly obese and can induce asthma.

The elevated body mass in the morbidly obese patient undergoing laparoscopic surgery increases the possibility of developing deep venous thrombosis (DVT) due to the high intraabdominal pressure that impairs the return of blood from the legs to the heart. Obesity may also promote the risks for DVT formation directly as a chronic inflammatory condition and indirectly through decreasing mobility.

The premise that laparoscopic surgery is better tolerated than open surgery in the obese relies on the fact that there is less operative trauma with laparoscopy and that the extent of pulmonary depression is related to the magnitude of operative trauma. This has been demonstrated in randomized trials comparing the impairment of pulmonary function after laparoscopic gastric bypass (GBP) and open GBP were a lower rate of segmental atelectasis and a higher forced expiratory volume in one second (FEV1) favor the laparoscopic approach.

Benefits of laparoscopy over conventional open surgery also includes a decrease in blood loss, shorter hospitalization time, less postoperative pain, and a minimized systemic inflammatory response. Many studies have demonstrated a more rapid resumption of daily activities and earlier return to work than open procedures. Improvements in quality of life can often be demonstrated months after surgery relative to those patients undergoing traditional open procedures. Other advantages of the laparoscopic technique include a decrease in the incidence of abdominal wall hernias and of wound infection because there is no large incision made there are also improved cosmetic results.

The main disadvantage of laparoscopy in the obese is that skills needed to perform these procedures safely is much higher than in the nonobese patient. Thus, complication rates may be higher when inexperienced surgeons are performing the procedures.

SEE ALSO: Gastric Bypass; Lap Band.

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Lap Band

THE LAPAROSCOPIC ADJUSTABLE gastric band (LAGB) is a device that can be used to cause significant weight loss in morbidly obese individuals. Gastric banding was first introduced in 1985 in Europe. Later modifications were introduced in Europe in 1993. The device received U.S. Federal Drug Administration (FDA) approval in June 2001.

The last decade has seen explosive growth in lap band procedures worldwide. Most of the growth is attributed to the development, maturation, and application of laparoscopic surgical techniques to the field of bariatric surgery. In the laparoscopic approach, approximately five small incisions (1 cm or less) are made in the abdominal wall instead of one big incision. This results in significantly less pain with fewer wound complications such as infection and hernias. The other advantages of the laparoscopic approach include faster recovery, shorter hospital stay, and less perioperative morbidity.

Numerous outcome-based studies have now clearly shown this operation to be safe and effective for long-term control of obesity along with resolving associated life-threatening conditions such as diabetes, high blood pressure, and sleep apnea.

The lap band might be considered by any patient who is morbidly obese and has tried and failed to lose weight with diet and exercise programs. The procedure is currently restricted to obese individuals who

have a body mass index (BMI) of at least 35 with medical problems relegated to obesity or any patient with a BMI of over 40.

The lap band is a silicone elastomer ring designed to be placed around the upper part of the stomach. The inflatable balloon on the inner surface of the band can be filled with saline. This balloon is connected by tubing to an access port that is placed beneath the skin during surgery. The surgeon can adjust the lap band by adding or subtracting saline inside the inner balloon through the access port.

The placement of the band creates a small pouch at the top of the stomach which holds approximately 30 milliliters volume. This pouch fills with food quickly sending this message to the brain. This sensation of fullness helps the person to eat smaller portions and lose weight over time. If the band is too loose, then the weight loss will be inadequate. Adding more saline can reduce the size of the stomach to further restrict the amount of food that can move through it. If the band is too tight, the patient may experience difficulty swallowing or reflux symptoms, and removal of some saline to loosen the band will reduce the amount of restriction. Correct adjustment of the band is essential for long-term success for weight loss. These adjustments are done in the office. A typical patient may require five to six adjustments during the course of the first three years. The first adjustment is not made until four to six weeks after the surgery.

There are many advantages of lap band surgery over other weight-loss surgeries. This surgery is very simple, it does not involve cutting or stapling of the stomach, which makes it reversible as the stomach returns to normal if the band is removed. It has a low mortality (1 in 2,000) with a short hospital stay and a quick recovery. The adjustment of the band does not require additional surgery. The procedure is purely restrictive with no malabsorption; therefore, there is less risk of developing vitamin or mineral deficiency.

Weight loss after lap band surgery is much slower compared to gastric bypass surgery. Sixty percent of every 100 pounds that the patient is overweight is expected to be lost after a three- to five-year period. The gastric bypass patient can expect 70 percent excess weight loss at the 18-month interval. Additionally, a majority of the associated comorbid conditions—particularly diabetes, blood pressure, high cholesterol, sleep apnea, fatty liver, and incontinence—are cured after the surgery.

Like any other procedure, lap band also has specific risks associated with it. Being a foreign body, there is always a risk of infection, which may require its removal. Sometimes the band may slip from its position or may erode into the stomach, requiring additional surgery. Malfunction of the port or leakage from the tube or the balloon has also been reported requiring the replacement of the device. However, with modifications in the device, these complications have been reduced to a minimum. Today, the lap band is considered the safest and least invasive of all weight-loss surgeries.

SEE ALSO: Bariatric Surgery in Children; Bariatric Surgery in Women; Multidisciplinary Bariatric Programs; Roux-en-Y Gastric Bypass.

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L.A. Weight Loss

L.A. WEIGHT LOSS is a commercial, nonmedical weight-loss program founded in 1987. The program promotes lifestyle change through intensive individual counseling and use of their packaged foods to supplement ordinary food consumed as part of an individualized diet plan. L.A. Weight Loss claims to be the fastest growing center-based weight-loss program in the world and has over 900 locations in the United States, Canada, Puerto Rico, Mexico, Australia, and Costa Rica. Individual L.A. Weight Loss centers are franchises and information about investing in a franchise, or working in one of the centers, is available through the program's franchise Web site.

Promotional materials for L.A. Weight Loss emphasize that their clients lose weight more quickly than participants in similar programs, and Womble and colleagues report that losses of two to three pounds per week during the initial phase of the program are advertised. Because this is more rapid weight loss than is commonly recommended, L.A. Weight Loss screens potential clients for contraindicated medical conditions, and clients with preexisting conditions must obtain approval from their primary care physician before enrollment. The program also has a medical director, currently (2007) Boyd D. Lyles, Jr., M.D. Intensive individual counseling is provided (at least three times per week during the initial phase). Counselors may be nurses, nutritionists, or other professionals, but most often are laypeople trained by L.A. Weight Loss. According to Womble and colleagues, participants are assigned a meal plan that provides 1,100–1,900 calories per day, devised by the staff dietitian with consideration to their age, sex, weight, and medical conditions. They are also counseled about serving and portion sizes and are told specific foods to eat or avoid; it is also recommended that they consume two soy-based supplement bars called L.A. Lites with each meal. Once reaching their goal weight, participants enter a stabilization phase in which they are encouraged to eat a greater variety of foods, and caloric levels are adjusted for weight maintenance.

Very little specific information about L.A. Weight Loss Centers is available from the program Web site. The main features in April 2007 were before and after pictures of actress and comedian Whoopie Goldberg with the endorsement "I lost 40 lbs in 23 weeks!" accompanied by the statement, "As people vary, so does their weight loss. You may lose more or less than Whoopie," a search engine to locate a center by ZIP code, and an 800 number to call for information. The franchise Web site offers more information about the financial success of the company, and investment and employment opportunities. According to Womble and colleagues, as of 2002, no scientific data were available evaluating the success or safety of L.A. Weight Loss programs.

The company has been accused of false advertising and high-pressure sales tactics, and legal judgments have been issued against it in several jurisdictions. In New York State, Attorney General Eliot Spitzer reached a settlement with the company in 2002 which required L.A. Weight Loss Centers to pay a $100,000 fine plus $10,000 to cover the cost of the investigation, post a performance bond, revise its contracts to disclose full costs of the program and make explicit the right of customers to cancel, and to make restitution to New York State customers who were refused refunds to which they were entitled. Among the charges were that L.A. Weight Loss Centers advertised that its program could be purchased "for only $7 per week," while the true cost was typically three to four times this amount and, in addition, the program could not be purchased on a weekly basis but required the annual fee to be paid in advance. The company also required participants to purchase a supply of L.A. Lites nutrition bars, which were originally described as optional.

In a similar judgment in Washington State in 2006, L.A. Weight Loss and NWM (a franchise which uses L.A. Weight Loss products) were ordered by the state Attorney General's office to provide $100,000 in refunds to customers who were misled or pressured into buying energy bars and other nutritional supplements, and to pay $90,000 for attorney's fees and future monitoring. This was the second judgment against L.A. Weight Loss in Washington State; the first, in 2005, required the company to pay $75,000 in legal costs and issue refunds to over 1,500 customers.

SEE ALSO: Atkins Diet; Jenny Craig; Medifast; NutriSystem; Slimfast; South Beach Diet; Weight Watchers; The Zone.

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LDL Receptors

THE DISCOVERY OF the LDL receptor over 30 years ago has helped to elucidate the complex mechanisms underlying cholesterol homeostasis in the body. The LDL receptor is a protein whose primary function is to transport cholesterol into cells. Low-density lipoprotein (LDL) is a protein that carries cholesterol and is often referred to as the bad or lousy cholesterol. The LDL receptor removes both LDL particles and intermediate-density lipoproteins (IDL) particles from the blood through a process known as receptor-mediated endocytosis. The LDL receptor will interact with an LDL particle at the cell surface. This is a favorable location for this interaction because the pH is neutral in this area. Once the LDL particle binds to the receptor, an LDL particle-receptor complex is formed and then the complex is brought into the cell by endocytosis.

Once the LDL particle-receptor complex enters the cell it is released as an endosomes, an organelle within the cell. Within the endosome, there is a decrease in the pH (it is more acidic than the outside of the cell) and this change in acidity results in the receptor releasing the LDL particle from the LDL particle-receptor complex. The LDL particle will then be further broken down for the cell to use the cholesterol. There are two different fates for the LDL receptor after it releases the LDL particle. The receptor may either be destroyed or recycled back to the cell surface to be used again to bring in more LDL cholesterol. This process occurs primarily in the liver, the major organ for the removal of cholesterol from the body.

The levels of free cholesterol within the cell can regulate the synthesis of the LDL receptor. When there are low levels of cholesterol in the cell, there is an increase in the transcription of gene that makes the LDL receptor. One factor that regulates the LDL receptor is known as sterol-responsive element binding protein-2 (SREBP-2). Statins are a class of medications that are widely utilized to lower total and LDL cholesterol. One way in which they work is to increase the synthesis of the LDL receptor. This will allow for the removal of excess cholesterol from the body. Conversely, when cholesterol levels are high in the cell, the cell decreases the synthesis of the LDL receptor.

It has been discovered that some people may have inherited mutations for the gene that makes the LDL receptor. This would result in a condition known as familial hypercholesterolemia (FH) or high cholesterol. Patients who suffer from FH have elevated levels of LDL cholesterol, sometimes reaching nearly 700 mg/dl (normal values of LDL cholesterol should be < 130 mg/dl). Patients may be homozygous or heterozygous for the defect in the LDL receptor. Patients who are homozygous will have much greater LDL levels and often die in childhood. This mutation is less common than patients who are heterozygous. Patients who are heterozygous for the LDL receptor mutation are at an increased risk of developing heart disease in adulthood. These patients are the ones who typically receive cholesterol-lowering medication.

SEE ALSO: Elevated Cholesterol; HDL Receptors.

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Leptin

LEPTIN IS A hormone that is produced in fat cells and functions to regulate body weight and eating. It was the first hormone discovered within fat cells and is part of the class of hormones now known as adipokines (hormones produced in the fat). Other hormones made in fat cells include adiponectin and resistin. In the mid-1950s, a mouse was discovered at Jackson Laboratories that ate a large amount of food and performed very little physical activity and became very obese. The gene required for the mouse to become obese was known as the ob gene. Mice that had two ob genes were the only mice to become obese and subsequently these animals were named the ob/ob

mice. Leptin was discovered in the mid-1990s as the hormone that was responsible for the obesity observed in the ob/ob mouse. The word *leptin* comes from the Greek word *lepthos*, which means thin.

Leptin was originally hypothesized to regulate how much food a person eats and how much physical activity he or she performs. The ob/ob mouse was very obese and had no leptin in its body. Once these leptin-deficient mice were given an infusion of leptin, they stopped eating and lost their extra body weight. It was later determined that when a normal mouse (not the ob/ob mouse) had a large amount of adipose tissue (fat cells), then it would have high levels of leptin. When leptin levels are high, the animals' appetite would decrease (they would eat less food), and the amount of physical activity they use would increase (they would move more in their cages). Alternatively, when leptin levels are low (in a thin animal), the animals' appetite would increase (they would eat more food) and the amount of physical activity they use would decrease (they would move less in their cages).

Leptin is believed to travel from fat cells to the brain. A leptin receptor is located on the brain which helps move leptin across the blood–brain barrier. Once in the brain, leptin is able to regulate if a person is hungry or if he or she satiated. Leptin works in the hypothalamus region of the brain and affects neuropeptide Y pathway, agouti related protein (AGRP) pathway, and the pro-opiomelanocortin (POMC) pathway as well as other pathways. Originally, leptin was believed to only be produced in fat cells, but small amounts of leptin are produced in other tissues as well such as the stomach, intestines, and the brain.

Once leptin was discovered in 1994, it was hypothesized that people who were overweight or obese must have had very low levels of this hormone and that is why people who are overweight or obese eat so much. Many biotechnology and pharmaceutical companies were interested in making a synthetic leptin as a treatment to control body weight. However, the opposite hypothesis was observed. People who were obese or overweight had more leptin than people who were thin. It was then proposed that overweight and obese people might be leptin resistant, that is their tissues were no longer responsive to the actions of leptin. Another proposed hypothesis was that leptin primarily works when people do not eat. When a person does not eat a sufficient amount of food, they may enter a starvation-like state. During these starvation conditions, the leptin begins to work and tells the body that it needs to eat.

Leptin deficiencies in humans, like the ones observed in the ob/ob mice, are very rare. There have been a few documented medical cases of members from the same family displaying similar symptoms that the ob/ob mice displayed. The children with the deficiency would eat large portions of food and do very little exercise. They were extremely obese. Treating these leptin-deficient patients with synthetic leptin alleviated all of their symptoms. These patients would have a reduction in body weight and normalization in the amount of food they ate and the amount of physical activity in which they engaged.

Additional studies were performed on the relatives of the children who were leptin deficient. These relatives were heterozygous for the leptin gene (i.e., missing some of the genetic information to make leptin). They had a lower amount of leptin levels compared to individuals with similar amount of body weight and body fat. When these individuals were given synthetic leptin as a treatment, they also had a reduction in the amount of food they ate, increase in physical activity, a decrease in body weight and decrease in body fat percentage. Overweight and obese subjects who were given very large doses of leptin had very little to modest effect in regard to changes in eating, physical activity, body weight, and body fat.

In addition to eating and physical activity, leptin has the ability to affect and/or regulate many other systems in the body. Leptin-deficient mice are often sterile. This is because leptin deficiencies may affect the synthesis of the sex hormones (i.e., testosterone and estrogen). When the leptin-deficient mice were given the synthetic leptin, the infertility disappeared. Leptin plays an essential role in the growth of neurons in the brain.

AMP-activated protein kinase (AMPK) is an essential molecule that is often said to be a master switch of metabolism. When it is turned on, the use of fatty acids in the body is increased and when it is shut off, fatty acid utilization decreases. Leptin plays a role in turning off AMPK. Leptin also plays a role in regulating glucose (sugar) and insulin metabolism in the body. Animals that were insulin resistant (prediabetic) and given leptin infusions had a normalization of their blood glucose and became more insulin sensitive. It is believed to affect glucose and insulin metabolism through its effects on the liver.

Since its initial discovery in the mid-1990s, leptin has proven to be a very interesting hormone to study. It is clear that it plays a role in regulating how much food a person may eat and how much energy they will use. Additionally, leptin can influence many other systems in the body and its function continues to be elucidated.

SEE ALSO: Adiponectin; Leptin Supplements; Ob/Ob Mouse.

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Leptin Supplements

OBESITY IS A major public health problem, and the pharmaceutical industry has recently rushed to find a scientific breakthrough in a cost-effective treatment for individuals who are already obese. For many obese individuals trying to lose weight, exercise and sound nutrition are not enough or are exceedingly difficult. A pharmaceutical approach, thus, is increasingly being sought as an adjunctive therapy to lifestyle changes.

DISCOVERY OF THE LEPTIN PROTEIN AND THE PROMISE OF LEPTIN REPLACEMENT

The leptin protein was discovered in the 1970s in different strains of mice that either had decreased production of, or a decreased response to, leptin. Researchers took a group of obese mice and noticed a mutation common among the mice that resulted in the inability to generate a protein called leptin. Further, subsequent studies in mice showed that when recombinant leptin was injected into these obese mice, there was a marked decrease in food intake and the mice lost weight.

This discovery sparked frenzy among the pharmaceutical industry. There was now a race to formulate a leptin pill that could ultimately reduce America's burgeoning public health problem. There was a problem, however. Some experiments in humans showed that after six months of giving leptin replacement, many of the subjects began to gain weight again. The roadblock was intensified by the fact that not all obese individuals, in fact only a small minority, were deficient in leptin. In addition, it appeared that obese humans were partially resistant to leptin, not just deficient in production to leptin. So, giving leptin as a supplement did not have much of an effect. In short, leptin was only part of the puzzle.

The advances made with leptin research have advanced current approaches. Current advances are focused on a more comprehensive approach. For example, Rimonabant, an experimental drug that has different biological actions than leptin, is being currently tested not only for the treatment of obesity but also for smoking, high cholesterol, and high blood pressure. The discovery of leptin was one impetus that increased the search for other medications that could either decrease appetite, increase energy expenditure, or a combination of both. The initial discovery of leptin came at the right time, just as public health professionals were really beginning to warn of the dangers of the increasing rates of obesity. Therefore, the research involving leptin had a unprecedented audience. While much of the research involving leptin did not hold the promise of its beginning, leptin research has paved the way for current advances.

SEE ALSO: Leptin; Rimonabant.

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Liking vs. Wanting

EATING FOOD IS normally regarded as a rewarding and satisfying experience. "Liking" and "wanting" are emerging constructs in a conceptual approach to food reward where separable processes of affect and motivation can be viewed as a major influence on en-

ergy intake. Liking and wanting achieve importance in light of the recognition of the contrast between homeostatic (regulation of appetite signals) and hedonic (of or relating to pleasure) processes that control eating. In our obesigenic environment there are good reasons for believing that nonhomeostatic processes linked with hedonics are responsible for overeating. Therefore, liking and wanting form part of the nonhomeostatic approach to the control of food intake.

Many people would assume that liking and wanting are identical phenomena, both of which signify a positive attraction to food. The logical view is that liking and wanting co-vary in a natural two-way sequence. In behavioural terms we assume that a change in liking will lead to proportional adjustments in wanting and, likewise, differences in wanting will predict changes in liking.

Therefore, some researchers suggest that a clear behavioural distinction might not be possible. However, there are strong grounds for recognising that liking and wanting can be clearly dissociated and have distinct identities. This means that they have much greater resolving potential for understanding the role of hedonics on eating and therefore on overconsumption. Thus, the issue of liking vs. wanting is concerned with the functional significance of these two distinguishable processes, operating within the non-homeostatic (hedonic) domain, for overconsumption and weight regulation in humans.

Liking and wanting appear to have separate and disproportionate roles in promoting overconsumption. In terms of liking, some individuals at risk of weight gain may experience an exaggerated hedonic response to palatable foods, so that foods are enjoyed more and therefore eaten in greater amounts for longer periods of time. Conversely, susceptible individuals may have a diminished ability to experience pleasure from food and therefore consumption of palatable food is driven up to satisfy an optimum level of stimulation. Processes of wanting may also bring about vulnerability to weight gain through increased reactivity towards cues signalling the availability of food (sometimes referred to as incentive salience). Moreover, a reduced ability to resist the motivation to eat when satiated may promote nonhomeostatic overconsumption.

A widely held notion is that wanting rather than liking may be the crucial process in maintaining an obese state. For example, research on chronic drug abusers indicate that repeated drug taking behaviour and strong motivation to obtain a "fix" can occur in the absence of any pleasant sensations during ingestion. Moreover, food liking is often a rather stable characteristic within an individual and appears relatively uninfluenced by increasing weight status. The implication is that liking may be important in establishing the motivational properties of food, but once these are retained it is the upregulation of wanting in an obesigenic environment—insensitivity to homoeostatic signals but overreactivity to external cues that promotes overconsumption by influencing what and possibly how much is eaten from moment to moment.

Finally, it should be noted that under normal circumstances, liking and wanting are assumed to covary, and in humans, few studies have been conducted to demonstrate that these processes can be differentiated. A convincing argument for the existence of two separate processes is that they can be influenced by distinct neural pathways in the brain.

SEE ASO: Addictive Behavior; Appetite Signals; Dopamine; Food Reward; Opioid Palatability; Taste Reactivity.

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Lipoprotein Lipase

LIPOPROTEIN LIPASE (LPL) is the enzyme responsible for the hydrolysis (breakdown) of triacylglycerols (TAG, or dietary fat) from lipoprotein carrying molecules into nonesterified free fatty acids and 2-monoacyglycerol for incorporation into tissues such as adipose, heart, and skeletal muscle. LPL plays an important role in lipid metabolism and

dysregulation of this enzyme has been implicated in pathologies associated with obesity, including cardiovascular disease.

Within adipose tissue, skeletal muscle, and heart, LPL is located on the capillary endothelium and is attached to the cell surface by heparin sulfate proteoglycan chains. LPL catalyzes the rate-limiting step in the breakdown of TAG from lipoproteins (proteins which transport lipids around the body) so that free fatty acids can be taken up by other target tissues. Two examples of lipoproteins that transport TAGs are chylomicrons and very low density lipoproteins (VLDLs). Dietary TAG are incorporated into chylomicrons and secreted by the intestine, while endogenous TAG is incorporated into VLDL and secreted by the liver. Upon secretion, the lipoproteins migrate to the skeletal muscle, adipose tissue, or the heart.

Apolipoprotein C2, which is present on the lipoprotein particles, serves as a cofactor for the TAG hydrolysis action of LPL. This reaction is necessary for cellular utilization of TAG as the lipoprotein molecules are normally too large to cross the capillary endothelium. Once inside the cell, the fatty acids and monoacylglycerol chains are either re-esterified (put back together) as TAGs for storage, or they are broken down further for energy needs.

Dysregulation of LPL in humans is associated with increased risk for coronary heart disease. Reduced activity of LPL is associated with hypertriglyceridemia, which is a risk factor for heart disease. Conversely, enhanced activity of LPL results in the increased release of remnant lipoproteins in circulation, which are subject for uptake by inflammatory macrophages, which may increase risk for atherosclerosis. In addition, LPL is expressed in macrophages from atherosclerotic lesions. Macrophage LPL activity is proposed to be proatherogenic, promoting uptake of lipids into the macrophages, which become incorporated into atherosclerotic lesions and subsequently increase the size of these lesions.

SEE ALSO: Atherosclerosis; Inflammation.

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Liquid Diets

LIQUID DIETS ARE diets that are almost exclusively used for medical purposes. They are diets that restrict intake to just liquids such as tea or juices.

Before and after medical tests, surgery, births, and in other situations, liquid diet can be very beneficial. In general, physicians do not recommend a liquid diet unless the patient is to be subject to procedures such as a colonoscopy or sigmoidoscopy.

A clear-liquid diet before surgery or tests can serve many functions. It aids in flushing the bowels. It also decreases the strain on the digestive system. It also supplies some nutrients. Physicians will usually supply patients with a fact sheet before beginning a liquid diet. The fact sheet will describe in detail how to proceed, what can be drunk, and the length of time to follow the diet.

Liquid diets are frequently used as a part of therapy following weight-loss (bariatric) surgery. This type of surgery uses a lap band or a gastric bypass to change the nutrition absorption of the patient. After the surgery, the patient will normally be given just liquids. On the first day, a liquid diet limits the intake to sips of clear liquids. The clear liquids are liquids such as apple juice, orange juice, water, flat diet sodas, sugar-free jello, broth, or other similar liquids. The next day, the patient normally moves to semiliquids.

Bariatric patients may be put on a liquid diet for as long as 10 days following the procedure. In very extreme cases, obesity may be treated with an all-liquid diet. Although successful at first, a liquid diet as treatment for obesity must be constantly monitored by a physician to prevent negative side effects from developing.

For long-term loss of excessive weight, liquid diets are not a very good option. A major reason is that it does not teach the obese person the actions and decisions that must be developed into habitual behaviors that are necessary for healthy eating. Obese individuals

typically perform better on diets that can more easily be incorporated into "real life" situations. Because consumption of all liquid diets, such as SlimFast or Optimum shakes, without intake of healthful meals as prescribed with these diets, do not train patients to eat regularly, they are not likely to be successful for the long-term.

Small weight losses are possible for short periods through the use of a clear liquid diet. Even more short-term losses are possible with the use of juices. However, a juice fast will lack proteins and fiber. The result is that some gains are made that may be quickly lost with the return of eating.

Experience suggests that the best that can be achieved by the use of liquid diets is the loss of a few pounds.

Protein shakes can also be a part of a liquid diet. These may help to eliminate weight in the short term. However, unless other tactics are employed such as more rest, more exercise, and a radical modification of a solid-food diet so that it is oriented toward moderate portion of vegetables, fruits, lean meats, and small portion of carbohydrates, the individual will rapidly regain weigh lost in the short term.

Experience suggests that the best that can be achieved for use of liquid diets is the loss of a few pounds. Several of these may be simply the weight of the passing of feces that is not replaced with new solid food.

Popular culture has promoted the idea that fluids can cleanse the body's systems of impurities. Claiming that the liver in particular can be purged of impurities encourages some to attempt liquid diets. Generally speaking, the elimination of most processed foods from a diet is beneficial. However, extreme use of a liquid diet will exclude nutrients including essential vitamins and minerals. These are required for good health.

Extreme dieting with a liquid diet runs the risk of negative health consequences. It opens the individual to reduced resistance to disease in the quest of a weight loss that is small compared to the possible negative consequences. A better strategy to employ is to use a liquid diet as a meal replacement. Regular meals in dietetically sound proportions can be balanced with a "meal" of liquids. However, the liquid meal replacement diet will fail it the solid meals are not regulated with good portion control.

SEE ALSO: Diet Myths; Dietary Restraint; Dieting: Good or Bad?

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Loneliness

Unlike related states of anxiety and depression, loneliness occurs within the normal range of functioning, across the life span.

FREQUENT, INTENSE, AND enduring loneliness can have far-reaching health implications. Many of its sequelae may complicate health conditions of obese patients through physiological, emotional, social, and behavioral means. Unlike related states of anxiety and depression, typically viewed as pathological, loneliness occurs within the normal and even healthy range of functioning, across the life span. Loneliness has been identified in children, adolescents, and adults. Males and females across every race and culture experience loneliness at similar rates, although some interesting variations have been identified, for example, coping strategies may vary.

Approximately 80 percent of survey respondents indicated being lonely occasionally, 15 percent much of the time, and 5 percent reported never feeling lonely. These rates corroborate the ubiquitous nature of loneliness and support the notion that it can be typical of the human condition. However, individuals who report being lonely much of the time may represent an unhealthy subgroup. A general definition is necessary, before further discussing pathological loneliness.

A definition of loneliness includes affective and cognitive aspects of the experience; feelings (e.g., emptiness, sadness), situations (e.g., being alone), and cognitions (e.g., I'm all alone). Here, loneliness will be defined as a negative subjective state associated with the perception that one's interpersonal relationships are inferior in number and/or quality desired for adequate social connection or belonging. This definition highlights the interplay of emotional and cognitive aspects of loneliness.

The cognitive aspect (i.e., discrepancy between desired and perceived relationships) helps distinguish it from other negative emotional states, such as depression. It is also important to note that the objective (or actual) number or quality of social relationships is not included in its definition. This emphasizes the phenomenological aspects of loneliness and differentiates it from solitude or simply being alone, in fact, time alone is not correlated with loneliness. It is the acceptability of the social network rather than the objective quality/quantity that determines the affective state.

Although present throughout the life span, loneliness may be more prevalent at different developmental stages. Some studies indicate adolescents report the highest rates, while others indicate young adults with commensurate rates. Increased focus on peer relationships and social status may raise both the expectations and value of social belongingness, creating higher rates of loneliness when these needs are not met. Cross-sectional studies found elderly community members report less loneliness, even though social network size may decrease in aging populations. A cognitive explanation for this resilience may be changes in expectations, cultivating some evolution of preparedness or acceptability. Another hypothesis proposes that the quality of relationships at this age is most critical, buffering against decreased quantity.

There may be a tendency to underestimate loneliness in males when using measures with high face validity (i.e., instruments including the word "lonely"), as males may be less likely to endorse such items. Overall consistent gender differences have not been found; males and females are similar in terms of perceived social connectedness. However, mixed results have at times been discovered, indicating small variations may be present at different ages. Few gender differences have been observed in children and adults, but

some studies indicate adolescent males may report more loneliness than females. Other studies indicate no differences; therefore, future studies are necessary before concluding adolescent males are the loneliest demographic.

The 20-item University of California, Los Angeles (UCLA) Loneliness Scale is the most common loneliness measure. A self-report questionnaire is well suited for measuring this construct. Some analyses support multiple dimensions of loneliness, but this scale is generally used to represent a single factor, trait loneliness, which is the frequent experience of loneliness. Trait measurement enables scientists to attribute characteristic thoughts, behaviors, and emotions to lonely "types," those experiencing loneliness across time and situation.

Lonely individuals' (i.e., those high in trait loneliness) number of friends and the amount of time spent in social situations may not distinguish them from their non-lonely counterparts. However, they may differ in other important ways. As already noted, lonely individuals are more likely to experience depressive symptoms. There is abundant data indicating that depression results in work absenteeism, decreased productivity, increased healthcare costs, is related to poorer prognoses of many conditions, and possibly plays a causal role in the development of certain diseases, such as coronary heart disease. Data indicate that loneliness is not only predictive of depressive symptoms, but also of many of the same deleterious health effects even when holding depressive symptoms constant. Impaired cardiovascular and immune function, higher rates of substance use (mixed results with alcohol), sleep inefficiency, anxiety, suicide, overutilzation of healthcare services, and fewer self-care behaviors may result from chronic loneliness.

Even though loneliness does not predict blood pressure differences, the mechanisms by which lonely and nonlonely achieve blood pressure may differ in crucial ways. Evidence suggests that high trait loneliness is indicative of higher total peripheral resistance and lower cardiac output. This finding may elucidate mechanisms of cardiovascular strain, which result in long-term health problems. There is further evidence that loneliness is predictive of blood pressure increases, as individuals' age. Therefore, high trait loneliness may warrant further study and clinical consideration.

The number of mechanisms responsible for the effects of loneliness is unknown. Unfortunately, regardless of whether the mechanisms are biological, social, behavioral, psychological, or a combination, there is a clear circularity of loneliness and its maladaptive sequelae. Loneliness may precede depressive symptoms, depressive social withdrawal behaviors lead to more social isolation and fewer opportunities to develop social skills, which in turn hinders social networking. This pattern of withdrawal increases isolation, which may lead to negative cognitive cycles.

Loneliness is also associated with negative thoughts about the self and others. The circularity of cognitions may maintain or exacerbate loneliness. As the lonely person cycles through the behavioral loop of withdrawal and isolation, it is reasonable to assume negative evaluations of the self and the future become more common as social failures accumulate. In fact, these evaluations, along with greater distrust and suspicion have been frequently observed in lonely individuals. This decreases the sense of group belonging, and social opportunities are more likely to be viewed as threatening and are therefore avoided. Lonely individuals become more distrustful and feel more threatened by socialization. Distrust and perception of threat further inhibits initiation of social activities, preventing opportunities for social skill development. Unfortunately this cycle may result in more threatened, anxious, isolated, and skill deficient lonely persons.

Loneliness has been associated with obesity. Recent research indicates that overweight children are more likely to become both the victims and perpetrators of "bullying" behavior. Unfortunately, both victims and perpetrators are likely to struggle with social rejection, which studies indicate lead to decreased will power. Successful diabetes management and caloric restriction, skills needed by many obese patients, may thereby become less likely.

Unlike obese females, obese males may experience loneliness at the same rates as normal-weight males; however, loneliness will still impact obese men's health differently. Studies indicate that when a nonrestricting person becomes lonely, he/she may eat less, while a restricting person (e.g., person on weight-loss plan) will overeat. Therefore obese men's weight loss may be impeded by loneliness even if it occurs at "normal" frequencies. In addition, it is clear that obese males

and females as children and adults face negative bias and discrimination, providing social, vocational, educational, and economic challenges.

Although both loneliness and obesity are predictive of a number of future negative outcomes, research has not determined the precise relationship obesity and loneliness in combination produce. One finding does suggest that depression is more likely when loneliness is attributed to physical appearance. In general it can be said loneliness and obesity increase the chance of negative medical outcomes, but to what extent there is overlap, additive, or synergistic effects remains unclear.

Loneliness poses a significant barrier to the treatment of obesity. It is one of the most common overeating triggers (antecedents), is associated with overeating for those attempting to restrict calories, and produces passive coping strategies. Most weight-loss treatments include both caloric restriction and active coping strategies. Therefore, loneliness may interfere with successful weight loss and maintenance. Given the paucity of research on loneliness and specifically its role in the development, maintenance, and treatment of obesity, further finding and research is clearly warranted.

SEE ALSO: Self-Esteem and Obese Women.

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Low Birth Weight

NEWBORNS WHO WEIGH less than 5 pounds, 8 ounces, are classified as being of low-birth weight (LBW). (A related category, small for gestational age [SGA], is sex specific and includes children who are less than the 10th percentile for birth weight and gestational age.) LBW, a marker for fetal underdevelopment, has been linked to a variety of causes, for example, maternal tobacco and alcohol use, maternal age, poor maternal nutrition, lower socioeconomic status, and so forth. Moreover, LBW newborns face significant health risks as adults, the most significant of these risks include comorbidities associated with adult obesity. Numerous studies, ranging from animal-based models to diverse population-based cohort studies that encompass decades, have addressed the health impacts of LBW throughout the life cycle.

Recent research has associated LBW-related obesity with several causes. Animal-based studies have highlighted the role of maternal nutritional and hormonal levels on predisposing factors such as tumor necrosis factor-α upon appetite levels and metabolic efficiency among offspring. Investigators have also examined the impact of caregiver practice among LBW infants. Healthcare professionals, significant others, and caregivers may overfeed LBW (or SGA) children to help him or her to achieve a normal developmental trajectory. Accelerated postnatal development, existing research suggests, increases subsequent risk for central obesity, dyslipidemia, Type 2 diabetes, insulin

The association of LBW and obesity raises important disease prevention issues for healthcare professionals.

resistance, cardiovascular disease, sympathetic nerve dysfunction, and sensorineural hearing loss, findings consistent across diverse populations in the Americas, Europe, Asia, and Australasia. The association of LBW and obesity raises important disease prevention issues for healthcare professionals. Assumptions about the benefits of developmental catch-up for LBW infants may require reconsideration in light of potential health outcomes in adulthood.

SEE ALSO: Infant Weight Gain and Childhood Overweight; Pregnancy.

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Low-Calorie Diets

A LOW-CALORIE DIET can be one of the simplest weight-loss strategies for a patient to follow by consuming less food without having complicated dietary restrictions. A successful low calorie diet can contribute to weight loss and health benefits, and decreases long-term health risks. The effectiveness of the low-calorie diet depends ultimately on the willingness of the patient to achieve weight loss.

As far back as 500 B.C.E., the Spartan culture utilized low-calorie diets to instill discipline and health. In order to maintain their lean athletic builds, the Spartans exhibited self-control and became immune to hunger cravings by eating less during times of restriction. Similarly, the Okinawans, a population in Japan known to be one of the longest-living populations on Earth, maintain the philosophy, "Hara Hachi Bu," which means "eat until the body feels 80 percent full."

Current popular diet trends tend to focus on restricting a certain food group such as carbohydrates, protein, or fats. Because the perfect balance of these macronutrients has yet to be established, it is difficult for dieters to ascertain which food group restriction may be the most beneficial. Therefore, limiting overall calories may be the simplest way to lose weight. In addition to reducing the number of calories, the quality of food consumed will contribute to optimal weight loss. This means that alcohol, high-sugar beverages, and other high calorie sweets should be eliminated because of their low nutritional value.

Before beginning a reduced calorie diet, a patient should seek the advice of a physician to take into consideration individual health precautions. A doctor can advise the dieter on how many calories he or she should limit in his or her personalized diet plan. Approximate caloric demands can be calculated for each individual on the basis of age, sex, and ideal weight.

To maintain a set weight, approximately 22 kilocalories per kilogram of body weight a day is necessary. Most low-calorie diets range between a low-end of 1,000 calories/day to approximately 1,500 calories/day. Anything less than 800 calories is considered a very low calorie diet (VLCD).

To further optimize and sustain weight loss on the low calorie diet, a patient should participate in an exercise regimen. An effective strategy would be to establish realistic goals for each patient. Next, a meal guideline should be established, with calories counted for all foods to be consumed. A food journal can be used to track the actual intake of the individual and used to assess progress.

By restricting calories, an individual has the ability to lose body mass. This body weight will be lost as lean muscle mass and body fat. Another benefit of this dieting method is decreased plasma insulin levels, improved glucose metabolism, and decreased blood lipid levels. Lower levels of total cholesterol, low-density lipoprotein, high-density lipoprotein, and triglycerides are found as body weight decreases. If the weight loss is maintained as a lifestyle by consuming fewer calories, the risks of heart disease, diabetes, and possibly cancer also decrease.

Positive reinforcement from friends/family can contribute to a dieter's success. A healthcare provider can also assist in providing the necessary motivation and monitoring. Psychologically, the patient may have to alter attitudes and feelings in regard to eating by self-restraint when food is available, positive thinking when the diet may be difficult, and ways to relax when stressful situations may cause a dieter to stray.

Often, dieters' expectations of weight loss are unrealistic, and dieters may be discouraged in the short term by the slow, gradual effects in physical appearance and body satisfaction. Because the amount of weight loss is dependent on multiple variables consisting of exercise, body type, genetic predisposition, number of fat cells, basal rate metabolism, hormones, and others, diets will have different effects on different people.

Another major problem lies in the physiological and psychological mechanisms with the feeling of satiety. If the body is consuming a smaller amount of calories, hunger may override the desire to eat less. Additionally, metabolism slows to compensate for the loss of caloric intake as a bodily defense mechanism.

In addition to reducing the number of calories, the quality of food consumed will contribute to optimal weight loss.

The daily calorie counts may also prove to be too tedious to follow. Patients may not realize that it takes time for the stomach to send a message to the brain that it is full, and tend to overeat. Although reducing calories is the best way to lose weight, it is often challenging due to the factors mentioned above.

SEE ALSO: High-Carbohydrate Diets; High Protein Diets; Low-Fat Diets; Prevention; Variety of Foods and Obesity; Very Low-Calorie Diet.

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Low-Density Lipoprotein

LOW-DENSITY LIPOPROTEIN (LDL) is cholesterol packaged in a protein and phospholipid coat. Cholesterol is insoluble, so it must be encased in this coat to facilitate transport in the blood. LDL is the major cholesterol carrier in the blood. It circulates throughout the body and is available to all cells.

Cholesterol is a waxy substance found in the cell membranes of all animal tissues. It is actually a steroid and not a fat. Cholesterol aids in the production of bile in the liver as well as being a component in the production of hormones including estrogen and testosterone. The body is designed to make adequate cholesterol, called endogenously produced cholesterol. However, additional cholesterol may come from dietary sources, including eggs, milk, cheese, butter, meat, fish, and poultry. Cholesterol is not found in plants, so high-fat vegetables and fruits like avocado and nuts do not have cholesterol.

As LDL circulates in the blood, it may adhere to arterial walls which feed the heart and brain. It forms plaque and may result in a blockage in the artery which may lead to atherosclerosis. A blockage in coronary arteries may result in a heart attack while a blockage in the carotid may result in a stroke.

Diets high in saturated fat, trans-fat, and cholesterol increase LDL. Saturated fats are primarily from animal sources of foods. Coconut oil, palm oil, and palm kernel oil are also saturated fats. Trans-fats are fats that have been hardened through hydrogenation, a process in which hydrogen atoms are forced onto the chains of carbon that make up fatty acids. They include stick margarine, shortening, commercial frying oil, and high-fat baked goods. Not all dietary cholesterol becomes blood cholesterol. It is the amount of total fat intake, especially saturated fats and trans-fat which may influence cholesterol levels.

Overweight and obese individuals tend to have increased LDL. Lifestyle and genetic factors are the primary contributors to elevated LDL. Lifestyle factors include smoking, overeating, and inadequate physical activity. Blood cholesterol may rise after the age of 20. Gender may play a role as menopause in women decreases estrogen production which aids in keeping LDL low.

It is recommended that total blood cholesterol be less then 200 milligrams per deciliter (mg/dl). LDL should be lower than 130mg/dl. An LDL lower than 100mg/dl is recommended if coronary heart disease (CHD) has been diagnosed. There are no symptoms presented with high cholesterol; therefore, a fasting blood test of serum cholesterol to determine the total lipid profile will provide information on HDL, LDL, and triglycerides.

Treatment for high LDL usually begins with lifestyle changes by increasing activity, making dietary changes, and losing excess weight. A drug classified as a statin may be prescribed by a physician to lower LDL. A statin works by blocking an enzyme used in cholesterol production. Lifestyle adjustments in conjunction with statins may lower LDL if lifestyle changes alone do not work or if there is a family history of high cholesterol.

SEE ALSO: Atherosclerosis; Blood Lipids; Coronary Heart Disease in Women; Elevated Cholesterol; High-Density Lipoprotein; LDL Receptor.

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Low-Fat Diets

A TYPICAL AMERICAN diet contains somewhere between 35 to 40 percent of calories from fat. The 2005 U.S. Department of Agriculture (USDA) Dietary Guidelines for Americans recommend that healthy adults keep total dietary fat intake between 20 to 35 percent of calories from fat. They define low fat intake as no more than 20 percent of calories from fat. There are many different types of low-fat diets, each with their own benefits and drawbacks. Studies show that low-fat diets may reduce the risk of coronary heart disease and may help people lose weight.

Dietary fat is an essential macronutrient that supplies energy and essential fatty acids and acts as a carrier for fat-soluble vitamins such as A, D, E, and K. Fat provides 9 calories per gram which is more than twice the caloric density of carbohydrates or protein. In adipose (fat) tissue, it serves as an important energy storage source. When the body has depleted energy from carbohydrates, it begins to burn fat in a process called fatty acid oxidation. This occurs during long fasts and after about 20 minutes of exercise.

Certain segments of the population need to consume a low fat diet due to health concerns. These groups include people with gallbladder disease, fatty liver, fat absorption disorders, and fatty acid oxidation disorders. People with elevated cholesterol, hypertension (high blood pressure), and/or a history of heart disease are advised by the American Heart Association (AHA) to reduce saturated fat and cholesterol intake and keep total fat intake below 30 percent of total calories.

There are several popular low-fat diets, many of which have been around for decades. Vegan, vegetarian, and macrobiotic diets are plant-based diets that are usually low in fat because most animal products are avoided, but they do not intentionally restrict calories from fat. Very low-fat diets (no more than 15 percent calories from fat) have been criticized as being "too restrictive" and depriving the body of essential fatty acids like omega-3 polyunsaturated fatty acid. It is possible that intake of large amounts of fiber from very low-fat diets diet could cause gastrointestinal distress such as diarrhea. Also, very restrictive diets are difficult to maintain for Americans who travel often and frequently eat out at restaurants. However, low-fat diets have documented clinical studies showing reversal of coronary blockage and weight loss. They are one of the few popular diet plans with a strong scientific basis.

The Ornish Diet is a well-known and well-studied very low fat diet. Dean Ornish is a cardiologist who is Clinical Professor of Medicine at the University of California, San Francisco. He created the Ornish Lifestyle Modification Program, based on: (1) a very low fat, high complex carbohydrate diet rich in fruits, vegetables, whole grains, beans, and legumes, (2) regular exercise, (3) stress management, and (4) family/community support systems to maintain desired healthy behaviors. The Ornish Diet restricts fat intake to 10 percent of total calories and promotes consumption of complex carbohydrates. According to Ornish's book *Eat More, Weigh Less*, the typical American diet contains 40 percent calories from fat, 20 percent from protein, and 40 percent from carbohydrates. Ornish recommends eating beans, fruits, grains, and vegetables whenever you are hungry. Nonfat dairy products, egg whites, and nonfat processed foods are allowed in moderation. Foods to be avoided include simple sugars, alcohol, meats, oils, avocados, olives, nuts, seeds, fat-containing dairy products, and any processed food containing more than 2 grams of fat per serving.

Similar very low-fat diets have been developed by Drs. John McDougall, Terry Shintani, and Gabe Mirkin. McDougall developed the 12-Day Diet and the McDougal Plan for Maximum Weight Loss (MWL). Both diets are almost vegan, emphasizing grains, vegetables, fruits, and beans. The 12-Day Diet restricts high-fat foods and caffeinated beverages for 12 days. After 12 days, some of these foods may be consumed occasionally. The MWL program has the same restrictions but also restricts fruit juices and dried fruits. It advocates consumption of whole grains, vegetables, legumes, fruit (limit to two servings per day) and sparing use of salt and sweeteners.

Terry Shintani's diet is based on the USDA food pyramid, but the top three tiers (oils, meats, and dairy) are replaced with nondairy calcium foods and noncholesterol protein/iron foods. A daily diet would consist of 8–13 servings of whole grains, 3–5 servings of vegetables, 2–4 servings of fruits, 2–3 servings of nondairy calcium foods, and 2–3 servings of noncholesterol protein/iron foods. Examples of the nondairy

Studies show that low-fat diets may reduce the risk of coronary heart disease and may help people lose weight.

calcium group include calcium-rich greens, seaweed, tofu, and sesame seeds. Examples of noncholesterol protein/iron foods include lentils, beans, high-iron greens, seaweed, nuts, and seeds. Gabe Mirkin recommends no more than 20 grams of fat per day (under 10 percent calories from fat). About 15 grams of fat ("the basic 15") should come from unlimited servings of fruits, vegetables, whole grains, and beans, and up to five servings of low fat dairy, seafood, or refined grains. The remaining 5 grams can come from personal favorite foods that may not otherwise be part of the diet. High fat foods should be avoided.

The Dietary Approaches to Stop Hypertension (DASH) clinical studies showed that dietary modification could reduce hypertension (high blood pressure). It is recommended by both the AHA and National Cancer Institute. Like a low fat version of the Food Guide Pyramid, the DASH diet is rich in fruits, vegetables, complex carbohydrates and low fat dairy products. It is lower in fat, saturated fat, cholesterol, and sodium, and higher in potassium, magnesium, and calcium than the typical American diet. High levels of potassium, magnesium, and calcium are thought to be partially responsible for the observed benefits. A typical daily DASH diet would include 7–8 servings of grains, 4–5 servings of fruits, 4–5 servings of vegetables, 2–3 servings of low fat or fat-free dairy products, under 2 servings of meat, and 4–5 servings of nuts and seeds per week. The Therapeutic Lifestyle Changes (TLC) diet was initially developed by the National Heart, Lung, and Blood Association's National Cholesterol Education Program (NCEP) as the Step I and Step II diets. The TLC diet is designed for people with elevated cholesterol. It recommends restricting saturated fat intake to under 7 percent of total calories, and reducing total cholesterol to less than 200 milligrams per day. Total fat intake can be maintained from 25 to 35 percent, with 15 percent of calories from protein and 50 to 60 percent from carbohydrates. This range of allowable total fat intake puts the TLC diet on the border of being low fat, even though saturated fat and cholesterol are restricted. It should be noted that the 25 to 35 percent fat recommendation is intended to allow for increased intake of unsaturated fat in place of carbohydrates in people with metabolic syndrome or diabetes.

Populations with diets naturally low in total fat and saturated fat have a reduced risk for heart disease. For example, native Japanese living in Japan derive about 15 percent of calories from fat, migrant Japanese living in Honolulu derive about 33 percent of calories from fat, and migrant Japanese living in California derive about 38 percent of calories from fat. The latter two groups have higher incidences of cardiovascular disease and increased body weight. Several studies have shown low-fat diets along with other lifestyle modifications, such as regular exercise and dietary counseling, result in weight loss and reduced risk of cardiovascular disease. Low-fat diets have reduced caloric density compared to high fat diets and often provide less total calories even when caloric intake is unrestricted. This factor may explain why many people lose weight on low-fat diets. However, some people on long-term low-fat diets may eventually adjust their caloric intake to that of a high fat diet, resulting in no weight loss or even weight gain. This is especially true when people consume highly processed fat-free products that contain large amounts of simple sugars with equal amounts of calories as their high fat counterparts. Some people in the medical community believe the low-fat diets and fat-free products developed in the 1990s contributed to the current obesity epidemic in America. Despite recent trends toward low carbohydrate diets that are high in fat, including Atkins, Mediterranean, and South Beach diets, low-fat diets are still the mainstay of most commercial weight loss plans like Jenny Craig and Weight Watchers. They are still highly recommended by the medical community for weight management and reducing the risk of cardiovascular disease. Low-fat diets are not a cure for obesity, but when accompanied by exercise and other lifestyle changes, they may be a valuable tool for weight management.

SEE ALSO: High-Carbohydrate Diets; High-Protein Diets; Low-Calorie diets; Macrodiets; Very Low-Calorie diets.

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Macrodiets

THE MACROBIOTIC DIET is defined by philosophy based on well-being and nutrition. The dietary component is similar to a vegetarian diet with emphasis on the quality of the food consumed. The health benefits of the macrobiotic diet have gained recent positive attention, but falsely spread health claims also detract from the benefits it has to offer.

The earliest advocate for the macrobiotic diet was Hippocrates, the founding father of Western medicine. He promoted a diet that was the basis for a long and healthy life. The Greek term *macro* meaning *large*, and *bio* meaning *life*, equate to lengthening the lifespan via principles of balance (yin and yang) and nutrition.

George Ohsawa, a philosopher from Japan, brought his teachings of Japanese folk medicine to North America in the 1950s. Ohsawa created the term *macrobiotic diet* as a natural way of living. His teachings emphasize eating less-processed, high-quality, and home-grown foods to build and strengthen the community. Initially, he stressed a 10-stage diet, with each stage more restrictive than the former, but later changed it based on criticism by governmental health agencies.

While the bulk of a macrobiotic diet is vegetables, this diet also incorporates limited amounts of white meat or fish, which should also be organic, free-range, and local. The diet consists of whole-grain products, which comprise 50 to 60 percent of the diet. Organic vegetables (20 to 30 percent), beans and seaweed (5 to 10 percent), and soups (5 to 10 percent) make up the rest of the diet. Beverages, condiments, and desserts should be less than 5 percent of dietary intake.

While no specific food item is prohibited, certain foods are described as overstimulating (purely yang) or very lethargic (purely yin) to the body and mind. Foods considered exhausting include sugar, alcohol, honey, coffee, chocolate, refined flour, and most commercial food products. Dense foods felt to contribute to stagnation include poultry, meat, eggs, and refined salt.

Interestingly, many of the macrobiotic diet components match the 2005 Dietary Guidelines for Americans. They comply with the recommended focus on plant foods as well as consuming whole grains while limiting saturated fat, added sugars, and sodium. This is preventative for chronic diseases and decreases all-cause mortality.

Epidemiological studies have shown obesity prevalence rates are 29 percent for semivegetarian and vegan women, while lactovegetarian prevalence is 25 percent versus 40 percent in omnivores.

Critics of macrobiotic diets in children claim that the diet (and similar vegetarian diets) are lacking in essential nutrients for proper growth and development. This is valid if the diet is poorly planned or executed. Parents who follow the guidelines and provide proper vitamin supplementation should have normal growth and development in their children. The following nutrients

should be especially monitored in children due to their importance in facilitating growth and function: calcium, protein, iron, zinc, vitamin D, vitamin B12, riboflavin, vitamin A, and omega-3 fatty acids.

Macrobiotic diets have been incorporated into complementary therapies for cancer treatment with little benefit. Difficulties arose in cancer patients due to weight loss, cost of required foods, and noncompliance to the diet. The American Cancer Society also does not credit the diet as a cure for cancer as some proponents of the diet have widely stated. No scientific evidence to date has shown that the macrobiotic alone has any therapeutic benefits in the treatment of cancer.

The Kushi Institute of Massachusetts promotes the macrobiotic diet as a tool to fight cancer. Other than the general nutritional benefits of an improved diet, no scientifically controlled studies confirm cancer remission based on methods that are being taught (for a price) at weeklong programs or private counseling sessions. The effectiveness of these is evidenced only by cancer patient and physician testimonials on the Kushi Institute's Web site. The governmental research that the Kushi Institute cites may be statistically irrelevant and is not widely supported by the scientific community.

SEE ALSO: Nutrition and Nutritionists; Prevention; Women and Dieting.

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Magnetic Resonance Imaging Scans

MAGNETIC RESONANCE IMAGING (MRI) is a clinical tool that allows for imaging of soft tissue, bone, and organs. MRI is a noninvasive test in which a person goes into a doughnut-shaped machine. It functions based on several principles of physical science including magnets and radio frequency. Inside the MRI machine, there is a very large magnet. Once it is turned on, it causes a change in the position of the atoms inside the cells of the body. Through the use of computers, these changed position of atoms will produce a picture of the inside of the body.

MRIs are generally safe exams. They do not expose patients to radiation compared to other tests such as computerized tomography (CT) scans or dual energy X-ray absorptiometry (DEXA) scans. This makes MRI scans a useful test for certain populations such as pregnant women and children. However, because MRI machines use a very strong magnet, certain patients are prohibited from having MRI scans performed such as those who have metal implants. It is also very important that no metal objects such as gas tanks are in the room with the MRI scanner as they can harm the patient in the scanner.

MRI is primarily used as a medical tool to assess muscle injuries or identify tumors. MRI is an excellent tool to use in the study of obesity as well. MRI

scans permit the study of body composition, an important component in the field of obesity. Whole-body MRI is used to measure total body fat and body fat distribution. This includes measuring the different types of fat such as subcutaneous fat (fat below the skin), visceral fat (fat around the organs), and intermuscular fat (fat between the muscles). Measuring the distribution of fat has been stated to be just as important as measuring the total amount of fat. Elevated amounts of visceral fat and intermuscular fat are associated with the development of many diseases such as heart disease and diabetes. Measuring fat with an MRI scanner produces better results than many other body composition tools such as skin folds, bioelectrical impedance, and underwater weighing. In addition to measuring body fat, researchers can measure total skeletal muscle and type of skeletal muscle (i.e., slow-twitch and fast-twitch muscles). MRI scans are also used for the measurement of organs such as the heart, liver, and brain.

Recently, MRI scanners have been used for more advance measures of body composition such as functional MRIs and spectroscopy. Functional MRI is used to obtain advanced images of the brain. It is being used to learn how certain tasks are related to different regions of the brain, specifically in the field of obesity functional MRI is being used to study eating behavior. Spectroscopy is used to measure nonvisible fat, known as ectopic fat, inside the muscle and liver. Like visceral and intermuscular fat, ectopic fat has been associated with increased risk of developing heart disease and diabetes.

SEE ALSO: Computerized Tomography; DEXA (Dual Energy X-Ray Absorptiometry).

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Maternal Influences on Child Feeding

MATERNAL FEEDING PRACTICES appear to influence children's eating habits and thereby children's weight. Maternal obesity is one of the greatest risk factors for a child's development of obesity. Researchers have proposed a number of explanations to account for this strong link. Association between child's and mother's weight may be explained through a shared genetic predisposition to a certain body structure or eating style, hormonal differences transmitted during pregnancy, or modeling of eating behavior.

Additionally, many propose that mothers have a direct influence on their children's eating habits through

Mothers tend to be more restrictive of daughter's intake if they are restrictive with their own intake, or if the daughter is overweight.

their feeding practices. Mothers are typically the primary caregivers and have traditionally assumed the greatest responsibility for feeding of a child. Thus, it would appear that they, more than any other person, would have a direct influence on a child's diet. This assumption is suggested by the fact that most studies on the association between parental feeding style and children's eating behavior and/or weight have been conducted solely or primarily on mother–child pairs.

Mothers first assume responsibility for the feeding of their child during pregnancy. Maternal intake directly affects the nutrition received by the fetus in utero. Studies have demonstrated that a mother's intake during this time may have long-lasting effects. For instance, children are more likely to be accepting of a flavor if the mother had consumed foods and/or drinks of this flavor during pregnancy. The assumption is that the flavor and/or smell of the mother's diet is transmitted a child. Research has in fact demonstrated that the smell of some foods, such as garlic, can be detected in the amniotic fluid.

During infancy, the mother typically controls the majority, if not all, of the feeding experiences, particularly if a child is breastfed. A number of studies have investigated the effect of breastfeeding versus bottle-feeding. While the research is somewhat mixed, it does seem to suggest that breastfeeding provides at least a modest degree of protection against childhood overweight. Studies indicate that breast- and bottle-fed infants are born at similar weights; however, bottle-fed infants tend to gain more weight throughout early childhood. As a result, research demonstrated that around age 5, bottle-fed children present with significantly higher body mass index (BMI), adiposity, and prevalence of overweight.

There may be a number of explanations for this phenomenon. Mothers who breast-feed may be more health conscious and therefore more likely to promote healthy eating behavior in children. Breastfeeding may transmit certain hormones or macronutrients which confer protection against the development of obesity. Further, breastfeeding may allow a child to respond to his or her own hunger and satiety cues in order to regulate energy intake. Bottle-feeding is generally more controlled by the mother and influenced more by external cues (i.e., the amount of formula in the bottle), which may promote eating past the point of satiety. Children who are breastfed also tend to show greater preference for flavors they have been exposed to through their mother's diet while breast-feeding and are more accepting of a range of novel flavors later in childhood.

A few studies have examined maternal feeding patterns in relation to disordered eating in infancy. Mothers of children with severe eating problems during infancy tend to display greater negativity and less responsiveness toward the child while feeding or playing with the child.

Several maternal feeding patterns later in childhood have been linked to children's patterns of eating and weight. One feeding practice that has received much attention is the mother's restriction of a child's food intake. Children of mothers who restrict food intake display poorer caloric regulation skills, more eating in the absence of hunger, and increased intake of restricted foods when restrictions are removed. These children tend to gain more weight over time than those whose mothers do not practice restrictive feeding habits. The relationship between restrictive feeding practices and eating behavior and weight appears to be strongest between mothers and daughter; it is not entirely clear that fathers' practices have the same effect or that sons are affected in the same way. Mothers tend to be more restrictive of daughter's intake if they are restrictive with their own intake, or if the daughter is overweight. Evidence suggests that restriction of a child's eating can perhaps be even more detrimental if the child is already overweight.

On the other hand, maternal pressure on the child to eat may also affect a child's eating habits. Children of mothers who deliver more eating prompts during meals tend to have greater caloric intake and spend a longer time eating. Mothers are more likely to place pressure on children with lower fat mass to eat and are less likely to pressure children who are overweight. Additionally, low-income mothers are generally more likely to pressure their children to eat. This suggests that this practice stems from fear of the child being underweight. However, this may promote unhealthy eating habits. Some studies have demonstrated that overweight children, but not normal-weight peers, eat faster, take larger bites, and accelerate eating more toward the end of the meal when their mother is present. This perhaps suggests that they have learned such behaviors through maternal prompts. By pressuring a child to eat, mothers may be encouraging eating be-

yond the point of satiety and therefore may be putting a child at risk for obesity later in life.

Mothers are generally encouraged to eat sensibly during pregnancy and while breastfeeding and to encourage healthy eating practices in children by allowing the child to respond to his or her internal cues for energy regulation. Overly controlling habits, such as restricting or pressuring children to eat, may make it more difficult for children to regulate their eating and body weight as they grow older.

SEE ALSO: Breastfeeding; Home and Parental Environment; Implications of Caloric Restriction of Food on Child Feeding Habits.

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Medical Interventions for Children

MEDICAL INTERVENTIONS FOR overweight/obese children (body mass index [BMI] above the 95th percentile) and children at risk for overweight/obesity (BMI above the 85th percentile but below the 95th percentile) begins with a clinical assessment, identification of the causes of weight gain, and changes in diet, physical activity, and lifestyle. When comorbidities pose significant health risk or if stabilizing or reducing fat mass in obese children through lifestyle changes is unsuccessful, further medical investigation and intervention are warranted. The targets of medical interventions of childhood obesity are to address causes of weight gain, prevent weight gain, initiate weight loss, and to treat the comorbidities.

Children's and adolescents' BMI should be attained during routine health visits, and the BMI should be plotted on the Centers for Disease Control and Prevention (CDC) age-for-BMI percentile charts. Information from a medical, family, nutrition, and physical activity history of obese children or children at risk for overweight/obesity will help determine the causes of obesity. Obesity is caused by a complex interplay of many factors, including genetic predisposition or abnormalities; environmental forces on nutrition and physical activity; sedentary behaviors; and medical reasons, such as disturbances to the hypothalamus (a region in the brain) or medications that stimulate weight gain. By understanding the reasons for obesity, further evaluation and recommendations can be made.

Screening for comorbidities, such as hypertension (high blood pressure), dyslipidemia (blood lipid abnormalities), nonalcoholic steatohepatitis (inflammation of the liver due to increased fat production and retention in liver cells), impaired glucose tolerance, and Type 2 diabetes (an inability to properly use insulin) may be indicated if the child has a BMI above the 95th percentile. Further reasons to screen for comorbidities include having a BMI above the 85th percentile and other comorbidities already present, a family history of comorbidities, or a child from an ethnic population with increased risk of comorbidity. A fasting blood lipids panel, a liver function test, and a glucose tolerance test are all examples of laboratory tests to screen for these comorbidities.

Weight maintenance is encouraged for children and adolescents with a BMI above the 85th percentile to prevent further weight gain. If obese children older than age 2 have a comorbidity or if children older than age 7 have a BMI above the 95th percentile, weight loss is recommended. Weight loss in obese children results in a reduction of fat mass, and many physical improvements throughout the body are attained. Often, weight loss in obese children can be difficult and many individuals undergoing substantial weight loss require the care of many healthcare specialists. The healthcare professionals involved in managing pediatric obesity should include a multidisciplinary team, including pediatricians, nurse practitioners, mental health specialists, nutritionists, endocrinologists, psychiatrists, pediatric geneticists, neurologists, school nurses, surgeons, pharmacists, and others.

Changes in diet, physical activity, and lifestyle are the primary methods to initiate weight maintenance and weight loss. The approach should be individualized; emphasis should be on the benefits of weight loss, setting realistic goals, and assessing motivation

Among the targets of medical interventions of childhood obesity are to address the causes of weight gain, and to prevent weight gain.

of the child and parents/caregivers. Family involvement in weight maintenance or weight loss is paramount, and the plan of action to maintain or lose weight must be catered to the family's willingness to make changes. Nutrition education for the family about portion sizes, caloric content of foods, and food choices are important to prevent hyperphagia (overeating). Emphasis of physical activity, a minimum of 30 to 60 minutes per day of moderate to vigorous intensity, is important for obese children. The choice of activity should depend on the child and his or her environment. Lifestyle changes, such as reducing sedentary activities, are important targets for weight management as well. Weight loss should not exceed two to four pounds per month, and monitoring this change is especially important for adolescents who have not yet achieved their final height.

If weight loss in morbidly obese adolescents cannot be achieved through diet and exercise alone, pharmacological modalities may be prudently considered. Weight loss in adolescents has been demonstrated with a variety of drugs; however, their effectiveness in long-term weight reduction and their long-term side effects are not well studied. The actions of drugs studied for weight management in adolescents include those that suppress appetite, limit fat absorption, and other drugs in which weight loss is a secondary effect.

Sibutramine, approved by the Food and Drug Administration (FDA) for use in adolescents older than age 16, suppresses appetite by inhibiting reuptake of the neurotransmitters serotonin, norepinephrine, and dopamine. Average short-term weight loss, measured at one year after starting use, ranges from 10–15 pounds, and this effect is greater than other medications. The effects of sibutramine on blood lipids, glucose, and insulin are unclear, but what is concerning is that sibutramine increases both systolic and diastolic blood pressure. Other side effects of sibutramine in adolescents include insomnia, dizziness, dry mouth, constipation, and its use is contraindicated with certain antidepressant drugs.

Orlistat, approved for use in adolescents older than age 12, is an intestinal lipase inhibitor, which effectively reduces digestion and absorption of dietary fat. Reductions in weight, BMI, and fat mass may be expected (although less than sibutramine); however, orlistat has no effect on glucose metabolism or blood lipids. Side effects include gastrointestinal discomfort, diarrhea, and gall bladder disease. Because orlistat limits the absorption of fats and fat-soluble vitamins, there is concern about deficiency of fat-soluble vitamins, particularly vitamin D, which is vital for adolescent bone health.

Metformin, approved for use in children older than age 10 with Type 2 diabetes, has primary effects on glucose metabolism, and has a secondary effect of weight loss and reduction in BMI. The primary action of metformin is to reduce the creation of new glucose from the liver, decrease absorption of dietary glucose, and improve insulin sensitivity. Modest weight loss at one year (8 pounds on average) may be expected as well as improvements in glucose metabolism. However, the long-term safety and effectiveness of metformin by starting its use in children early in life is not known.

Indeed, some rare genetic abnormalities result in childhood obesity, and pharmacological treatment can be successful in reducing body weight and fat mass. Children lacking the gene for leptin, a hormone secreted by the adipose tissue which has effects on

hunger and satiety, overeat and become obese early in childhood. Treatment with recombinant human leptin reverses this deficiency and normal weight may be attained as the child ages.

Leptin and other hormones affect energy intake by modulating hunger and satiety at the level of the hypothalamus. Thus, damage to the hypothalamus as a result of a tumor, surgery, trauma, or irradiation may result in childhood and adolescent obesity. The damage may result in weight gain through hyperphagia, reduced thermogenesis (heat production by increasing metabolism), or greater insulin secretion. This condition is referred to as hypothalamic obesity. Octreotide, a drug that suppresses insulin secretion, may be used for weight loss in children with hypothalamic obesity.

Other drugs that have weight-loss effects are either unapproved by the FDA for use in children and adolescents (such as rimonabant), or weight loss is a secondary effect of a medication treating another pathology unrelated to energy metabolism. Such modalities should not be considered for weight loss.

Bariatric surgery is an uncommon method of weight loss in adolescents, but the rate of procedures being performed is rapidly rising. Adolescents with a BMI at least 40 with complications are candidates for bariatric surgery. Additionally, the adolescent should have completed most of his or her physiological growth, have psychological stability, demonstrated diet and physical activity change, and have family support.

There are three major types of bariatric procedures, and their actions include reducing absorption of nutrients by bypassing parts of the stomach or intestines; restricting the size of the stomach to limit dietary intake; and a combination of reduced absorption and restriction. Bypass surgeries often result in the greatest amount of weight loss when compared to other interventions. Restrictive procedures, such as laparoscopic banding, are considered safer because of their adjustability and reversibility. All procedures have considerable side effects, namely nutrient deficiency in bypass surgeries. As with many of the pharmacological treatments for weight loss in adolescents, the long-term efficacy and side effects of these procedures are unknown.

In addition to promoting weight loss in the obese child and adolescent, treating the comorbidities is another important target to consider in the medical setting. Obesity in children and adolescents has many comorbid conditions including insulin resistance and Type 2 diabetes, renal complications, cardiovascular disease, liver disorders, orthopedic complications, and neurological complications.

The incidence of pediatric Type 2 diabetes parallels that of obesity, as most new cases of Type 2 diabetes in children are associated with obesity. The trend is alarming, as Type 2 diabetes was once considered to be an adulthood disease. Type 2 diabetes is the body's inability to properly use insulin, the hormone responsible for stimulating glucose uptake by many body tissues. Screening for Type 2 diabetes should be completed if the child is older than age 10 (or has begun puberty), has a BMI above the 85th percentile, and has other risk factors, such as family history of diabetes, belonging to certain ethnic groups (American Indian, African American, Latino, Asian American, Pacific Islanders), maternal history of gestational diabetes (a diabetic state experienced during pregnancy), or other conditions including acanthosis nigricans (dark patches of the skin due to high insulin levels), hypertension, dyslipidemia, and polycystic ovary syndrome.

A diagnosis of Type 2 diabetes can be given from a casual glucose concentration (test given at any time) above 200 mg/dL a fasting plasma glucose (no food eaten for at least eight hours) greater than 126 mg/dL, or an oral glucose tolerance test (another screening test for diabetes) greater than 200 mg/dL. Following a diagnosis, education to the child and the family about the disease is fundamental. In addition to nutrition therapy, self-management education, and family involvement, medical treatment of Type 2 diabetes may include the use of metformin and exogenous insulin. As mentioned previously, metformin helps glucose metabolism and weight loss. Exogenous insulin might be necessary to normalize other metabolic processes.

Renal complications may be associated with Type 2 diabetes. Microalbuminuria, high levels of albumin in the urine but not high enough to be detected by dipstick, may be presented with Type 2 diabetes and may be an indication of end-stage renal disease. Low-dose angiotensin-converting enzyme (ACE) inhibitors, a class of antihypertension drugs, are recommended for Native-American children with microalbuminuria.

Cardiovascular complications are often seen in obese children and adolescents. These complications include hypertension, dyslipidemia, left ventricu-

lar hypertrophy, and cardiomyopathy. Hypertension (high blood pressure) and dyslipidemia (abnormal blood cholesterol and triglycerides) are risk factors for cardiovascular disease and they are both associated with obesity in children and adolescents. Diet, exercise, and lifestyle therapy have been demonstrated to improve these parameters in the obese pediatric population, but medical treatment may be indicated in some obese individuals.

Children and adolescents with hypertension (age-specific systolic or diastolic blood pressure above the 95th percentile) may be treated with antihypertensive medications if they have symptomatic hypertension, secondary hypertension, hypertensive target-organ damage, diabetes, and persistent hypertension despite nonpharmacologic measures. Dyslipidemia can include blood triglycerides over 150 mg/dL, high-density lipoprotein (HDL) cholesterol ("good" cholesterol) below 40 mg/dL, and low-density lipoprotein (LDL) cholesterol ("bad" cholesterol) over 130 mg/dL. A feature of chronic dyslipidemia is inflammation, which can lead to atherosclerosis. Pharmacological treatment of dyslipidemia in children may be indicated if LDL cholesterol remains over 190 mg/dL after significant attempts at diet therapy. Cholestyramine, which binds bile acids, and statins, which inhibit an enzyme in cholesterol metabolism, are drugs that may be used in children.

Weight loss is the primary approach to improve left ventricular hypertrophy (increased left ventricular mass) and cardiomyopathy (heart failure).

Obesity in children and adolescents may lead to serious liver complications, most notably the spectrum of complications resulting from fat deposits in the liver. Fat may accumulate into liver cells (nonalcoholic fatty liver disease [NAFLD]), become oxidized, cause inflammation (nonalcoholic steatohepatitis [NASH]), cause fibrosis (excess fibrous connective tissue), and result in cirrhosis (scarring that results in a nonfunctioning liver). NAFLD and NASH are referred to as "nonalcoholic" because the deposition of fat and cirrhosis is similar to that observed in alcoholics, even without consuming alcohol. Liver function tests (tests that detect certain liver enzymes), imaging tests (such as magnetic resonance imaging), and liver biopsy can all lead to the diagnosis of NAFLD, NASH, or cirrhosis. Although no drugs are currently prescribed for these conditions in children and adolescents, gradual weight loss and control of diabetes, if present, will help these conditions.

Excess weight also has effects on the bones of growing children. One common orthopedic problem is slipped capital femoral epiphysis (SCFE). This condition happens when the growing end of the femur (the large bone in the thigh) slips from the hip. Pain in the knee, groin, hip, or thigh and a limp are symptoms of SCFE. Immediate consultation with an orthopedic specialist is necessary, and pinning of the hip is one treatment. Another orthopedic condition is Blount's disease, or bowing of the tibia (shin bone). Surgery may be an appropriate intervention.

A neurological complication of obesity is pseudotumor cerebri. This condition is characterized by increased intracranial pressure and an association with papilledema (swelling of the optic disc). Treatment includes weight loss and the drug acetazolamide.

SEE ALSO: Atherosclerosis in Children; Bariatric Surgery in Children; Behavioral Treatment of Childhood Obesity; Child Obesity Programs; Childhood Obesity Treatment Centers in the United States; Cost of Medical Obesity Treatments; Drug Targets that Decrease Food Intake/Appetite; Family Therapy in the Treatment of Overweight Children; Fatty Liver; Future of Medical Treatments for Obesity; Hypertension in Children; Medication Therapy after Bariatric Surgery; Metabolic Disorders and Childhood Obesity; Metformin; Multidisciplinary Bariatric Programs; Orlistat (Xenical); Pharmacological Therapy for Childhood Obesity; Sibutramine (Meridia).

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Medications that Affect Nutrient Partitioning

THE CHEMICAL ACTION of some medications may have side effects of increasing or decreasing body weight. The body handles nutrients through nutrient partitioning by storing nutrients, using nutrients to convert food to chemical components, releasing the heat created from biochemical processes, or excreting. Energy taken into the body through foods and beverages must go through the digestion process. Carbohydrates provide the sugars that create energy in the body, proteins are broken down to individual amino acids for the building of muscle, fats are the primary long-term energy source and are stored largely unchanged in adipose tissue.

Nutrient partitioning can be affected by metabolic changes, blocking or inducing storage, and increasing or decreasing excretion. Medications may have unintentional side effects of increasing metabolism, stimulating the thyroid, or working directly in the digestive tract. Medications that can affect nutrient partitioning include corticosteroids, lithium, tranquilizers, phenothiazines, some antidepressants, and medicines that increase fluid retention. Side effects for prescription medications taken for other illnesses may reduce or increase weight and interfere with the absorption or excretion of nutrients.

MEDICATIONS WITH THE POTENTIAL FOR CAUSING WEIGHT GAIN

Adrenocorticosteroids used for the treatment of inflammatory and immunologic disorders affect carbohydrate, protein and fat metabolism. Even within therapeutic dosage, adverse effects include fluid imbalance, increased glycogen production, and stimulation of insulin release resulting in increasing fat deposits.

Anabolic steroids are used medically for androgen replacement in males, to reverse protein loss due to severe illness or trauma, and to treat aging and as a growth stimulant. Circulating testosterone in the body is reduced in obese individuals. The abuse of anabolic steroids in sports to "bulk up" to create large muscles may be due to fluid retention in the muscles.

MEDICATIONS THAT MAY CAUSE WEIGHT GAIN BY EDEMA (FLUID RETENTION)

- Clonidine is used to treat hypertension by inhibiting function of the sympathetic nervous system stimulates alpha adrenoreceptors in the arterioles.
- Phenothiazine drugs are used to treat serious mental and emotional disorders. As a group, they may cause loss of appetite, fever, or low body temperature in addition to retention of urine.
- Lithium is used for mood stabilizing for bipolar affective disorder. The mode of action is not definitive although it affects electrolytes, neurotransmitters, and converts inositol monophosphate to inositol. Lithium may cause weight gain through fluid retention or through another undefined mechanism.
- Estrogens is used for hormone replacement, as part of contraception, or to suppress ovulation may cause weight gain by increasing triglyceride levels and altering carbohydrate metabolism in addition to fluid retention.
- Indomethacin is a nonsteroidal antiinflammatory used in specific cases of acute gouty arthritis, ankylosing spondylitis, pericarditis, and pleurisy.
- Methyldopa is used to treat hypertension by inhibiting function of the sympathetic nervous system stimulates central alpha adrenoreceptors.

MEDICATIONS TO DECREASE NUTRIENT ABSORPTION

Medications prescribed for obesity are most commonly appetite suppressants and work by affecting the satiety centers of the brain and resulting in less food being consumed. Unique in action, orlistat is a prescription medication to treat obesity by blocking intestinal action of gastric and pancreatic lipases, the enzymes to break down and absorb fats. The result is a decrease in absorption of ingested fat by approximately 30 percent. In doing so, it may increase excretion of fat soluble vitamins A, D, E, and K.

HERBAL AND DIETARY SUPPLEMENTS

Many dietary supplements purport to cause weight loss and include information that appears to boost metabolism and indicate ingredients have been scientifically studied. The Food and Drug Administration (FDA) regulates the labeling of dietary supplements and suggests watching for potentially fraudulent claims including cures, scientific-sounding terms

Medications prescribed for obesity are most commonly appetite suppressants and work by affecting the satiety centers of the brain.

fat-burning or *energizing* should be substantiated with scientific evidence, treatments for many symptoms and testimonials or claims the medical community is suppressing product benefits.

SCIENTIFIC AND CLINICAL STUDIES

Studies of beta-agonists show some modification of body fat content and body weight in the absence of nutritional reduction although side effects make them unsuitable as medications. Future research will provide better understanding with the relationship of medication on energy metabolism. In addition, genetic studies to isolate genes for obesity make the potential for identifying receptors and compounds to bind to those receptors may increase the medications available specific to nutrient partitioning and weight modification.

SEE ALSO: Adrenergic Receptors; Blood Lipids; Metabolic Disorders and Childhood Obesity; Orlistat.

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Medications that Increase Body Weight

TWO CLASSES OF medications may increase body weight. One class is designed to increase body weight or increase appetite in cachexic patients, and this class includes four approved products (oxandrolone, dronabinol, megestrol, and growth hormone). The second class of medications increases body weight as a side effect of treating other conditions, and this class includes many antipsychotics, certain antidepressants, certain antiepileptic agents, insulin, and peroxisome proliferator-activated receptor (PPAR) gamma agonists.

The anabolic steroid oxandrolone is a synthetic derivative of testosterone, which was approved for the treatment of wasting in 1964. The weight gain indication is primarily based on a six-week clinical trial in a population with heterogeneous clinical backgrounds. Longer-term clinical data have not been published. Dronabinol is the synthetic version of the cannabinoid tetrahydrocannabinol, the major active ingredient in *Cannabis sativa*. Dronabinol was first approved for the treatment of nausea and vomiting and later extended to anorexia associated with acquired immunodeficiency syndrome (AIDS) in 1992. The appetite-enhancing effect of marijuana smoking is also well documented

Megestrol is a synthetic progesterone derivative, and it is also indicated for the treatment of anorexia,

cachexia, and weight loss in AIDS patients. In several clinical trials where change of body composition was determined, megestrol appears to increase fat mass.

Growth hormone is approved for the treatment of wasting and cachexia in AIDS. A small increase in lean body mass may be associated with growth hormone treatment.

There are other medications officially approved to treat psychiatric disorders but are associated with significant weight gain as a major side effect. For example, many atypical antipsychotics such as clozapine, olanzapine, and risperidone and some typical antipsychotics such as thioridazine and chlorpromazine cause severe weight gain on chronic use. The mechanism of antipsychotics-induced weight gain is not entirely clear, although it has been proposed that multiple brain receptors targeted by antipsychotics mediate the weight gain side effect. Only a few antipsychotics (such as zaprasidone and molindone) appear to have minimal effect on body weight.

Some antidepressants such as amitriptyline and lithium are associated with long-term weight gain. The antidepressant-induced weight gain cannot be explained solely by the improvement in depressive symptoms. In the case of some selective serotonin reuptake inhibitors, short-term weight loss is often followed by small or inconsistent weight gain. The exact mechanism of antidepressant-induced weight gain has not been elucidated.

Several antiepileptic agents such as valproate, carbamazepine, and gabapentin are associated with weight gain. However, topiramate and zonisamide cause weight loss. The divergent effects of different antiepileptic agents on body weight may be related to the different mechanisms of these agents affecting neural activity.

Antidiabetic treatment based on intensive insulin therapy and insulin secretagogues such as sunfonylureas or thiazolidinedione (TZD) is generally accompanied by weight gain. The insulin-induced weight gain may be attributed to the anabolic effect of insulin and increased appetite. TZD-induced weight gain may be attributable to TZD's ability to promote adipocyte differentiation.

Drug-induced weight gain can be a serious side effect and may jeopardize effective medical therapy. Weight gain may be lessened or prevented by diet, exercise, or selection of an effective therapy with less weight gain potential.

SEE ALSO: Cannabinoid System; Serotonergic Medications.

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Medication Therapy after Bariatric Surgery

BARIATRIC SURGERY FOR weight loss is a successful method to improve the quality of life for patients who suffer from morbid obesity. Successful procedures typically result in significant weight loss and improvement of obesity-related comorbid conditions. Unfortunately, bariatric surgery can result in the development of multiple complications including those that occur from use of medications that can be harmful to patients post surgery.

An area of research that is significantly lacking is the management of medications in patients following bariatric surgery. Unfortunately, due to the lack of studies available, general guidelines must be developed from applying knowledge of specific medication characteristic and what is known about nutrient malabsorption following bariatric surgery.

The changes in the gastrointestinal tract following bariatric surgery significantly effect medications that are administered orally. The significant reduction in the area available for medications to come in direct contact with the gastrointestinal surface for absorption effect the amount of medication entering the blood stream. The changes in the acidity of stomach fluids can also affect medication efficacy. The amount of medication that enters the blood stream following oral administration is highly dependent on its ability to enter liquid form, the amount of time it has

in direct contact with the gastrointestinal tract, and blood flow to various regions of the gastrointestinal tract where medications are absorbed.

The chemical structure of each medication differs and dictates the type of environment it requires to enter the blood stream. Medications with acidic characteristics are readily absorbed in the acidic environment of the stomach. Medications with slightly acidic properties or those that are alkaline are readily absorbed in the slightly less acidic environment of the intestines. Following malabsorptive/restrictive bariatric procedures such as Roux-en-Y procedures, the stomach's ability to produce stomach acid is reduced. Medications that require an acidic environment may not be fully absorbed and higher doses may be required to improve the medications effectiveness.

The change in acidity may also affect medications that are enteric coated. Enteric coating is designed dissolve in an alkaline environment such as the intestines. The coating prevents medications that may be deactivated from the stomach acid from being released into the stomach or provides a protective barrier against medications that directly irritate the stomach lining. Post surgery, the reduction in stomach acidity may result in a premature dissolution of the enteric coating, potentially exposing the stomach to medications that may harm the integrity of the stomach lining. Similarly, medications that are fat soluble are also readily absorbed in the stomach. The reduction in contact time due to a shortened gastrointestinal tract following surgery may result in decreased absorption and efficacy of fat soluble medications. Higher doses of medications that are fat soluble may be required to provide a therapeutic effect.

Medications administered orally in a solid dosage form are required to dissolve into a solution form prior to being absorbed into the blood stream. Hence, utilization of medications that are commercially available in a liquid or elixir formulation may improve absorption following surgery. Oral formulations that are used to control the rate a medication is absorbed typically require more time in the gastrointestinal tract prior to dissolving into a solution. Medication formulations such as extended-release, delayed-release, and sustained-release formulations can take anywhere from two to 24 hours to be completely absorbed through the gastrointestinal tract and should be avoided due to a possible decrease in medication efficacy.

Most medications enter the blood stream via the small intestine. Bypassing the duodenum and early part of the jejunum not only results in significant deficiencies of nutrients absorbed in these locations, but also decreases the absorption of many medications. A specific subset of medications that may be directly affected are those that undergo first pass metabolism. The process of first pass metabolism involves a medication to initially enter the blood stream, then proceed through an initial metabolic pathway, then be released into the duodenum for reabsorption prior to delivering its therapeutic effect. Post surgery, the duodenum is bypassed limiting the ability for it to absorb medications that undergo first pass metabolism. Postsurgery medications that undergo first pass metabolism should be assessed for possible dose increases or replaced with appropriate alternative medications to achieve therapeutic goals for each individual patient.

Appropriate medication use includes ensuring medication efficacy and safety. Medications that can directly affect the integrity of the stomach lining can leaded to an increase in stomach ulcer formation in patients post bariatric surgery. Nonsteroidal antiinflammatory drugs (NSAIDs) decrease the prostaglandins that form a protective barrier on the stomach lining, but can also directly irritate the stomach lining. Newer agents select inhibit cycloxigenase 2 (COX-2) to prevent prostaglandin depletion and are believed to be more protective but may still directly irritate the stomach lining.

The use of NSAIDs should be avoided in patients post bariatric surgery. Protective agents such as misoprostol and carafate should be coadministered if the use of these agents is necessary. Patients post surgery may be at high risk for developing osteoporosis due to the potential for deficiencies in calcium absorption. In addition to NSAIDs, bisphosphonates also have the ability to produce gastric ulceration. Patients should be encouraged to discuss alternative pharmacotherapeutic options for osteoporosis prevention and treatment with primary care physicians.

Evaluation of medication therapies should be performed routinely following bariatric surgery due to anatomical changes and rapid weight loss that occurs. Specific medication characteristics should be taken into considerations when selecting or adjusting a patient's medication therapy.

Potentially harmful medications such as NSAIDs and bisphosphonates should be avoided. Patients should be encouraged to discuss medication concerns with not only their bariatric surgeon, but also their primary care physician and pharmacists to ensure appropriate medication use and prevent medication adverse effects.

SEE ALSO: Calcium and Dairy Products; Lap Band; Multidisciplinary Bariatric Programs; Osteoporosis; Roux-en-y Gastric Bypass; Vertical Banded Gastroplasty.

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Medifast

MEDIFAST IS LOW-CALORIE weight-loss and weight-maintenance program which relies on a combination of prepackaged Medifast meals and conventional food. Medifast is also the name of the publicly traded company (listed on the New York Stock Exchange) which conducts operations primarily through two wholly owned subsidiaries. Medifast products are produced by Jason Pharmaceuticals, a company founded by William Vitale in 1980, in a production facility in Owings Mill, Maryland, and are marketed by Jason Pharmaceuticals and Take Shape for Life. The latter, founded in 2002, is a network of health advisers supervised by physicians which market Medifast products as part of a wide-ranging "healthy lifestyle" program.

The Medifast Web site emphasizes the use of Medifast products within a healthy lifestyle including exercise, and the maintenance of weight loss through permanent behavioral changes. There is no charge for joining the Medifast program, but following the program requires purchasing a substantial number of Medifast packaged meals. Like the Atkins Diet, the Medifast diet is low in calories and carbohydrates

In the case of some selective serotonin reuptake inhibitors, short-term weight loss is often followed by small or inconsistent weight gain, but the exact mechanism of antidepressant-induced weight gain has not been elucidated.

and is intended to put the body into a state of ketosis which is believed to aid in burning fat. During the dieting phase of the Medifast program, an individual will consume 800–1,000 calories per day. Medifast encourages medical supervision of the Medifast diet, although this is not a requirement as the products may be purchased directly through the Medifast Web site. In addition, dieters have the option of enrolling in a Medifast Weight Control Center. These are franchise operations which currently (2007) exist in Texas and Florida and offer medical supervision and counseling for individuals following the Medifast diet.

The basic Medifast diet program is called the "5 & 1 Plan" because it is based on the daily consumption of five prepackaged Medifast meals plus one "lean and green" meal of conventional food. Following this plan, a person eats a small meal every two to three hours, a plan which is intended to keep them from becoming hungry and thus encouraging adherence to the diet. Medifast meals must be purchased from the company and are available in over 50 varieties which are interchangeable; they include shakes, bars, oatmeal, and scrambled eggs. Supplemental snacks such as soup and chips are also available, and there are meals designed specifically for diabetics, and separate meal packages for men and women. Costs for Medifast meals vary, but in May 2007, the discounted cost from the company Web site for four weeks worth of meals was $275. The "lean and green" meal consumed daily consists of conventional food chosen and prepared by the dieter, and is made up of a lean protein such as chicken, fish, or egg whites, and a salad or low-carbohydrate green vegetable.

The Medifast program is divided into three segments: the actual dieting period, the transition period, and the maintenance period. Common to all phases is an educational component focused on making dieters conscious of their food consumption and dietary requirements; in the second and third phases, they must adopt an individualized diet which will allow them to maintain a constant weight. After dieters achieve their target weight, the transition period begins. During this period, dieters begin to eat more normal foods and fewer Medifast meals, gradually increasing their caloric intake, and includes in their diet higher-carbohydrate fruits and vegetables which were avoided during the initial period. During the transition period, dieters continue to eat five or six small meals per day, made up of a combination of conventional food and Medifast meals: the assumption is that dieters are now accustomed to that schedule and has also been trained to choose small portions of food similar to that provided by the packaged meals. The company Web site recommends eight weeks in the transition period for people who lost less than 50 pounds, 12 weeks for those who lost 50–100 pounds, and 16 weeks for those who lost over 100 pounds. During the maintenance period, dieters consume approximately the same number of calories as they burn; frequent consumption of portion-controlled meals remains a cornerstone of this phase (which could last the rest of the person's life) although the use of Medifast meals is optional.

As of 2007, no peer-reviewed studies of the Medifast program appear in Medline. However, it has aspects in common with other diets including the Atkins Diet (high protein, low calorie), Slimfast (meal replacement), and numerous low-calorie and very-low-calorie diets. In general, such programs have often been found effective in initial weight loss attrition and with weight regain after one year variable and is highest for those following very-low-calorie diets. Further study is needed to establish the long-term effectiveness of Medifast and similar plans.

SEE ALSO: Atkins Diet; Caloric Restriction; Low-Fat Diet; Low-Calorie Diet; Portion Control; Very Low-Calorie Diet.

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Melanocortins

MELANOCORTINS ARE A group of pituitary peptide hormones including adrenocorticotropin (ACTH) and the alpha-, beta-, and gamma-melanocyte-stimulating

hormones. Five melanocortin receptors (MC1 through MC5) have different physiological functions.

MC1R expression occurs in macrophage/monocytic cells, lymphocytes with antigen-presenting and cytotoxic functions, neutrophils, endothelial cells, astrocytes, and fibroblasts. MC2 is also known as the adrenocorticotrophic receptor because it selectively binds ACTH. The MC3R gene encodes a G protein-linked receptor, coupled to both cAMP- and inositol phospholipid-Ca^{2+}-mediated signaling systems. MC3R expression occurs in brain, placenta, and gut, but not in melanoma cells or in the adrenal gland.

Melanocortin-4 receptor is expressed in the brain, including in hypothalamic nuclei known to be involved in feeding behavior. It is involved in integrating long-term adipostatic signals from leptin and insulin. MC5R contributes to regulation of exocrine gland function and to certain immune responses. It is expressed in peripheral tissues and occurs in the adrenal glands, fat cells, kidney, liver, lung, lymph nodes, bone marrow, thymus, mammary glands, testis, ovary, pituitary testis, uterus, esophagus, stomach, duodenum, skin, lung, skeletal muscle, and exocrine glands. Melanocortins are known to modulate fever, inflammation, and immunity by acting both on peripheral targets and within the brain. Data from both rodent models and humans suggest that intact neuronal melanocortin signaling is essential to prevent obesity. Melanocortins are also involved in the pathogenesis of disorders at the opposite end of the spectrum of energy homeostasis, the anorexia and weight loss associated with inflammatory and neoplastic disease processes.

Decreased melanocortin signaling via pharmacological or genetic means results in increased food intake and weight gain. Activation of central melanocortin receptors inhibits feeding and leads to weight loss, whereas blockade of the central melanocortin signaling pathway increases food consumption and promotes weight gain. Melanocortin system agonism promotes weight loss through decreasing appetite, increasing sympathetic nervous system activity, and modulating thyroid-releasing hormone, corticotropin-releasing hormone, brain-derived neurotrophic factor, melanin-concentrating hormone and orexin. Recent studies suggest that melanocortins could be involved in mediating the effects of leptin, and in controlling the expression of neuropeptide Y (NPY).

SEE ALSO: Agouti and Agouti-Related Protein; Hormones.

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Mendelian Disorders Related to Obesity

IT HAS BEEN confirmed that genetic factors have a significant role in the pathogenesis of obesity. Genetic disorders related to obesity can be categorized as Mendelian syndromes associated with obesity such as Bardet-Biedl syndrome, multifactorial obesity, and single-gene disorders with obesity as an isolated or predominant presentation. Mendelian disorders are diseases that are inherited according to Mendel's laws. In some Mendelian disorders, obesity is one of the clinical presentations. More than 30 Mendelian disorders with obesity as a clinical presentation have been reported. These Mendelian disorders include autosomal dominant ones such as Prader-Willi syndrome, Albright hereditary osteodystrophy, fragile X syndrome, ulnar-mammary syndrome; autosomal recessive ones such as Bardet-Biedl syndrome, Alstrom syndrome, Cohen syndrome; and X-linked ones such as Borjeson-Forssman-Lehmann syndrome, Mehmo syndrome, Simpson-Golabi-Behmel Type 2, and Wilson-Turner syndrome.

The causative mutations behind the Mendelian disorders related to obesity have been identified, but there is still no clear information regarding the mechanisms underlying these mutations which lead to metabolic disturbances and obesity. In these syndromes, the product of the defective gene is an intracellular protein, but the function of such proteins is still unknown. In other words, there is no explanation about the role of these proteins in energy imbalance leading to obesity.

Prader-Willi syndrome (PWS) is the most common Mendelian syndrome related to obesity. The clinical features of this autosomal-dominant disorder include reduced fetal activity, obesity, muscular hypotonia, mental retardation, short stature, hypogonadotropic hypogonadism, and small hands and feet. In most of the cases, a deletion or disruption of the genes on the proximal long arm of the paternal chromosome 15 causes PWS. In other cases, there is a maternal disomy.

The Albright hereditary osteodystrophy (AHO) is an autosomal-dominant Mendelian disorder. Its clinical features consist of obesity, round facies, short stature, brachydactyly, subcutaneous calcifications, hypocalcaemia, increased serum parathyroid hormone (PTH) level, and parathyroid hyperplasia. In some cases, it as accompanied by mental retardation. AHO is caused by parental imprinting of mutations in the GNAS1 gene (guanine nucleotide-binding protein, a-stimulating activity polypeptide 1).

Fragile X syndrome is an autosomal-dominant disorder and is caused by mutation in the FMR1 gene. The most dominant clinical feature of this syndrome is mental retardation. There is a Prader-Willi-like subphenotype of the fragile X syndrome whose clinical presentations consist of extreme obesity with a full and round face.

Ulnar-mammary syndrome is caused by mutations in the TBX3 gene and is an autosomal-dominant disorder. Some of its clinical features include ulnar ray defects, small penis, delayed puberty, and obesity.

The Bardet–Biedl syndrome (BBS) is an autosomal-recessive syndrome which is characterized by obesity, mental retardation, pigmentary retinopathy, polydactyly, and hypogenitalism. As this Mendelian disorder is genetically heterogeneous, it is linked to at least seven loci.

Alstrom syndrome is an autosomal-recessive disorder due to mutation in the ALMS1 gene. It is characterized by retinitis pigmentosa, deafness, obesity, and diabetes mellitus like Bardet-Biedl syndrome, but there is no mental defect, polydactyly, or hypogonadism in this syndrome.

Cohen syndrome is a rare autosomal-recessive disorder overrepresented in the Finnish population which is due to mutations in the COH1 gene. Obesity is seen in this syndrome, but it has been mentioned as an insignificant presentation.

Mehmo syndrome (MEHMO) is an X-chromosomal mental retardation syndrome characterized by obesity, mental retardation, epileptic seizures, hypogonadism and hypogenitalism, and microcephaly.

Borjeson-Forssman-Lehmann syndrome (BFLS) can be caused by mutations in the PHF6 gene and is an X-linked disorder with clinical features such as severe mental defect, epilepsy, hypogonadism, hypometabolism, marked obesity, swelling of subcutaneous tissue of face, narrow palpebral fissure, and large but not deformed ears.

Wilson-Turner syndrome is an X-linked disorder characterized by some features resemble to BFLS such as obesity, mental retardation, and gynecomastia.

Simpson-Golabi-Behmel Type 2 syndrome is another X-linked disorder related to obesity which is caused by a mutation in the CXORF5 gene.

Besides the above syndromes, there are other Mendelian disorders in which obesity is presented. In the online Mendelian Inheritance in Man (OMIM) database, these syndromes have been described. The nature of processes causing obesity in these syndromes is still a question.

SEE ALSO: Bardet-Biedl Syndromes; Down's Syndrome; Genetic Mapping of Obesity Related Genes; Genetics; Genomics; Inherited Taste Preferences; Obesity Gene Map; Prader-Willi Syndrome.

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Menopause

MENOPAUSE IS A natural event in women aged 49–55. Menopause is not a disease but a normal biological transition and inevitable process of change. Major hor-

monal shifts occur which may affect a woman's entire being. In many ways, this hormonal shift is likened to puberty as it is a time of symptoms before a change. Like puberty, the hormonal shifts of menopause mark a rite of passage and an entrance to a new life phase.

Menopause begins once a woman has ceased to menstruate for 365 days. The years preceding are referred to as perimenopause or the climacteric, commonly referred to as "the change." The duration may last up to 10 years. Each woman is different and there is no way of predicting how long this phase may be. It is possible that a woman is thrown into a surgical or chemical menopause. A hysterectomy that includes removal of both ovaries will result in immediate menopause. Chemotherapy and radiation may cause a premature menopause. Stress, anorexia, and intense exercise may stop menstruation, but if it returns, a natural menopause may still occur.

The ovaries are an endocrine organ the size and shape of an almond. The role of the ovary is to produce eggs and hormones including estrogen, progesterone, and androgen, which affects libido. The glands of the endocrine system are the main hormone producers. The pituitary gland secretes hormones that control ovulation and menstruation. The pituitary and the ovaries respond to each other by instructing the rise and fall of hormone levels. At menopause, these levels fluctuate; less progesterone is produced in the ovaries followed by a decrease in estrogen. The ovaries cease egg production.

Symptoms of the climacteric may include hot flashes, irregular periods, fuzzy thinking, insomnia, headaches, vaginal dryness, irritability, and mood swings. Decreased ovulation causes an increase in estrogen relative to progesterone manifesting in the onset of symptoms. Because each woman is individual and unique, many of the symptoms may be absent or mild. In others, they may be devastating and disruptive to a woman's overall health and well-being. There are options to ease the discomfort and disturbances, but what works for one woman may not work for another. It is important to identify your particular issues and address them with a combination of remedies including a frank discussion with your physician.

The least invasive option is to make positive lifestyle changes. These changes may have the added benefit of increasing your quality of life physically, emotionally, and cognitively.

Menopause is best treated by taking into account a woman's health history, her family history, and her individual symptoms.

Exercise is an important element before, during, and after menopause. Women who exercise often experience fewer symptoms. They have the added benefit of muscle strength which helps strengthen bones, aid in balance, and protect the heart muscle.

Hot flashes and night sweats are a common complaint. It is advisable to avoid spicy food, caffeine, and alcohol. Keep rooms cool and dress in layers. Exercise and stress reduction may also help. Adding one to two daily servings of soy may be beneficial. Soy contains phytoestrogens which mimic estrogen. Good sources include soy milk, tofu, tempeh, and soy nuts. Black cohosh is a supplement that may combat hot flashes. It has been endorsed by the North American Menopause Society for a duration of up to six months.

Metabolism slows in midlife, requiring fewer calories to maintain weight. This is an important time to increase exercise and lower total calories. Keep blood sugars stable by eating smaller, balanced meals frequently, about every three to four hours. The DASH (Dietary Approaches to Stop Hypertension) diet decreases sodium intake and promotes eating healthy proteins, whole grains, fruits, and vegetables, and is endorsed by the

National Heart, Lung, and Blood Institute. A healthy diet may ease the transition to menopause.

Stress is a major contributor to adverse symptoms. It is important to understand that stress cannot be eliminated, but coping mechanisms may be learned. Deep breathing is a technique which can be done anywhere with no expense. Meditation and yoga are ways to learn to focus. It is vital to create self time, even if only for 15 minutes a day. Laughter is great medicine as it releases endorphins, or "happy hormones." Massage therapy and visualization are techniques that may also help.

Mood swings, irritability, and insomnia may be interrelated and elevated. The body and mind need sleep as a time to rest, heal, and recharge. Without it, functioning is less than optimal and often difficult. Exercise may help with insomnia and mood swings as does decreasing or eliminating alcohol and caffeine. Keeping the bedroom dark and cool, using it only for sleeping at night or sex, may ease symptoms. Avoid eating several hours prior to bedtime. Using cotton sheets may keep the body cool. A cup of chamomile tea at bedtime may induce sleepiness. Mood swings may regulate once sleep improves. Irritability may be greatly reduced. Exercise, yoga, stress reduction, and support groups may ease the tension. If these do not help, a physician may prescribe SSRIs (selective serotonin reuptake inhibitors) such as Paxil®, Prozac®, or Effexor®.

Fuzzy thinking may be a by-product of the normal aging process, not just menopause. Keeping the mind sharp by doing word challenges and/or puzzles may encourage focus by stimulating thought process. A daily cup of ginseng tea or ginkgo biloba supplement may be beneficial to clear thinking.

Once the aforementioned remedies are tried, there is the option of hormone therapy (HT). HT has replaced the term *hormone replacement therapy* as it is no longer only estrogen replacement but rather an enhancement of what the female body requires to operate optimally. This is usually an estrogen, progesterone, or combination. There are several delivery systems including orally, vaginally, or transdermally by creams, patches, or gels. These hold risks as well as benefits especially to those women with a history of heart problems and/or breast cancer. HT may cause an increase in blood clots, heart attack, stroke, and breast cancer. The benefits may cause a decrease in hot flashes, slower bone loss, and improved sleep. Vaginal dryness may benefit by HT as may applying a sexual lubricant during intercourse.

Bioidentical hormones are hormones most like the hormones made in the body. They are synthesized in a lab from hormone precursors found in wild yams or soy. They are chemically converted to be identical to what the body produces naturally.

Osteoporosis, or thinning of the bone, causes bone to become brittle, porous, and susceptible to fracture. HT may help as may a healthy diet, exercise, and calcium supplements.

This time of transition usually occurs in conjunction with other life stressors often including empty nest, loss of parents or spouse, career changes, and changes in physical appearance. There are options to explore to ease the transition. Women with menopausal symptoms are advised to speak with their physician regarding management strategies. Treatment is best prescribed by taking into account a woman's health history, her family history, and her individual symptoms.

SEE ALSO: Exercise; Hormones; Osteoporosis; National Heart, Lung, and Blood Institute; Stress.

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Menstrual Problems

MENSTRUATION IS THE periodic discharge of blood and mucosal tissue from the uterus, which occurs approximately monthly in nonpregnant women from the onset of puberty to menopause and is necessary for female reproduction. Menarche, or the first menstrual period, occurs during puberty and signals the onset of the reproductive years of a woman. On the other end of the spectrum, the beginning of menopause is when there have been no menstrual periods for 12 consecutive months and no other biological or physiological cause can be identified. Between these two end points, women would ideally have normal,

regular menstrual cycles, or eumenorrhea, that range from 21 to 35 days in length.

Menstrual cycles are counted from the first day of menstrual bleeding because the onset of menstruation corresponds closely with the hormonal cycle. The menstrual cycle may be divided into several phases and the length of each phase varies from woman to woman and cycle to cycle. During the follicular phase, estrogen slowly builds up. Meanwhile, the follicle matures. Estrogen peaks right before ovulation, or the release of the egg from the ovary. The surge of luteinizing hormone (LH) triggers ovulation. The egg travels through the fallopian tube and may be fertilized by a sperm in the process. If the egg is not fertilized within a day of ovulation, it dies and is resorbed. During the luteal phase, the corpus luteum, or the remainder of the follicle postovulation, produces progesterone, causing the intrauterine lining to accumulate. Once the corpus luteum degenerates and progesterone levels decline, the uterine lining is shed, indicating the start of the menstrual cycle.

Menstrual problems are characterized by the irregularity of menstrual cycles, or the unpredictable variability of intervals, duration, or bleeding. With regard to irregularities in ovulation, oligoovulation is the term for infrequent or irregular ovulation, defined as cycles of more than 36 days or fewer than 8 cycles a year. Anovulation is the absence of ovulation when it would be normally expected, such as in a postmenarchal, premenopausal woman. Patients with anovulation usually present with the irregularity of menstrual periods. Additionally, anovulation can also cause cessation of periods, also called secondary amenorrhea, or excessive bleeding, as characterized by dysfunctional uterine bleeding.

In terms of menstrual cycles, polymenorrhea refers to cycles with intervals of 21 days or less. Another condition is oligomenorrhea, defined as infrequent or light menstrual cycles with more than 35 days. Occurring in approximately half of patients with bulimia nervosa, the mechanism of oligomenorrhea appears to be related to hypothalamic-pituitary function.

Similarly, amenorrhea is a diagnostic criterion for anorexia nervosa. Amenorrhea is the absence of a menstrual period in a woman of reproductive age. Physiologic states of amenorrhea are seen during pregnancy and lactation, or breastfeeding.

In patients with anorexia, the exact mechanism of amenorrhea has yet to be elucidated. Nonetheless, severe caloric restriction suppresses the hypothalamic–pituitary axis, possibly mediated by cortisol, leptin, growth hormone, and insulin-like growth factor I, resulting in the suppression of the pituitary production of LH and FSH. Abnormally low levels of LH and FSH cause low levels of circulating estrogen, thereby blocking ovulation.

Because estrogen is essential for the incorporation of calcium into bone, patients with eating disorders such as anorexia are at high risk of developing osteopenia and osteoporosis. Particularly, certain patients with eating disorders may also exercise excessively, increasing their risk of stress fractures. The female athlete triad consists of a menstrual disorder, an eating disorder, and osteoporosis.

A common cause of ovulatory infertility is polycystic ovarian syndrome (PCOS), affecting 1 to 5 percent of women. PCOS is characterized by hyperandrogenism and chronic oligoanovulation. Approximately 50 percent of PCOS women are overweight or obese, with adipose tissue distribution predominately around the abdomen as a common clinical presentation. Because the history of weight gain often precedes PCOS, obesity may play a pathogenic role in the development of the syndrome. Administration of insulin-sensitizing agents, such as metformin and troglitazone, was associated with improved menstrual cyclicity in women with PCOS.

Menstrual problems are characterized by the irregularity of menstrual cycles, or the unpredictable variability of intervals, duration, or bleeding.

Both insulin and insulin-like growth factors (IGFs) seem to be involved in the interruption of the normal follicle maturation favoring the formation of atretic follicles. Therefore, obesity seems to amplify the degree of insulin resistance and hyperinsulinemia in PCOS, whereas abnormalities of the IGF–insulin-like growth factor binding protein (IGFBP) system may be important in normal-weight PCOS women.

Additionally, women with PCOS may have hyperandrogenism, which is worsened by particularly central obesity, through the reduction of sex hormone-binding globulin (SHBG) serum levels, which increases the delivery of free androgens at the level of peripheral tissues. High levels of estrogens have been detected in women with PCOS. The acyclic production of extraglandular estrogen may lead to a positive feedback on LH secretion and a negative feedback on FSH secretion, resulting in an increase of the circulating LH/FSH ratio. The elevated levels of LH substantially contribute to the development of hyperplasia of the ovarian stroma and thecal cells, further increasing androgen production and, in turn, providing more substrate for extraglandular aromatization and perpetuation of chronic anovulation.

With respect to nutrition and environmental factors, diet is a well-established factor in the regulation of sex steroid metabolism. Research has shown that the high-fat, low-fiber diet is related to an increase in androgen circulating levels. Additionally, evidence finds that very high lipid intake decreases SHBG blood levels and increase free androgen index. Therefore, a high-fat, lower-fiber diet may impair sex steroid metabolism by increasing androgen availability while favoring the promotion and maintenance of obesity in certain groups of women with PCOS.

SEE ALSO: Anorexia Nervosa; Bulimia Nervosa; Eating Disorders and Athletes; Eating Disorders and Obesity; Early Onset Menarche and Obesity in Women; Menopause; Metformin; Polycystic Ovary Disease.

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Metabolic Disorders and Childhood Obesity

METABOLISM IS THE regulation of energy throughout the body. Obesity is a metabolic disorder in which excess fat is accumulated in the adipose tissue, or fat tissue. In children, this can lead to a number of other disorders in metabolism throughout the body. Such abnormalities include increased inflammation, decreased insulin sensitivity, Type 2 diabetes, cardiovascular complications, and problems with liver function. Understanding the genetics and lifestyle factors of obesity in youth will provide important insight on the progression and treatment of metabolic disorders associated with childhood obesity.

Obese children are described to be in a chronic state of inflammation. This is primarily because of increased fat mass. Adipocytes, or fat cells, are not simply storage sites for fat. Rather, they secrete a variety of hormones and other molecules that modulate many metabolic processes, including inflammation. In obesity, the adipocytes can be greater in number and be greater in size when compared to normal weight individuals. The hormone leptin is secreted from the adipcytes, and higher leptin levels is observed with higher body fat. Leptin has effects on the hypothalamus and plays a role in food intake. Conversely, adiponectin (an antiinflammatory hormone secreted by the adipose tissue) is lower in obese children compared

to normal weight children. High levels of leptin and low levels of adiponectin are associated with inflammation. Additionally, other molecules that stimulate inflammation are higher in obese children, including tumor necrosis factor-α (TNF-α), interleukin-6, and c-reactive protein. These hormones may have effects on liver, muscle, kidneys, heart, and brain. It is suspected that fat deposits in the visceral region, or abdominal fat, is responsible for most of the inflammation, however, other fat depots, such as those found underneath the skin (subcutaneous) in other regions of the body may also have an important effect on inflammation. One of the consequences of chronic inflammation is the body's decreased ability to use the hormone insulin.

The primary action of insulin is to allow cells to uptake glucose. Insulin is a peptide hormone that is secreted by the pancreas in response to eating food. The digestion product of many foods containing carbohydrate is glucose, a free sugar, and glucose is found in the blood after digestion. Under normal circumstances, the pancreas detects glucose in the blood and it then releases insulin into the blood. Insulin then signals other organs, such as adipose, muscle, and liver, to uptake the free glucose.

In some obese children, the ability of organs to uptake glucose with normal insulin levels is impaired. Impairment in glucose uptake by tissues is known as insulin resistance. For the obese, this may be due to high levels of fat within muscle or from high levels of circulating free fatty acids. Increased levels of lipid in the muscle probably interfere with the effectiveness of the insulin receptor, thereby preventing the muscle to recognize insulin. To compensate for insulin resistance, the pancreas may release even more insulin. The prevalence of insulin resistance is greater in obese children than normal weight children. In fact, a high body mass index (BMI) in childhood is linked with decreased insulin sensitivity, and this association is followed into young adulthood. Interestingly, the insulin sensitivity appears to have different effects between boys and girls during puberty, with boys having decreased sensitivity and girls have increased sensitivity.

Insulin resistance can be a chronic condition and it may be lead to Type 2 diabetes. Type 2 diabetes is the result of insulin resistance and a defect in insulin secretion from the pancreas. Although the pancreas attempts to maintain high insulin output in response to the insulin resistance in the peripheral tissues, eventually it cannot keep up with insulin production. In this state, insulin has lost its normal physiological function, and high blood sugar (hyperglycemia) and high blood insulin levels (hyperinsulinemia) can be observed. Obesity can further stimulate high blood sugar because the liver increases its output of sugar from other precursors (gluconeogenesis). Hyperglycemia and hyperinsulinemia can be observed even when the individual has not been eating (fasted), a hallmark of Type 2 diabetes. Thus, glucose metabolism has been deranged.

Although genetic factors play an important role in the development of Type 2 diabetes, obesity is becoming a clear risk factor for the disease. The prevalence of Type 2 diabetes in children and adolescents has increased dramatically in the last three decades, and this is often attributed to the increased prevalence of obesity. Once thought to be a disease in adulthood, Type 2 diabetes is becoming a metabolic disorder of the pediatric population. In fact, in some populations such as Native Americans, Latinos, and African Americans, Type 2 diabetes is more common than type 1 diabetes in children.

The reason for abnormalities in insulin and glucose metabolism in obese children may be related to the distribution of fat. As with inflammation, visceral fat, or abdominal fat, is associated with insulin resistance. Furthermore, there is also an association between visceral fat and blood lipid abnormalities. It is important to note, however, that further research is necessary to determine if visceral fat is solely responsible for these associations, or if other fat depots, such as subcutaneous (beneath the skin) and fat below the waist, contribute to the metabolic abnormalities.

Dyslipidemia refers to abnormal lipids (including fats and cholesterol) in the blood. Overweight children with visceral fat tend to have elevated low-density lipoprotein cholesterol (LDL-C, the "bad" cholesterol), high blood triglycerides, and decreased high-density lipoprotein cholesterol (HDL-C, the "good" cholesterol). Some of the greatest concerns for dyslipidemia are the development of atherosclerosis, the metabolic syndrome, and the progression of nonalcoholic fatty liver disease.

Atherosclerosis is the inflammation and stiffening of the arteries that involves the progression of many steps. It begins with an infiltration of fat into the lining of arteries (formation of fatty streaks), oxidation

of lipid, an inflammatory response, and the stiffening of the arterial wall. This could lead to total closure of the artery or rupture, which is a cardiovascular event. Atherosclerosis is typically associated with older people, but early development of atherosclerosis has been observed in the obese pediatric population. In fact, it is estimated that up to 50 percent of the obese pediatric population have already developed fatty streaks.

In adults, a conditioned referred to as the metabolic syndrome is characterized with increased waist circumference, low HDL-C, high triglycerides, high blood pressure, and high levels of blood glucose. It is often used in adults as a marker for increased risk for cardiovascular disease. Although the use of the term in children is not standard practice, it is estimated that as much as 30 percent of obese children have the metabolic syndrome.

The liver is an important organ involved in many metabolic processes. Its metabolic roles include the processing of absorbed nutrients from foods and the storage of energy. However, hyperinsulinemia and hyperlipidemia due to obesity may have negatives effects on the liver. High levels of insulin in the blood increases the production of fat from other precursors in the liver.

Additionally, excess fat might be already present in the circulation. The excess fat can be deposited and retained in the liver. When large amounts of fat are stored in the liver of obese people, this is called nonalcoholic fatty liver disease (NAFLD); it is called nonalcoholic because the fatty infiltration of the liver is similar to that observed in alcoholics. The fat can be stored or processed for energy in the liver (via β-oxidation), and oxidative stress insults the liver. The oxidative damage can be accompanied with an inflammatory response and this can progress to nonalcoholic steatohepatitis (NASH). The liver can then have connective tissue buildup (fibrosis) and scarring can occur (cirrhosis). Once scar tissue has been established, liver failure is a major concern.

Obesity is unlikely to be the only factor that leads to metabolic abnormalities in childhood, as it is possible for an individual to be obese and have no derangements. Genetic susceptibility appears to play an important role in metabolic changes as well. Research describing single- or multigene susceptibility to metabolic derangements will provide further insight on the pathology of obesity in youth. Also, the impact of lifestyle factors on a metabolic profile appears to be very important, as increasing physical activity can improve insulin resistance, decrease inflammation, and increase adiponectin levels.

SEE ALSO: Adipocytes; Adiponectin; Atherosclerosis in Children; Blood Lipids; C-Reactive Protein; Cytokines; Fatty Liver; Hypothalamus; Insulin; Interleukins; Leptin; Physical Activity in Children; Type 2 Diabetes; Visceral Adipose Tissue.

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Metabolic Rate

METABOLISM IS THE physical and biochemical modifications of compounds in living organisms. For human beings, metabolism primarily deals with processing food, along with the atmospheric gas oxygen, into energy needed to maintain a living state. Metabolic rate refers to the speed or rate at which this process happens. Metabolic rate is also known as energy expenditure.

Metabolic rate is made up of four processes: (1) basal or "resting" metabolism, which includes energy needed to maintain electrochemical balance in cells, energy for growth and repair of structural components of the body, and energy expended during the mechanical work of the cardiovascular and respiratory systems at rest; (2) energy needed for exercise or physical work; (3) energy needed to maintain a "core" body temperature; and (4) energy used during the eating and digestion of food (sometimes called the thermic effect of food).

At rest, internal organs such as the liver, brain, heart, and kidneys together can account for about 60 percent of metabolic rate, and muscle accounts for about 15 percent. During exercise or physical work, the energy requirements of muscle can increase manifold. The thermic effect of food can account for ~10 percent of

metabolic rate. The rest is made up by metabolism of other organs. Because modern persons typically live in a thermoneutral environment, the energy needed to maintain body temperature is very little and thus contributes very little to daily metabolic rate.

There are several ways to directly measure metabolic rate. Heat production (a by-product of metabolism) can be measured by placing a person in a suit or chamber that measures heat production. This method is called direct calorimetry, and requires expensive and sophisticated equipment. A simpler method is to measure oxygen consumed over a time period. This method is called indirect calorimetry. These are direct ways to measure metabolic rate.

There are several "field methods" used to indirectly measure metabolic rate. Behavioral observation and time and motion analysis can be used for supervised subjects. Questionnaires and interviews of subjects can be used. Movement assessment devices such as pedometers and accelerometers can be worn by subjects. These methods and devices sacrifice accuracy for ease and/or decreased cost. Metabolic rate can be affected by a number of factors, including age, gender, amount of fat and muscle mass, time of day, heredity, certain drugs and disease states, and exercise or physical work.

To affect the metabolic rate, changes must occur in one of the four processes listed above. For the U.S. population, the energy needed to maintain core temperature and for eating and digestion of food cannot be altered much. Thus, the two ways to alter metabolic rate are to change basal metabolism, and/or change the amount of exercise and muscular work.

The largest contributor to basal metabolism that can be reasonably and voluntarily altered is the amount of muscle mass a person has. By incorporating resistance training, muscle mass can be increased which should in theory raise basal metabolic rate. Conversely, when people consume fewer calories, or diet, typically both muscle and fat mass are lost. Thus, dieting tends to lower basal metabolic rate, contributing to an overall reduced daily metabolic rate. The simplest thing to do to increase metabolic rate is regular exercise. The benefits of modern life have contributed greatly to reductions in muscular work, and thus metabolic rate. Regular exercise can counteract this reduction in metabolic rate.

SEE ALSO: Dieting: Good or Bad?; Doubly Labeled Water; Energy Expenditure Technologies; Exercise; Indirect Calorimetry; Metabolic Disorders and Childhood Obesity; Set or "Settling" Point.

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Metformin

METFORMIN IS A drug used in the management of Type 2 diabetes mellitus (T2DM) and is part of the biguanides class of medicine. Other classes of medications for T2DM include sulphonylureas, thiazolidinediones, acrabose, incretin mimetics and insulin therapy. Metformin is sold commercially as Glucophage®, Glucophage XR®, Fortamet® or Riomet®. Metformin is taken orally and has hypoglycemic effects (lowers blood sugar). Metformin has been approved for use in the United States since the 1990s.

The mechanism for metformin is not fully known. However, it is believed that metformin reduces the amount of glucose (sugar) that the liver releases into the body. Metformin also decreases the amount of glucose that the body will absorb from the diet. Additionally, metformin can increase the insulin sensitivity of other tissues such as muscle. Metformin has several advantages compared to other drugs. Metformin does not require the pancreas to produce more insulin (like sulphonylureas do), but rather, it works to make insulin receptors more sensitive to the actions of insulin. This will preserve the pancreas's ability to make insulin. Also, metformin does not promote weight gain (sulphonylureas and thiazolidinediones do); in fact, metformin results in weight loss.

Metformin acts as an appetite suppressant and the weight loss associated with metformin resulted in mainly the loss of body fat and little loss of muscle. Patients who took metformin had improvements in their lipid profile. These patients had decreased their total and low-density lipoprotein (LDL) cholesterol (bad cholesterol) levels and decreased their triglyceride

levels. It is not known if metformin directly improves the lipid profile or if this improvement is secondary to the weight loss associated with metformin.

Some of the side effects of metformin use include nausea, cramping, diarrhea, and anorexia; however, these side effects disappear at lower dosages. Also, these side effects are milder if metformin is taken immediately before or after a meal. Patients with kidney disease should not use metformin as it may result in a condition known as lactic acidosis.

Metformin is also used for the treatment of several other medical conditions including insulin resistance and polycystic ovary syndrome (PCOS). Insulin resistance is a prediabetic condition in which some of the body's tissues, such as the adipose tissue (fat) and muscle, become less sensitive to the actions of insulin. Because metformin is an insulin sensitizer, it can reduce the severity of the insulin resistance. PCOS is a condition in females that is associated with abnormal ovulation and elevated levels of testosterone.

In addition, many women with PCOS are insulin resistant and exhibit symptoms of the metabolic syndrome. Women who have PCOS tend to be overweight or obese. Metformin is a treatment prescribed to women with PCOS as it can alleviate the insulin resistance. In addition, the use of metformin decreases testosterone and luteinizing hormone levels and promoted weight loss and resulted in improvement in patients' ability to ovulate. The use of metformin also increases the likelihood of patients becoming pregnant and reduces the likelihood of miscarriages.

SEE ALSO: Polycystic Ovary Disease; Type 2 Diabetes.

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Mexican Americans

IN THE UNITED States, overweight and obesity are common in many minority ethnic and racial groups. The populations where this is especially true are among Mexican American and black (non-Hispanic) adults. They are considerably more likely to be overweight or obese than white (non-Hispanic) adults. In adult men, Mexican Americans have the highest rate of overweight and obesity among ethnic and racial groups in the United States. In adult women, blacks (non-Hispanic) are the only racial ethnic group that have a higher rate of overweight and obesity than Mexican Americans. The problem of obesity is made worse due to the fact that abdominal fat is the most common fat distribution pattern among Mexican Americans. This fat distribution pattern is more commonly associated with the obesity comorbidities. This is especially true for metabolic syndrome and Type 2 diabetes. Even leaner subjects with this fat distribution pattern have been found to be at higher risk for these comorbidities.

This may be one reason why overweight and obese Mexican Americans are at a greater risk for comorbidities. Being overweight or obese and being Mexican American has been shown to be a greater risk for developing Type 2 diabetes. In addition to the fat distribution pattern, recent research has shown that certain single nucleotide polymorphisms are associated with Type 2 diabetes. These single nucleotide polymorphisms have been found at a greater rate in certain Mexican American populations. This suggests that there is a genetic component that increases the prevalence of Type 2 diabetes among Mexican Americans.

It is not only Mexican American adult populations that have a higher rate of overweight and obesity. Analysis of the National Health and Nutrition Examination Survey found that Mexican American adolescents are at a greater risk for adverse health outcomes, compared to youth of other racial and ethnic groups. The prevalence of overweight was 40.9 percent and obesity 22.9 percent in Mexican-Americans ages 12 years to 19 years. For most ages, males had a higher prevalence of overweight and obesity than females. One in every three Mexican-American male adolescents was obese. Some of the factors associated with overweight and obe-

sity among Mexican Americans include being from a low-income family, having a history of asthma, high blood pressure, perceiving oneself as being less physically active compared to one's peers, and not being in excellent health. Overweight adolescents often reported a lowered caloric intake than their non-overweight counterparts. However, people who are overweight or obese often underreport their caloric intake. There is no evidence to suggest that this is not the case in Mexican American adolescents. Difficulty in accurately counting calories may be a reason that people become overweight or obese.

Previous studies concerning immigrant populations have suggest that American cultural norms, values, and practices, may contribute negatively to several health outcomes, one example of which is poor eating habits. Compared to second- or first-generation Mexican American adolescents, third-generation Mexican American adolescents experience excessive health disparities. These findings have been shown to be true even when controlling for economic circumstances. These findings present an epidemiological paradox.

Living in the United States is associated with having better access to healthcare and health insurance. Therefore, one would assume that this would result in a decrease in adverse health outcomes, including overweight and obesity. In general, however, Mexican American self-report comparatively poor access to health care services, regardless of health insurance status. One measure of acculturation, speaking English at home, is correlated to higher prevalence rates of chronic illness than Mexican Americans who do not speak English at home.

Increased acculturation has been correlated to a decline in healthy behavior among Mexican Americans. For both men and women, the likelihood of having a high BMI was greater among those who were more acculturated. A traditional Mexican diet consists mainly of low-fat foods, such as beans, rice and vegetables.

More acculturated Mexican Americans were found to consume a great quantity of more calorically-dense foods. Even when compared to other Hispanic racial and ethnic groups, Mexican Americans has a higher total and saturated fat and lower carbohydrate intake as percent of calories than Puerto Rican and Cuban Americans.

One theory that that attempts to explain the high prevalence of overweight and obesity is the cultural changes that Mexican Americans have undergone in the past few years. In the past few generations, Mexican Americans have undergone a rapid nutritional transition. Their ancestors consumed most grains, fruit, and vegetables. However, by migrating from rural to urban regions, there has been an increased consumption of fats and refined sugars. This migration also resulted in the decrease of physical activity and a more sedentary lifestyle. Genetic characteristics that enabled Mexican American to thrive in a less calorically dense, more physically demanding environment may have negative effects on them once their environment has changed. The higher prevalence of

Obbesity is made worse due to the fact that abdominal fat is the most common fat distribution pattern among Mexican Americans.

Type 2 diabetes in Mexican-origin populations could be related, in part, not only to Native American genetic admixture but partly to Spanish admixture, as a consequence of its historical contribution to the Mexican gene pool

Evidence that Mexican Americans are at a genetic predisposition for becoming overweight and obese and developing some of the comorbidities can be gathered from examining the prevalence of these diseases in a urban Mexican population. There is a higher prevalence of obesity, high blood pressure, Type 2 diabetes and dyslipidemia. Mexico is ranked third in the world for obesity prevalence, only behind Kuwait and Samoa. It has also been observed that a large percentage of Mexican obese individuals had abdominal fat distribution. Fat distribution is often genetic.

SEE ALSO: Hispanic Americans; South America.

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Microarray Analysis

A MICROARRAY IS an assay that is used to determine which genes are expressed in a sample. They can assess thousands of genes in a single experiment; therefore, the process is often known as gene expression profiling. A gene chip is used which holds a collection of microscopic probes attached to a solid surface such as glass, plastic, or silicon. Depending on the type of microarray, the probes consist of either DNA, proteins, antibodies (a separate type of protein microarray), or tissues. The most common type is the DNA microarray, which may consist of either cDNA or oligonucleotide probes. Each microarray experiment relies on the principle that expression is determined by the amount of hybridization of a labeled RNA to a complementary probe. Once the microarray is constructed, the target mRNA population is labeled, typically with a fluorescent dye, so that hybridization to the probe spot can be detected when scanned with a laser.

Measuring gene expression using microarrays is relevant to many areas of biology and medicine, such as studying treatments, disease, and developmental stages. For example, microarrays can be used to identify disease genes by comparing gene expression in diseased and normal cells. In terms of obesity, it is often used to determine changes in expression of genes related to adipocytes. Newer methods, however, are looking at gene expression in the brain to examine psychological factors affecting eating habits.

The microarray can be used with different interventions to determine what sets of genes are activated by the intervention. Once those genes are identified, all data from the microarray should be verified with a method such as reverse transcriptase polymerase chain reaction (RT-PCR). It is important to remember that microarrays are qualitative, not quantitative. Microarrays only tell a researcher whether a gene is activated; they cannot be read to determine to what extent it is activated (i.e., whether one gene is activated two times more than another). Because of the inability to conclude quantitative data as well as sources of potential error, the importance of the use of RT-PCR as verification cannot be overstated. One potential downside to the microarray is that it gives so much information that it blurs the line between hypotheses and data collection. Researchers fear that

it will encourage data fishing, with no proper scientific rationale to explain results.

With the current epidemic of obesity in America, the field of microarray research in regards to obesity is wide open. While epidemiological obesity problems are intricate, with psychological, socioeconomic, and cultural causes among others; genes also play a major role. Major questions that may be answered using microarray technology include what genes may predispose people to obesity and what effects upstream behavioral effects such as overeating and sedentary lifestyle play on gene expression.

For example, in a study conducted by Samuel Nadler and associates, a microarray examination comparing lean and obese mice showed a reverse gene expression profile between the two groups. Genes which had previously been shown to increase during adipocyte differentiation were decreased in obese mice, while genes previously shown to decrease during adipocyte differentiation were increased in the obese mice.

The use of microarray technology is fostering the growth of a new field known as nutrigenomics. Nutrigenomics focuses on the interactions between the nutrients persons consume in the diet and specific genes in their DNA. Much research has been completed, with varying results, on nutrient interactions with human adipose tissue. Other factors possibly contributing to obesity are now being examined. For example, Permana and colleagues propose that because food intake is controlled by processes in the brain, obesity is a primarily neurological disorder with genetic causes.

This same group, therefore, designed experiments to examine differential gene responses to nutritional intervention in obese versus lean individuals. Some preliminary data taken from human hypothalami revealed differential gene expression of 26 transcripts in obese versus lean participants, including histidine carboxylase and neuropeptide Y2 receptor. Although these findings need further research, these transcripts show promise because obesity is essentially an imbalance of energy intake/output, and these genes contribute to energy homoeostasis in animals.

As a relatively new method of research, microarray analysis holds enormous potential in battling the obesity epidemic. In the coming years, as nutrigenomics and translational research become even more prevalent, hopefully the microarray will help provide answers to the questions about genetic predisposition to obesity.

SEE ALSO: Genetics; Genomics; Translational Research.

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Middle East

OBESITY HAS BECOME an increasing problem in the Middle East with the change in diet—partly through prosperity—the decrease in exercise, and also the more widespread use of the motor car and availability of public transport.

In Turkey, European writers from the early modern period wrote of obese Ottoman Sultans, officials, and courtiers as well as members of the sultan's harem. Even allowing for literary license, the problem does seem to have been a significant one, but was restricted to the extremely narrow ruling class. Most of the problems that Turkey has faced regarding obesity have been during the 20th century when there has been a decrease in manual labor and an increase in sedentary occupations, a decline in the amount of exercise, and a dramatic change in the local diet with the introduction of many foods that contain saturated fats.

Associate Professor Volkan Yumuk of Harbiye, Istanbul, from the Turkish Association for the Study of Obesity is the national representative on the International Association for the Study of Obesity

A Middle Eastern meal of broiled fish. Obesity has become an increasing problem in the Middle East due to a change in diet.

(IASO) and responsible for increasing the public awareness of the problem of obesity in Turkey.

In a very recent survey carried out in Izmir City, Drs. F. Sarac, S. Paryldar, E. Duman, F. Saygyly, M. Tuzun and C. Yylmaz of the Department of Endocrinology and Metabolism, Ege University Hospital, Bornova, Izmir, distributed survey questionnaires to 1,752 obese and 400 normal-weight adults in the city, and from the replies that they received, they were able to show that 45 percent of obese women wrote that they experienced a reduced quality of life owing to their obesity, while 41.3 percent of men felt the same.

The results also showed that obesity is closely associated with poor levels of general health. Another survey led by Dr. D. Ozmen from the Department of Public Health Nursing, School of Health, Celal Bayar University, Manisa, found that low self-esteem and depression was more common among obese Turkish adolescents than their nonobese school contemporaries. The health complications coming from obesity were clearly shown to result in a lower level of disease-free and distant disease-free survivals in cancer patients being treated for operable breast carcinomas.

In Syria, there has also been a rise in obesity, but the better diet of many people in the country has led to lower rates than in Turkey. One study in 2004 of secondary schoolchildren in Damascus and nearby areas found that more than 35 percent of the children had not eaten meat in the week prior to being interviewed, and worryingly, more than half had not eaten green vegetables during that time, with only 11.9 percent having eaten fruit three or more times a day. Although the lack of meat eating has reduced the prevalence of childhood obesity, the lack of vegetables and fruit will result in other medical problems. In another study, which was into the cardiovascular health of adults in Aleppo, obese people were found to be more at risk of problems and complications than others. There is also a difference between obese people of different genders. A much earlier study, undertaken by S. Hadidy, L. Fahdi, H. Tabbakh, and M. Deebo and published in 1983, showed that the serum level of triglycerides was significantly higher in obese males than in females also suffering from obesity.

Lebanon, to the south of Syria, went through a period of prosperity from the 1930s, which ended with the civil war in 1975, and once again is going through rapid social change and modernization. During the 20th century, the diet of many people in Lebanon has significantly changed, with more dairy products and saturated fats being consumed every day. It is estimated that possibly a fifth of the current population of Lebanon are overweight, but no recent figures were collected until Drs. F. G. Haddad, H. Brax, E. Zein, and T. El Abou Hessen, of the Service de Médecine Interne, Centre Hospitalier et Universitaire Hôtel-Dieu de France, in Beirut, carried out a survey assessing the prevalence of obesity among Lebanese patients in hospitals in Beirut from 2000, analyzing material on 313 patients aged 13 years and older. They found that 22.1 percent of these were overweight, and 18 percent were obese, with the percentage considerably higher for those aged 40 years and older. The figures are very high, but given that patients suffering from obesity have a higher prevalence to have other medical complications, the figures for the overall population would be considerably lower.

To ascertain the reasons for obesity, there was another survey into household consumption in 2001. Using a random sample of 444 Beirut residents aged 25–54 years, it was quickly discovered that fat contributed 38.9 percent of the average daily energy intake, with carbohydrates providing 47.2 percent and protein 13.4 percent. The average consumption was 3,030 grams per day, with 45.3 percent of subjects eating less than the recommended 400 grams of fruits and vegetables each day. With most cooking not using olive oil, it was also found out that women consumed more dairy products, milk, vegetables, and coffee than men, with women generally eating a lower-fat diet than men. People aged 25–34 consumed more meat, more sugar, and considerably more alcoholic beverages and soft drinks than older people. With a low consumption of fish and heavy intake of bread, the study highlighted some of the problems that may well lead to higher rates of obesity in Lebanon in the years to come. Indeed, in a study of 12,299 adolescents at private schools in Lebanon, carried out by H. Chakar and P. R. Salameh of the Faculty of Medicine, University of Balamand, 7.5 percent were found to be obese, with 24.4 percent at risk of obesity.

In Israel, although the prosperity of the population has led to increasing numbers of people being overweight, the strict diet followed by many people has reduced the prevalence of obesity, although there have been some notable exceptions such as Ariel Sharon, the prime minister from 2001 to 2006, who suffers from obesity and developed cardiovascular problems, eventually suffering from a stroke. The Israel Society for the Study and Prevention of Obesity has campaigned for greater awareness of obesity in the country, with Professor Maximo Maislos, Head of the Atherosclerosis and Metabolism Unit, Soroka UMC Department of Medicine, Ben Gurion University, Beer-Sheva, serving as the national representative on the IASO.

Obesity has not been much of a problem in Jordan, nor in neighboring Iraq, although the current Iraqi president, Jalal Talabani, has made occasional jokes about his own obesity, and there have been studies of rising levels of obesity among children, many of whom have been housebound for several years with their parents afraid to let them go to school or to sports activities because of the insurgency.

In the countries of the Persian Gulf, the incredible affluence brought by oil has resulted in a change in diet, a decline in exercise, and the increase in sedentary occupations, all of which have led, as they have done in so many other countries, to a rise in obesity levels. Studies have shown this to be the case in Kuwait. In Bahrain a study carried out at the Naim Primary Health Care Center by F. A. Alnasir and E. M. Masuadi of the Department of Family and Community Medicine, Arabian Gulf University, Manama, Bahrain, related obesity to the level of lipids in the bodies of the people being studied. With so little known about the diets of schoolchildren in the Gulf States, a 2006 study of children in Qatar, published in 2007, shows that the levels of obesity there were 1.6 percent for males and 5.4 percent for females, with 54.8 percent of males and 23.1 percent of females being underweight. This certainly indicates that the Gulf States probably have a much lower rate of childhood obesity than many other countries in the world, although figures collected in the United Arab Emirates at the same time show that 13.7 percent of schoolchildren were obese, again with girls much more likely to be obese than boys.

There have also been surveys undertaken in Oman by J. A. Al-Lawati and P. Jousilahti from the Department of Non-Communicable Diseases Surveillance and Control, in Muscat, who studied Omanis in Nizwa. In Yemen, Y. A. Raja'a and M. A. Bin Mohanna of the Department of Community Medicine, Faculty of Medicine and Health Sciences, Sana'a University, studied 1,253 school children in 2002–03 and found the percentage of children aged 10–14 who were overweight was only 6.2 percent, with those obese being 1.8 percent.

SEE ALSO: Africa, North; Prevalence of Childhood Obesity Worldwide.

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Monogenic Effects that Result in Obesity

AS HUMAN GENES have not changed appreciably over the last few decades, the recent increase in the prevalence of obesity has been attributed to obesigenic environmental and lifestyle factors, namely, excessive caloric intake and reduced physical activity. However, the significant variability between humans in their propensity to gain weight suggests that susceptibility to obesigenic environmental challenges is largely determined by genetic factors.

"Common" obesity is thought to arise from mutations in multiple genetic loci (polygenic obesity), each with modest effects, which interact with each other (gene–gene interactions) and/or with obesigenic environmental factors (gene–environment interactions) to increase body weight. Obesity resulting from mutations in a single gene (monogenic obesity) is comparatively uncommon, but has increased our understanding of the complex pathways that regulate appetite and body weight homeostasis in humans. Much of the research in this area has concentrated on the leptin-melanocortin pathway, the components of which are important determinants of energy balance in animals and humans.

THE LEPTIN-MELANOCORTIN PATHWAY

Leptin is a fat-derived hormone, or adipokine, the circulating levels of which correlate with the degree of adiposity. Activation of proopiomelanocortin (POMC) neurones in the hypothalamic arcuate nucleus by leptin (and insulin) results in the proteolytic cleavage of POMC by prohormone convertase-1 (PC-1), yielding the anorectic peptide α-melanocyte stimulating hormone, an endogenous ligand at melanocortin 4 receptors (MC4R) in the paraventricular nucleus. Concurrently, leptin inhibits the release of the orexigenic neuropeptide Y. Activation of MC4R has a number of downstream effects, culminating in inhibition of food intake. Humans with monogenic forms of obesity because of mutations in a number of components of this pathway have been identified over the last decade or so, highlighting the importance of the leptin-melanocortin pathway in mediating energy balance and appetite regulation in humans. With the exception of MC4R deficiency, only a small number

of humans with mutations in leptin, leptin receptor, POMC, and PC-1 have been identified. Other genetic syndromes, in which obesity is one of many characteristic features, such as Bardet-Biedl, Prader-Willi, and Alstrom syndromes, will not be discussed further in this entry.

MUTATIONS IN LEPTIN AND LEPTIN-RECEPTOR

Following their initial description of two severely obese Pakistani cousins from a consanguineous family, who were homozygous for a frameshift mutation in the leptin gene, Farooqi and O'Rahilly have identified a number of other individuals homozygous for the same mutation. Congenital leptin deficiency results in extreme and early-onset hyperphagia and obesity. Affected individuals exhibit marked increases in body fat (body fat around 57 percent of weight), delayed puberty with hypogonadotrophic hypogonadism and abnormalities in T-cell number and function, with increased rates of childhood infection and mortality.

The Cambridge group has also reported that treatment of leptin-deficient individuals with subcutaneous leptin therapy led to a significant reduction in body weight (almost entirely due to a reduction in body fat), associated with a reversal of hyperphagia. Furthermore, treatment with leptin led to the onset of appropriately timed puberty and an improvement in abnormalities of immune function.

Homozygous and compound heterozygous mutations in the leptin receptor have also recently been described in severely obese individuals. Although humans with leptin receptor-deficiency exhibit similar features to those with congenital leptin deficiency, there is some recent evidence to suggest that the clinical phenotype may be less severe.

MUTATIONS IN POMC AND PC-1

The clinical phenotype of human POMC deficiency is characterized by red hair and pale skin (due to lack of signaling at the MC1R in cutaneous melanocytes), neonatal adrenal crisis (as POMC is a precursor of ACTH in the pituitary), and hyperphagic obesity (due to lack of MC4R signaling in the hypothalamus).

Mutations in PC-1 have been described in two humans to date. PC-1 cleaves POMC and other pro-hormones. The reported phenotype includes juvenile-onset obesity, hypogonadotrophic hypogonadism, hyperinsulinemic hypoglycemia, ACTH deficiency, and interestingly, defective small intestine absorption.

MUTATIONS IN MC4R

By far the most common form of monogenic obesity in humans is due to mutations in the MC4R gene, a seven-transmembrane G-protein couple receptor predominantly expressed in the central nervous system. The prevalence of MC4R deficiency is up to 5 to 6 percent in severely obese children and up to 2 to 3 percent in obese adults, making this disorder one of the most common known monogenic diseases. MC4R deficiency is (co)dominantly inherited, with homozygotes more severely affected than heterozygotes. A correlation between the degree of in vitro receptor dysfunction and the clinical phenotype has been described.

Humans with MC4R deficiency exhibit the following clinical features: early-onset hyperphagia and obesity, increased fat and lean masses, hyperinsulinemia, increased linear growth, and increased bone mineral density. The growth hormone axis and pubertal development and timing are normal. Unlike mice with MC4R deficiency, no defect in energy expenditure has yet been identified in humans. Interestingly, the phenotype appears to be most prominent in childhood, with many features, particularly hyperphagia, hyperinsulinemia, and accelerated linear growth becoming less marked with age.

SEE ALSO: Genetic Influences on Eating Disorders; Genetic Mapping of Obesity Related Genes; Genetic Taste Factors; Genetics.

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Mood and Food

EMOTION-INDUCED EATING HAS been implicated as a risk factor for the development of obesity. Numerous studies have shown that some people overeat as a way to control negative emotions. One reason for this is that eating during times of emotional distress acts to reduce negative emotional experiences. When most people think about mood and eating, they think about the phrase *emotional eating*. This can be defined as eating in response to negative emotions which may include stress, anger, anxiety, boredom, sadness, and loneliness. Food can be used to produce feelings of comfort, connection, and love.

The opposite may also occur. For many, mealtimes during childhood may be remembered as a time of family conflict and criticism. As children, many people were rewarded with sweets for being "good." In attempt to replicate these positive feelings, some people unconsciously or consciously reward themselves with food. Most food rewards are comfort foods, ones that are high in calories and usually filled with sugar and fat. The use of food to manage mood becomes a self-reinforcing habit. Comfort food helps a person feel good in the moment, but it is usually temporary. Once finished overeating, thoughts return to problems or worries. Additionally, many people report having the additional worry of guilt about their overeating. Thus, the result is a vicious cycle.

Emotional eating occurs on a continuum. For most people, infrequent times of emotional eating are normal. We all celebrate with food during the holidays and on special occasions. We also tend to mourn with food and use food to comfort others when something sad occurs, like an illness or a death. Further along the continuum are those who use food as a primary coping mechanism and as a strategy for mood regulation. For these individuals, food is used in reaction to, and as a defense against, any intense feeling or stressful life situation. At the far end of the spectrum are individuals for whom emotional eating has greatly begun to interfere with daily functioning and with life in general. These individuals may have a diagnosable eating disorder, such as bulimia nervosa or binge-eating disorder, and they may also suffer from clinical depression or another mood disorder.

There are both psychological and biological explanations to help explain why people turn to food, even when it causes problems such as depression and obesity.

STRESS

One of the biggest contributors to negative mood states is stress. Stress can be defined as the types of things that produce negative emotions. There are two different types of stressors. The first are stressful events—major ones such as a job change or unemployment, a divorce, or a medical problem. The other type of stressors are considered daily hassles, which can be defined as repetitive but chronic annoyances of everyday life such as waiting in line, sitting in traffic, or bad weather. Either type of stress can trigger emotions that lead to overeating. As a result, people under long-term stress tend to gain weight over time. This might be related psychologically to learned coping behaviors (eating in response to stress as a way to cope with negative moods) as well as to underlying biological changes.

One thing that varies greatly among emotional eaters is how a person perceives stressful situations and how that individual has learned to respond to stress. One individual may feel major stress from a particular situation, whereas another person will handle it better by using the event as an opportunity to improve himself or herself. In general, those on the lower end of the emotional eating continuum have higher perceived stress thresholds (meaning a situation has to be fairly severe to be perceived as being stressful) and stronger coping mechanisms for dealing with stress. On the other hand, individuals farther along on the spectrum, such as those with eating disorders, tend to perceive even mildly stressful events as being more stressful.

When considering the biological contribution of stress to emotional eating, cortisol, a hormone secreted during stress, is a key player. Cortisol is involved in regulating caloric consumption and has been shown to increase appetite and food intake. Thus, individuals who produce excess cortisol, such as those with binge-eating disorder and clinical depression, might be more susceptible to overeating following stress. Recent studies in rats have led scientists to speculate that the use of comfort foods in humans may be stimulated by elevated cortisol in response to stress, which could also be related to increased obesity.

BINGE EATING

Binge eating is defined as overeating a large amount of food while experiencing a sense of loss of control during the eating episode. Not all emotional eating is binge eating, but binge eating plays an important role in the relationship between mood and food. In fact, some researchers have shown that negative mood predicts overeating and can lead to the development of binge-eating disorder in females. In fact, many people report that negative mood states occur directly before a binge.

Affect regulation theory suggests that heightened emotional disturbances increase the likelihood of binge eating because an individual feels that eating will soothe his or her painful negative emotions. Food then serves as a tension reliever, distracting a person from uncomfortable feelings. Eating food takes the edge off any feeling that a person would rather not feel or tolerate. Numerous researchers have suggested that negative affect (negative moods and/or feelings) increases risk for certain addictive behaviors, including binge eating. In more severe eating disorders, such as bulimia nervosa, individuals attempt to get rid of their calories (by vomiting or laxative abuse) to reduce their anxiety about impending weight gain consequent to overeating. For these individuals, the purging itself provides an emotional relief, whereas for others, overeating provides the relief from negative emotions.

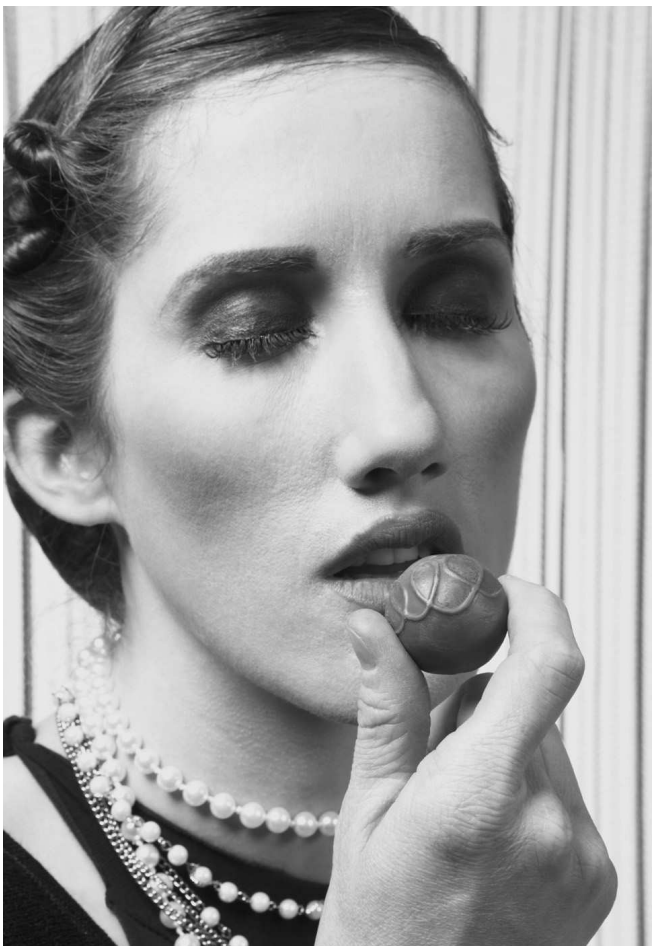
DEPRESSION

Depressed mood and negative affect have been found to predict future increases in symptoms of disordered eating, including binge eating. Specifically, depressed individuals may consume food to improve their mood or as an attempt to escape from adverse emotions. The pleasure they feel after eating may serve as a reinforcement, which in turn leads to continued overeating. Often, this develops into a cycle which can be difficult to break.

Binge eating severity and degree of depression are strongly related. Higher rates of clinical depression have been found in obese binge eaters compared to obese individuals who are not binge eaters. Furthermore, overweight and normal-weight binge eaters often have similar levels of depression, suggesting that binge eating, rather than body weight, is related to depression. In other words, of people who are obese, those who are obese and also binge eat are more likely to also be depressed, possibly as a result of their binge-eating behavior. It has been suggested, then, that binge eating might therefore be link between depression and obesity, because often binge eating starts before a person becomes obese. Depression might also help explain why people become binge eaters. However, scientists are not really sure if depression comes first and leads to binge eating or if people begin binge eating and then become depressed because they cannot control their food intake.

MOOD REGULATION

Many researchers are focused on the biology and brain chemistry of mood regulation and overeating. The limbic system is the area of the brain that regulates many activities, including emotions and the stress response. The hypothalamus, located at the base of the brain, controls many basic functions, including appetite and



Eating comfort foods help a person feel good in the moment, but it is temporary—once finished overeating, thoughts will return to problems.

stress reaction. Moreover, it regulates the function of the pituitary gland, which in turn, regulates key hormones including cortisol. Disturbances or abnormalities in the limbic system, including how neurotransmitters function, could affect mood and behavior.

Serotonin, norepinephrine, and dopamine are neurotransmitters that have been implicated in depressed mood and other mood disorders. Additionally, high cortisol levels might also contribute to depressed mood. Interestingly, both food and mood regulation share similar biological pathways and thus help describe the mood and food relationship. For example, when food is consumed, changes in serotonin, endorphins, and dopamine levels occur in the brain. These are very powerful chemicals, which also affect mood. In turn, food can greatly affect mood. Caffeine consumption mimics the body's stress response, increasing, and causing a release of stress hormones. In small doses (about two cups of coffee), caffeine acts as a mild stimulant. However, overconsumption can cause anxiety and physical symptoms such as headaches and mild tremors.

Macronutrient content of food can also influence mood. When carbohydrates are eaten, the brain's production of serotonin is temporarily increased. Serotonin reduces anxiety and has a calming effect. Thus, many people use carbohydrate-rich foods for comfort and to feel good. Fat, however, does not influence brain chemistry in the same manner. Protein acts opposing with carbohydrate metabolism and prevents this soothing effect from carbohydrates from occurring. Protein increases the brain's production of the neurotransmitter dopamine, which temporarily boosts concentration and alertness. This might help explain why most people prefer to eat sweet, carbohydrate-rich food when they are stressed. So, people not only use food to psychologically distract themselves from bad moods, but there is physiological evidence that eating the food actually makes them feel better.

ADDICTION MODEL

In addition to coping mechanisms, the reasons why people who are chronically stressed and eat in response to their negative moods are also related to the idea that some foods have seemingly addictive qualities. From a biological perspective, some of the chemicals produced during stressful times help determine what one eats and how one stores fat in the body. Recent studies have used scans of the brain to investigate similarities in the brain chemistry of drug addicts and chronic overeaters. Evidence shows that several chemicals in the brain can influence the motivation to eat. One class of drugs is the endogenous opioids. Drugs of abuse strongly stimulate this system either directly or indirectly. For example, when a person eats palatable foods (foods that taste good), such as chocolate, the body releases trace amounts of opioids, which in turn help elevate mood. That "reward" may reinforce a liking for foods that remind one of this satisfying and good feeling.

Serotonin has been coined the "feel-good" hormone and also plays an important role in the regulation of mood and appetite. It influences the intake of certain substances such as alcohol and cocaine. Excessive consumption of carbohydrate-rich foods has been found to be associated with negative moods. Similarly, recent evidence has shown that consumption of carbohydrates is often used as self-medication because sugar-filled, carbohydrate-rich foods trigger the release of serotonin and, in turn, temporarily elevate mood.

The brains of obese individuals may also be lacking brain receptors for dopamine, a chemical that is part of the brain's motivation and reward system. Dopamine, like serotonin, makes a person feel good and could be strongly related to the urges one has to eat something in order to make one feel good. In a study comparing the brain scans of obese and normal-weight volunteers, researchers observed that obese individuals had fewer dopamine receptors compared to lean individuals. Thus, the brains of obese people and drug addicts both have fewer dopamine receptors than normal subjects. Whether this suggests a propensity to addiction (drugs in one case, food in the other) for these individuals or a reaction to drug use and overeating is not clear. However, low dopamine has been related to emotional eating scores on a questionnaire and to frequency of food cravings.

Several studies of the brain related to drug craving in addicted subjects suggest that similar brain circuits underlie the enhanced motivational drive for food in obese subjects that are found in drug-addicted subjects. Some obese people get more pleasure from eating than do people of normal weight. These individuals might be more likely to overeat in response to negative moods, because they get more pleasure from food than others.

In summary, the relationship between mood and food is circular, and comprises both psychological and biological pathways. As a result of either negative events or decreased levels of chemicals in the brain, people experience negative mood states. As a result, in response to past learned behaviors, a person overeats, choosing primarily sweet and fatty foods that produce chemicals that make him or her feel better. Simultaneously, this eating also distracts a person from negative moods. Once the eating episodes are finished, the person often feels very guilty and ashamed. This produces a brand-new cycle of negative moods and result in changes in brain chemistry.

BREAKING THE CYCLE

Although admittedly difficult, it is possible to break the cycle of emotional eating. A few tips are as follows:

Managing stress. This is an important first step. Prioritize tasks and learn to say no. Many emotional eaters are also caretakers, often finding time to take care of everyone but themselves. It is okay to decline invitations and to ask others for help.

Emotion regulation. Learn to deal with your emotions by talking about your feelings with others, rather than trying to feel better by overeating. Ultimately, you must learn to stop using food to cope with your feelings. One way is to practice becoming more tolerant at dealing with negative emotions, rather than quickly trying to soothe them with food.

Eat only when you are hungry. This is tough to master but can be achieved with practice. Rate your hunger on a scale from 1 to 10 before you eat, with 10 being famished and 1 being stuffed. If you are not actually hungry, engage in an activity that does not involve food. You can reevaluate your hunger 30 minutes to one hour later.

Journal. Make a list of situational and emotional triggers. Keep a food and emotion journal that includes what you eat, how much you eat, when you eat, how you are feeling when you eat, and how hungry you are. This will help reveal patterns that might not be obvious. In the future, you will be better able to avoid situations that result in negative emotions or trigger overeating.

Distraction. Instead of eating, take a walk, listen to music, read, or call a friend. Pamper yourself. Try engaging in activities that either make you feel good or serve as good distractions from food.

Clean out the cabinets. Although people are constantly bombarded with trigger foods in the environment, you have the ability to limit the types of foods kept in your homes. Avoid having an abundance of high-calorie comfort foods in the house. Talk to family members to obtain support and permission to remove some of those favorite foods from the home. Instead, choose healthier snacks. Over time, these will begin to satisfy cravings.

Exercise. Exercise burns calories and reduces stress. It can also distract a person from whatever is causing stress in his or her life, and over time, it can take the place of food. It stimulates the production of the feel-good brain chemicals such as endorphins and serotonin and therefore can make one feel better, much in the same manner as food.

SEE ALSO: Binge Eating; Compulsive Overeating; Depression; Food Reward; Stress.

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Morbid Obesity in Children

IN PEDIATRICS, THE term "obese" generally refers to children whose weight weight for height (or Body Mass Index—BMI) is greater than the 95th percentile for their age and gender (notably, some institutions instead refer to this group as "overweight"). The complications of morbid obesity span multiple

organ systems and include such disorders are hypertension, Type 2 diabetes mellitus, obstructive sleep apnea, and many others. Treatments for morbid obesity include lifestyle modification, pharmacologic therapy, and in the most extreme cases, bariatric surgery.

DEFINITION

In pediatrics, the term *obese* generally refers to children whose weight is greater than the 95th percentile for their age and gender (notably, some institutions instead refer to this group as "overweight"). While no specific percentile is delineated to classify a child as "morbidly obese," the term is generally used to identify those children whose risk of obesity-related illnesses is dramatically increased compared to other obese children.

PREVALENCE

The term *morbid obesity* is not officially recognized in the pediatric population by the U.S. Centers for Disease Control and Prevention (CDC). Thus, accurate statistics to measure the prevalence of morbid obesity in U.S. children are not available. However, based on statistics from 1999 to 2004, the prevalence of BMI above the 95th percentile (referred to by the CDC as "overweight" and by other institutions as "obese") in children and adolescents increased from 13.9 percent in 1999–2000 to 17.1 percent in 2003–04. Research indicates that the prevalence of morbid obesity increases significantly faster than obesity alone (this research was compiled from adult research subjects), so the prevalence of morbid obesity in children is likely increasing as fast, if not faster, than obesity.

Epidemiologic data on morbid obesity are not currently available. However, the data for obesity alone in 2003–04 indicate that 16.3 percent of Caucasian children, 19.2 percent of Mexican-American children, and 20.0 percent of African-American children were above the 95th percentile for BMI.

CAUSES

The cause of morbid obesity in childhood is unclear. However, many researchers believe that morbid obesity is simply an extreme form of obesity and is thus subject to many of the same risk factors. The specific risk factors for childhood obesity are innumerable, but can be easily grouped into categories of genetic or inborn versus environmental risks. The genetic contribution to obesity is estimated to be anywhere from 25–70 percent, while the environmental contribution can vary in the same manner. However, most experts agree that much of the increase seen in obesity over the last 30 years has been due environmental risk factors as sedentary lifestyle (television watching, video game playing, etc.), large portion sizes, and the consumption of high-calorie, high-fat food. Limited education, the cessation of smoking, and lower socioeconomic status are also associated with increased risk of obesity.

Genetic and endogenous factors also appear to play a role in obesity. Various family, adoption, and twin studies have demonstrated a strong genetic component to becoming obese. Mutations in certain genes such as the beta-3-adrenergic receptor gene and the peroxisome-proliferator-activated receptor (PPAR) gamma 2 gene have been associated with altered protein function that is believed to contribute to weight gain. In addition, metabolic, neurologic, and endocrine factors are also associated with obesity; those individuals with lower metabolic rates, lower levels of sympathetic nervous system activity, problems with insulin resistance, and dysfunction of the dietary feedback control system (involving such chemicals as ghrelin, leptin, and melanin-concentrating hormone) are all at an increased risk of becoming obese.

Notably, certain genetic and endocrine disorders are also associated with severe obesity. One of the most common genetic disorders associated with obesity is Prader-Willi syndrome, a neurodegenerative disorder that, among other things, is characterized by hyperphagia (increased appetite and food consumption) and generalized obesity by 2–3 years of age. Endocrine disorders associated with obesity include hypothalamic deficiency, hypothyroidism, Cushing syndrome, and growth hormone deficiency.

COMPLICATIONS

The term *morbidly obese* is generally used to describe people who are at a highly increased risk of developing weight-related health consequences. The following is a description of the many complications for which morbidly obese children are at high risk.

Cardiovascular Complications. The two main cardiovascular complications of childhood obesity are hypertension and dyslipidemia. Hypertension, or high blood pressure, is diagnosed when a child's blood pressure is above the 95th percentile based on their age, height, and sex. Dyslipidemia is characterized by an imbalance of one's cholesterol and triglcyeride levels. Most commonly, obese children will have a high level of low-density lipoprotein (LDL, aka "bad") cholesterol and a low level of high-density lipoprotein (HDL, aka "good") cholesterol. Both hypertension and dyslipidemia are significant risk factors for developing atherosclerosis, which is a major cause of heart attacks and strokes later in life.

Endocrine Complications. One of the most common complications of morbid obesity in children is impaired glucose tolerance, which is defined as a blood glucose level between 140 and 199 mg/dl at the two-hour mark of a 75 gram oral glucose tolerance test (in which serial blood glucose levels are measured after the patient drinks a concentrated sugar solution). If no changes are made to the child's lifestyle and weight, this condition may progress to full-blown Type 2 diabetes mellitus. Diabetes is a very serious condition with its own associated complications, including cardiovascular disease, eye disease, kidney disorders, and nerve problems. Because of these severe, yet preventable, health consequences, both the American Academy of Pediatrics and the American Diabetes Association recommend screening certain overweight children (those with other risk factors in addition to their obesity) for Type 2 diabetes every two years starting at age 10.

Endocrine complications of obesity also have an effect on the reproductive system. In males, obesity has been associated with delayed onset of puberty. However, the opposite is true in obese females, who tend to experience an earlier onset of puberty. Obesity is also a significant risk factor for polycystic ovarian syndrome, a condition in females that is associated with infertility and an abnormally high level of androgens in the bloodstream.

Gastrointestinal Complications. Cholelithiasis, or having gallstones, is relatively uncommon in children as compared to adults. Of those children with cholelithiasis, obesity is the most common cause. The presence of gallstones can be completely asymptomatic or can be associated with a number of symptoms including fatty food intolerance, jaundice, and severe abdominal pain. If a patient's gallstones present any health risks or are the source of frequent pain, surgical removal of the gallbladder may be indicated.

Obesity is also associated with the most common liver disease in children, nonalcoholic fatty liver disease (NAFLD). Most children with this condition do not have any symptoms, but undetected liver damage can be severe; if untreated, some children may even progress to liver failure.

Neurologic Complications. Pseudotumor cerebri, or idiopathic intracranial hypertension, is a condition in which the blood pressure in the cranium is elevated for unexplained reasons. Approximately half of the children who present with this condition are obese and obesity is believed to be a risk factor for the disease. Typically presenting with headache, the condition can lead to severe visual impairment or even blindness. Management typically involves both an ophthalmologist and a neurologist.

Orthopedic Complications. One of the most common orthopedic complications of obesity is a slipped capital femoral epiphysis (SCFE). Most common in obese adolescents, this condition involves the displacement of the "cap" of the femur (the portion that contributes the "ball" to the hip's "ball and socket" joint) from the femoral shaft. Typically presenting with hip or leg pain and an altered gait, this condition requires immediate intervention by an orthopedic surgeon to prevent future bone complications.

Tibia vara, or Blount disease, is a condition in which excessive weight bearing on the legs of obese children causes the legs to become bowed and the tibias to become improperly rotated (tibial torsion). This condition also requires management by an orthopedic surgeon.

Pulmonary Complications. The most common pulmonary complication of childhood obesity is obstructive sleep apnea (OSA). Obesity, by decreasing the size of the airway and/or increasing its collapsibility, is a significant risk factor for this condition, which is characterized by "missing breaths" during sleep. OSA is believed to be a risk factor for hypertension and possibly other cardiovascular conditions. Children with OSA may re-

quire continuous positive airway pressure (CPAP) during sleep.

TREATMENT

A number of treatments for morbid obesity exist today, but each is of limited success. Lifestyle interventions such as decreasing one's calorie and fat intake, instituting structured physical activity into daily life, and reducing sedentary behaviors (such as watching television) have all been demonstrated to achieve short-term weight loss. However, as these routines are often difficult to initiate and even more difficult to maintain, they are of limited effectiveness in the long term. Pharmacologic interventions are also used for morbid obesity. Medications such as orlistat and sibutramine have been widely studied and are shown to have modest effectiveness in promoting weight loss over the long term. Newer medications are being developed every day.

Bariatric surgery is a form of obesity treatment that is typically undertaken only after fervent attempts at lifestyle modification and pharmacologic interventions have failed. The pediatric patients who are typically recommended for bariatric surgery are adolescents who have failed prior interventions of at least six months in duration, have a BMI of at least 40, and who have obesity-related comorbidities that would benefit from weight loss. Bariatric surgery has proven to be considerably more effective than lifestyle and medical interventions at achieving weight loss and improving comorbid conditions. However, it has numerous potential complications and is only generally undertaken in the most dire of circumstances.

SEE ALSO: Bariatric Surgery in Children; Behavioral Treatment of Childhood Obesity; Hypertension; Orlistat; Polycystic Ovary Disease; Sibutramine; Sleep Apnea.

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Morbid Obesity in Men

OBESITY HAS BEEN labeled a public health crisis with nearly one-third of adults in the United States classified as obese. Morbid obesity (also referred to as clinically severe obesity) affects about eight to 10 million American adults (about five to 10 percent) and represents the fastest-growing group of obese individuals in the nation. It is estimated that about 8 percent of all adults, and specifically 2 percent of men, are morbidly obese. In addition to increased risks for heart disease, hypertension, Type 2 diabetes, and other obesity-related conditions experienced by both obese men and women, severely obese men are also at higher risk for aggressive prostate cancer and premature mortality.

Life expectancy may be as much as 20 years less for severely obese men compared to that of males at a healthy weight. Although a modest weight loss of 5 to 10 percent can reduce risks of comorbid conditions and mortality, diets and medically supervised weight-loss programs have proven to be largely ineffective for treating morbid obesity. Surgical treatment may represent a more effective option, given the currently available outcomes data that demonstrate the improvement or resolution of comorbid conditions, a reduction in mortality risk, and sustained long-term weight loss.

Body mass index (BMI) is the most commonly accepted measure used for categorizing individuals as under- or overweight, obese, or severely obese. BMI is calculated by dividing a person's weight in kilograms by his or her height in meters (weight [kg]/height [m^2]). BMI is not gender-specific and does not differentiate the amount of weight from muscle mass versus fat, so very muscular individuals could be misclassified by

using BMI alone. A person with a BMI below 18.5 is considered underweight, while a healthy-weight individual has a BMI in the range of at least 18.5 to 25. A BMI of at least 25 to 30 indicates that a person is overweight. Individuals with a BMI of at least 30 are considered obese. A person with a BMI of at least 40 (or at least 35 with a significant comorbid condition) is classified as morbidly or severely obese. Waist circumference or waist-to-hip ratio and percent body fat are also sometimes used to classify the degree of overweight and obesity. Men with a waist circumference of over 40 inches, or who have more than 25 percent body fat, are considered at risk for complications of obesity. In addition, an individual may be considered severely obese when he or she is 100 pounds or more over ideal body weight, as specified by the Metropolitan Life Insurance Company standard height and weight tables for men and women. The tables were originally developed to indicate a range of desirable weights associated with the lowest mortality rates.

Although BMI does not take into account gender differences, studies have demonstrated variability as to where fat accumulates (regional fat distribution) and its associated risks between males and females. Women tend to accumulate larger amounts of gluteal fat, resulting in a larger hip circumference (creating an "apple shape"), while men tend to store fat in the abdomen, resulting in a larger waist circumference ("pear shape"). Abdominal (upper body or visceral fat), has been correlated with a greater risk for cardiovascular disease, diabetes, hypertension, and hyperlipidemia.

Morbidly obese men may experience more carbohydrate intolerance and decreased endurance, and in turn, be less physically fit than severely obese women. In addition, morbidly obese men are also more likely than women to have sleep apnea, obesity hypoventilation syndrome, and venous stasis disease. Severely obese men are also two and a half times more likely than women to have nonalcoholic steatohepatitis, an obesity-related condition that interferes with liver function. Furthermore, the accumulation of abdominal fat may play a role in the development of colon, rectum, and aggressive prostate cancer. Recent studies have suggested that severe obesity may double the risk of developing prostate cancer. Finally, in economic terms, healthcare costs are estimated to be 69 percent higher for severely obese men than for healthy-weight males.

Life expectancy may be as much as 20 years less for severely obese men compared to that of males at a healthy weight.

Severe obesity also substantially increases the risk of premature mortality in men. The risk of mortality for severely obese men is approximately 12 times higher than healthy weight males of the same. In addition, life expectancy for severely obese males may as much as 20 years less as compared to a reduction of five years for morbidly obese females. Although data on racial/ethnic differences are sparse, research indicates that there may be a difference between populations in ideal BMI ranges for optimal life expectancy. The ideal BMI range is estimated to be 25–30 for African Americans, about 23–25 for Caucasians, and much lower for Asian Americans.

The prevalence of severe obesity has increased at a rapid rate over the past 20 years. From 2000 through 2005, the number of individuals classified as morbidly obese (BMI of at least 40) increased by

50 percent. Since the late 1980s, the prevalence of severe obesity has risen over 500 percent as compared to moderate obesity and overweight. About 2 percent of adult males, over two million individuals, are severely obese. Although a modest weight loss of 5 to 10 percent can reduce the risk for and improve obesity-related conditions, commercially available diets and medically supervised weight-loss programs have, for the most part, proven ineffective in treating morbid obesity. Surgical treatment for severe obesity may be a more effective option in that outcomes data demonstrate a reduction or resolution of many comorbid conditions, a reduced risk for mortality, and sustained long-term weight loss for many individuals.

SEE ALSO: Assessment of Obesity and Health Risks; Body Mass Index; Mortality and Obesity.

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Morbid Obesity in Women

ESTIMATES BY THE World Health Organization (WHO) show that in 88 nations, more than half the population is overweight—1.6 billion adults worldwide. Ten million Americans are morbidly obese. The degree of obesity is frequently discussed in terms of body mass index (BMI). BMI is a measurement of how healthy one's weight is in relation to one's height. A BMI of 40 or greater represents morbid obesity. For a woman, this is approximately 80 pounds over her ideal body weight. The height of the average female in the United States is 5' 4" For this same female, she would enter the morbidly obese range at a weight of 234 lb. Recent studies indicate the life expectancy of morbidly obese women to be eight years less than in normal-weight women. The increase in morbid obesity has a direct correlation with increases in cardiovascular disease, Type 2 diabetes, joint problems, and infertility. Once obesity has progressed to the morbid range with a BMI greater than 40, the weight is at a point to prevent normal function or to predispose one to developing additional pathologic conditions. As the BMI climbs higher, the mortality risk increases.

Many factors influence weight. It is not as simple as a lack of willpower. Simply stated, the cause of obesity is greater energy (calories) taken in than energy (calories) out. There are a multitude of influences as to why we may take more energy in than we expend energy out. Excess fat storage results from an abnormal conversion of calories to fat. Many women have experienced what is frequently termed *yo-yo dieting*: you go on a diet, lose weight, go off the diet, and regain the weight plus additional weight. When the body is placed into starvation mode during a low-calorie diet, it remembers the starvation period when resuming a more normal calorie intake and subsequently institutes an abnormal conversion of calories to fat to store for the next starvation period.

While genetics can predispose to obesity, environmental factors such as a more sedentary society; television; automobiles; low-cost, high-fat, energy-dense foods; larger servings/high snack consumption; and overconsumption of liquid calories certainly increase the likelihood of progression to morbid obesity. Satiety is the sensation of fullness experienced after eating. If this feeling is impaired, then consumption of calories beyond what is necessary may result. Sa-

tiety is influenced by many variables; two commonly discussed are leptin, a hormone secreted by adipose tissue, which acts on the hypothalamus which is responsible for the sensation of satiety, and ghrelin, the "hunger hormone" which is produced in the stomach and upper portion of the small intestine and is responsible for escalating the sensation of hunger. We cannot downplay the influence of convenience and fast foods on our weight. Many inner-city consumers do the bulk of their grocery shopping at convenience stores, which lowers the quality and variety of available foods. Additionally, many medications can influence hunger; antidiabetics, antipsychotics, antidepressants, antiepileptics, steroids, and antihistamines can all influence hunger and, therefore, weight gain.

A profile of the typical morbidly obese woman may include a history of multiple attempts to lose weight via diet and exercise; they are typically on five or more medications with multiple obesity-related health conditions such as diabetes, hypertension, obstructive sleep apnea, arthritis or joint pain, gastroesophageal reflux disease, menstrual irregularity, polycystic ovarian disease or fertility issues, and frequently psychosocial and emotional issues.

Morbid obesity is costly on many levels. It increases the individual's healthcare costs and medication costs more than smoking. Compared to other chronic diseases such as Type 2 diabetes, coronary artery disease, hypertension, arthritis, and breast cancer, the cost of obesity is the highest. However, obesity can be a direct contributing factor to each of these other diseases. The economic costs of morbid obesity reach farther than the individual woman; estimates from the U. S. Department of Health and Human Services (HHS) place the direct and indirect costs at more than $117 billion each year. Additionally, the healthcare costs increase further as the degree of obesity increases. The costs reach beyond dollars and cents. Women with morbid obesity have more lost work days, more restricted activity days, and higher levels of disability than those with a normal weight.

Morbid obesity is a chronic disease and must be treated as such. It may be viewed as a behavioral issue by many who are not well educated on the topic. In general, morbidly obese individuals are considered a distinct group of patients who require more aggressive approaches to weight loss. When you treat the morbid obesity, you treat the related health conditions.

Morbid obesity is a chronic disease but may be viewed as a behavioral issue by many who are not well educated on the topic.

SEE ALSO: Bariatric Surgery in Women; Body Mass Index; Ghrelin; Leptin; Mortality and Obesity.

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Mortality and Obesity

OBESITY HAS BEEN associated with physical disabilities at least since the days of Shakespeare when he penned the line "Falstaff sweats to death, and lards the lean earth." Yet, it was not until the 1950s that life insurance companies began to recognize that obesity was an independent risk factor for death and diminished life expectancy and began to price policies accordingly. Since the 1950s, a multitude of

epidemiologic studies have shown that being underweight, overweight, obese, or morbidly obese causes significant health problems and affects human longevity. These associations are complex, and whenever possible, the effects of age, gender, race, smoking status, and preexisting disease must be factored in to understand of the role of body mass index (BMI) on mortality. Understanding the impact of obesity on health and mortality is of interest for both medical science and public health.

Older studies found little or no association with being overweight and the increased risk of death. However, recent studies have shown that the risk of death from all causes is increased at or above the BMI of 28 (overweight BMI ranges from 26–30). This is true for men and women in all age groups. The risk of death increases as the BMI rises.

However, these studies did not address smoking and chronic disease as independent causes of mortality in obese populations. It was thought that the connection between overweight individuals and death risk was lessened partially due to improved medical management of chronic disease such as coronary heart disease, cardiovascular disease, and Type 2 diabetes mellitus. When these confounding variables are accounted for in the analysis, the higher risk of death for patients with a BMI over 28 still remains. However, it is important to realize that eliminating preexisting disease from a study ignores the fact that some chronic disease is the direct result of the health-diminishing effects caused by increased adiposity.

It is estimated that 280,000 excess deaths in the United States annually can be attributed to obesity and morbid obesity.

Because smoking and preexisting disease are predictive of low BMI and poor survival, selecting out individuals with smoking history and/or preexisting disease at the onset of a long-term study strengthens the association between being overweight and death risk. The risk of death is increased by 20 to 40 percent in the overweight category when compared to similar ideal weight patients when smoking and preexisting diseases are accounted for. When analyzed in this way, excess weight accounts for 18.1 percent of premature deaths in men and 18.7 percent of premature deaths in women. If individuals with any smoking status and with any amount of preexisting or chronic disease are included, the impact of excess weight is seemingly diminished. Analysis in this way shows that excess weight accounts for 7.7 percent of premature deaths in men and 11.7 percent of premature deaths in women. Further clarification of these associations by eliminating confounding variables will help establish more accurate guidelines for BMI that are tailored to the individual and not to humans in general.

It is estimated that 280,000 excess deaths in the United States annually can be attributed to obesity and morbid obesity when compared to cohorts who have normal BMI. The obese category (BMI 30–35) alone is estimated to contribute to nearly 112,000 of those deaths. It is thought that the impact of obesity on mortality may actually have decreased over time with improvements in public health and medical care. Even so, as the prevalence of obesity in the United States continues to increase, so does the concern for the understanding of the association between obesity and mortality.

Obesity is associated with an increased risk of death for individuals of any age, but the relative risk actually lessens as one gets older. For example, obese men with a BMI ranging from 30–31.9 have relative risks of death of 1.62 (30–64 years old), 1.42 (65–74 years old), and 1.16 (75 years and older). For women of the same BMI range, the relative risks of death are 1.51 (30–64 years old), 1.32 (65–74 years old), and 1.25 (75 years and older). The lowest risk of death for any age is generally within the normal range of BMI.

Another way to relate age and obesity to mortality is to follow the effects of elevated BMI at the age of 50. Excess weight at midlife has a strong influence on decreased life expectancy. Individuals who were over-

weight at the age of 50 have a risk of death that is 20 to 40 percent higher than those who are normal weight. Those who were obese or morbidly obese at the age of 50 have two to three times and three to four times the risk of early death, respectively.

Gender also influences the risk of death due excess weight. The BMI ranges associated with the lowest overall risk of mortality are higher for men. The optimal BMI range for men is 23.5–24.9 and 22.0–23.4 for women. Despite this, men tend to have a higher risk of mortality than women for above-normal ranges of BMI regardless of race or ethnicity.

Race and ethnicity play an important role in the outcomes of excess weight on mortality. The associations between obesity and mortality risk are slightly stronger among Hispanic, Asian, Pacific Islander, and Native-American men and women than white or black men and women. This is true whether or not smoking and preexisting disease are eliminated from a study population. Whether these issues are culturally related or genetically determined has yet to be proven.

Mortality seems to be less influenced by elevated BMI in black men (relative risk of death is 1.35) than for white men (relative risk of death is 1.66) for BMI ranges from 32 to 34.9. This fact is true regardless of smoking history or disease status.

With a BMI of 35 or higher, black women have a 20 to 30 percent increased risk of death, while the same BMI group of white women have a 75 to 100 percent increase risk of death. It is thought that this is due to the tendency of black women to have a more peripheral distribution of fat than white women. This distribution has been shown to be less associated with atherogenic risk factors such as increased levels of cholesterol, triglycerides, and degree of peripheral insulin resistance.

Another example of this is seen when comparing Asian and Western populations. Asians generally have higher percentages of body fat for the same BMI levels when compared to Western populations. On average, an Asian's BMI is lower by 1.9–3.2 for the same percentage of body fat. This is usually attributed to Asians having a more slender build and less muscle mass and connective tissue. It is because of these differences that the World Health Organization has recommended that cutoff values for the overweight and obese categories of BMI be lower for Asian populations than for Western populations.

The relationship between excess body fat, early death, and risk of developing comorbidities has been supported by numerous studies. The medical risks of overweight, obese, and morbidly obese persons include cardiovascular disease, coronary heart disease, pulmonary disease, hypertension, Type 2 diabetes, and cancer. The mechanisms for disease that link excess body fat and the risk of death include lipid abnormalities, hormonal alterations, insulin resistance, glucose intolerance, and chronic inflammation.

A high BMI is most predictive of death from cardiovascular disease. This is a great concern when considering that cardiovascular disease and coronary heart disease are leading causes of death within most industrialized countries. This is especially true for men whose risk is nearly three times that of men within the normal category of BMI. Even a BMI as low as 26.5 for men and 25.0 for women puts an individual at increased risk of mortality from cardiovascular causes.

The risk of cancer-related death increases 40 to 80 percent for morbidly obese men and women. Current estimates attribute 14 percent of all U.S. deaths from cancer in men and 20 percent of those in women to elevations in BMI above normal. Eliminating those who have a history of smoking strengthens the relationship between obesity and cancer death in both men and women. There is no relationship between low BMI and increased risk of cancer death, highlighting the fact that obesity is the major cause of numerous cancer deaths in the United States.

In men, positive increases in death rates are associated with BMI for all cancers (relative risk: 1.52 for BMI at least 40), as well as liver cancer (4.52 for at least 35), pancreatic cancer (2.61 for at least 35), stomach cancer (1.94 for at least 35), esophageal cancer (1.91 for at least 30), colorectal cancer (1.84 for at least 35), gallbladder cancer (1.76 for at least 30), multiple myeloma (1.71 for at least 35), kidney cancer (1.70 for at least 35), non-Hodgkin's lymphoma (1.49 for at least 35), prostate cancer (1.34 for at least 35), and all other cancers.

In women, positive increases in death rates are associated with BMI for all cancers (relative risk: 1.88 for BMI at least 40), uterine cancer (6.25 for at least 40), kidney cancer (4.75 for at least 40), cervical cancer (3.20 for at least 35), pancreatic cancer (2.76 for at least 40), esophageal cancer (2.64 for at least 30), gallbladder cancer (2.13 for at least 30), breast cancer (2.12 for at least 40), non-Hodgkin's lymphoma (1.95 for at least 35), liver cancer (1.68 for at least 35), ovarian cancer

(1.51 for at least 35), colorectal cancer (1.46 for at least 40), multiple myeloma (1.44 for at least 35), and all other cancers (1.88 for at least 40).

Being overweight (BMI at least 28) or obese elevates mortality risk and decreases longevity. As BMI increases, this risk can worsen. While all ethnicities are affected, for some such as African-American and Hispanic, the implications of obesity can be worse. In order to combat these trends, it is important that individuals take steps to maintain healthy weights, and treat current obesities and co-morbid conditions that arise.

SEE ALSO: Cytokine; Morbid Obesity in Men; Morbid Obesity in Women; National Cancer Institute.

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Multidisciplinary Bariatric Programs

OBESITY SURGERY HAS proven a remarkable tool in the hands of surgeons skilled at the procedures. No other tool or medication can prevent cancer and result in the resolutions of diabetes, hypertension, sleep apnea, arthritis, and polycystic ovarian syndrome. This list could go on and on. However, in 1991, the National Institutes of Health (NIH) recognized what so many surgeons were also realizing—that surgery alone did not work in everyone and that some patients would need more help than others. To do this, a more systematic approach to bariatric surgery would be needed.

Up until 1991, there was an ad hoc way of approaching the bariatric patient in the United States and around the world. Surgeons from all over the world often approached the workup for bariatric patients very differently. After the surgery, the care of the post-bariatric surgical patient varied from none to an extensive education and dietary program. It was not until the 1991 NIH consensus conference that surgeons, patients, and hospital systems began to demand that both surgeon and patient be part of programs that integrated multiple levels of care. For the NIH consensus conference, this meant a program with an integrated dietary regimen, appropriate exercise instruction, behavior modification, and psychological support.

After 1991, insurers also began to demand that any patient operated on by a surgeon must show that the NIH requirements were possible in their program prior to giving approval for an operation. This resulted in more programs around the country that integrated dietary, psychological support, exercise, and behavior modification.

These requirements worked well for a time. However, many surgeons, insurers, and patients realized that further work must be done to ensure good-quality outcomes both in surgery and after surgery. In 2002, the American Society of Bariatric Surgery formed the Surgical Review Corporation (SRC). This independent nonprofit group made up of surgeons, hospitals, and insurance providers would monitor all bariatric programs that voluntarily participate. Those who demonstrate that they are truly multidisciplinary and have excellent safety profile would be designated a Center of Excellence (COE).

Among the unique requirements that the SRC proposed was that both the surgeon and the hospital (or outpatient surgical center) must submit applications together to form a single Center of Excellence. Thus, tacitly admitting the fact the good outcomes happen not only because the surgeon is good but because the hospital systems are also important.

Currently, the SRC monitors all aspects of participating surgeons programs. When patients are seen in

the hospital or the clinic, they verify that weight-appropriate furniture is available, and that there is adequate space for the handicapped. All people in the hospital and office who come in contact with these patients must show that they have received sensitivity training, that every patient has a psychological evaluation, and that each patient meets the standards for surgery outlined by the NIH. Additionally, complications are tracked in the hospital and weight-loss data are followed in the clinic. There must also be provisions for psychological support using support groups and each support group must be run by trained professionals. Some type of dietary program must exist. There are many more data points, but these highlight some of the most important ones.

Truly, bariatric surgery has come a long way from its origins in the 1950s. No longer can surgeons operate without the benefit of a multidisciplinary team that helps ensure quality outcomes in this difficult patient population.

SEE ALSO: American Society for Bariatric Surgery; National Institutes of Health; Bariatric Surgery in Children; Bariatric Surgery in Women.

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National Association to Advance Fat Acceptance

THE NATIONAL ASSOCIATION to Advance Fat Acceptance (NAAFA), founded in 1969, is a nonprofit human rights organization whose purpose is to improve the quality of life for fat people. NAAFA has five primary purposes, as stated on its Web site: to fight discrimination and work for equal opportunity for fat people; to disseminate information about the consequences of being fat, including the psychological, sociological, legal, medical and physiological aspects; to sponsor research; to encourage obese people to accept themselves and to promote their acceptance in society; and to serve as a forum to address the issues of fat people. In 2007, NAAFA had 10 regional chapters as well as the national office in Oakland, California.

NAAFA promotes its goals through advocacy, education, and support. In the area of advocacy, NAAFA fights size discrimination and represents the interests of fat people in many venues. NAAFA's Fat Activist Task Force conducts letter writing campaigns to combat negative media representation and offensive advertising. NAAFA members have participated in rallies and demonstrations against Southwest Airlines policies regarding fat passengers and at a conference funded by the commercial weight loss industry. NAAFA educates lawmakers about legal protection needed by fat people, and acts as a legal clearinghouse for attorneys involved in size discrimination cases. An annotated list of recent lawsuits concerning fat discrimination is also available from the NAAFA Web site. NAAFA also represents consumers in hearings concerning the commercial weight loss industry and represents the interests of fat people at obesity research congresses, including conferences at the National Institutes of Health.

NAAFA's educational activities focus on reducing prejudice against fat people. NAAFA representatives have appeared on television programs including *60 Minutes*, *Larry King Live*, and the *Oprah Winfrey Show*; have been featured in newspapers including the *New York Times* and *USA Today* and have appeared on many radio programs and in magazines. The NAAFA Hall of Fame, accessible through the NAAFA Web site, honors fat people who made important contributions to society. A number of informational brochures are available from the NAAFA Web site: topics covered include *Airline Tips for Large Passengers, Dispelling Common Myths about Fat People, Guidelines for Therapists Who Treat Fat Clients,* and *Weight Loss: Fact and Fiction.*

In the area of support, NAAFA seeks to raise the self-esteem of fat people and to create a fat subculture. One action toward this goal is the distribution of information and expression of fat-positive attitudes through the NAAFA newsletter, pamphlets, position statements, and conferences. Creation of a fat subculture is facilitated through NAAFA social events and

special interest groups. NAAFA conferences and the annual convention also create an environment where people can explore their issues about fatness, and network with others who have similar experiences.

Special interest groups (SIGs) within NAAFA allow people with common interests to exchange information about and work together on issues of mutual concern, and meet other people with whom to share interests and problems. Many SIGs sponsor discussion groups and put on workshops at regional events and the annual NAAFA convention, and many produce publications and mailings. Current SIGs include the Big Man's SIG (for large men and those attracted to them), the Supersize SIG (for women size 48 and above), the Sleep Apnea SIG, the Weight Loss Surgery Survivors SIG, the Military Issues SIG, the Teen/Youth SIG (for members through age 19), the Young Nation SIG (for member aged 19–29), and the Lesbian Fat Activist Network.

NAAFA has issued a number of policy statements which are available through its Web site. Topics covered include fat admirers (persons who prefer fat people as sexual partners), obesity research, weight loss drugs, the diet industry, weight loss surgery, employment discrimination, physical fitness, adoption, activism, education discrimination, and feederism (intentional weight gain for the purpose of sexual pleasure to oneself or one's partner).

SEE ALSO: Advertising; Body Image; Fat Acceptance; Feminist Perspective and Body Image Disorders; Stereotypes and Obesity.

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National Cancer Institute

THE NATIONAL CANCER Institute (NCI) in the United States is part of the National Institutes of Health (NIH), which is located within the Department of Health and Human Services. NCI was established as an independent research institute in 1937 by the National Cancer Institute Act; NCI became part of NIH in 1944 and its scope was broadened by the National Cancer Act of 1971. NCI coordinates the National Cancer Program, also created in 1971, which conducts, supports, and coordinates research, training, and information dissemination concerning cancer and the care of cancer patients and their families.

NCI budget request for 2007 was for approximately $5.9 billion. Most of that money was allocated for research, with 42 percent for research project grants, 13 percent for intramural research, and 12 percent for research centers and SPOREs (Special Programs of Research Excellence, projects that promote interdisciplinary and translational research). The largest nonresearch-funded area was cancer prevention and control, which received 10 percent of the allocation.

Approximately 60 major academic and research institutions in the United States are designated NCI Cancer Centers or Comprehensive Cancers. Institutions are selected for either designation through a peer review process, receive financial support from the NCI, and must reapply every three or five years. Cancer Centers generally conduct basic, population, and clinical research, and may provide clinical care. Comprehensive Cancer Centers conduct research and provide patient services, and must demonstrate expertise in each of laboratory, clinical, and behavioral and population-based research. Comprehensive Cancer Centers must also conduct activities in outreach and education.

NCI position on obesity and cancer, as stated in the "Obesity and Cancer" fact sheet at the NCI Web site, is that the percentage of overweight and obese children and adults has been steadily increasing in the United States, that obesity is associated with lack of physical activity and consumption of a high-calorie and/or high-fat diet, and that obesity is associated with a number of diseases. Those diseases include colon cancer, breast cancer in postmenopausal women, eudiometrical cancer, kidney cancer, and esophageal cancer; there is also evidence that suggests that obesity may be related to gastric cardia cancer and gallbladder cancer, while evidence relating obesity and prostate cancer, ovarian cancer, and pancreatic cancer is contradictory. The fact sheet also states that observational studies have shown that avoiding weight gain is associated with lowered risk of cancers of the colon,

breast (postmenopausal), endometrium, kidney, and esophagus; that weight loss is associated with reduction in risk of breast cancer; and that physical activity seems to be related to a reduction of risk for colon and breast cancer. Several NCI-funded studies are currently investigating the relationship between obesity and breast, ovarian, endometrial, prostate, colorectal, and esophageal cancers.

SEE ALSO: Breast Cancer; Colon Cancer; Endometrial and Uterine Cancers; National Institutes of Health; Obesity and Cancer; Ovarian Cancer.

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National Center for Health Statistics

THE NATIONAL CENTER for Health Statistics (NCHS) is a Center within the Centers for Disease Control and Prevention (CDC), which is located within the Department of Health and Human Services of the United States federal government. NCHS works with other federal, state, and private agencies to obtain and compile data on a variety of health topics, and is the principal United States federal agency responsible for collecting, analyzing, and disseminating statistical information relevant to the health and well-being of the American public. The ultimate purpose of NCHS efforts is to monitor the health of Americans and support decision making and research. The NCHS budget request in 2007 was $109 million.

NCHS administers the National Vital Statistics Systems, which collects information on births, deaths, and marriages in the United States, and conducts numerous annual, periodic, and longitudinal sample surveys. Among the best-known NCHS surveys are the National Health and Nutritional Examination Survey (NHANES), the National Health Interview Survey (NHIS), the Longitudinal Studies on Aging (LSOAs), and the National Survey of Family Growth (NSFG). NCHS also produces a number of publications and reports, including the *Advance Data* report series which provide timely analyses of data from current surveys; the *National Vital Statistics Reports* series, which provides timely reports of birth, death, marriage, and divorce statistics; and the *Vital Health and Statistics Series* which cover a range of topics from technical descriptions of the survey design and data collection methods of NCHS surveys to data analysis.

Because overweight and obesity have become major foci of public health efforts in the United States, the NCHS has collected, analyzed, and disseminated information on this topic. The NCHS fact sheet on overweight and obesity draws on information from the NHANES, which is considered more accurate than many surveys because it is based on measured (rather than self-reported) data on height and weight. This fact sheet reports that the number of overweight and obese children and adolescents in the United States was stable from 1960 to 1980, but has increased sharply since 1980. The NCHS fact sheet also reports racial/ethnic disparities in overweight among children, with the lowest rates in non-Hispanic whites and the highest in non-Hispanic blacks and Mexican Americans. The NCHS reports that the prevalence of obesity has been increasing among American adults as well, from 15 percent in 1976–80 to 30 percent in 1999–2002. In addition, the NCHS reports that 9.9 million physician office visits in 1999–2002 included a diagnosis of obesity, that in about 23 percent of those visits an anti-obesity drug was prescribe, and that 75 percent of obese patients received either weight reduction or diet counseling.

SEE ALSO: Center for Disease Control; Department of Health and Human Services; Prevalence of Childhood Obesity in the United States; Prevalence of Obesity in U.S. Women.

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National Eating Disorders Association

THE NATIONAL EATING Disorders Association (NEDA) is dedicated to preventing eating disorders, expanding public understanding of eating disorders, and promoting access to quality treatment for those affected. It is the largest not-for-profit organization in the United States devoted to eating disorders and serves individuals and their families affected by anorexia, bulimia, and binge eating disorder as well as individuals with weight and body image issues. NEDA was formed in 2001 through the merger of Eating Disorders Awareness and Prevention (EDAP) and the American Anorexia Bulimia Association (AABA). NEDA headquarters are in Seattle, Washington, and the organization also has an office in New York.

The Media Watchdog Program of NEDA includes over 1,000 volunteers who monitor media, including TV, radio, and magazines, for positive and negative body images presented in advertising. The program includes guidelines, available through the NEDA Web site, for critically evaluating the media and sending reports of positive or negative ads to NEDA headquarters. NEDA staff reviews the submissions and writes letters of praise and/or protest to the advertisers; in the case of letters of protest, NEDA continues corresponding with the advertisers until they change their advertising strategies and messages. NEDA considers positive ads to be those that display a variety of natural body shapes and sizes, attribute positive characteristics to both heavy and thin people, incorporate images of people eating balanced meals, and include women as more than objects of beauty. They consider ads to be negative if they include excessively thin models or models with computer-enhanced features, portray large people negatively, and glamorize people on diets or present people relying on food as a response to stress, frustration, or loneliness.

The Parent and Family Network (PFN) established by NEDA is a free resource offering support to parents and family members of people with eating disorders; PFN also helps affected families connect with each other and with advocacy opportunities. Resources for family members are also available through the NEDA Web page, including information about eating disorders, available treatments, insurance benefits, online information and support services, and links to advocacy opportunities.

The States for Treatment Access and Research (STAR) program of NEDA advocates for improved insurance coverage for the treatment of eating disorders. In addition, NEDA is a founding member of the Eating Disorders Coalition for Research, Policy, and Action, based in Washington, D.C., whose goals include increasing funding for research and education, improving access to treatment for eating disorders, and promoting national awareness of eating disorders as a public health problem.

A list of healthcare providers who treat eating disorders is available through the NEDA Web site and may be searched by state; types of providers listed include physicians, nutritionists, counselors, and inpatient and outpatient facilities. The NEDA Web site also includes lists of support groups for people with eating disorders, and research studies currently recruiting participants. In addition, NEDA maintains a help line.

SEE ALSO: Advertising; Eating Disorders and Gender; Eating Disorders and Obesity; Eating Disorders in School Children.

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National Heart, Lung, and Blood Institute

THE NATIONAL HEART, Lung, and Blood Institute (NHLBI) is one of the National Institutes of Health (NIH), which are located within the United States Department of Health and Human Services. The Mission of the NHLBI is to provide leadership for diseases of the heart, blood, lungs, blood resources and sleep disorders, and since 1997 for the NIH Women's Health

Initiative. The NHLBI supports research, conducts education activities for health professionals and the public, and supports training and career development for health professionals. Five National Education Programs are coordinated by the NHLBI: the National High Blood Pressure Education Program, the National Cholesterol Education Program, the National Asthma Education and Prevention Program, the National Heart Attack Alert Program, and the Obesity Education Initiative.

The NHLBI is one of the members of the NIH Obesity Task Force, along with 24 other components of the NIH, including the National Cancer Institute, the National Institute on Aging, and the National Institute of Mental Health. The NIH *Strategic Plan for Obesity Research*, which is intended to help coordinate obesity research across the NIH and identify new areas of research, is available for download in PDF format from the NHLBI Web site. The Strategic Plan's goals and strategies are organized around four themes: lifestyle modification for obesity treatment and prevention; pharmacologic, surgical, and other medical approaches to obesity treatment and prevention; the link between obesity and associated health conditions; and cross-cutting research focusing on topics such as health disparities, translational research, and education and outreach.

The NHLBI, in cooperation with the National Institute of Diabetes and Digestive and Kidney Diseases, produced the publication *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report*, which may be downloaded in PDF format from the NHLBI Web site. The *Clinical Guidelines* state the NHLBI position that obesity is the second leading cause of preventable death in the United States and is a multifactorial disease that develops from an interaction of genetics and the environment. The Guidelines present the scientific literature concerning obesity found in MEDLINE, 1980–97, as summarized by a 24-member Expert Panel. Results are presented in the following major categories: reduction of disease risk through weight loss, methods of measuring the degree of overweight or obesity, goals for weight loss, methods to achieve weight loss, goals for weight loss maintenance, and weight loss for smokers, older adults, and diverse patient populations. Easy access to the content of the NHLBI Clinical Guidelines is available through *Guidelines on Overweight and Obesity: Electronic Textbook,* which allows browsing through chapter outlines or by links to related topics, as well as free text searching.

Dissection: The NHLBI provides leadership for diseases of the heart, blood, lung, blood resources and sleep disorders.

SEE ALSO: Future of Medical Treatments for Obesity; Government Agencies; National Institutes of Health.

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National Institutes of Health

THE NATIONAL INSTITUTES of Health (NIH) is the primary federal agency for conducting and supporting medical research. The NIH is located within the U.S. Department of Health and Human Services and is composed of 27 Institutes and Centers; some of the best known of these are the National Cancer Institute, the National Heart, Lung, and Blood Institute, the National Institute on Aging, and the National Library of Medicine. Today, the NIH has over 18,000 employees at its main campus in Bethesda, Maryland, and satellite locations, and receives over $28 billion in Congressional appropriations, approximately

80 percent of which is awarded as research grants. The goals of the NIH are to protect and improve the nation's health by fostering innovative research and its applications, developing scientific and human resources, and expanding the knowledge base in medicine and related sciences.

The roots of NIH date back to 1887, when the Laboratory of Hygiene was created as part of the Marine Hospital Service. In 1930, the Laboratory changed its name to the National Institute (singular) of Health and began granting fellowships for basic research. Much of the early focus of the NIH was on infectious diseases, which paralleled the focus of medicine and epidemiology of the time; for instance, NIH workers conducted a landmark study in 1906 in which they identified the milk supply as the source of a typhoid epidemic in Washington, D.C. NIH scientists also demonstrated that rodents were a reservoir of bubonic plague, and that lice spread typhus fever. In the area of nutrition, Joseph Goldberger proved that pellagra was caused by a vitamin deficiency rather than an infectious agent.

The National Cancer Institute, which opened in 1937, was the first institute within the NIH that focused on a disease rather than a field of study such as chemistry, and also represented a broadening of interest to include chronic as well as acute diseases. Major expansion of the NIH took place after World War II, as the budget expanded from $8 million in 1947 to over $1 billion in 1966. A hospital opened on the NIH campus in 1953 to promote collaboration between clinicians and research scientists.

The NIH Obesity Research Task Force includes representative from 25 Divisions, Offices, and Centers within the NIH. The Task Force developed a *Strategic Plan for NIH Obesity Research*, published in August 2004, which was created with input from scientists, leaders of health organizations, and the general public. The *Strategic Plan* includes short-term, intermediate, and long-terms goals for basic, clinical, and population-based research, within the themes of 1) behavioral and environmental approaches to modifying lifestyle; 2) pharmacologic, surgical, and other medical approaches to preventing and treating obesity; and 3) breaking the link between obesity and related diseases such as cancer and heart disease.

Because obesity is a major health concern in the United States, many institutes within the NIH conduct research related to obesity and overweight, and only a few can be named here. The Obesity Education Initiative (OEI), launched by the National Heart, Lung, and Blood Institute (NHLBI) in 1991, has the goals of reducing the prevalence of physical inactivity and encouraging healthy eating habits to reduce the number of overweight Americans, and thereby to reduce morbidity and mortality from coronary heart disease and other conditions such as Type 2 diabetes.

The OEI has two approaches to these goals: the *population approach*, which focuses on reducing the prevalence of overweight and physical activity in the general population through education and intervention programs, and the *high-risk approach* which targets individuals at high risk for, or already experiencing, medical complications associated with overweight. In June 1998, an expert panel issued the *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: Evidence Report*, which is the first federal clinical practice guidelines adopting an evidence-based approach to obesity and overweight issues. The guidelines provide recommendations for weight loss and weight maintenance, provide the scientific basis for those recommendations, and offer practical strategies for implementing them.

The Office of Obesity Research (OOR) within the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) coordinates obesity research within NIDDK; it is led by codirectors from the Division of Digestive Diseases and Nutrition and the Division of Diabetes, Endocrinology, and Metabolic Diseases. The NIDDK Obesity Research Working Group consists of representatives from seven divisions and offices within NIDDK and is responsible for coordinating obesity research within NIDDK and across the NIH, identifying research opportunities and initiatives, identifying and planning workshops and conferences, and preparing reports and inquiries related to obesity.

The Weight Control Information Network (WIN) within the NIDDK provides scientific information on obesity, physical activity, weight control, and nutrition to the news media, health professionals, the general public, and Congress. Many publications written in English and Spanish that explain the health risks of obesity and methods to avoid or treat it are available for viewing through the WIN Web site, and hard copies may also be ordered through the Web site. Basic statistics about obesity are also available through the

Web site, as are links to obesity-related research, NIH obesity activities, and other organizations concerned with obesity.

SEE ALSO: Fitness; Future of Medical Treatments for Obesity; Governmental Agencies; Health Disparities—NIH Strategic Plan.

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National Weight Loss Efforts for Children

CHILDHOOD OBESITY IS a major public health problem and poses important challenges for both health care and school-centered environments. Three modalities currently available for the treatment of overweight in children and adolescents include behavioral approaches, pharmacologic approaches, and surgical approaches. Therefore, national weight loss efforts for children have focused on the establishment of healthy habits in youth that may help prevent many chronic health problems later in life. The subsequent problems are attributable to unhealthy eating, sedentary lifestyle, and overweight. However, food and eating environments seem to contribute to the increasing epidemic of obesity and chronic diseases, beyond the individual factors such as knowledge, skills, and motivation.

For many individuals, there is 'tracking' of metabolic and lifestyle factors from early age to adulthood, with some individuals genetically predisposed. However, for the youth, obesity and dyslipidemia are generators of hypertension, glucose intolerance and complications of atherosclerosis in adulthood. Indeed, childhood obesity seems to substantially increase the risk morbidity regardless of obesity persistince into adulthood. With 97 percent of U.S. children attending school daily, the school environment provides a rich arena for the delivery of standardized health messages. However, the role of parents in the development of healthy eating and physical activity patterns is critical from the earliest stages of life and warrants further attention. Aside from excess kcaloric intake and inactivity, genetics and environment predispose a child to be obese. Accordingly, any program or treatment plan must include the guardians, who may also be overweight or obese. There is a need of ongoing input of experts and leaders from all sectors and fields to promote healthy lifestyles at schools and within the home, while respecting each child's need for safety, security, and respect.

Community-based interventions are built on theory, receive input from community members and may produce potent, sustainable change. The community involvement can mobilize inherent community assets while being able to pinpoint specific needs. Environmental and policy interventions may be among the most effective strategies for creating population-wide improvements in eating. The Centers for Disease Control and Prevention (CDC) includes a Division of Adolescent and School Health which funds collaborations between state education and health agencies to promote coordination of school health programs.

Also, the CDC's Prevention Research Centers (PRCs) engage public health organizations, academic institutions, and communities in partnerships to develop, test, and disseminate programs to improve health outcomes. The American Dietetic Association (ADA), takes the position that pediatric overweight intervention requires a combination of family-based and school-based multi-component programs that include the promotion of physical activity, parent training/modeling, behavioral counseling, and nutrition education.

SEE ALSO: Childhood Obesity Treatment Centers; Federal Initiatives to Prevent Obesity.

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Native Americans

STUDIES OF NATIVE-AMERICAN health in the United States are complicated by several facts. One is that there is wide variation among people who define themselves as Native Americans, including genetic heritage, degree of assimilation to mainstream American culture, geographic residence (both region of the country and urban versus rural), and standard of living (considering factors such as poverty, level of education, and usual occupation). Another is that even when all Native American groups are lumped together, they still constitute a small minority of the U.S. population, limiting the accuracy of any estimates made from survey data. For this reason, in many surveys, data are not released specifically on Native Americans as they are, for instance, for Hispanic Americans. In addition, when data have been collected on members of particular tribes, that information is sometimes not released because the small number of individuals involved raises confidentiality concerns.

American Indian and Alaska Native (AI/AN) is one of the six racial/ethnic categories used in many U.S. federal government surveys. AI/AN includes people whose heritage can be traced back to any of the original peoples of North, Central, or South America, and who maintain tribal affiliation or community attachment. Approximate 2.9 million people, or 0.9 percent of the U.S. population, identified themselves as AI/AN in the 2000 U.S. Census. There are 569 federally recognized tribes, each with their own culture, beliefs, and practices, as well as a number of tribes that are not federally recognized. The largest concentrations of AI/AN individuals are in the West, Southwest and Midwest, and more than half of the AI/AN population does not permanently live on a reservation. In general, AI/AN people have some of the worst health outcomes of any ethnic group in the United States due to many influences including poverty, low levels of education, geographic isolation, cultural barriers, and mistrust of the U.S. government and Western medicine. Several of the leading causes of death for the AI/AN population are related to obesity, including heart disease (first), cancer (second), and diabetes (fourth).

According to the National Heart, Lung, and Blood Institute, using 1987 data, the percentage of AI/AN adults (aged 18 and over) who were overweight (defined as body mass index [BMI] in at least the 85th percentile) and obese (BMI in at least the 95th percentile) was greater for both males and females overall and in all age categories when compared to the U.S. population as a whole, and most of the differences were statistically significant. Of AI/AN males, 33.7 percent were overweight, compared to 24.1 percent of the U.S. population, and 40.3 percent of AI/AN females were overweight, compared to 25.0 percent of females in the general population. Of AI/AN males, 13.8 percent were obese, compared to 9.1 percent of the entire male population, and 16.6 percent of AI/AN females were obese, compared to 8.2 percent of the overall female population. The highest rates of overweight and obesity were in the 45–54 age group; 45.5 percent of AI/AN males in this age group were overweight and 28.2 percent were obese, while 54.0 percent of AI/AN females in this age group were overweight and 18.7 percent were obese.

One explanation offered for the higher rates of obesity among AI/NA people is the "thrifty genotype" theory which suggests that some ethnic groups became particularly efficient over time in storing caloric intake as fat, which increases the probability of survival when food is scarce but can lead to obesity and related diseases such as Type 2 diabetes when food exists in abundance. Food scarcity is theorized to have been prevalent for hundreds or thousands of years in regions where many AI/NA people have historically lived, but which are currently characterized by the availability of large amounts of food, particularly foods high in fat and refined carbohydrates, turning this former evolutionary advantage into a liability. This theory has also been proffered as an explanation for rapidly rising rates of obesity throughout the world, but is not entirely accepted as proven within the scientific community.

The Pima Indians of Arizona and northern Mexico are often offered as a case study in the thrifty genotype hypothesis. Pimas living in the United States have one of the highest rates of diabetes in the world: half of adult Pima Indians in the United States are diabetic,

and 95 percent of those with diabetes are overweight. In contrast, diabetes is rare among Pimas living in Mexico, and that population as a whole is not overweight. Because the two groups of Pimas are genetically similar but differ in lifestyle (including in food intake and exercise), they have been heavily studied for clues to the influence of diet and exercise on obesity and insulin function. Successful interventions to prevent and control Type 2 diabetes among the Pima have included reduced-calorie and reduced-fat intake, increased fiber and fruit and vegetable consumption, and increased physical activity.

SEE ALSO: Ethnic Disparities in the Prevalence of Childhood Obesity; Ethnic Variation in Obesity-Related Health Risks; Pima Indians; Western Diet.

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Neuropeptides

A NEUROPEPTIDE IS a peptide, a molecule formed by sequential linking of amino acids in a genetically defined order. A peptide joined to other peptide units or that has adopted secondary structure is a protein. Neuropeptides are made by neurons (nerve cells in the brain), but the term refers to peptides secreted from the nerve terminal upon stimulation. Over 100 different neuropeptides are known to be released by different populations of neurons in mammalian central and peripheral nervous systems. Many neuropeptides, particularly those made in the brain and in nerves associated with the gastrointestinal tract, have important roles in controls of food intake and energy expenditure, and are therefore relevant to the regulation of body weight and adiposity.

Neurons use chemical signals to communicate information, including neurotransmitters, neuropeptides, and even gases. Neurotransmitters and neuropeptides are distinguishable by chemical content, site of synthesis, and general effects of neural communication.

Whereas neurotransmitters are made in nerve terminals by enzyme actions on simple precursor molecules, and packaged into small vesicles locally, neuropeptides are made in the cell body. They are encoded by sequences of DNA (deoxyribonucleic acid), transcribed to mRNA (messenger-ribonucleic acid), and translated into an amino acid sequence (peptide). Because the neuropeptide is to be secreted, it is initiated by a short sequence of amino acids that permits packaging into large vesicles that are transported from the cell body down to the terminal. Sometimes the mature neuropeptide arises from a pre-propeptide that is cleaved and otherwise processed after translation. Beta-endorphin and alpha-melanocyte-stimulating hormone, both from proopiomelanocortin (POMC), are good examples.

Upon vesicle fusion with the plasma membrane at the nerve terminal, the neurotransmitter or neuropeptide is released into the space, called a synapse, between the terminal and the next cell (neuron, or other type). Neurotransmitters generally interact with receptors that contain ion channels on the postsynaptic membrane, affecting the cell's excitability by depolarizing or hyperpolarizing the plasma membrane. Neuropeptides have more diverse effects, including altering cell excitability, gene expression of other peptides, local blood flow, cell morphology, and other features and processes. Whereas neurotranmitters usually have short-term effects, neuropeptides tend to have prolonged action. Generally, neuropeptides act at receptors that are coupled to G-proteins, and affect the activity of specific enzyme cascades that lead to gene expression and other changes. These "metabotropic" receptors have subtypes that are expressed on select populations of neurons or other cells. Neuropeptides can thus act as specific signals between one population of neurons and another. Neurons often make both a neurotransmitter and one or more neuropeptides, which are released

differentially. Neuropeptides are often associated with specific behaviors. For example, neuropeptide Y (NPY) is a neuropeptide that acts in certain brain regions to increase the organism's food intake.

SEE ALSO: Agouti and Agouti Related Protein; Appetite Control; Appetite Signals; Bombesin; CART Peptides; Central Nervous System; Cholecystokinin; Corticotropin-Releasing Hormone; Ghrelin; Hypothalamus; Melanocortins; Neurotransmitters; NPY (Neuropeptide Y); Oxytocin and Food Intake; Peripheral Nervous System; POMC (Proopiomelanocortin).

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Neurotransmitters

NEUROTRANSMITTERS ARE GENES that promote or inhibit the release and transmission of electrical impulses between a neuron and other cells. These chemicals include around 10 "small-molecule neurotransmitters" (e.g., acetylcholine, dopamine, norepinephrine, serotonin) and over 50 neuropeptides (such as neuropeptide Y [NPY] and agouti-related protein [AgRP]), and are related to the control of a subject's physiological processes. They, therefore, play an important role in normal functioning. As many neurotransmitters are involved in food intake and in energy expenditure, they are also implicated in eating disorders and obesity.

Indeed, humans have an excellent system which allows them to conserve energy in time of plenty, storing it in the form of body fat, but very few mechanisms aimed at decreasing unnecessary food intake. In fact, as humans are more dependent than most animals on social cues and timetables, sometimes they eat even if they are not hungry. However, as a general rule, food intake is related to the perception of hunger and satiety, which originate in the brain through processing signals which can be activated by dietary breakdown, or produced by distension of the stomach and intestines. This process is regulated by interactions between neuronal networks and neurotransmitters.

Neurotransmitters are produced in the cell body of the neuron and then transported to the end of a presynaptic axon or synthesized directly within the axon terminal. When nerve impulses reach this point, the small vesicles that contain the molecules of neurotransmitters fuse with the membrane of the axon terminal, so that the molecules of neurotransmitters are released into the synaptic cleft. Here, they can bind to specific receptors and start their excitatory/inhibitory action. In fact, some neurotransmitters (e.g., glutamic acid) promote the transmission of electrical impulses, while others (e.g., gamma aminobutyric acid) discourage it.

Various mechanisms have the task of stopping this stimulating/inhibitory action. The neurotransmitter can be removed from the synaptic cleft, its structure can be changed by specific enzymes, or the whole neurotransmitter molecule can be re-taken up into the axon terminal which released it. Furthermore, the effectiveness of the neurotransmitter action can be hindered by vesicles which are too small, and/or by their too rapid closure.

Many neurotransmitters are involved in the process of food intake. Some of them (e.g., dopamine, norepinephrine, and serotonin) suppress the appetite, while others (e.g., neuropeptide Y) stimulate it and promote overeating. Overeating, which may lead to obesity, could thus be caused by a lack of anorexigenic (food intake reducing) neurotransmitters, or a failure in the reception mechanism of their signals.

As neurotransmitters can easily be modulated for treatment purposes, scientists are developing antiobesity medications whose goals are to increase the level of anorexigenic neurotransmitters in the body (enhancing their production and preventing their destruction or reabsorption by the nerve endings after they have performed their function) and optimize the reception mechanisms of their messages.

SEE ALSO: Dopamine; Food Intake Patterns; Leptin; Neuropeptides; Norepinephrine.

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New Candidate Obesity Genes

MODERN MOLECULAR GENETICS is a fast-growing tool to understand different diseases and clinical conditions. Understanding genetic basis of obesity is yet to be achieved, although this field has been increasing tremendously in recent years. Several single-gene mutations have been linked to obesity in animal models but not in humans because it is considerably more complicated. It is expected that obesity arise from the interactions of multiple genes which makes the search for obesity genes a real challenge. Anonymous major genes accounting for as much as 40 percent of the variation in body weight and the same percentage of the variation in fat mass have been reported in addition to major genes influencing adipose tissue distribution.

Identifying the genetic determinants influencing obesity development needs pulling resources and knowledge from different laboratories around the world comprising both academic and industrial partners. It is important to know that the association of a gene with complex traits indicates a possible causative gene, but it should be clear that no single SNP (single nucleotide polymorphism) could be linked to obesity alone but a combination of different genes exposes an individual to develop obesity. It is also clear from several studies that certain SNPs may work as a protecting gene against obesity. Transgenic mice molecular biology technique, quantitative traits loci (QTL), gene mapping, and chromosomal scanning has been widely used to look for obesity candidate genes. Gene linkage analysis can be performed with candidate gene markers or with a variety of other polymorphic markers, such as microsatellites. Evidence for linkage becomes more apparent as the marker loci get closer to the true locus that cosegregates with the phenotype. The bulk of the ongoing research focuses on the molecular mechanisms of appetite and satiety regulation, energy metabolism, nutrient partitioning, and adipose cell differentiation and enlargement.

As of early 2007, several obesity candidate gene have been reported in the form of single-gene mutation and many expressed genes resulted in a phenotype that affect body weight and adiposity. The numbers of QTLs human obesity-related phenotypes are increasing which widen the genomic regions exploration. Recently, there have been many studies reporting associations between DNA sequence variations and obesity phenotypes.

Different international laboratories carry out large-scale genomic studies with an aim to look for geneto-environmental determinants susceptibility to obesity development. The new approach described by Herbert et al. will provide a template for future association studies. Using a multistage design, without sacrificing genome-wide significance, the authors selected the top 10 SNPs for further analysis, and only one SNP variant near the INSIG2 gene was associated with obesity.

The human obesity gene map 2005 update presents the latest 12th update of the human obesity gene map, which incorporates published results up to the end of the year 2005. In that report, 176 human obesity cases due to single-gene mutations in 11 different genes and 50 loci related to Mendelian syndromes relevant to human obesity have been mapped. The number of QTLs reported from animal models currently reaches 408. The number of human obesity QTLs derived from genome scans continues to grow, and we now have 253 QTLs for obesity-related phenotypes from 61 genome-wide scans. A total of 52 genomic regions harbor QTLs supported by two or more studies. The number of studies reporting associations between DNA sequence variation in specific genes and obesity phenotypes has also increased considerably, with 426 findings of positive associations with 127 candidate genes. The electronic version of the map with links for useful publications and relevant sites can be found at http://obesity gene.pbrc.edu.

The most recent important findings in obesity genetic were exposed by a genome-wide search for Type 2 diabetes-susceptibility genes identified a common variant in the *FTO* (fat mass and obesity associated) gene that predisposes to diabetes through an effect on body mass index (BMI). Half of White Europeans have one defective

copy of the gene *FTO* that is located on chromosome 16, which carries a 30-percent increase of obesity, and 16 percent hold two altered copies which carries a 70-percent increased obesity risk per studies published in 2007 from British and Finnish research groups. Further studies on this gene are needed in other ethnic groups.

Although research into *FTO's* role is in its early stages, understanding how variation in the FTO gene region is associated with adiposity may provide insight into novel pathways involved in the control of adiposity.

SEE ALSO: Animal Models of Obesity; Db/Db Mouse; Genetic Influences on Eating Disorders; Genetic Taste Factors; Genetics; Genomics; Monogenic Effects that Result in Obesity; Ob/Ob mouse; Quantitative Trait Locus Mapping; SNP Technologies; Thrifty Gene Hypothesis; Tubby Candidate Gene.

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New Drug Targets that Prevent Fat Absorption

NEW DRUG TARGETS that prevent fat absorption primarily act by inhibiting lipases in the stomach and small intestine which break down fat into single monoglyceride units which can subsequently be absorbed into the intestinal cells and then be transported to other parts of the body for storage. Its implications in the treatment of obesity have shown that it can function to induce weight loss in individuals in combination with other diet and lifestyle interventions, as well as improvements in other health parameters of the obese state.

Dietary fat is ingested in the form of triglycerides, which are three fatty acid molecules that are attached to a glycerol carbohydrate backbone. Upon ingestion, these triglycerides are normally broken down by lipases, or enzymes, which break the triglyceride into two fatty acid chains and the glycerol backbone still attached to one fatty acid chain in the upper portion of the small intestine (duodenum) as well as in the stomach itself. The signal that fat is in the gastrointestinal tract also causes the enzyme cholecycstokynin to excrete bile acid from the gall bladder. The broken-down fat emulsifies with the bile acid, thereby protecting the polar nature of the fatty acid chains from coming in contact with the aqueous water environment of the digestive tract.

Bile acids and free fatty acids combine to form the circular molecular known as the micelle, where the polar ends face inward and the nonpolar end of the fatty acid faces the aqueous water environment of the digestive tract. Consequently, they travel to the edge of the intestinal tract where finger-like intestinal villi cells where the micelle breaks down the free fatty acids are actively absorbed into the intestinal villi cells. Here, they are transported through the lymph fluid to the liver. In the liver, the fatty acid chains may be repackaged with the glycerol backbone and subsequently sent to other tissues in the body such as the fat storage tissue adipose, or they may be directly used for energy as their breakdown generates the energy molecule adenosine triphosphate (ATP). ATP subsequently is broken down to release energy that the body uses to function.

The primary drug that has been synthesized to work by inhibiting fat absorption is Orlistat. Other polymers that inhibit fat absorption by inhibiting lipases are still in development and have not been developed into actual pharmaceutical therapies. Orlistat exerts its influence by selectively inhibiting the gastric lipase and lipase from the pancreas. It does so by binding to the active site of lipase enzymes, thus preventing their action in breaking down triglycerides into the fatty acids chains that can be absorbed into the intestinal villi cells. Consequently, the undigested triglycerides are excreted in the feces and are not actively absorbed by the body where they can be stored as fat. Alli is a form of Oristat that is available without a prescription. Alli causes similar side effects as Orlistat (anal leakage, gas, loose stools), but is half the dose. Long-term studies are not yet available on the effectiveness of Alli.

In clinical trials, orlistat-treated patients had significantly higher weight loss at one year and subsequently maintained that weight loss at two years with continuation of the drug. Studies have shown that approximately 30 percent of the ingested fat is lost in the feces by the action of orlistat. The drug as well has been shown to exert itself in a dose-dependent manner, meaning the higher the dose, the greater the fat malabsorption. The U.S. Food and Drug Administration (FDA) approved a prescription version of the drug marketed as Xenical®. Its usage has been recommended as three 120 milligram tablets to be taken along with meals. The highest recommended dosage of orlistat is 400 milligrams daily.

Orlistat also had a beneficial effect on a number of other unhealthy parameters of the obese state. The total serum cholesterol of patients taking orlistat was significantly lower than patients taking a control drug. Furthermore, a significant reduction was found in low-density lipoprotein cholesterol (LDL-C). LDL-C has been implicated as the leading cause of cardio disease, and the subsequent lowering by orlistat might signal its use not only to reduce fat absorption but also in the treatment of high cholesterol levels. Orlistat has also been implicated as an effective lowering agent of blood pressure. Additionally, orlistat studies in diabetic patients have shown improved control of their postmeal glycemic response, characterized as a much higher spike in diabetic patients in blood glucose upon ingestion of food.

Side effects of orlistat include abdominal pain, oily stool, increased defecation, and fecal incontinence as well as flatulence. In addition, it has been suggested that absorption of fat-soluble vitamins such as vitamins A, D, E, and K might be significantly inhibited because they require fat to be actively absorbed into the body. Consequently, decreases in blood coagulation associated with decreased vitamin K absorption have been suggested as possible with orlistat usage.

Alternatively, another approach to reduce absorption of ingested fat has been the development of a synthetic fat to be incorporated into food products. This synthetic fat subsequently cannot be readily broken down by gastric lipases and will simply be excreted intact. Olestra, a sucrose carbohydrate conjugated with six to eight fatty acid chains has the physical properties of fat but subsequently cannot be broken down in the intestine. Adding olestra to a reduced-energy diet has resulted in improved weight loss.

New drug targets induce weight loss in individuals in combination with other diet and lifestyle interventions.

SEE ALSO: Adipocytes; Cholecystokynin; Drugs that Block Fat Cell Formation; Fat Intake; Orlistat (Xenical).

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New Drug Targets to Improve Insulin Sensitivity

INSULIN IS A hormone having multiple functions which include regulating sugar, protein, and fat metabolism. Insulin resistance is defined as a state of having a reduction in biological activity of insulin. Insulin

resistance has multiple causes including obesity and excess caloric intake. The major health implications of insulin resistance include Type 2 diabetes mellitus (T2DM), hypertension, obesity, polycystic ovary syndrome, metabolic syndrome, and cardiac and vascular diseases. While exercise and diet managements improve insulin sensitivity, they are sometime not adequate in helping patients with T2DM controlling their blood sugar. Drugs are sometimes needed in combination with diet and exercise managements. Many drugs work to improve insulin sensitivity and help patients manage a normal blood sugar level. The biguanides and the thiazolidinediones are two classes of drugs that work to improve insulin sensitivity.

Insulin is synthesized by beta cells of the pancreas with its main function in regulating whole-body sugar (glucose) metabolism. Insulin is released into the bloodstream after every meal to lower the spiked blood sugar level that accompanies such meals. It accomplishes this task by stimulating proteins that transport glucose inside the cell where it can be used for energy or storage. Insulin's main targets include the liver, skeletal muscles, and fat tissues; however, there are insulin receptors on virtually all cells in the human body indicating that insulin exerts its effect basically on all cells.

Once inside the cell, sugars are destined for different metabolic fates. One of these fates for sugars is to be broken down via a series of reactions to harness energy (in the form of ATP) for cellular usage. Glucose within the cell could also be converted into glycogen (a storage form of sugar) or transformed into fat to be used in the future. In fat metabolism, insulin inhibits fat breakdown and stimulates fat synthesis. Insulin achieves these tasks by controlling the genes involved in fat synthesis and degradation. In protein metabolism, insulin helps maintain an equilibrium protein turnover rate—a balanced state of having the rate of proteins degradation equal to the rate of protein synthesis. It achieves this task by stabilizing various components in the protein synthetic machinery. Insulin also prevents protein breakdown.

No matter which drugs are used, diet and exercise managements are recommended along with drug treatments for insulin resistance.

In insulin resistance, insulin essentially loses its biological effects, and cells and tissues no longer become sensitive to it. Insulin resistance has multiple causes including obesity and having too much caloric intake. Some of the consequences for insulin resistance include T2DM, hypertension, dyslipidemia, polycystic ovary syndrome, and cardiac and vascular diseases. On a cellular level, insulin resistance causes a decrease in glycogen synthesis as well as sugar transport.

Two families of drugs—targeting to improve insulin sensitivity and helping patients with T2DM managing a normal blood sugar level—include the biguanides and the thiazoglitazones. Many other drug families are available to treat T2DM including the alpha-glucosidase inhibitors, glucagon-like peptide 1s, sulfonylureas, and meglitinides. However, they do not work by directly increasing insulin sensitivity like the biguanides and the thiazoglitazones families mentioned. No matter which drugs are used, diet and exercise managements are recommended along with such drugs treatments for insulin resistance and T2DM.

The biguanides family includes metformin, phenformin, and buformin. Metformin can be utilized by itself along with diet and exercise to improve insulin sensitivity and to lower the high blood sugar associated with T2DM. It is the first line of defense for overweight patients with mild to moderate T2DM demonstrating insulin resistance. Metformin can also be used in combination with other antidiabetic drugs such as sulfonylureas or thiazolidinediones. Furthermore, metformin can be taken in combination with nateglinide (a meglitinides antidiabetic agent) when metformin in combination with exercise and diet management are no longer effective. Phenformin, another biguanides, was used briefly in the United States; however, it was taken off the market because it causes acid buildup in the blood. Buformin, the third biguanides introduced, had limited use.

The thiazolidinediones family, commonly referred to as the "glitazones," includes rosiglitazone, piogli-

tazone, and troglitazone. Patients with T2DM who benefit most from the glitazones are those with the most insulin resistance. Along with diet and exercise management, rosiglitazone can be taken alone as monotherapy or in combination with other antidiabetic drugs including sulfonylureas, metformin, or insulin. For patients who do not respond well to sulfonylureas or metformin separate monotherapies, rosiglitzone is used in combination with metformin and sulfonylureas. The combination therapy of rosiglitazone and metformin serves as a second line of defense for patients who cannot properly control their blood glucose with separate monotherapies. Along with diet and exercise, pioglitazone (a glitazone) can be taken as monotherapy or in combination with other antidiabetic agents including glimepiride (a sufonylureas), metformin, or insulin. Troglitazone, another member of the glitazone family, was taken off the market because it has toxic effects on the liver.

SEE ALSO: American Diabetes Association; Hypertension; Insulin; Metabolic Rate; Metformin; NIDDK; Ovarian Cyst; Type 2 Diabetes.

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New Drug Targets to Increase Metabolic Rate

IN GENERAL, DRUG targets for the treatment of obesity suppress appetite, prevent absorption of food in the gut, or increase energy expenditure. Drugs that reduce appetite or inhibit absorption ultimately reduce caloric intake, which can lead to a loss of both fat and lean tissue in the body and a slowing of metabolic processes. On the other hand, metabolic drug targets have the potential to function like exercise; the body uses fuel instead of storing it, and weight lost is primarily fat. Drugs targeting metabolism may aid in preventing weight regain that is typically seen after long-term caloric restriction. Furthermore, many drugs that increase energy expenditure also improve the body's response to insulin and are used to treat diabetes. Drugs that alter metabolism fall into the following categories: (1) drugs that enhance use of fuel, or (2) drugs that prevent storage of fat.

Typically, new drug targets for enhancing metabolism are found using knockout mouse models. This process involves finding or producing mice with either a lean or obese body type, then determining which genes are responsible for the phenotype, and designing drugs to either enhance or inhibit the relevant proteins. The hope is that the mice closely model human metabolism and that effective treatments in mice will also work in humans.

Drugs that imitate the sympathetic nervous system, which is responsible for the fight-or-flight response, enhance the release of noradrenaline, leading to thermogenesis (the production of heat), and breakdown of fats in addition to acting as an appetite suppressant. However, they have also been known to cause cardiovascular side effects such as elevation of blood pressure and rapid heart rate. Newer drug targets specifically activate β_3-adrenergic receptors, which in rodents does not cause these undesirable side effects and stimulates thermogenesis via uncoupling proteins.

Uncoupling proteins are of interest because they produce heat instead of energy. While some forms of these proteins such as UCP-1 and 2 have been effective in mouse models, they are less effective in humans. UCP-1 disappears in humans after birth, while UCP-2 appears to improve insulin sensitivity. UCP-3, however, is of interest as a new target because of its prevalence in skeletal muscle.

Mitochondria are the "power house" of the cell organelles because they both produce ATP, the body's form of energy, and are involved in the oxidation or breakdown of fats. One specific protein of interest is called PGC-1α and works with transcription factors to increase the consumption of oxygen, as well as

increase the production of mitochondria. Although PGC-1α itself does not have natural binders, it activates estrogen-related receptor α which is a potential target for drug ligands. PPARδ, on the other hand, is a protein that increases the breakdown of fats and is another mitochondrial protein of interest.

The leptin pathway is another metabolically linked process that has generated much interest as a target for treatment of obesity. Leptin is a hormone released from adipose tissue that indicates a high energy level in the body, causing decreased food intake and increased energy expenditure. Treatment with leptin is not effective in most obese subjects; however, other molecules and proteins in its signaling pathway, such as neuropeptide Y, melanocortins, axokine, and adiponectin, also appear to enhance energy expenditure, likely via enhancement of the sympathetic nervous system, and are targets of interest for drug therapy.

Growth hormone is a hormone released by the hypothalamus, a center in the brain, and stimulates growth along with increased use of fuel. While the hormone itself is problematic, shortened analogues of the protein have promise as enhancers of use of fuel. Thyroid hormone has long been known to activate metabolism; however, treatment with the hormone itself has been shown to cause increased heart rate and breakdown of protein, but other targets downstream of this hormone may be better options to pursue.

Other metabolic proteins that are potential targets are involved in the production of lipids for storage. For example, ACC2 is a protein that senses high energy states and is involved in transporting fats for storage. Blocking this enzyme will prevent storage and lead to prevention of obesity. Fatty-acid synthases are enzymes that produce chains of fat for storage, while triacylglycerol, the final storage form of fat, is produced by an enzymatic pathway that includes enzymes such as diacylglycerol acyltransferase (DGAT). Drugs that block these not only prevent fat storage, but may also be thermogenic.

There are many issues in the search for antiobesity drugs that target metabolic pathways. Findings in rodent models do not necessarily correlate to human metabolism. Furthermore, molecules that enhance metabolism in one tissue may have side effects in other tissues, or may act centrally to increase food intake. However, there are few currently approved drugs for prevention or reversal of obesity, and metabolic enhancement has many avenues that can be pursued.

SEE ALSO: Adiponectin; Adrenergic Receptors; Leptin; Noradrenergic Drugs; Norepinephrine; NPY (Neuropeptide Y); PPAR (Peroxisome Proliferator-Activated Receptors); Thyroid Gland; Uncoupling Proteins.

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NIDDK

THE NATIONAL INSTITUTE of Diabetes and Digestive and Kidney Diseases (NIDDK) is one of 27 institutes and centers within the National Institutes of Health (NIH) focused on reducing the burden of disease and improving public health. The NIDDK conducts and supports basic, translational, and clinical research in a number of areas including diabetes and other endocrine and metabolic diseases, human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), kidney and urologic diseases, liver and digestive diseases, hematologic diseases, nutritional disorders, weight control, and obesity. In addition to supporting and conducting research, the NIDDK provides research training and mentoring opportunities and disseminates knowledge through outreach and communications programs. The NIDDK serves as the government's lead agency for obesity research.

The NIH Obesity Research Task Force was established in 2003 to coordinate obesity-related research across the institutes. The Task Force is cochaired by

directors from the NIDDK and the National Heart, Lung, and Blood Institute (NHBLI) and includes representatives from the other NIH institutes. The Task Force, with the help of representatives from other NIH institutes, external scientists, and members of the public, developed the Strategic Plan for NIH Obesity Research, released in 2004. This dynamic multidimensional plan for addressing obesity represents a collaborative effort between the NIH, other federal agencies, public and private organizations, and community members. It provides a guide for coordinating clinical and population-based obesity-related research activities across NIH institutes.

The plan's strategic goals are organized into four areas. The first, research toward preventing and treating obesity through lifestyle modification, is intended to identify behavioral and environmental factors contributing to childhood and adult obesity and to test potential intervention strategies. The second, research toward preventing and treating obesity through pharmacologic, surgical, and other medical approaches, is designed to illuminate molecules and biological pathways responsible for regulating fat storage, appetite, and the balance between energy intake and expenditure. The third theme, breaking the link between obesity and associated health conditions, encompasses research to understand biological connections between obesity and health conditions such as cardiovascular disease, diabetes, cancer, and nonalcoholic fatty liver disease. The fourth theme, cross-cutting research topics, includes technology, translational research (translating basic and clinical research results into practical applications), education and outreach efforts, and research on health disparities.

Additionally, as part of the Strategic Plan, the NIDDK supports several clinical research centers with complementary objectives. Clinical Nutrition Research Units (CNRUs) and Obesity/Nutrition Research Centers (ONRCs) located at university-based centers throughout the country, conduct basic, clinical, multidisciplinary, and translational research on obesity, eating disorders, and weight management. Specifically, CNRUs integrate research, educational, and service activities related to human nutrition, health, and disease, and engage in interdisciplinary research.

ONRCs are sponsored by both federal and nonfederal agencies and provide coordinated support for interdisciplinary research through the provision of funding for facilities and staff shared across projects. Specific goals of ONRCs include, among others, creating and strengthening multidisciplinary biomedical research in obesity and nutrition; developing new knowledge about the development, treatment, and prevention of obesity; and understanding and treating disease and disorders related to energy metabolism and imbalance.

The NIDDK also helps fund the Longitudinal Assessment of Bariatric Surgery (LABS), a consortium of six clinical centers conducting epidemiological, clinical, and behavioral research on bariatric surgery as a treatment for severe obesity. It is expected that data from LABS will provide insight into the risks and benefits of bariatric surgery. In addition, the consortium members will help to standardize definitions and data collection instruments to develop evidence-based recommendations for selecting and evaluating patients and providing follow-up care. LABS members will also engage in basic and clinical research to investigate how bariatric surgery affects obesity-related conditions, and to better understand behaviors and psychosocial variables.

In addition to supporting research, NIDDK provides a wide variety of educational materials for researchers, healthcare providers, policy makers, and the public. The Weight-control Information Network (WIN), for example, is an information service established in 1994 by the NIDDK. WIN provides science-based information on obesity, weight control, physical activity, and nutrition via fact sheets, electronic newsletters, and other publications distributed to health professionals, members of the public, the media, and Congress. One example is *WIN Notes*, an electronically distributed newsletter that informs healthcare professionals about obesity-related activities, including those of the NIDDK-sponsored Clinical Obesity Research Panel (CORP). CORP is an advisory group comprised of leading external obesity researchers and clinicians who provide input to the NIH and to WIN.

Statistics on overweight and obesity, including definitions, prevalence, and economic costs, are also available through WIN. Furthermore, the NIDDK has developed national initiatives as a part of WIN, such as Sisters Together: Move More, Eat Better, which encourages African-American women to maintain a healthy weight via diet and exercise.

Another important NIDDK initiative developed to address overweight and obesity is We Can! Ways to Enhance Children's Activity and Nutrition. We

Can! is a national education program for preventing overweight and obesity in youth aged 8–13 by focusing on improved food choices, increased physical activity, and reduced screen time (the amount of time spent watching TV, playing video games, and sitting at the computer). The program is designed to help parents and caregivers, as the primary influencers of this age group, to understand the risks of obesity-related health conditions, to choose healthy foods and limit calories from fat and sugar, and to encourage at least 60 minutes of physical activity per day for children.

The We Can! program offers a variety of resources for the home and community settings including posters, ads, wristbands, parent handbooks, community tool kits, and animated presentations on the importance of preventing overweight and obesity. We Can! is supported collaboratively by the NIDDK, NHLBI, National Cancer Institute (NCI), and National Institute of Child Health and Human Development (NICHD).

SEE ALSO: Federal Initiatives to Prevent Obesity; Government Policy and Obesity; National Heart, Lung, and Blood Institute; National Institutes of Health.

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Night Eating Syndrome

NIGHT EATING SYNDROME (NES) is conceptualized as a circadian disorder, manifested by a delay in the daily intake of food. Persons with NES must report evening hyperphagia (consuming at least 25 percent of the total daily calories after the evening meal) and/or nocturnal awakenings with ingestions of food (three or more episodes per week). Associated characteristics include morning anorexia (fasting or undereating), insomnia, depressed mood, and life stress.

Most persons with NES feel a compulsion to eat before bed and during nocturnal awakenings to help return them to sleep.

The prevalence of NES increases with adiposity and with psychiatric comorbidity. Prevalence studies have found rates of 1.5 percent in the general population, 9 to 14 percent in obesity treatment clinics, 4 percent in an older type 2 diabetic population, and 12 percent in outpatient psychiatric clinics. The breakdown of NES among males and females and non-Hispanic white and black participants does not differ from the distribution of these demographics in the samples of recent prevalence studies.

ASSESSMENT OF NES

The Night Eating Questionnaire (NEQ) is a 14-item self-report inventory which assesses the pattern and timing of food intake, hunger and cravings for food, and mood and sleep difficulties. A score of 30 or greater has a positive predictive value of 73 to 77 percent. The Night Eating Syndrome History and Inventory (an unpublished structured clinical interview) provides a more thorough assessment of NES; it is available from the authors. In addition, food records noting the time, type, and amount of food consumed throughout the day and night is invaluable in both diagnosing and treating NES.

TREATMENT OF NES

The increased research interest in NES over the past decade has led to some promising therapies. Three studies have found that the serotonin reuptake inhibitor (SSRI) sertraline significantly reduces evening

hyperphagia, nighttime awakenings, and nocturnal ingestions of food, as well as body weight.

Cognitive behavioral therapy has also shown promise for treating NES. In a pilot study, patients have shown a benefit, including a weight loss of six pounds for completers, comparable to that seen with sertraline. The combination of pharmacotherapy and psychotherapy has not been tested but may prove useful in the future.

Investigators have also reported some success with progressive muscle relaxation, topiramate, paroxetine, and light therapy. Further research is necessary to confirm these findings, and to determine whether behavioral weight loss treatment would be effective for reducing weight and NES.

DIFFERENTIAL DIAGNOSIS

NES should be differentiated from binge eating disorder (BED) and sleep-related eating disorder (SRED). The main aspect of NES is the delay in the timing of eating. Most persons with NES feel a compulsion to eat before bed and during nocturnal awakenings to help return them to sleep. However, the amount of food that they eat is usually not as large as a binge; the average nocturnal ingestion is 300 to 400 calories. In contrast, the hallmark feature of BED is the consumption of a large amount of food at one sitting, often 2,000 or more calories. Also, with BED, binges do not generally take place during nighttime awakenings, although they may take place in the evening.

SRED is a parasomnia where sufferers are sleepwalking and eating and have no recollection of their nocturnal eating episodes until they see the evidence the following morning. Persons with NES have awareness and recollection of their nocturnal eating episodes.

SEE ALSO: Anorexia Nervosa; Binge Eating; Bulimia Nervosa; Compulsive Overeating.

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Non-Diet Approaches

NON-DIET APPROACHES TREAT compulsive overeating without restricting types or amounts of food. Proponents say that the poor success rate for weight-loss diets is due to basic flaws in the concept of restrictive dieting: limitation of food causes a feeling of deprivation, which results in bingeing behavior, and the body has physiological mechanisms which keep it within a limited range of weights. As an alternative to repeated dieting and regaining, the non-diet approach, also known as health at every size, advocates stopping dieting behavior; distinguishing between weight loss, fitness, and healthfulness; learning to eat based on internal rather than external cues; making choices about nutrition for health rather than for weight loss; exercising for health rather than for weight loss; accepting one's natural body size; and improving one's health and well-being through means other than weight loss. Preliminary research has shown that non-diet approaches may be effective in improving health on a long-term basis.

HISTORY

A major impetus for the non-diet movement was the realization that diets were ineffective. Albert Stunkard's studies in the late 1950s showed that very few people were able to take off weight and keep it off. The National Institutes of Health, looking at all the major studies done up to that point, reaffirmed those findings in its Technical Assessment Conference in 1992. No known methods of weight loss were found to have a statistically significant success rate when dieters were followed for several years.

Dr. William Bennett and Joel Gurin first propounded the setpoint theory. Bodies have fairly stable setpoints that can be adjusted downward temporarily with restrictive dieting and vigorous exercise, but when the dieting and exercising are discontinued, the large majority of people regain all the weight they lost, and many gain more than they lost. Jane Hirschmann and Carol Munter offered the first non-diet system for ending compulsive overeating. They advised people to end all restrictions on food, to figure out which foods one's body is craving, and to make those foods available at all times. By eliminating the concepts of "good" and "bad" foods and giving oneself permission to eat exactly what is most desired, the forbidden quality and

therefore the irresistible allure of food would be reduced. This would interrupt the cycle of dieting, feeling deprived, giving up, and bingeing, and the person would learn to eat normally.

APPROACHES

Non-diet practitioners—counselors, therapists, and dietitians—provide their clients with alternatives to dieting. Practitioners help clients learn how their bodies process foods and how to make decisions concerning amounts and types of foods based on how different foods make their bodies feel. Two important aspects of the non-diet approach are intuitive eating and mindful eating. Intuitive eating, also called attuned eating, means recognizing hunger and finding the exact food that will satisfy that hunger. People are advised to learn to recognize the external cues they have been following, and to replace them with internal cues.

Examples of external cues include counting calories or grams of fat or carbohydrates; obeying rules set down by diet programs; listening to criticism from family, friends, or strangers; paying attention to food promotion and advertising; or comparing one's body size, shape, or weight with those on television or in magazines. Instead of eating according to such external cues, the non-diet approach advises people to learn to listen to their own internal cues, which might include varying degrees of hunger, appetite, bodily reactions to different foods, or cravings for specific foods.

Intuitive eating also involves distinguishing between "stomach hunger" and "mouth hunger" (also called "emotional eating"). As the shame around food and eating lessens, non-dieters are better able to feel internal hunger cues. If there are deep-seated emotional issues that are causing compulsive eating, more constructive coping mechanisms should be explored.

Mindful eating means learning to be aware of how one's body feels while eating certain foods; learning to enjoy food without guilt; and learning to recognize the effect specific types and amounts of food have on one's body. Those who eat mindfully figure out which foods have negative effects on their health and well-being, and can choose to reduce or eliminate such foods without feelings of deprivation. Learning to eat in a mindful way also helps a person recognize how much food it takes to feel full. For those who have lived on a controlled diet for years, it may take time to learn what fullness feels like, and the learning process may involve occasionally eating until uncomfortably overfull to find the level of satiety that provides the most well-being.

The non-diet approach takes nutritional information into account, but always tempered by attention to individual needs. Non-dieters are advised to increase their consumption of fruits, vegetables, and whole foods, but only to eat those foods that they find delicious and satisfying. Just as calorie restriction can lead to bingeing, eating unpalatable foods because of nutritional pronouncements is considered counterproductive.

Once foods are no longer thought of as good or bad and eating is normalized, former dieters can make rational choices about which foods work best for their bodies. Non-dieters are advised to find health-care practitioners with a weight-neutral approach to health, and to pay attention to risk factors such as blood pressure, blood glucose, cholesterol, and cardiovascular fitness. Deciding not to eat a particular food for reasons of health or well-being creates less of a feeling of deprivation and more of a feeling of self-empowerment and self-care.

In feeding children, Ellyn Satter has offered a modified non-diet approach in which parents decide when and what to eat, and children are given the choice of whether to eat and how much to eat. This approach offers structure but allows the children to determine for themselves how hungry they are.

EXERCISE

The most common reason people start exercise programs is for weight loss, or as part of a weight-loss regime. When the weight loss stops, or the weight is regained, the dieter gives up on the exercise as well. In the non-diet approach, physical activity is promoted not for weight loss, but rather for the sake of health, vitality, and pleasure. Research has clearly shown that exercise provides substantial health benefits even without weight loss. Perhaps most importantly, those who increase their physical activity for health and well-being are more likely to continue to exercise than those whose exercise programs are connected to weight-loss efforts. Since the goal is not thinness, exercisers are able to work towards more attainable goals such as strength, stamina, balance, flexibility, and stress reduction.

SIZE ACCEPTANCE

An essential part of the non-diet method is learning to accept one's body as it is, or teaching clients to do so. The non-diet, health at every size approach may lead to weight loss (because it helps eliminate compulsive overeating), but responsible advocates emphasize that weight loss must not be the goal. Normalized eating will help the body settle on its natural weight as determined by genetic makeup, neither raised nor lowered artificially by dieting. In a culture that idealizes thinness, that weight may be higher than the "desired" weight. Getting rid of judgments and self-criticism is important for quality of life. The health at every size approach advises people to stop thinking that weight loss will solve all their problems and to treat themselves as well as they would treat other people, without judgment or criticism.

QUESTIONING THE RESEARCH

Non-diet advocates question the validity of the claim that weight is a primary cause of poor health. They point out that while the average weight of the population in the United States and around the world has risen consistently, longevity has increased at the same time. A smaller percentage of people die of heart disease each year, and a higher percentage of older people live active lives with fewer infirmities and disabilities.

Health at every size proponents point to flaws in much obesity research. Some scientific research concerning obesity fails to distinguish between correlation and causation. Showing a correlation is insufficient evidence to prove a causal relationship. To prove that a behavior causes obesity, researchers must not only show a strong correlation, but must also consider all possible confounding factors. For instance, if a study finds a correlation between television watching and childhood obesity, the researchers must determine whether the television watching causes the obesity, whether the obesity causes more television watching, or whether an outside factor (such as socioeconomic level, lack of safe outdoor play environments, or fear of harassment by peers in outdoor physical activity) might be causing both.

The same problems are found in research on the effects of obesity. Confusion of correlation and causation has led to the widespread belief that obesity is a major cause of serious health problems. This belief is challenged by proponents of health at every size. For example, while there is a high correlation between body weight and hypertension, studies have failed to prove that the weight causes the hypertension. Other risk factors, such as sedentary lifestyle, high stress levels, and poverty have a much greater correlation with hypertension, and more research separating out and adjusting for those factors needs to be done. The fact that blood pressure can be lowered through exercise without weight loss shows that weight is not the only factor.

Non-diet proponents also criticize research that fails to take dieting history into account, saying that chronic dieters often have more health problems than those who maintain a high but stable weight. Dieting may be a risk factor in itself; weight cycling, or yo-yo dieting, is strongly correlated with increased risk of cardiovascular problems, Type 2 diabetes, eating disorders, and depression.

Although most people assume that heavy people with health problems need to lose weight, research has failed to prove that weight loss per se results in substantial health improvement. This may be true because researchers fail to separate out the effects of weight loss from the effects of improved nutritional and exercise habits, so it is impossible to tell what actually caused the health improvement. It is also very difficult to find control groups of successful dieters who have maintained their weight loss over time.

Careful analysis of studies has shown that moderate levels of overweight are not associated with increased risk of disease and death. Katherine Flegal's landmark study showed that the federal government's definition of the healthiest weight did not coincide with actual statistics of morbidity and mortality. Those with a body mass index (BMI) that defined them as overweight (but not obese) actually lived longer and healthier lives than those whose weight was supposedly ideal. The estimated number of excess or premature deaths due to obesity had to be revised from 400,000 down to 26,000 as a result of this analysis.

NON-DIET RESEARCH

A large portion of the health risks typically attributed to obesity may actually be due to sedentary lifestyles regardless of body size. In studies where weight and exercise were treated as separate factors, researchers at the Cooper Institute kept subjects' weight stable and increased their cardiovascular fitness through exercise. The subjects' blood pressure improved. The researchers also showed that cardiovascular fitness was a better predictor than BMI for overall risk of death.

Other research includes Linda Bacon's two-year comparison of groups using diet and non-diet approaches. The dieting group was given a standard weight-loss diet as well as ongoing support. The non-diet, health at every size, group was taught intuitive eating and self-acceptance. Both dieters and non-dieters showed improvements in health markers such as blood pressure and cholesterol level. The dieters lost more weight initially, but regained it all by the end of the two years, whereas the non-diet group's weight remained stable.

While both groups increased their physical activity, only the non-diet group maintained that increase. The most significant difference was that 92 percent of the non-dieters stayed with their new regime of good nutrition, intuitive eating, exercise for health, and size acceptance. In contrast, the dropout rate for dieters was 41 percent. Weight loss through dieting was not sustainable. The health-centered approach made permanent, positive changes in the lifestyles of those who learned it.

PUBLIC POLICY

Non-diet, health at every size advocates favor public policies that have health rather than weight loss as their goal. Weight-centered policies such as BMI report cards for schoolchildren create embarrassment and shame, and often result in harassment and bullying as well as eating disorders and dangerous weight-loss practices. Such policies also fail to address the health needs of thin or average-size children who may not get enough exercise or eat adequately nutritious food. The "health at every size" approach recommends initiatives that do not mention weight at all. Such programs would help people of all sizes lead healthier lives, whether they lost weight or not.

Exercise facilities and parks, advocates say, should be made safer and more accessible, and exercise programs for health rather than weight loss should be encouraged. A program's success should not be measured in pounds lost, but rather in actual improvements in health or risk factors.

Schools should budget for physical education, and should make gym classes enjoyable, so that children of all sizes and abilities can work toward their own personal best. Instead of a "war on obesity," non-diet, health at every size supporters advocate public policies with titles such as "commitment to health."

CRITICISM

Some critics of the non-diet approach say that allowing any food in any quantity will result in overeating. Others note that the system might not be effective for those whose satiety mechanisms or metabolisms are permanently damaged from years of dieting and regaining. Still others say that nutritional and exercise guidelines must be followed, rather than allowing the body to self-regulate. Proponents of Health at Every Size respond by saying that methods based on rules and restrictions have not worked, and it makes sense to try a different approach.

SEE ALSO: Bariatric Surgery in Children; Bariatric Surgery in Children; Exercise; Lap Band.

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Noradrenergic Drugs

NORADRENERGIC DRUGS ARE those that are intended to influence norepinephrine, which is a chemical neurotransmitter or some other related chemicals. By manipulating noradrenergic neutrons, noradrenergic drugs affect the activity of the brain and the central

nervous system (CNS) and it is possible that this will lead to positive health outcomes. One such outcome is the suppression of appetite for food which leads to feelings of satiety and reduction in intake of food. The consequence, necessarily, is weight loss in the short term or medium term.

Several problems, of course, exist with the use of such drugs as a form of sustainable weight-loss regimen. These include the possible side effects of the extended use of drugs having a significant impact upon the higher functions of the body and the difficulty in sustaining weight loss simply by eating less—this tends to become counterproductive in the long term (after perhaps six months or longer, depending on individual characteristics of the patient) without behavior modification and physical exercise regimen examination. However, owing to the powerful forces giving rise to obesity in the adult populations of so many countries, it is unrealistic to imagine that cures for the condition can occur in large numbers. Nevertheless, palliation (long-term weight reduction to a less unhealthy body state) is a reasonable and achievable outcome in a large number of cases and, consequently, drug use continues to be a useful tool for medical practitioners.

Early manifestations of noradrenergic drugs included amphetamines and related drugs, which exhibited the desired appetite-suppressant qualities but which also produced euphoric effects which led to a potential problem with abuse. The use of narcotics such as amphetamines and, more recently, cocaine as a suppressor of appetite and, hence, means of maintaining a very low weight, persists in the various entertainment industries of the Western world and helps to explain the prevalence of extraordinarily and unhelpfully slender fashion models. There is no clinical need for the use of drugs of this type in combating obesity, but their effectiveness, or perceived effectiveness, has led to the acquisition of a bad reputation for noradrenergic drugs in all their various manifestations.

A second generation of antiobesity drugs included products such as fenfluramine and fluoxetine, which had similar effects as those of the amphetamine family of pharmaceuticals but largely avoided the abuse problems. These drugs influenced the creation and absorption of serotonin in different ways so as to create the appetite suppressant effect, and this has been shown to be effective to the medium term (approximately 16 weeks, although this length varies significantly in the case of individual cases). These drugs tend to work along the interface between depression (or at least stimuli giving rise to symptoms of depression in some people) and food ingestion—food provides a respite from depressive effects in the short term and promoting well-being in various ways—consequently has a tendency to reduce obesity in many patients. However, these drugs are typically not noradrenergic in nature.

The complexity of many drugs and their impact upon the equally complex actions of the human body are such that what was once popular as a means of medication becomes unpopular as new evidence is unearthed but those same drugs are suggested for treating different conditions. At the same time, understanding of the nature and treatment of side effects is continually developing and this has the effect of making once-disregarded treatments viable once more. Research continues with noradrenergic drugs to determine whether the early weight-loss aspects can still be employed or whether different effects can be successfully deployed.

SEE ALSO: New Drug Targets that Prevent Fat Absorption; New Drug Targets to Improve Insulin Sensitivity; New Drug Targets to Increase Metabolic Rate; Pharmacological Therapy for Childhood Obesity.

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Norepinephrine

NOREPINEPHRINE IS A catecholamine which functions to regulate many metabolic processes in the body. Norepinephrine is made from the neurotransmitter dopamine and can be converted into another catecholamine, epinephrine. Dopamine, norepineph-

rine, and epinephrine are considered catecholamines because they are derived from the same compound, catechol. Despite both being part of the catecholamine class, norepinephrine is classified as a neurotransmitter while epinephrine is classified as a hormone. Norepinephrine is made and released in the brain and the kidneys. It is released from these organs when blood pressure is low and during stressful conditions. During exercise, catecholamine levels increase. Norepinephrine and epinephrine are responsible for the "fight or flight" response during stressful conditions.

Norepinephrine has several effects on metabolic pathways in the body. Once it is released into the body, norepinephrine will bind to specialized receptors, known as adrenergic receptors, which are found on most tissues in the body. There are two classes of adrenergic receptors: alpha and beta. When norepinephrine levels are increased, it can activate enzymes which promote the utilization of stored energy. Glycogen and triglycerides are the storage forms for sugar and fat, respectively, and norepinephrine enables the release of these stored nutrients in metabolic processes called glycogenolysis and lipolysis.

Norepinephrine will also stimulate sugar production in the body, a process known as gluconeogenesis. Norepinephrine can also affect the heart by increasing the frequency and the strength of the heart beat. It will also increase blood pressure. As a result of norepinephrine stimulatory actions, it will increase the metabolic rate of a person. Norepinephrine also works on other systems in the body such as pupil dilation, sodium and potassium balance, and thyroid function.

Norepinephrine has the opposite effect of insulin on many metabolic pathways. Insulin promotes the storage of sugar and fat as glycogenesis and lipogenesis, respectively, while norepinephrine promotes the breakdown of sugar and fat. Within the brain, norepinephrine has many affects. Norepinephrine can affect feeding behavior and mood. Low levels of norepinephrine are associated with depression and increased food intake.

At pharmacological doses (very high), norepinephrine will cause the blood vessels to constrict and thus reduce blood flow to many tissues. This would subsequently lead to a decrease of norepinephrine's effects on tissue (i.e., decreased glycogenolysis and lipolysis). Agonists and antagonists of catecholamines are used as pharmaceutical agents. Agonists are used as appetite suppressants, general stimulants, asthmatic medication, and nasal decongestants. Antagonists are used to treat hypertension, coronary artery disease, and complications of hyperthyroid diseases.

SEE ALSO: Adrenergic Receptors; Dopamine; Metabolic Disorders and Childhood Obesity; Neurotransmitters.

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North America

OBESITY HAS LONG been a problem in North America, especially in the United States. Although specific data have only been collected in recent times, of the 10 largest people for whom their "peak" weight is known—most from the 20th century—eight were from the United States, one from Mexico, and the other, Mohamed Naaman, being from Kenya. Of the 10 next largest people, one was from the United Kingdom, with all the others being from the United States.

In 2007, the United States had one of the highest rates of obesity in the world, with its occurrence in the adult population doubling between 1980 and 2002, and tripling in children during the same period. In 2003–04, children (aged 2–19) had very high levels of obesity, with 17.1 percent of them found to be overweight, and for adults (aged 20 and over), 32.3 percent were also found to have been overweight.

In the Native-American population in the pre-Columbian era, there was little obesity. Many of the tribes of North America, because of their active lifestyle, had few people who were even overweight, let alone obese. However, there was clearly a problem in nonnomadic societies, such as the Aztecs who lived in modern-day Mexico. Their general beliefs included a view that obesity was an affliction from which

some gods suffered. For people living in Teotihuacán, the Aztec capital, and elsewhere in their empire, the Aztecs developed an extensive vocabulary relating to overweight in different parts of the body. The term *quechtzotzol* referred to people who were "flabby" and had a double chin; *puchquiyotl* referred to people who were also "flabby" with their fat distributed widely around their body; *cotztzotzol* referred to people who had fat calves; *eltzotzolli* referred to people who had fatty tissue across their chests; and *ititzotzolli* referred to people who had larger stomachs.

It was not long after the European settlers arrived in North America that obesity started to become a problem with some people. This became more noticeable as diet improved and the level of exercise declined. Although there are no instances of obesity among the Pilgrim Fathers, Boston, Lincolnshire, the place from where many of them came, has the highest rate of obesity in Britain, with one in three residents now being regarded as clinically obese.

John Ratcliffe, the captain of the *Discovery* that sailed to Virginia in 1607, and the second president of the colony, is often portrayed as overweight. Soon after U.S. independence, in 1799, John and Mary Darden of Rich Square, North Carolina, had a son, Mills, who became a farmer and then is believed to have established a saloon. In about 1830, he moved to Henderson County, Tennessee, and there, some local villagers claimed that they measured his weight by testing him sitting on a one-horse cart which operated with springs, and seeing how far the springs were depressed. They then put weights on the empty cart until it reached the same level, thus ascertaining Darden's weight. The claim was that he was 7 feet 6 inches tall and weighed 1,020 pounds (463 kilograms), making him 30 percent taller and six times heavier than the average American at the start of the 21st century. He died in Lexington, Tennessee, on January 23, 1857; no photograph of him is known to survive. His wife was said to have weighed 98 pounds and she died in 1837 after bearing him either three or five children.

Johnny Alee, born in 1853, was also from North Carolina, living in Carbon (modern-day Carbonton). When he turned 10, he apparently became a voracious eater, and when he was 15, he could barely stand up, with men hardly able to reach around his thighs. It was said that his main activity was going from his arm chair to the dinner table. Although

Obesity has become much more serious in recent times, and the United States now has the highest rate of obesity out of any of the major countries in the world—the percentage of obese children and adolescents tripled between 1980 and 2002.

his story has not been fully verified, he died in 1887 after having been said to have fallen through the floor of his cabin. His body was then weighed on the scales owned by the local coal company; it was said he weighed 1,132 pounds.

As is common with most severely obese people, one of the largest people in recorded history, Carol Yager (1960–94) also died young. She was only 5 ft. 7 inches tall, and had been overweight since childhood. In 1993 when she weighed 1,189 pounds—at her heaviest, she weighed more than 1,600 pounds—she was admitted to the Hurley Medical Center in Flint County,Michigan, suffering from cellulitis. She was placed on a strict 1,200 calorie diet and succeeded in losing 500 pounds, although much of it was fluid, which she regained soon after her discharge. She has appeared on Jerry Springer's talk show, and later succumbed to incipient kidney failure. Another well known obese individual, Jon Brower Minnoch (1941–83) was from Bambridge Island, Washington State, and being 6 feet 1 inch tall, he was believed to have weighed more than 1,400 pounds. He was eventually treated at the University Hospital, Seattle, and died aged 42.

Other extremely obese people include Roselie Bradford (b. 1944) who is recognized by the *Guinness Book of Records* as weighing up to 1,200 pounds, putting on much of her weight after she married in 1973. Of a similar size, Michael Edleman (1964–92) from Pomona, New York, had reached 154 pounds at age 11, having to leave school when he was 10 because he could not fit into the school desks. His mother, herself 700 pounds, looked after him, and Edelman spent much of his time in bed during most of the day, refused to take part in any commercial dieting programs, and stopped eating, dying from lack of food at age 28.

Although these people, and many others, are clearly suffering from major medical problems, there are many other people from North America who have suffered from obesity to a much lesser extent, but still one which has raised the likelihood of diabetes, kidney problems and other complaints, including depression. Great attention was given to the problem surrounding obesity by the documentary film *Super Size Me* (2004), which was nominated for an Academy Award. Starring Morgan Spurlock, an independent U.S. filmmaker, who spent a month in 2003 living exclusively on food from McDonald's and stopped any form of regular exercise. Eating 5,000 calories a day—the equivalent of 9¼ Big Macs—his health was tracked by medical professionals after having been given a health check by a cardiologist, a gastroenterologist, and a general practitioner. He started with a clean bill of health and over the month, he lost much of his energy, and ended up with serious medical complications. The film was criticized for the fact that Spurlock undertook not to walk more than 5,000 steps a day, with McDonald's (and others) pointing out that anybody eating the quantity he did, without exercising, would also face similar problems. However, Spurlock's film caught the attention of many who detemined that easy access to food with a high fat content may be the major cause of obesity in the United States and in other Western societies.

The rising levels of childhood obesity, as well as obesity in adults, has led to some politicians being keen to introduce measures to encourage exercise and reduce the easy availability of fatty food. Deborah Ortiz, a state senator in California, has been one of those who has actively campaigned on the issue.

In Hawaii, Guam, and other parts of the Pacific, there has been an extensive prevalence of obesity with the Hawaiian King, Kamehameda V (reigned 1863–72) certainly being overweight. Mention should be made of James Buchanan "Diamond Jim" Brady (1856–1917) who became a millionaire from railroad development and was well known as one of the most famous gourmands (or gluttons) of the period. He was noted as eating up to three dozen oysters, six crabs, six lobsters, a sirloin steak and vegetables, as well as cakes and candy in a single meal. One of the U.S. naval commanders at Pearl Harbor often complained about having the crew for some of his ships being sailors who were overweight, even though he himself was obese.

The research on obesity in the United States has led to many attempts to try to reduce it, with the U.S. neurosurgeon Harvey Cushing being involved in diagnosing what became known as Cushing's syndrome, a metabolic disorder which is caused by the overactivity of the adrenal cortex, resulting in obesity. Cushing's work in medicine was celebrated with a 45-cent postage stamp which was issued by the U.S. postal authorities in June 1988 as part of a large series of stamps of "Great Americans."

In addition to medical treatments, there have also been many diets which have been introduced in North

America and elsewhere, with health clinics offering different treatments. Indeed, following the publicity after Michael Edelman, weighing 994 pounds, was evicted from his house, having to leave on a forklift truck, several clinics offered to treat him or get them on their weight-loss programs for publicity. When Walter Hudson (c.1944–91), who weighed 1,197 pounds—even though the industrial scales broke while attempting to confirm his weight—went on his "Bahamian Diet," it was claimed that he lost some 200 pounds, threatening to sue *Newsday* which said that Hudson did not appear to have lost much weight. Robert Coleman Atkins (1930–2003), an American cardiologist, became well known for his Atkins Nutritional Approach to diet, well known as the "Atkins Diet." What was less well known was that Dr. Atkins himself was obese at the end of his life, weighing 255 pounds although his family stated that much of this was owing to fluid retention during his period of hospitalization just before his death.

During the 1920s and the 1930s, the increased use of the motor car, and also the rise in sedentary jobs, led to a consequent rise in obesity. After World War II, with the growing prosperity in the United States and also Canada and Mexico, the increase in the number of office-based jobs and further reliance on the motor car led to a significant decline in the amount of exercise many people were getting, and obesity came to be a major medical problem for the nation.

Charles "Tubby" Curnan, the chauffeur for Eleanor Roosevelt, weighed 300 pounds, and could barely fit into some of the cars he had to drive for her; and New Orleans attorney Dean Andrews, made famous by Oliver Stone's film *JFK* (1991) were only two examples of the rise in obesity levels among people in public life during the 1950s and 1960s. Indeed, John Candy (1950–1994), the actor who played Andrews in the film, has had many similar roles playing obese characters. Candy died from a heart attack during the night of March 4, 1994, after a complete blockage of one of his coronary arteries, his medical condition having been made worse by his obesity.

There were also many other overweight people connected with the film industry including director Alfred Hitchcock (1899–1980), and actors Charles Laughton (1899–1962) and Sydney Greenstreet (1879–1954), who were all born in England and moved to Hollywood, Laughton playing Quasimodo in *The Hunchback of Notre Dame* (1939), and Greenstreet playing many roles of obese men in films starting with *The Maltese Falcon* (1941). Tol Avery (1915–73) played many obese characters in more than 100 screen appearances. A glutton during this period was Edward Abraham "Bozo" Miller, living in San Francisco, who in 1963, was said to have eaten 27 two-pound pullets at a single sitting. In more recent years, several other actors have suffered from obesity including Jerry Messing (b. 1986) and Jerry Nachman (1946–2004), who appeared on a primetime show, while editor-in-chief of MSNBC also suffered from obesity, succumbing to cancer.

Such had become the growing concern over obesity, especially among children, that in 1956 the President Dwight D. Eisenhower founded the President's Council on Youth Fitness which subsequently became the President's Council on Physical Fitness and Sports. The problem with obesity has become much more serious in recent times, and the United States now has the highest rate of obesity out of any of the major countries in the world. The percentage of children and adolescents who are obese has tripled between 1980 and 2002, with the percentage of adults doubling during the same period. In figures made available for 2003 and 2004, of all children and adolescents between the ages of 2 and 19, some 17.1 percent of them are overweight, with the figure for adults (aged 20 years or older) during the same period being 32.2 percent.

Part of the major worry among health professionals is not just that the rise in obesity has contributed to a significant rise in problems over cardiovascular disease, diabetes, and general health problems, but that with childhood obesity becoming more important, the rates are set to rise even more. Shops selling clothing have had to make allowances with sizes increasing markedly since they were introduced. Airlines have also had to make adjustments to their load-bearing calculations.

In Mexico, the increase in desk jobs, other sedentary occupations, and the use of the motor car have resulted in a higher percentage of Mexicans being clinically obese. The famous muralist Diego Rivera (1886–1957) was clearly heavily overweight, but during his lifetime, his size was exceptional. Nowadays, the rise in adult and also childhood obesity in Mexico will result in a serious strain on the health budget of

the country in years to come. Indeed, the Mexican, Manuel Uribe, in 2006, was claimed to be the most obese human in the world, weighing 1,235 pounds.

In Canada, H. B. Sokar-Todd and A. M. Sharma of the Hamilton General Hospital, Hamilton, Ontario, conducted a study to identify research in obesity in Canada using databases of serious medical research. They found that there were only 17 articles published in the 1970s, 136 during the 1980s, 687 during the 1990s, and 346 from 2000 until 2003. Of the total of 1,186 articles, 816 were original works and these included 29 percent that were based on animal experiments, 16 percent on experimentation involving humans, 14 percent being surveys of the population in a given area or of a particular group, 13 percent were obesity-related comorbidities, 11 percent were to do with diagnostic and surgical issues, 2 percent to do with the impact of weight loss, and only 1 percent to do with the healthcare cost of the problem. Of all the research, 34 percent was conducted in Quebec and 33 percent in Ontario. Mention should be made of Fishka Rais (d. 1974), a Canadian actor who was born in South Africa and who died during surgery to treat his obesity.

The increased awareness of obesity in the United States and Canada has led to a number of journals being published in the United States especially about the issue of obesity. *Obesity Research* was published by the North American Association for the Study of Obesity, and is now issued as *Obesity,* produced in Silver Spring, Maryland. Kelly D. Brownell, a U.S. professor and scientist, and an expert on obesity and weight control was appointed Director of the Rudd Center for Food Policy and Obesity at Yale University, being named in 2006 as one of the World's "100 Most Influential People" in *Time* magazine.

There have also been many private diet clinics opened throughout the United States and Canada, with new diets regularly being promoted in the press and on television. The Academy of the Sierras in Fresno, California, is a secondary school which is devoted to the education and treatment of obese students, and is run by Ryan David Craig, the president of Healthy Living Academies. The American Obesity Association, a nonprofit organization, was founded in 1995 by Dr. Richard L. Atkinson and Dr. Judith S. Stern; and the Canadian Obesity Network was founded in March 2006.

Two diets that have gained notoriety both internationally and in the U.S. are the F-plan Diet from British author Audrey Eyton, and the Beverly Hills Medical Diet from Arnold Fox. Mention should also be made of the book *Awaken the Diet Within* by Julia Griggs Harvey (b. 1962) who started researching after experiencing her own battle with obesity. As with all diets, both relied on restrictions on what you could and could not eat, and these and many other diets have made more people aware of the calorie content of particular types of food, resulting in many people either losing weight or not putting on weight. Critical of many of these diets, Paul Campos, a law professor on the faculty of the University of Colorado in Boulder, was the author of *The Obesity Myth* (2004) which was critical of the hysteria surrounding the concern over dieting in the Western world. Even with these dieting plans, and the greater awareness of obesity, with the availability of much healthier processed food, the obesity rates in the United States, Canada, and Mexico has continue to rise.

SEE ALSO: Atkins Diet; North American Association for the Study of Obesity; President's Council on Physical Fitness and Sports; Prevalence of Obesity in U.S. Women.

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North American Association for the Study of Obesity

THE NORTH AMERICAN Association for the Study of Obesity (NAASO), also known as the Obesity Society, promotes research, education, and advocacy to

prevent and treat obesity, to improve the lives of affected individuals, and to better understand obesity. NAASO was founded in 1982 and in 2006 had over 2,000 members, primarily researchers and clinicians engaged in obesity research and treatment. *Obesity* is the official, peer-reviewed journal of NAASO and is published 12 times per year.

NAASO has issued a number of position statements on topics related to obesity, including approval for Orlistat® as an over-the-counter drug, insurance coverage for bariatric surgery, and obesity as a public health problem. The NAASO position on weight bias is presented in the fact sheet *Obesity, Bias and Stigmatization*, issued by the Weight Bias Task Force of NAASO and available from the NAASO Web site. This publication examines the social consequences of being overweight and defines weight stigma, describes where it occurs and its consequences, discusses how children are affected by weight stigma, suggests strategies to reduce weight stigma, and includes a bibliography of relevant articles and a link to further resources on weight stigma. NAASO has also testified before Congress on topics related to obesity; summaries of these presentations are available online.

Goals for continuing medical education (CME) within NAASO include increasing physicians' knowledge about obesity, disseminating the results of scientific research about obesity, providing guidance about the prevention and treatment of obesity, translating research into practical applications, and supporting management of obese patients by healthcare practitioners. *Obesity Online*, which may be accessed through the NAASO Web site, is NAASO's primary vehicle for professional education and includes continuing medical education activities, downloadable slides, virtual meetings, and commentaries on research. Slide collections within *Obesity Online* include references linked to PubMed and links to additional slides on related topics, so users can create and download a customized presentation, and are organized into the categories Principles of Obesity, Office Management of the Obese Patient, Therapies for the Management of Obesity, and The Metabolic Syndrome. Virtual Meetings are presentations from professional meetings on current issues in obesity, including streaming media with synchronized slides and CME activity information.

NAASO sponsors an annual Scientific Meeting; the 2007 meeting was held October 20–24 in New Orleans, Louisiana. The meetings are intended primarily for scientists, physicians, and other healthcare professionals involved in obesity research and treatment or related diseases such as diabetes and hypertension and include oral and poster presentations of current obesity-relate work and an exhibition of programs, products and services relevant to obesity treatment, and research. The NAASO Web site includes a list of meeting held by other organizations relevant to obesity professionals.

The NAASO Web site also includes information about obesity written for the general public. Five fact sheets, each of which summarize information on basic topics and provide a bibliography and links to further information. Besides the above-mentioned fact sheet *Obesity, Bias and Stigmatization*, fact sheets are available for the subjects *What Is Obesity?, Obesity and Diabetes, Obesity and Cancer,* and *Childhood Overweight.* Information, including statistics and graphical presentations, are available on U.S. obesity trends, estimated medical expenditures due to obesity, and comparisons of NIH funding for different research and disease areas.

SEE ALSO: Body Image; Fat Acceptance; Future of Medical Treatments for Obesity; Stereotypes and Obesity.

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NPY (Neuropeptide Y)

NEUROPEPTIDE Y IS the most abundant peptide released from the hypothalamus and it is found not only in the brain, but also in the autonomic nervous system. Thirty-six amino acid residues including a tyrosine at each end make up the structure of NYP. NYP

is known to be one of the most potent stimulators of food intake. Not only does NPY function in feeding behavior, but it also functions in several other physiologic roles such as circadian rhythms, sexual function, anxiety responses, vascular resistance, memory and learning, and epilepsy. There is evidence to suggest that NPY is also involved in the regulation of feeding behavior including food intake and carbohydrate preference as well as metabolic and lipogenic rates. Therefore, NPY may be involved in regulation of body fat and development of obesity.

NPY's role in regulating energy balance is well known. It forms part of the "lipostat" system along with leptin and corticotropin-releasing hormone (CRH). The lipostat system is a system whereby our energy stores generate signals that are compared with targets encoded in the brain, and differences between these drive our food intake levels, activity patterns, and resting and active metabolisms. In relation to feeding, high NPY levels in the cerebrospinal fluid are associated with high food intake and decreased physical activity. Leptin, produced by adipocytes in response to high fat levels is detected by the arcuate nucleus in the hypothalamus. The arcuate nucleus is a collection of neurons (nerve cells) in the hypothalamus of the brain. Increased arcuate nucleus activity acts on the paraventricular nucleus to inhibit the production of NPY at that site, thus reducing feeding behavior. Arcuate nucleus activity also stimulates the release of CRH which further decreases feeding and increases energy expenditure.

NPY operates on the G-protein coupled receptor. This kind of receptor causes metabolic changes in the target cell. It contains seven membrane spanning domains and five subtypes. Four of the five subtypes are functional in humans. Subtype Y1 and Y5 have known roles in the stimulation of feeding and Y2 and Y4 have known roles in inhibiting appetite.

Injection of NPY into cerebral ventricles or directly into the hypothalamus of rats potently stimulates food intake and decreases energy expenditure, while simultaneously inducing lipogenic enzymes in liver and white adipose tissue. Consequently, continuous or repeated central administration of NPY leads readily to obesity.

SEE ALSO: Appetite Control; Appetite Signals; Corticotropin-Releasing Hormone; Leptin.

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Nutrient Reward

THE TERM *nutrient reward* is an attempt to measure the benefit a person gains by eating a particular food product. Unlike a measure of calories, this obviously varies from person to person, making the study harder but also, in many ways, more worthwhile as it will help identify why people continue eating particular products even when they are aware that it may cause them to put on weight.

To study the nutrient reward of particular products, it is very important to research into the eating habits of people. The concept of this is that medical professionals believe that there must be a way of getting a high nutrient reward for the individual, yet at the same time lose weight, or at any rate not put on weight. They also believe that there would be a ready market for manufacturers to make such products. Indeed, some manufacturers have produced products for which the aim is to have a similar nutrient reward as the original product, yet at the same time, the individual consumes far few calories.

Because of the idiosyncrasies in the tastes of individuals, it is often impossible to make generalizations. However, to draw up a regimen by which an individual may lose weight, this is much easier. In consultation with a dietitian, a list is drawn up of products that the individual likes and how much he or she likes them. The dietitian may suggest not eating particular products, or eating them in different quantities; however, many have found it far more effective to use product substitution by which an individual can continue with much the same diet but either lose weight or not put on weight by substituting products that have a similar level of nutrient reward

to that person in terms of flavor or texture, yet have far lower number of calories. Because dietitians have found that the changes needed to be made are often similar between individuals, this information has been passed on to manufacturers, with several major companies using this information in planning their range of products. The most readily available examples are soft drinks such as Diet Coke and Diet Pepsi. However, many companies and brand names now have a range of products including similar ones that are low sodium, low-fat, fat free, or have some health information or message displayed on the product.

SEE ALSO: Nutrition and Nutritionists; Nutrition Education.

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NutriSystem

NUTRISYSTEM, INC. IS a publicly traded company which offers a weight-loss program based on the use of prepackaged meals. The company also offers free individual counseling to clients (i.e., those who purchase NutriSystem foods) by telephone and internet, and sells vitamins and dietary supplements as well. The company was founded in 1972 as Nutri/System, Inc., a chain of commercial weight-loss centers, and has offered a number of different approaches to weight loss over the years. Nutri/System, Inc. filed for bankruptcy in 1993, and closed all its weight-loss centers and reemerged in 1999 as an entirely online weight-loss and weight-management service. NutriSystem is currently a publicly traded company listed on NASDAQ and sells its products online and through the QVC network.

The basis of the NutriSystem program is the use of prepackaged meals purchased from the company. An individual who wishes to join the program places an order through the company Web site or toll-free telephone number, or through the QVC network for a supply of prepackaged meals. Each meal can be selected individually or the dieter can opt for a selection of the most popular meals. Different meal packages are available for men and women, for diabetics, for vegetarians, and for older men and women (the "silver" packages). The sets of prepackaged meals, which include breakfast, lunch, dinner, and snacks, are delivered to the dieter's home, along with a meal planner which includes directions on grocery items to add to the meals (primarily fresh fruits and vegetables and dairy products). Purchase of NutriSystem foods also allows the dieter free access to unlimited nutritional and weight-loss counseling online or by telephone.

NutriSystem claims that its NutriSystem Nourish program, which is the basis of the prepackaged meals, is based on foods that have a low glycemic index. This means they contain primarily carbohydrates which break down slowly in the body and do not cause spikes in insulin levels, as do foods with a high glycemic index. Scientific evidence for the usefulness of the glycemic index in diet or weight-loss programs is equivocal: some studies have supported their benefits, while others have not. According to the company Web site, the NutriSystem meal plan meets the United States Department of Agriculture 2005 Dietary Guidelines, including the following points: lower-than-recommended amount of sodium; less than 5 percent of calories from trans- and saturated fats; inclusion of lean meats and whole and enriched grains; and inclusion of at least 4.5 cups of fruits and vegetables per day.

The NutriSystem program recommends beginning with a 28-day supply of food; other plans are also available, including a two-week supply and a two-week, weekdays-only supply. Costs vary according to the specific package selected, but in May 2007, the discounted cost on the QVC Web site for a 28-day package was just under $300. A *Forbes* magazine article in 2005 found NutriSystem to be the second most expensive of 10 popular diet plans at 108.5 percent of the median cost for food in the United States; only Jenny Craig was more expensive, while programs such as Slim-Fast and the Zone Diet cost considerably less.

NutriSystem has many characteristics in common with other prepackaged meal programs such as Jenny Craig, and meal replacement programs such as Slim-Fast. Such programs have been demonstrated to be

more effective in the short term than diets in which individuals are assigned a total amount of calories to consume but are allowed to select their own food and judge portion sizes. This is probably in part because most people are very poor judges of portion size and obese people in particular tend to underestimate how much food they consume, so that the use of prepackaged meals simply assures that the individual consumes the number of calories intended by the diet.

The provision of prepackaged meals removes the guesswork from food selection and also trains the individual to become accustomed to smaller portions, with the idea that they will continue to eat less when they resume eating conventional food. The use of prepackaged meals is also associated with improved maintenance of weight loss, probably for this reason. In addition, use of prepackaged meals removes many of the social and emotional connotations from food and cooking, which may make it easier for an individual to change his or her dietary habits. Unlike the Web sites of other meal replacement programs such as Medifast, there is no discussion on the NutriSystem Web site of transitioning from a diet based primarily on NutriSystem products to one based primarily on conventional food selected and prepared by the individual.

Pavlou and colleagues conducted an early randomized experiment in which 49 overweight women were assigned to follow a 1,000-calorie/day diet using NutriSystem foods, and were randomly assigned to exercise or no-exercise conditions. After eight weeks, there was no statistically significant difference between the two groups: Those who exercised lost 10.9 percent of initial body weight, while those who did not exercise lost 7.9 percent of initial body weight. Although this study demonstrated substantial weight loss using NutriSystem portion-controlled meals, it is unclear if the same results would be achieved using current NutriSystem meals or if they would be achieved if the subjects were not taking part in a structured weight-loss experiment.

NutriSystem was a pioneer in the use of the internet to promote dieting and weight loss. It differs from programs such as eDiets.com, however, in that NutriSystem makes its money by selling packaged meals; nutritional and dietary counseling is provided free of charge to dieters who buy NutriSystem foods. In contrast, eDiets.com charges a monthly fee, in return for which it provides members with dietary and fitness advice, customized diets and grocery lists, and an e-mail newsletter. However, the efficacy of the internet to promote weight loss has not been thoroughly studied: advice-only self-help sites such as eDiets.com have not been found effective, but behaviorally based diet sites have achieved more success. However, no recent studies have looked specifically at NutriSystem or at the relative contribution to weight loss of the availability of online support versus the use of meal replacements.

SEE ALSO: Low Calorie Diet; Low Fat Diet; Portion Control; Very Low Calorie Diet.

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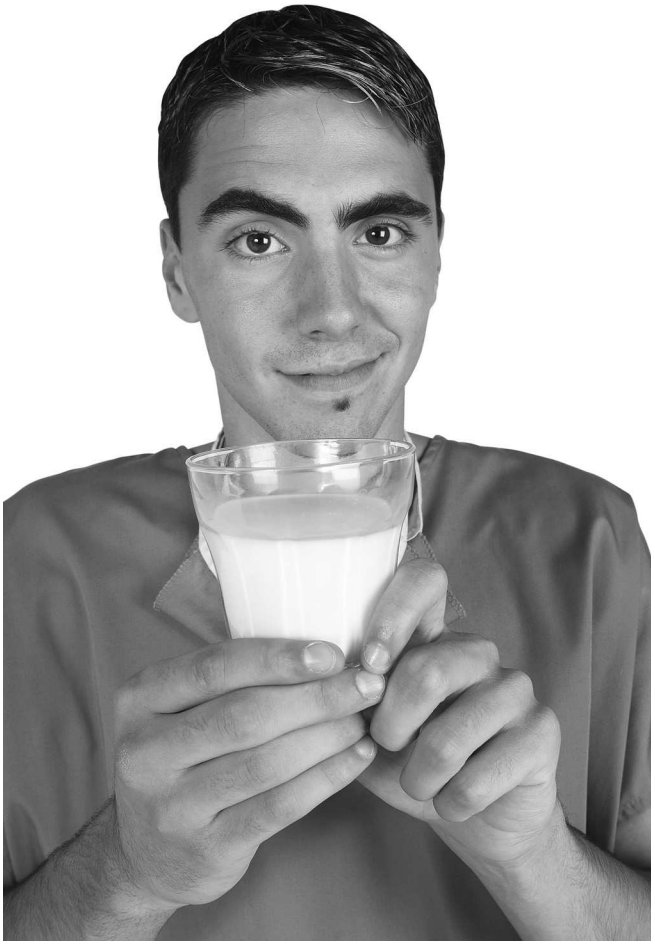
Nutrition and Nutritionists

NUTRITION IS THE science of food, nutrients, and their effect on health. The field of nutrition is dynamic. The breadth of knowledge is perpetually increasing and opinions as to what constitutes ideal nutrition advice are constantly changing. Despite the faddism that is often synonymous with people's perception of nutrition, there are basic tenets that are the foundation of nutrition. Judith Brown, in her book *Nutrition through the Life Cycle*, defines these basic principles as follows:

- Food is a basic human need.
- Growth and health require energy, nutrients, and other substances that are provided by food.
- Nutrition-related health problems begin on a cellular level.
- Both inadequate and excessive levels of nutrient intake can result in malnutrition.

- Humans are capable of managing fluctuations in food intake.
- Inadequate consumption of nutrients, disease states, genetics, and any combination of these factors may result in malnutrition.
- Certain groups of people are at greater risk than others of suffering from poor nutrition.
- Nutrition can affect the development of certain chronic diseases.
- A healthy diet is defined by adequate intake and balance.
- Foods cannot be characterized as "good" nor "bad."



A registered dietitian (RD) has a dietetic-specific degree and has completed a dietetic internship.

The six basic categories of nutrients provided by food are carbohydrates, proteins, lipids, vitamins, minerals, and water. Nutrients are defined as essential or nonessential. Essential nutrients either cannot be synthesize or produced in adequate amounts by the human body and are therefore required in the diet. Nonessential nutrients, while present in food and utilized by the body, do not have to be included in the diet as sources of energy, components of larger molecules, or for physiological functions. The six basic categories of nutrients can be further categorized into macro- and micronutrients.

Carbohydrates, protein, and lipids are considered macronutrients. Macronutrients are larger molecular structures found in both plants and animals that can be ingested, digested, absorbed, and utilized by other organisms for energy and for the synthesis of the consuming organism's own carbohydrates, proteins, and lipids. Micronutrients are organic compounds, specifically vitamins and minerals, that are distinct from macronutrients. Micronutrients are natural components of food and are usually found in trace amounts. They are required for normal physiological functions, and inadequate quantities can cause specific deficiency syndromes.

Predominantly utilized as a source of energy, carbohydrates are essential macronutrients. Per gram, carbohydrates provide 4 calories of energy. Carbohydrates are chemical compounds comprised of sugar molecules, all of which have the basic molecular formula $(CH_2O)_n$ where n = any integer between 3 and 8. Monosaccharides are single-sugar carbohydrates such as glucose, fructose, galactose, and mannose. Of all the naturally occurring monosaccharides, only a fraction can be absorbed by humans. Disaccharides, such as sucrose, maltose, and lactose, are the result of two monosaccharides linked together. Oligosaccharides are carbohydrates that contain 2 to 20 sugar molecules and have low molecular weight. Polysaccharides are also known as complex carbohydrates. They have elaborate structures, higher molecular weight than the other types of carbohydrates, and are either digestible, partially digestible, or indigestible. Starch, glycogen, and fiber are all polysaccharides.

Proteins, also essential macronutrients, are complex chemical compounds whose primary structure is defined by a specific sequence of amino acids. These amino acids serve the primary function of building and maintaining the various tissues of the body. While protein can provide the same amount of energy as carbohydrates, 4 calories per gram, this function is secondary. Humans require 20 amino acids for the synthesis of all proteins. All amino acids have the same

general structure: a carboxyl group (COOH), one carbon atom attached to an oxygen atom by double bond and to a hydroxyl group by a single bond, and a side chain (R group). It is the side chain that is different for each amino acid, dictating its identity and function. With the exception of nine, the human body is able to synthesize the required 20 amino acids. These nine amino acids are known as essential amino acids and are obtained from the diet. Food sources are often described by their dietary protein quality, based on their amino acid profile. Foods with all essential amino acids or greater amounts of the most required amino acid have higher protein quality.

The term *lipid* encompasses fats, oils, and related compounds such as cholesterol. Generally, fats are solid and oils are liquid at room temperature. Structurally, though, dietary fats and oils are the same: one glycerol molecule with three attached fatty acids, a structure known as a triglyceride. Fatty acids are carboxylic acid heads with hydrocarbon chains that vary in length from 8 to 22 carbons, in the number double bonds, and in the location of the first double bond. Fatty acids are often described as saturated, monounsaturated, or polyunsaturated. This description indicates the number of double bonds in the hydrocarbon chain. Saturated fatty acids do not have any double bonds, meaning that all carbons in the hydrocarbon chain are bound on either side to another carbon atom and their other two binding sites are linked to hydrogen atoms. Monounsaturated fatty acids contain one double bond, meaning that two adjacent carbon atoms have each lost a hydrogen atom and share two binding sites instead of one. Polyunsaturated fatty acids contain more than one double bond.

Fatty acids are also described as *cis-* or *trans*-fatty acids. This description only applies to fatty acids that have double bonds. *Cis*-fatty acids have the hydrogen atoms attached to the double-bonded carbon atoms on the same of the double bond, forming a bend in the hydrocarbon chain. *Trans*-fatty acids have one hydrogen atom on opposite sides of the double bond, giving it a structure more similar to saturated fatty acids. The summation of the characteristics of each fatty acid of a triglyceride molecule defines the character of the fat or oil. In general, oils have shorter fatty acid chains or fatty acid chains with more double bonds while fats have longer fatty acid chains or fewer double bonds.

Lipids are essential macronutrients as well. Fats and oils are concentrated sources of energy, providing 9 calories per gram. Because they can be stored and provide the greatest amount of energy per gram of all the macronutrients, fat stores are a survival mechanism, sustaining the body in times of inadequate food supply. Fat deposits also have structural, thermal, and protective functions. Recent evidence indicates that in addition, fat deposits have an endocrine function. On a molecular level, fatty acids are the precursor for cholesterol and steroid hormones, essential components of cellular membranes and specialized lipids, and the vehicle for certain vitamin absorption.

Vitamins are micronutrients, chemical substances that perform specific functions in the body. These functions can be generalized into four categories: membrane stabilizers, hydrogen and electron donors and acceptors, hormones, and coenzymes. There are 13 vitamin groups that are categorized according to their solubility. Fat-soluble vitamins are A (retinol), D (cholecalciferol), E (tocopherol), and K. Water-soluble vitamins are C (ascorbic acid), B_1 (thiamine), B_2 (riboflavin), B_3 (niacin), B_6, folic acid, biotin, pantothenic acid, and B_{12} (cobalamin). Each vitamin group has different isomers and active analogues known as vitamers. The Institute of Medicine, Food and Nutrition Board has determined dietary reference intakes for each vitamin group and these are intake standards for healthy people.

Minerals are also micronutrients. Individually, they are single atoms that exist primarily in an ionic state: calcium, phosphorous, magnesium, iron, zinc, fluoride, iodine, selenium, copper, manganese, chromium, molybdenum, sodium, potassium, and chloride. Due to the charge that they carry, minerals are able to complex with other minerals or with organic compounds. Whether they exist as ions or as part of more complex molecules, the functions of minerals are essential. They are structural components of body tissues; they are involved in growth and development; they have immune function; they regulate enzyme activity, acid-base balance, osmotic pressure, the transport of other molecules, and muscle and nerve irritability. While there is debate whether certain trace and ultra-trace minerals are essential, all of these minerals have a defined function within the human body.

Nutrition has an enormous and ever-expanding impact on health. The field of nutraceuticals has become more recognized as different compounds in foods are

found to have pharmaceutical effects and significant influence on a broad range of health issues. Nutrigenomics is a field that explores the effects of nutrition on gene expression. As the prevalence of obesity increases, though, more attention has been placed on the energy equation, the balance of energy taken in and energy expended. To lose weight, the amount of energy taken in as calories from food must be less than the amount of energy expended as calories used for basic metabolic functions and physical activity. To maintain a healthy weight, the two must be equal.

As stated earlier, inadequate intake of vitamins and minerals can result in specific deficiency syndromes; therefore, food must be considered in terms of both energy and nutrients. This conundrum returns us back to the ninth principle of human nutrition: A healthy diet is defined by adequate intake and balance. With factors such as age, gender, body size, genetics, life stage, and health status affecting the amounts of all macro- and micronutrients required and factors such as culture, socioeconomic status, psychology, and marketing affecting our food choices, the decisions about what we eat become more difficult to make. If we are limited in the number of calories we should take in, we need to make each calorie provide as many nutrients as possible. As food becomes more processed, determining which foods are nutrient dense becomes more convoluted.

To navigate their complicated nutrition needs, many people turn to professionals for guidance. Whose help do you seek—a dietitian's or a nutritionist's? The difference between the two is not obvious and the terms are often used interchangeably. A registered dietitian (RD) has a dietetic-specific degree and has completed a dietetic internship. A dietitian is certified, registered, or licensed according to his or her state's requirements and the profession is regulated. The use of the term *nutritionist* does not mandate any specific training, degree, or licensing nor does the term imply any professional regulation. Essentially, anyone can call himself a nutritionist. Therefore, people seeking clinical advice about how to manage their health through nutrition should seek the advice of a registered dietitian.

SEE ALSO: Nutrition Education; Nutrition Fads.

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Nutrition Education

NUTRITION EDUCATION IS a critical component of health promotion and disease prevention programs to help participants to adopt and maintain healthy eating patterns. Ideally, nutrition education should take place in a variety of venues, including, but not limited to, homes, schools, physician offices, hospitals, community settings, and media.

To change behaviors for the broadest population, effective nutrition education should be culturally appropriate, participatory, and behaviorally-focused. Given the recent surge in obesity, effective strategies for weight management are required in addition to general nutrition messages. Overweight and at-risk-for-overweight individuals should be provided with the behavioral skills necessary to consume a healthful diet and perform adequate physical activity within their environmental context

Promoting weight management behavioral change tends to be a gradual process and focuses on skill building. The Dietary Guidelines for Americans play a critical role in shaping nutrition education messages targeted toward skill-building. Common nutrition education messages targeted to obesity reduction include eating a variety of fruits and vegetables, limiting high-fat foods and energy-dense foods, controlling portion sizes, substituting water and fat-free or low-fat milk for sweetened beverages, engaging in

moderate physical activity most days of the week, and reducing sedentary activity.

SCHOOLS

The federal child nutrition programs (National School Lunch Program, School Breakfast Program, Summer Food Service Program, and Child and Adult Care Food Program) provide nutritious foods and nutrition education, most commonly in school-based settings. Most nutrition education occurs through bulletin boards with nutrition displays or during school lunch week. However, few school meals programs offer nutrient information on food labels, give tours of their facilities, or provide nutrition input to newsletters. Only a minority of school meals programs provide classroom nutrition education.

When nutrition education is offered in the classroom, it is often integrated into core subjects to complement an emphasis on core standards. Most schools focus on increasing students' knowledge about what is meant by good nutrition, rather than influencing students' motivation, attitudes, and eating behaviors. Recently, schools have moved to promote more behaviorally focused messages and to reinforce classroom education with involvement by students' caregivers, changes in school meal programs, and food-related policies, including reducing snack bars, school stores, and vending machines.

MESSAGES TARGETED TO CAREGIVERS

In response to the surge in obesity, many schools have begun to involve caregivers in nutrition education efforts. Messages targeted to caregivers emphasize the need to promote healthful eating behaviors and regular physical activity for children outside of the school setting. Caregivers are provided with information that encourages them to actively participate in their children's behavior changes by increasing the availability and accessibility of healthy foods; limiting the availability and accessibility of sweetened beverages and high-fat, calorie-dense, nutrient-poor foods; controlling portion sizes; supporting and enabling regular family physical activity; and limiting television and recreational screen time. Although controversial, some schools have begun to provide students' body mass index (BMI) assessments to caregivers and encourage them to discuss weight issues with their children's healthcare providers.

COMMUNITY

Communities consist of multiple components, including individuals, families, interest groups, faith-based groups and work sites, and government. According to the Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity, communities must play an important role to solve the obesity epidemic. Although community involvement is still a small component of nutrition education programs, communities have begun to develop obesity prevention and nutrition education programs for children and adults.

PARTICIPATORY EDUCATION

Nutrition education commonly employs a participatory approach to instruction, particularly in the community setting. This method of instruction encourages participants to actively engage in identifying prior positive and negative eating experiences and areas for intervention, to determine the best ways to change their own behaviors. Interactions occur as exchanges between participants and dialogues between educator and participants.

With a participatory approach, the educator acts as a facilitator committed to fostering behavior change rather than an outside expert providing only knowledge-based information. Nutrition concepts are elaborated in terms that are appropriate to the audience and based on what makes sense within the context of the audience's cultures, values, and beliefs using language and experiences that match that of the audience. Educational materials illustrate messages about food consumption and preparation and help participants to identify needs and problems so that they may envisage nutritional well-being and discuss appropriate nutrition decisions and actions.

PEER EDUCATORS

Peer education is an effective behavioral change strategy that is commonly used in nutrition education, especially in community-based efforts. Peer education typically involves using members of a group to promote healthy behaviors among other members of the same group, thereby providing a link between program staff and participants. The main role of peer educators is to help group members define their concerns and seek solutions through the mutual sharing of information and experiences. Peer educators tend to be able to empathize and understand the emotions, thoughts, feelings, and language of the participants

and thus are able to relate to challenges confronted and inspire and encourage their peers to adopt health-seeking behaviors. Peer education is often used to effect change at the individual level by attempting to modify an individual's knowledge, attitudes, beliefs, or behaviors, and at the group or societal level by modifying norms and stimulating collective action that leads to changes in programs and policies.

MEDIA

The media can act as a nutrition education tool to raise consumer awareness about the negative health effects of being overweight and to promote healthy eating and fitness. A range of public health service messages exist, yet the amount of airtime they receive is limited compared to the quantity of commercial advertisements for unhealthy "junk foods" and "fast foods."

A second media education technique beginning to be used is the incorporation of nutrition and physical activity messages into story lines. These methods can help to mitigate the negative effects of excess screen time through the promotion of healthy messaging.

SEE ALSO: Changing Children's Food Habits; Expanded Food and Nutrition Program; Food Stamp Nutrition Education Program; School Based Interventions to Prevent Obesity in Children.

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Nutrition Fads

A NUTRITION FAD is a particular food or supplement which is rendered fashionable for the moment by general public acceptance and practice. A nutrition fad may be a style of cooking, a specific food, or inclusion, exclusion, or combination of foods. A fad is short-lived because it may fall short of its expectations. The perceived benefits of a nutrition fad may be detrimental to one's health or create an economic burden.

There are three factions of food faddism. First, a food or nutrient promises to cure specific diseases or problems. The second is that eating a certain food is an expression of a particular lifestyle. Third, certain foods should be eliminated as their nutrients may be harmful.

There is a massive variety of food which is not only promoted as the latest and greatest, but is widely accessible in stores and via the internet. Along with the abundance of choice is an abundance of half truths and exaggerated claims which become momentarily fashionable. Colorful and often misleading information abounds, yet fads thrive on those seeking a quick and easy fix. Many of these purported claims are scientifically unsubstantiated and may in reality cause or initiate health problems or nutritional deficiencies. Pieces of a scientific study may be cited, but it may

The low-carbohydrate, high-protein Atkins diet is one of the many nutrition fads that may fall short of its expectations in the long-term.

omit pertinent information for marketing purposes. Scientific concepts may be filtered to sell the product. A study may be interpreted or manipulated in a way to appeal to the consumer's hopes and expectations.

The public generally acknowledges the link between health and nutrition, thus allowing a plethora of nutritional information, both good and bad to be disseminated. The desire for more information opens the door to more misinformation and misinterpretation. To determine viable information, one may look to the source by checking the author's credentials, training, and qualification in nutrition. One must ask why is this product being marketed and what is the evidence that it works. The gold standard of research studies is the randomized trials that are large studies performed over time. Viable conclusions may be drawn from more than one of these studies. When the results of these studies are picked up by the media and broadcast to the public the information may be accurate but incomplete. The benefits of the product being researched may require unrealistic amounts to be used to have a positive effect. The media's report may present a biased view to effectively endorse a product.

Sales of dietary supplements reached $13.9 billion in 2004. A supplement is not reviewed by the U.S. Food and Drug Administration (FDA) prior to its marketing. The manufacturer is responsible for its safety and that the claims are truthful and accurate. These claims may be exaggerated and based on small biased studies. The FDA has recourse against a manufacturer if false representation can be proven.

People may cause themselves harm by delaying or avoiding a consult with a qualified health professional. A person may be unaware of the potential toxicity between a prescription drug and a vitamin or supplement. For example, when taking Coumadin®, a prescription blood thinner, it is advised to avoid vitamins E and K, as well as aspirin and gingko biloba. If used together, severe internal bleeding may occur.

Celebrities and public figures are often employed to market a food or supplement. The "Got Milk" campaign exhibits a variety of role models and is a positive statement of a viable nutrient. However, celebrities may endorse a supplement simply to sell a product where they may have no significant education to substantiate the claim. Still others with no training in science and nutrition may write a book based on individual testimonials with little or no scientific evidence. These celebrities endorse and encourage product identification by preying on the emotions of the public that lauds them. This may create a false assumption that because the celebrity endorses it, it must be good.

Good health requires work and discipline. There are no quick fixes. The American Dietetic Association's campaign for National Nutrition Month 2007 is, "The best path to fitness and health is to be 100 percent fad free." If products claims look too good to be true, they most likely are.

SEE ALSO: Diet Myths; Food Labeling; Supplements and Obesity.

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Obese Women and Marriage

THE WESTERN MODEL of beauty values thinness and physical attractiveness frequently at the expense of health. In this thin-obsessed culture, one of the biggest concerns women express about their bodies is the belief they are fat or obese. Obesity is not gender specific, and is an excess in body fat. Assessment of overweight and obesity is based on the body mass index (BMI) calculated by dividing one's weight in kilograms by the square of height in meters. The World Health Organization defines overweight as a BMI of 25 to 30 and obesity as a BMI over 30. Morbid obesity is defined as a BMI of 40 or above or a BMI or 35 and above, in conjunction with comorbid health conditions. The precise percentage of married women with obesity is unknown and a rough estimate is difficult to speculate.

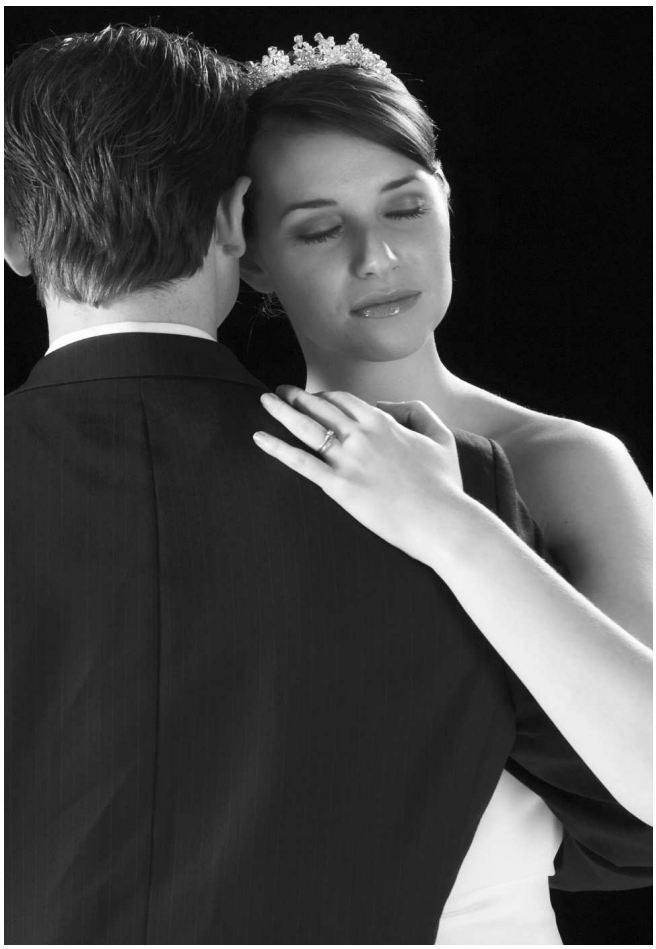
Based on these figures, roughly 25 percent of women in the United States are classified as obese. Some estimates place the prevalence of obesity closer to 34 percent for women in the United States with 50 percent of women being overweight. In women with too much body fat, obesity is associated with many comorbid health-related conditions including, but not limited to, osteoarthritis, breast cancer, endometrial cancer, and cardiovascular disease. Gallbladder disease, urinary stress incontinence, infertility, miscarriage, and menstrual abnormality and disturbance correlate with increased body fat. The incidence of birth defects, specifically neural tube defects, occur more frequently with morbid obesity as well as increased mortality poor quality of life.

Obesity is a complex condition of multiple etiologies. Many obese women face the stigmatization of blame for their weight which promotes poor self- and body image. An overfocus on weight is a risk factor for developing an eating disorder. This may be attributable to the powerful social stigma associated with obesity. There is ample evidence that obese women tend to internalize the pervasive anti fat bias that exists in this society. For many women, poor body image and body dissatisfaction results in dieting. For obese women dieting is a way of life. The term "yo-yo" dieting" relates to the cycle of repeated weight loss followed by repeated weight gain. Some research suggests that the negative effects of "yo-yo" dieting increases the risks of obesity and are more harmful to women's health than being overweight.

The prevalence of obesity rates in individuals having chronic psychiatric illness is documented to range from 26 to 62 percent; however, research does not support the finding that the majority of obese women have a diagnosable psychiatric illness. It is estimated that approximately 30 percent of obese individuals seeking treatment for obesity may have a form of an eating disorder termed *binge-eating disorder* (compulsive eating). Binge eating is prevalent with Caucasian

women but is present in other ethnic groups. According to the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV-TR), binge eating is an episode of uncontrolled eating of large amounts of food rapidly on a regular basis when not hungry. The episode is followed by feelings of disgust and embracement as a result of the behavior.

There are no compensatory behaviors for the extreme caloric intake. These periods of bingeing occur on the average two days per week for a period of six months. Many obese women binge eat without meeting the full criteria of binge-eating disorder which falls under the classification category of eating disorder not otherwise specified (EDNOS) as outlined in the DSV-IV-TR. While not all obese women binge, chronic binging without compensatory behavior leads to weight gain and contributes to obesity.



Fantasy and reality: For many obese women, the prospects for marriage and the selection of partners may be reduced.

Not all women seek treatment to deal with their obesity. Obesity surgery, however, is quickly becoming the treatment of choice for many obese women who have experienced chronic diet failure. The overall research findings suggest bariatric surgery has a favorable impact on personal relationships. Many women experience a marked increase in self-esteem, paralleled by improvements in physical, social, and occupational functioning. Self-confidence, assertion, improvement in social activity and the alleviation of depression and anxiety are experienced following surgery. Longitudinal data indicate that an immediate rise in physical and psychosocial well-being is a common phenomenon following weight-loss surgery. Findings related to obese women suggest that marital satisfaction increases after weight loss if marital satisfaction existed prior to weight-loss surgery. Other findings suggest weight loss following bariatric surgery may lead to an increased rate of separation and divorce.

These findings may relate to the quality of the marital relationship prior to surgery. According to the health-at-every-size paradigm, health status is not based primarily on BMI, but rather being healthy and fit at every size. Some researcher and clinicians subscribe to the supposition that there are benefits to obesity including decreased risks for multiple diseases including cancer, infectious disease, osteoporosis, respiratory disease, and mitral valve prolapse. Empirical data focusing on the risks versus the benefits of obesity are substantial. The relationship between marital status and obesity is complex and not clearly established. For many obese and morbidly obese women, however, the prospects for marriage are decreased and the selection of potential partners may be reduced or limited. Obese women tend to marry later in life and date and marry heavier partners. Some research supports findings that in general, men are less tolerant of their partners being overweight and are less comfortable dating obese women. Other findings suggest that body weight is not associated with marital quality and does not predict marriage or divorce.

Health problems, however, may impact the quality of the marriage and contributed to marital disruption. In the United States, approximately 92 percent of adults will get married. The expectation for many couples is that their union will provide an ongoing opportunity for meeting emotional, physical, economic, and social needs as well as providing an outlet for

sexual fulfillment. Studies focusing on marital status and health report that healthier individuals are more likely to be married. For women in particular, those in quality marriages are 20 percent less likely to die than single women from a variety of illnesses including suicide and cardiovascular disease and experience better mental health. Several studies looking at older cohorts suggest findings that widowed women are more likely to become obese than married women.

Researchers have noted a positive association between weight gain and marriage and while both men and women gain weight, men are more likely to reach obese status based on BMI definitions. This in part may be due to a change in eating behaviors as a couple. Several studies have found a correlation between the couple's dietary behavior and the development of similar eating patterns. This tendency to merge eating patterns can either promote unhealthy behaviors or reinforce health-promoting behaviors as the couple working together. A critical factor to any permanent behavior change, specifically weight loss and a healthy eating habit, is support. Several studies have reported the negative correlation between weight loss and weight-loss maintenance and partner sabotage. Partner support is a documented benefit for obese married women attempting to adapt healthier eating behaviors and lose weight. Finally, it is important for obese women to set realistic weight-loss goals.

SEE ALSO: Eating Disorders and Gender; Ethnic Disparities among Obesity in Women; Women and Dieting.

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Obese Women and Social Stigmatization

ANECDOTAL EXPERIENCE AND empirical research demonstrate that obese individuals face a great deal of stigmatization in a variety of domains. The prevalence of obesity in the United States, with some statistics reaching as high as 65 percent of adult Americans being overweight or obese, makes such stigmatization an important focus of study because of the substantial number of people who face such stigmatization as well as the unfortunate consequences that such stigma brings to affected individuals. Research studies reveal that stigmatization experienced by obese individuals includes negative judgments/evaluations, social rejection, and physical obstacles. Such negative evaluations include descriptions of obese individuals as lazy, dishonest, stupid, and self-indulgent. Numerous studies have revealed that such stigmatization can lead not only to marginalization and low self-esteem, but also to outright discrimination. Furthermore, research has also documented that in many areas of stigmatization, obese women are subjected to even greater stigma than obese men. An overview of the stigma faced by obese men and women will be discussed with a specific emphasis on the prejudice experienced by obese women.

Many studies have replicated findings of stigmatization of obese children and adults, placing obesity among other targets of overt stigmatization including acquired immunodeficiency syndrome (AIDS), criminality, and drug addiction. A landmark study conducted by Rich-

Evidence suggests that obese individuals receive lower wages compared to their nonobese counterparts.

ardson and colleagues in the 1960s shed light on the early onset of obesity stigma in American society. In this study, elementary school-age children were shown pictures of children with various physical disabilities and asked to rank each picture according to how much they liked each one. The majority of children in this study ranked the obese child as "least liked." This finding was replicated in a young adult population in a study conducted by Latner et al. in 2005. In this study, college-age men and women were shown pictures of six adults, four of whom had physical disabilities, one of whom was obese, and one of whom was "healthy"; the obese picture was given the second lowest rank in reference to whom the rater liked the most.

The obesity stigma has also been identified in romantic relationships where it has been shown to specifically affect obese women. Chen and Brown asked participants to rank order preferences for a sexual partner in which their choices included a "healthy individual," a physically disabled individual, an obese individual, an individual with a history of mental illness, and an individual with a history of curable sexually transmitted diseases. The obese individual was found to be the least favorable among both male and female participants. In addition, a gender difference was found with regard to the obesity stigma in that men ranked obese women as considerably less preferable. This provides support for the belief that men rely more on weight when making judgments about sexual attractiveness.

Additional research has found the obesity stigmatization to be particularly salient for obese women. Research conducted on overweight adolescents has found that overweight and obese adolescent girls are more likely than overweight adolescent boys to report being teased by peers and family members. Similar research also reports that obese adolescent boys and girls are less likely to date than healthy weight adolescents and that obese girls are less likely to date than obese boys. Studies have also found that overweight and obese women are less likely to be married than obese men. One study reported that men are more likely to choose a female romantic partner from a newspaper advertisement who is recovering from a drug dependency than one who is obese. Such findings suggest that women are more strictly judged by narrow cultural standards of appearance than men are, highlighting the increased stigma that obese women must face.

Empirical research has demonstrated that the obesity stigma has widespread tangible effects on the lives of obese individuals, especially women. Specifically, Puhl and Brownell report findings of discrimination against obese individuals in the workplace, in medical/health settings, and in educational settings. Stigmatization and discrimination against obese individuals in the workplace appears to be quite pervasive, beginning in the interview and hiring process and enduring throughout the individual's employment, affecting aspects such as promotions and salary increases.

Evidence suggests that obese individuals receive lower wages compared to their nonobese counterparts and that fewer obese individuals are employed in high-level positions and/or receive promotions. Of note, it appears that in the workplace obese women specifically are subjected to a disproportionate amount of discrimination; obese women have been found to be paid less than their nonobese female counterparts and to be more likely than thin women to be employed in low-paying jobs.

Weight stigmatization has also been documented in medical/healthcare settings. Research has found negative perceptions of obese people among medical students, dietitians, nurses, and doctors. Specifically, a recent study conducted by Schwartz et al. found that health professionals who treat and study obesity associated the stereotypes of lazy, stupid, and worthless with obese people. Research also suggests that while healthcare professionals recognize the health implications of obesity, many are ambivalent about and hesitant to specifically discuss and treat obesity. Negative attributions on the part of healthcare professionals are important because of their implications for the medical care that obese individuals receive both in the care offered to the patient and in the effect that such attitudes have on the individual.

Specifically, research suggests that these negative attitudes may have a particularly strong effect on obese women. Research has found that obese women are more likely than other populations to postpone medical care. One study found very overweight women to be much less likely than average weight and moderately overweight women to get annual pelvic examinations. Obesity in women has also been found to be associated with appointment cancellations, and a study conducted by the National Health Interview Survey found that among women, higher body mass index (BMI) was related to less use of preventative healthcare services, including breast examinations and Pap smears. While it is difficult to assess the degree to which health professionals' weight stigma affects the quality of healthcare that obese individuals receive, research suggests that obese individuals, specifically women, avoid healthcare as a result of their weight. Whether this is caused by obese women's personal insecurities regarding their weight, by such individuals' perception of being stigmatized by their healthcare providers, or a combination of these, it appears that obese women are not using healthcare as often as other populations.

Obesity stigmatization has also been identified in school settings, both in the subjective experience of teasing and treatment by teachers and peers and in the more objective domains of college acceptance and academic performance. Research has documented the incidence of elementary school-age children associating negative stereotypes to obese children and adults. Such negative stereotyping persists into adolescence and adulthood, and additional research has found that college students also associate negative stereotypes such as being lazy, self-indulgent, and sexually incompetent to obese individuals.

Research comparing obese and nonobese high school students with comparable application rates and academic performance has found that nonobese students are more likely to be accepted to colleges compared to their obese counterparts. In addition, studies have found that overweight men and women are underrepresented in college student populations and that overweight students receive less financial support from their family than normal-weight students. Consistent with findings of increasing stigmatization against obese women, research findings demonstrate that obese women have lower rates of college acceptance compared to obese men and that obese women receive less parental financial support for college than obese men.

While it is difficult to determine the exact cause of the obesity stigma, many theorize that the Western emphasis on individualism, the belief that individuals are in control of their own destiny, and with proper effort and determination can alter their life outcome may play an important role. Regardless, it seems that the complex causes of obesity have not yet permeated the minds of the majority of Americans who, consciously or unconsciously, stigmatize obese individuals, leading many obese individuals to endure extensive pain, humiliation, and injustice as a result of being overweight. Even more unfortunate, it appears that this stigma has an even stronger effect on women who, as a group, are judged by many according to their shape and weight.

SEE ALSO: Self-Esteem in Obese Women; Stereotypes and Obesity; Stigmas against Overweight Children.

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Obesity Action Coalition

THE OBESITY ACTION Coalition (OAC) is a national 501(c) (3) nonprofit, patient-based organization dedicated to educating and advocating for all those affected by obesity, morbid obesity, and childhood obesity. The mission of the OAC is to elevate and empower those affected by obesity through education, advocacy, and support. It aims to educate patients, family members, and the public about all forms of obesity. In addition, the OAC will increase obesity education, work to improve access to medical treatments for obese patients, advocate for safe and effective treatments, and strive to eliminate the negative stigma associated with all types of obesity.

The OAC offers many valuable educational and advocacy resources, such as the OAC Web site; the "Understanding Obesity Series," which details obesity, childhood obesity, and morbid obesity; the OAC Insurance Guide, "Working with Your Insurance Provider: A Guide to Seeking Weight-Loss Surgery"; the OAC's advocacy guide, "Your Voice Makes a Difference: A Guide on How You Can Help Fellow Patients Affected by Obesity"; and others.

Obesity is a complex disease. Patients often experience a wide variety of other health conditions (comorbidities), which include diabetes, heart disease, cancer, sleep apnea, and many more. Adding to the challenge, patients are often denied access to much-needed medical treatment (such as medically managed weight loss or bariatric surgery) as some payers and employers still do not recognize obesity and morbid obesity as a disease.

The OAC encourages patients to become advocates for change. To assist patients in accomplishing this change, the OAC provides many advocacy-related resources. Located under the "Advocacy" section of the OAC Web site, visitors have the ability to view national- and state-level issues that may affect them, OAC Public Policy Statements, Facts and Figures on obesity, and much more. Together, education and advocacy are an incredible combination of tools that can positively affect the obesity community, and proactively promote access to safe and effective treatment options for those affected by this disease. By building a coalition of members ranging from patients and their family members to healthcare professionals, the OAC will focus national attention on obesity; organize those concerned as advocates for action, advances, and change; and visibly affect and impact the healthcare community and the public.

SEE ALSO: American Diabetes Association; American Dietetic Association; American Medical Association; American Society for Bariatric Surgery.

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Obesity and Academic Performance

WHILE RESEARCH IS generally consistent regarding findings of poorer academic achievement among obese children and adolescents compared to their normal-

weight peers, a causal relationship between obesity and academic performance cannot be assumed. Thus, research suggests that obesity is associated with, but not a cause of, low academic performance.

Numerous research studies have identified a difference in academic achievement between obese and normal-weight children and adolescents. A review of studies on this topic conducted by Taras and Potts-Datema reported that for the most part, research findings indicate an association between low academic achievement and obesity despite differences in the assessments used, populations studied, and magnitude of findings across various research studies. For example, a study on primary school children in Thailand found that being overweight was associated with having a lower grade point average (GPA) in children in seventh through ninth grades.

Another study by Mikkila et al. found an association between poor school performance and obesity in Finnish adolescents. In addition, Li found that obese children had significantly lower IQs than normal-weight children in China. Campos and colleagues reported that obese Brazilian children had a lower IQ than normal-weight children. Laitinen et al. reported that in Finland, being obese at age 14 was a strong predictor of low school performance at age 16 and low level of education through age 31. In the United States, Falkner and colleagues found that obese middle school children were more likely to be held back in school and consider themselves poor students compared to average-weight adolescents. Furthermore, Greenfield and Fellner found that obese undergraduate women, but not obese men, had lower GPAs than their healthy-weight counterparts. Together, these studies all suggest a relationship between obesity and academic performance.

Academic performance is less widely researched in adults because most are no longer involved in formalized education; however, a study by Karnehed et al. looked at the relationship between obesity and attained education in Swedish men. They found that even when taking into account intelligence level, parental education, and socioeconomic status, obese men were doing worse than normal-weight men in the educational system. They reported that men who were obese when they were 18 years old had obtained lower grades in the ninth grade than normal-weight men. According to these findings, obese men were also less likely to attend university and even less likely to graduate from university compared to their normal-weight counterparts. This was an especially interesting finding because, unlike in the United States, education in Sweden is free of charge.

In this study on Swedish men, ninth-grade academic marks were found to be better predictors of educational attainment than an intelligence test taken when the students were 18 years old. This suggests that obese students were receiving lower marks than was predicted by their intelligence tests scores. The reasons for this were not examined; however low self-esteem as well as possible stigmatization by peers and teachers were identified as possible explanations. Research done by Falkner and colleagues, which found that obese adolescent boys were more likely to believe that they were poor students, more likely to predict that they would drop out of school, and more likely to be held back, raises interesting questions regarding the cause of poor academic performance found in obese individuals.

Unfortunately, this study only surveyed obese men and consequently these results cannot be generalized to the obese population as a whole. However, despite this significant limitation, this study highlights the importance of academic achievement and its relationship to life outcomes. Educational attainment has been found to be associated with occupation and income, and thus, while a causal connection cannot be assumed, poor academic achievement in obese individuals may be the first indicator of lower achievement and lower earning potential later in life.

While most studies agree that there is a relationship between obesity and low academic performance, the multitude of differences between obese and normal weight youth make it difficult to determine the cause underlying this disparity. Specifically, a study conducted by Datar et al. on the academic performance of American kindergartners and first-graders found that overweight children had significantly lower scores on math and reading standardized tests compared to children who were not overweight. However, compared to nonoverweight children, the overweight children in this study were also more likely to be poor, Hispanic, have mothers who were less educated, have a higher birth weight, and watch more hours of television or videos. When statistical analyses were run, which took the differences between the overweight and nonoverweight population

such as socioeconomic status into account, the differences in test scores were no longer significant.

In fact, children whose family incomes were above $75,000 and whose mothers graduated from college were significantly less likely to be overweight and were significantly more likely to have higher test scores. In addition, race/ethnicity and mother's education were found to be better predictors of test scores than weight. Similarly, a study conducted by Sigfusdottir et al. found that overweight Icelandic middle schoolers had lower self-esteem, worse grades, higher depressive mood, less educated parents, less physical activity, and poorer nutrition than their normal-weight peers. These studies echoed the complexity of the relationship between weight and academic achievement and concurred with other findings that weight is related to but not a cause of poor academic performance.

Childhood obesity is also associated with health risk factors, such as gastroenterological and endocrine conditions; high rates of psychopathology, such as anxiety and depression; and psychosocial problems, such as low self-esteem, low self-worth, and adjustment difficulties. All, some, or a combination of these factors may have an effect on academic performance or, alternatively, may have an effect on obesity/weight gain.

Research conducted by Schimmer et al. introduces another interesting variable which further highlights the complex relationship between obesity in children and low academic performance. This study evaluated the health-related quality of life, a construct which incorporates physical, psychological, social, and school functioning, of obese 5- to 18-year-olds. They found that obese youth were four times more likely to report impaired school functioning than normal-weight youth and had missed an average of about four days of school, which was about three more days than healthy youth, in the month prior to their participation in the study. While the reasons for and results of this high rate of absenteeism were not investigated, one might speculate about the relationship between truancy, the physical and psychological problems that tend to be associated with obesity, and the lower academic performance found in obese children/adolescents. However, again, the casual relationship between these factors cannot be determined.

While research on the effects of overweight on children and adolescents has found an association between obesity and low academic performance, a causal link between these two factors has not been empirically supported. Rather, research suggests that obesity is a marker and not a cause of low academic performance. Despite the complexity of the relationship between body weight and academic achievement, the many physical, psychological, and economic consequences of obesity in childhood make a strong case for initiatives on the part of the educational system to target obesity at a young age.

SEE ALSO: Morbid Obesity in Children; Obesity in Schools; Overweight Children and School Performance; Physical Activity and Obesity; Physical Activity in Children; Prevalence of Childhood Obesity in the United States; School Based Interventions to Prevent Obesity in Children; School Initiatives to Prevent Obesity; Self-Esteem and Children's Weight; Self-Esteem and Obesity; Stigmas against Overweight Children.

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Obesity and Cancer

THE TERM *cancer* refers to all malignant neoplasms or new cell growth. The molecular basis of cancer begins with nonlethal genetic damage to an individual cell. The processes through which a normal cell undergoes neoplastic transformation, deviates from its normal growth pattern, and progresses to a malignant phenotype have been separated into four phases. They are: (1) change in the deoxyribonucleic acid (DNA) pattern or its expression in the target cell; (2) growth of the transformed cell; (3) invasion into adjacent structures; and (4) metastases and relocation to distant tissues.

Cell division in the normal cell is remarkably accurate, and have multiple redundant systems that monitor cell integrity and detect DNA damage. Tumor suppressor gene products can arrest cell division until damage is repaired. Carcinogens exert damage by binding directly to the DNA or by modifying host detoxification enzyme systems that convert procarcinogens to more polar metabolites. Sometimes a metabolite becomes an "ultimate carcinogen" capable of damaging DNA.

Several cancers develop because the host has inherited mutations (germ-line mutations) that increase DNA susceptibility to damage. If acquired or inherited, mutations limit the ability of the cell to detect and correct DNA damage, or if proliferative signals from mediators and mutant oncogenes stimulate cell growth in spite of growth controls, the damaged cell will divide and produce daughter cells that contain "heritable" defecta that can be transmitted to all cell progeny. Further proliferative signals to cells with heritable DNA defects have potential to generate further mutations in the growing clone. Fortunately, genes that code for proteins make up only a small portion of the total genome. The efficiency of DNA repair, combined with the small number of scattered targets throughout the genome explain how organisms can survive "in a sea of carcinogens" without developing the disease.

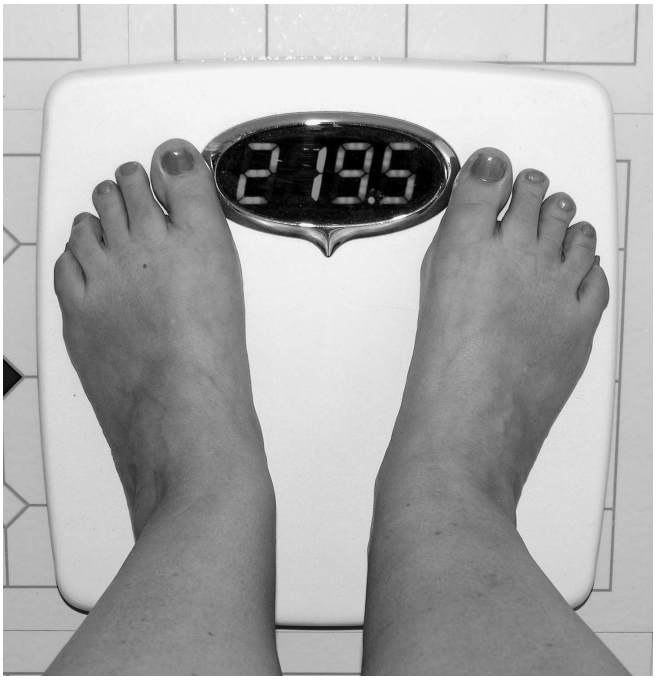
Heritable damage at a functional DNA locus is termed *initiation*. This damage modifies the behavior of the cell in one of two ways. It enhances the ability of the cell to (1) reproduce without consideration of normal cell restraints and (2) invade other tissues and travel to distant sites reserved for other cell types. Promoters are agents that, while not carcinogenic themselves, facilitate the growth of initiated cells by globally stimulating proliferation in the tissue or organ. In normal cells, the effects of growth promoters, hormones and other promoters are reversible. The life cycle of a cell proceeds through an orderly progression from birth to death, leaving no progeny. If the promoting influence is removed from normal tissue, proliferation stops, cells progress through their life cycle and die, thus hyperplasia can regress. In contrast, the progeny of transformed (initiated) cells stimulated to undergo unrestrained proliferation do not die but accumulate until clinically detected as a tumor.

As the transformed cell proliferates in defiance of DNA repair and normal cell cycle controls, it accumulates additional DNA damage that further augments its aggressive behavior. A transformed clone overgrows its neighbors, takes up essential nutrients, and prospers at the expense of the organism. Several physiological changes are characteristic of the mutant clone: (1) self-sufficiency in growth signals, (2) insensitivity to growth inhibitory signals, (3) evasion of apoptosis, (4) defects in DNA repair, (5) limitless replicative potential, (6) sustained ability to stimulate ingrowth of new blood vessels (angiogenesis), and (7) ability to evade immune defenses and rejection. As the mutant clone expands, it acquires subpopulations with additional phenotypic attributes including altered hormonal responsiveness and reduced susceptibility to antineoplastic drugs (termed *progression*). Thus, despite its origin as a single initiated cell, considerable genetic and phenotypic heterogeneity is observed in a tumor while it is still small and clinically undetectable. Ominously, some transformed cells acquire the ability to invade and metastasize at an early, undetectable stage.

RISK DUE TO CARCINOGENS, PROMOTERS, AND ANTICARCINOGENS IN FOOD

Environmental carcinogens, including chemicals, radiation and viruses, have potential to damage DNA and transform cells leading to cancer in animals. While some of the most potent carcinogens are produced in the extraction and incomplete combustion of fossil fuels, or are synthetic products created by industry, an enormous number of potentially carcinogenic chemicals are produced naturally by plants as defense against predators, as by-products of food processing, and by microorganism consumed in food. Because obesity results from storage of food ingested in excess of requirements, the obese subject may be at greater risk for cancer secondary to ingestion of a greater quantity of carcinogens in the food supply. At the same time, social, psychological, and biological factors that influence food intake can result in consumption of foods that contain high levels of carcinogens.

Some of the more common carcinogens in food include nitrostable amines and nitrates used to preserve food. Sodium nitrite is used to preserve bacon and processed meats such as ham and salami and is converted by gut bacteria to N-nitroso-compounds, carcinogenic in all species studied. Aflatoxin produced by the fungus *Aspergillus flavus* grows on improperly stored grains, nuts, and legumes and has been implicated as a hepatocarcinogen. Additionally, food preparation methods that expose food to high heat for a long time is known to alter protein structure and create carcinogenic heterocyclic amines, lipid peroxides and other mutagens. Meat also contains heme iron and other components implicated in production of endogenous N-nitroso compounds and in increased risk for several cancers including prostate, colon, and rectum.



Increased body size (BMI) and obesity is associated with moderately increased risk for cancers of all types.

Dietary fat is also implicated in cancer risk. In 1982, the National Academy of Sciences reviewed studies relating tumor growth to diet in experimental animals and large population studies demonstrating increased rates of breast, colon, prostate, and endometrial cancers correlated with per capita animal fat consumption. Based on this evidence, guidelines recommending reduction of fat intake to 30 percent of daily calorie intake were developed. Since then, a large body of experimental evidence has revealed that the relationship of dietary fat to cancer is more complex than originally thought.

The influences of total fat intake and total energy intake have not been firmly distinguished in animal models or in human studies, nor have the carcinogenic effects of red meat been separated from the effects of meat fat. Additionally, individual dietary fatty acids, even within a fatty acid type, may have different effects on carcinogenesis, tumor growth, and metastasis. Dietary fatty acids modulate cancer risk and metastasis through their role as substrate for local synthesis of eicosanoid metabolites such as prostaglandins and leukotrienes. These hormones modulate processes such as tumor-endothelial cell adhesion, proteolytic enzyme activity, and other biological cascades that facilitate tumor growth and spread.

While the mechanisms through which a specific dietary fatty acid type modulates risk have not been fully elucidated, the efficiency of membrane fatty acid conversion to eicosanoids may be involved. Fatty acids of the omega-6 type, derived from grains, produce eicosanoids implicated in cancer risk. In contrast, fatty acids of the omega-3 type, derived from green plants and cold-water fish, replace omega-6 fatty acids in the cell membrane. Eicosanoids produced from different fatty acids have different structure and may account for the observed difference in cancer risk.

Other dietary components also influence risk. Over 250 epidemiologic studies conducted in numerous countries with diverse diets support the association between fruit and vegetable consumption and reduced risk. In addition to fruits and vegetables, other whole foods including whole grains, legumes, tea, coffee, and chocolate contain a myriad of components including carotenoids, vitamins C, E, and K, dietary fiber, flavanoids, indoles, procyanidins, isoprenoids, and other bioactive components. Phytochemicals as well as substances in animal products are under intensive investigation for their ability to reduce DNA damage, modulate cell cycle activity, modify detoxifying enzymes that convert procarcinogens to ultimate carcinogens, mimic estrogens and other hormones by binding their receptors, and other activities that inhibit the initiation, promotion, and progression of cancer.

RISK FROM CHRONIC POSITIVE ENERGY BALANCE

The possibility that chronic caloric overload increased risk for cancer has been investigated for over 50 years. Experiments with overfed animal models demonstrated increased numbers of cancers, while chronically restricted animals lived longer and were cancer free. Epidemiological studies that measured body size and height subsequently revealed that growth exerts a modest influence on risk for all cancers, especially for hormone-dependent breast, uterine, and prostate cancer. Possible mechanisms for this increased risk include growth promoters and stimulatory hormones such as sex hormones, glucocorticoids, and insulin, as well as inflammatory mediators associated with obesity.

Obesity has been generally associated with insulin resistance and elevated plasma insulin levels. Experimental and epidemiologic studies support the hypothesis that the growth promoting effects of insulin increase risk for colon carcinogenesis. Insulin may directly activate its own receptor, or the receptors for insulin-like growth factor (IGF) expressed on normal colorectal cells and on cancer cells. Since insulin is only weakly mitogenic, its action is likely to be indirect, mediated through the growth-promoting effect of nonesterified fatty acids released from adipose tissue, which potentiate insulin resistance. Alternatively, elevated insulin levels may lead to increased concentrations of IGF and its binding proteins. These peptides are involved in regulation of cell growth and proliferation as well as cell transformation and death. Thus, the interactions of insulin, IGF, and IGF-binding proteins provide a mechanism linking excess energy intake with processes underlying the development and spread of cancer.

Obesity and chronic hyperinsulinemia are also associated with changes in total and bioavailable plasma sex steroid levels in both pre- and postmenopausal women. Increased bioavailability of sex steroids, especially estrogens, may result from several interactions with insulin and IGF. Increase in insulin and bioactive IGF concentrations in the obese organism inhibit the hepatic synthesis of sex-hormone–binding globulin (SHBG).

Insulin and IGF can also enhance the synthesis of androgens by the gonads and adrenal glands. Finally, androgens undergo increased conversion to estrogen metabolites by aromatization in adipose tissue. Thus, in postmenopausal women, the body mass index (BMI) is positively related to plasma levels of estrogen metabolites as well as to levels of bioavailable estrone not bound to SHBG. The "unopposed estrogen" hypothesis proposes that mitogenic effects of estrogens, when insufficiently counterbalanced by progesterone can result in growth promotion in hormone dependent tissues. Recent investigations have revealed that estrogen can also act as a "complete" carcinogen, capable of inflicting DNA damage by oxidative mechanisms.

Finally, adipose tissue accumulation is associated with the production of several proinflammatory factors including tumor necrosis factor alpha (TNF-α), interleukin-6 (IL-6), monocyte chemotactic protein 1, inducible nitric oxide synthase, and transforming growth factor ß1. Macrophages enter adipose tissue where they scavenge large dying adipocytes and release reactive oxidative products and other mediators.

While the link between chronic exposure to inflammatory mediators and cancer has not been clearly developed, inflammatory peptides induce rapid cell proliferation and produce DNA-damaging free radicals. Rapid cell division increases the likelihood of replication errors and ineffective DNA repair at critical regulatory sites. In an inflammatory environment, key enzyme cascades are upregulated, resulting in synthesis of inflammatory eicosanoids and other mediators known to facilitate tumor growth, invasion, and metastases. One molecular basis for this relationship is through the nuclear factor kappa beta

($NF_K\beta$) pathway that regulates apoptosis, cell proliferation, and cell growth arrest and enhances growth of new blood vessels by inducing vascular endothelial growth factor expression.

Clinical evidence for the link between chronic inflammation and cancer is seen in the development of hepatic cancer in chronic hepatitis and colon cancer in chronic colitis, and in the inverse association between long-term use of nonsteroidal antiinflammatory drugs (NSAID) and reduced risk of several cancers. In the endometrium, unopposed estrogens, as well as other established risk factors, induce an inflammatory effect. Insulin and estrogen exposure, in conjunction with inflammatory mediators, have been implicated in the development of endometrial cancer. A proinflammatory milieu can initiate and promote neoplastic transformation directly. It can also increase estrogen production, which may facilitate carcinogenesis by disrupting the estrogen-progestogen balance.

In summary, increased body size (BMI) and obesity is associated with moderately increased risk for cancers of all types. The data are confounded by difficulties in measurement and the multifactorial nature of the risk factors. The food supply contains both carcinogens and anticarcinogens, thus the choices made by the obese subject may play a major role in actual risk. Accumulation of metabolically active adipocytes and associated macrophages have a significant impact on whole body homeostasis. Further, there is evidence that dietary and lifestyle patterns modify the influence of adipose tissue on metabolic parameters. Mechanistic and clinical investigations are needed to guide recommendations.

SEE ALSO: Breast Cancer; Colon Cancer; Endometrial and Uterine Cancers; National Cancer Institute; Ovarian Cancer; Prostate Cancer; Uterine Cancers.

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Obesity and Drug Use

IN GENERAL, THE majority of obese adults tend not to be substance abusers. In fact, the diagnosis of a mental health disorder such as depression or anxiety tends to be higher for the obese population, whereas a lifetime risk of a substance use disorder tends to be relatively low. Alcohol abuse is relatively low in the adult obese population, as is marijuana, cocaine, and heroine abuse. It appears that for obese individuals, the drug of choice is food. The statistics point to an increase in substance use over the past decade in the obese adolescent population. Exact percentages of substance use, substance abuse, and dependency regarding obese adults, adolescents, or younger children are relatively scarce.

In general, 8.2 percent of the U.S. population aged 12 and older are illicit drug users. Of this number, 2.7 percent abuse prescription drugs. There are three categories of prescription drugs most commonly abused. The first category is opioids. Opioids are most commonly prescribed for pain and include oxycontin, vicodin, and demerol. The second category of abused prescription drugs includes central nervous system depressants. These drugs are primarily prescribed for sleep disorders and anxiety and include valium, xanax, nembutal, and mebaral. The last category of prescription drugs that tends to be highly abused by obese adults is stimulants. Stimulants are most commonly prescribed for obesity, attention deficit hyperactivity disorder (ADHD), and narcolepsy and include adderall, ritalin, dexadrine, and meridia. Stimulants including adderall and ritalin are being used by some pediatricians in the treatment of childhood obesity. The Food and Drug Administration, however, has not approved this "off-label" use of stimutants for the treatment of childhood obesity.

Similarly, off-label use of other medications, such as antidepressant medications that have weight loss side effects, may be prescribed to obese children and adults. The prescription use of stimulant drugs to treat adult obesity is more common. Physicians are required, though, to monitor adult patients taking obesity-related medications on a monthly basis. Legislation is currently looking into ways to monitor the abuse of prescription medications. Prescription drug abuse is becoming a large societal problem as abusers search for means to obtain their drug, from

stealing prescriptions to having multiple physicians prescribe them.

There are many causes of obesity, including a genetic predisposition, metabolism, a medical condition, a lifestyle issue, or any combination of the above. Another cause is thought to be brain chemistry and its link to addictive and compulsive behavior. Serotonin levels in the brain affect mood. Serotonin also causes the body to crave carbohydrates. Carbohydrates, in conjunction with the smell or taste of food, can stimulate dopamine which is the reward system in the brain. Dopamine is a naturally occurring substance that regulates feelings of pleasure. Reduced activity of dopamine in the brain may contribute to addictive behavior and obesity.

Compensating for low levels of serotonin, individuals consume more foods high in carbohydrates to temporarily feel better. This process slows down dopamine and the individual needs more of the same substance to feel pleasure. As a result, the individual begins to gain weight. Viewed from an addiction perspective, obesity has similar characteristics to other addictive disorders including drug addiction and compulsive gambling. These similarities include cravings for, obsessive searching for, and overconsumption of the substance (food). Research has demonstrated that obese adolescents admitted to a rehabilitation facility for drug dependence gained an average of 11 pounds after 60 days in treatment.

New research is now focusing on the notion of addiction transfer. Many compulsive eaters who have undergone bariatric surgery can no longer consume the large portions of food they were previously able to consume prior to their weight-loss surgery. According to addiction transfer, if food cannot be consumed, the addictive behavior will be transferred to a different substance or behavior. Addiction transfer specifically refers to patients who have undergone bariatric surgery and have developed new addictions to shopping, gambling, alcohol, or some other substance. Anywhere from 5 to 20 percent of post-bariatric surgery patients develop new compulsive behaviors. Research has found that after bariatric surgery, transfer of addiction from food to other substances, including alcohol and other drugs, does tend to increase.

The incidence of alcohol abuse tends to be the highest after bariatric surgery compared to other substances. Following gastric bypass surgery, many individuals are unable to tolerate alcohol as a result of the physiological changes from the surgery. With several types of bariatric surgery, a large portion of the stomach is bypassed resulting in the creation of a small pouch. When alcohol is consumed, it passes through this pouch to the intestines and is absorbed more quickly into the bloodstream. This may result in more rapid high blood alcohol levels and intoxication.

One component of treatment for individuals with addiction is to engage them in regular exercise. Exercise increases the number of dopamine receptors and the individual naturally feels good. The added bonus for the obese is that exercise is an effective way to lose weight and can decrease appetite.

Diet pills are advertised on television and sold on the Internet. At varying prices, anyone can purchase "Chinese diet pills," "Brazilian diet pills," Hoodia Gordonii, and so forth. These pills claim to produce quick, long-lasting weight loss. The obese consumer is eager to try anything to see quick results and will spend tremendous amounts of money in desperation. Without any type of federal regulation, consumers have no guarantee as to the contents of these "diet pills" or their potential harmful side effects. A safer method is through careful monitoring of a drug therapy program with a physician specializing in obesity.

Drug therapy is recommended for individuals who have a BMI of at least 30 and no obesity-related complications, or those with a BMI of at least 27 with two or more obesity-related complications. Currently, there are two long-term weight-loss drugs approved by the Food and Drug Administration for treating obesity—orlistat and sibutramine. The newest drug, orlistat, works as an inhibitor of pancreatic and gastrointestinal lipases. Orlistat, also known as Xenical®, blocks the absorption of approximately 30 percent of dietary fat. Orlistat tends to have a low abuse potential because it is not a central nervous system anorectic agent. Orlistat is also the only drug used specifically for obesity that is not currently a controlled substance. Weight loss associated with this medication is linked to a reduction in blood pressure. Sibutramine, also known as Meridia®, was introduced in 1997. It inhibits norepinephrine and serotonin neuronal reuptake. Originally developed as an antidepressant, it was found effective in reducing body weight and appetite, as well as increasing satiety.

There are three approved short-term medications currently on the market for obesity. All three are appetite suppressants. They are classified as stimulant

drugs that affect neurotransmitters in the brain, namely dopamine, epinephrine, and norepinephrine. Diethylpropion is also known as Tenuate® or Tenuate Dospan®. Phendimetrazine has trade names of Bontril®, Plegine®, Prelu-2®, X-Trozine®, and Adipost®. Phentermine is also known as Adipex-P®, Fastin®, Ionamin®, Oby-trim®, Pro-Fast®, and Zantryl®. Phentermine is half of the "fen/phen" combined drug previously used for weight loss that was taken off the market several years ago due to serious heart and lung complications experienced in patients. All three drugs are designed to be used for only two to three weeks. The average weight loss for individuals using these types of obesity medications is an average of 5–22 pounds. Some studies report up to a 10 percent loss from their baseline body weight following the use of these medications.

The growing understanding of the physiological mechanisms that regulate body fat content will allow for the development of new drugs to combat obesity. Currently, there are two new drugs in clinical trials. Rimonabant, which affects brain chemistry, and ciliary neurotrophic factor, which affects hormone levels to control appetite. Meanwhile, the primary focus is on the prevention of obesity. The management of obesity remains a multidisciplinary intervention: dietary management, an exercise program with regular follow-up, and pharmacological agents. Drug therapy is decided upon on an individual basis.

SEE ALSO: Drug Targets that Decrease Food Intake/Appetite; Drugs and Food; Drugs that Block Fat Cell Formation; Orlistat (Xenical); Pharmacological Treatment of Childhood Obesity; Rimonabant; Sibutramine (Meridia).

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Obesity and Socioeconomic Status

OBESITY FOLLOWS A socioeconomic gradient in which disease burden is greatest among those with limited economic resources. Regardless of race or ethnicity, obesity disproportionately affects individuals of lower socioeconomic status (SES). Findings from the *National Health Interview Survey*, reported that 26 percent of the population below the poverty level is obese, compared with populations in the highest income bracket, which had a 15 percent prevalence of obesity.

The inverse relationship between SES and obesity begins in adolescence and continues through the life span. In limited-resource women, the weight gradient actually increases with age. Limited-resource women tend to gain more weight each year than comparable populations of women with higher SES. A number of potential mechanisms have been suggested to explain the relationship between limited-resource individuals and obesity, including overconsumption of inexpensive, high-energy foods; periods of overeating followed by periods of deprivation, also known as the "food stamp cycle"; and limited access to recreational resources.

OVERCONSUMPTION OF INEXPENSIVE FOODS

In terms of total food purchases, limited-resource families spend nearly 34 percent of their disposable income on food, compared with 8.7 percent spent by

higher-income families. The disparity between the percentage of money spent by limited-resource populations compared with higher-income populations is great enough such that food spending effectively functions as an indicator of affluence. Although limited-resource families spend a higher percentage of their incomes on food, these families actually spend less total money on food. If limited-resource families spent as much per person on food as their higher-income counterparts, they would outlay more than 54 percent of their total disposable income on food alone. Instead, limited-resource families appear to maximize their money by purchasing less costly foods.

It is well established that higher diet quality is associated with higher incomes and with lower rates of obesity. When financial resources are lacking, nutrition and health tend to function in a secondary role to economic considerations. Households reduce food spending by changing the quality or variety of foods consumed before they reduce the quantity of food eaten. As a result, while families may get enough food to avoid feeling hungry, they may be poorly nourished because they cannot afford a consistently adequate diet that promotes health and averts obesity. The need to satisfy hunger overrides considerations for health.

INEXPENSIVE FOODS: ENERGY DENSE AND LESS NUTRITIOUS

Energy density is defined as the quantity of energy per unit of edible weight and is often expressed in kilocalorie per kilogram (kcal/kg) of food. The energy density of foods is a function of their water content. Low energy-dense foods are heavily hydrated and tend to be rich in nutrients. These include lean meats and fruits and vegetables. High energy-dense foods are dry, contain large quantities of fat and sugar and tend to be nutrient poor. Examples include refined grains, most fast foods, soft drinks, and snack foods, such as potato chips and cookies.

High energy-dense foods usually cost less than low energy-dense foods. A unit of energy from high-fat, high-sugar foods, such as potato chips and cookies, tends to be less expensive than a unit of energy from fruit and vegetables. For example, consider the relationship between energy density (kcal/kg) of the following foods and their "energy cost" as expressed in kcal/$. The energy cost of potato chips is 1,200 kcal/$ and the energy cost of soft drinks is 875 kcal/$. In contrast, the energy cost is approximately 250 kcal/$ for fresh carrots and approximately 170 kcal/$ for frozen orange juice. The difference in cost per calorie between the high energy-dense foods and those of lower density is more than sixfold.

High energy-dense foods provide a consumer with more calories per dollar than low-calorie food choices. The discrepancy in pricing increases between low and high energy-dense foods can be attributed to U.S. agricultural policy. Energy-dense foods, such as corn (added sugars) and soy (added fats), tend to be much more heavily subsidized than low energy-dense foods such as fresh produce. Fruits and vegetables are not governed by the same farm polices that encourage the overproduction of corn and soybeans. The result of subsidization of particular crops is reflected in retail trends. Pricing increases are lower for sugars/sweets and fats and oils than for fruits and vegetables.

ENERGY-DENSE FOODS CONTRIBUTE TO OBESITY

Consumers of higher SES have higher quality, healthier, and more varied diets which include more high-quality meats, seafood, fruits, and vegetables. Lower SES diets tend to include more low-cost meats, inexpensive grains, added sugars and added fats, and few fruits and vegetables. For example, limited-resource shoppers on average pay less per pound for ground beef, yet they shop in stores where ground beef is costlier. (Ground beef costs are directly related to its quality and fat content.) This discrepancy can be explained by recognizing that limited-resource shoppers tend to buy low-quality, high-fat beef, because they cannot afford more expensive cuts. Despite the benefits of increased fruit and vegetable consumption in combating obesity, individuals with lower household incomes (below 130 percent poverty) consume the fewest fruits and vegetables of any SES group.

Even individuals taking part in government supplemented food programs consume less than the recommended servings of fruits and vegetables a day. As such, limited-resource populations fail to meet recommendations made by U.S. Department of Agriculture's (USDA's) Dietary Guidelines Advisory Committee to consume a minimum of three servings of vegetables and two servings of fruits each day and the National Cancer Institute's 5-a-Day for Better Health Campaign, which recommends eating at least five servings of fruits and vegetables each day.

Limited-resource populations consume more calories per dollar than higher-income populations. The consumption of high energy-dense foods can contribute to weight gain and obesity. This is so, because individuals eat a constant weight of food. If individuals consume foods that contain a greater amount of energy per unit of weight, the result may be an overall increase in energy intake that can result in subsequent weight gain. Refined grains, added sugars, and added fats are among the lowest-cost sources of dietary energy, but tend to be nutrient poor. These foods tend to be low cost, taste good, and are convenient.

ACCESS TO HEALTHY FOODS

Income can be associated with both the location of food outlets as well as the selection of food that is available. Both of these factors influence an individual's ability to follow dietary recommendations put forward by the USDA.

Higher SES neighborhoods tend to have greater access to supermarkets, convenience stores, and more diverse food choices than their lower SES counterparts. Consequently, individuals living in low SES neighborhoods are more reliant on smaller shops, convenience stores, and bodegas to acquire food. Not only do these smaller stores offer a more limited selection of foods, but their prices are typically higher than those of larger supermarkets. The presence of large neighborhood supermarkets is associated with better diet of neighborhood residents. Moreover, more healthful products in stores are associated with increased consumption of more healthful foods by individuals living near to the stores. Those who live and shop in wealthier neighborhoods tend to be less obese than those who shop in more limited-resource areas.

Many limited-resource populations reside in urban centers. Urban dwellers tend to pay more for food in their local community compared to suburban residents, who buy the same goods at larger supermarkets for a lower cost. Due to the higher food prices and limited resources, inner-city residents may seek less expensive food outside of their neighborhoods. Unfortunately, these groups can experience greater difficulties obtaining access to large (and distant) shopping facilities because they may lack private transportation or live in areas where public transportation is inadequate or nonexistent.

ACCESS TO RECREATION FACILITIES

Obesity is a result of not only consuming excess calories, but also of expending too few calories through activity. SES is related to obesity and physical activity. Limited-resource populations tend to be less physically active and more obese than their more advantaged counterparts. Commonly, lower SES populations have reduced access to recreational facilities. Reduced access to recreational facilities is frequently associated with decreased physical activity and increased probability of obesity. Other factors that influence a community's engagement in physical activity include affordability, quality and accessibility of facilities, crime, safety, social cohesion of the neighborhood, street connectivity, sidewalks, and number of recreation areas.

SEE ALSO: Access to Nutritious Foods; Food Insecurity and Obesity; Income Level.

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Obesity and Sports

PHYSICAL ACTIVITY DECREASES in direct relationship to the degree of obesity. Obesity has been defined as being 20 percent or more overweight, using the concept of relative weight. Obesity in younger individuals

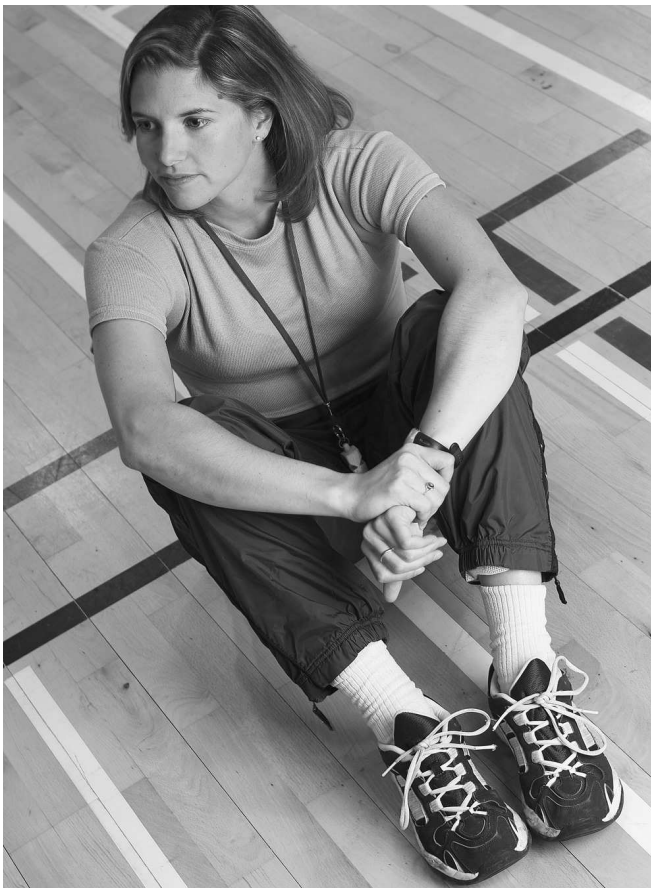
is generally characterized by elevated levels of fat mass and fat-free mass, whereas obesity in the older adults is reflective of high levels of body fat, but low quantiles of fat-free mass. There is positive energy imbalance between energy intake and energy expenditure in obese individuals and the most variable component of the daily energy expenditure is the physical activity.

Although the etiology is multifactorial, it is readily accepted that low levels of physical activity are one of the leading risk factors for the development of obesity and its attendant comorbidities. Many obese individuals are sedentary, have little training or skills in physical activity, and are difficult to motivate toward increasing their activity. Young children who have a family history of obesity and also have low levels of physical activity are at increased risk for development of childhood obesity The loss of fat-free mass and gain in body fatness—preferentially stored in central regions of the body—in the adults are at least partially attributable to physical inactivity. The decline in physical activity may accelerate the accumulation of central body fat in women during the menopause transition.

Exercise and sports can be described as a subcategory of physical activity defined as any voluntary movement produced by the skeletal muscles that results in increased energy expenditure. Although the line between sports and exercise is certainly not clear, exercise is planned, structured, and repetitive movements and sport is a physical activity or skill carried out by a set of rules and engaged in recreatively or competitively.

Physical activity, regardless of what kind, expends energy. The average sedentary person usually expends only 300–800 calories a day in physical activity, most of this from informal, unplanned types of movement. On the other hand, athletes usually match their resting metabolic rate energy expenditure through hard, intense exercise. Increased levels of physical activity are associated with decreased risks for obesity. Those engaging in vigorous exercise that causes heavy sweating or large increases in breathing or heart rate, one to three hours a week, or walking for more than four hours a week, are better able to ward off weight gain than their more sedentary counterparts.

Physical activity can reduce the risk for obesity for several reasons. First, it can vary widely from person to person, and thus when it is increased or decreased, it can have large influences on total energy expenditure, and consequently, energy balance.



Low levels of physical activity are one of the leading risk factors for the development of obesity and its attendant comorbidities.

Second, physical activity can increase the amount of fat free mass FFM (or lean muscle mass) that one has. FFM is the primary determinant of resting metabolic rate, and has long-term consequences for energy balance. Third, exercise training can influence substrate (nutrient) utilization, thereby playing a role in how ingested nutrients are partitioned into fat and FFM. Finally, there is some suggestion that exercise can increase endorphins and mood, and reduce feelings of hunger in the short term, so an increase in exercise might lead to reduced energy intake.

In recent studies it is reported that the risk of obesity determined by body mass index in certain sports such as football and wrestling is higher, whereas those sports such as tennis, volleyball, baseball, and softball have no significant association with overweight. The sports require knocking opponents down, encourage the youth to maintain a large body mass index as a playing advantage. Additionally, the risk of being

overweight is lowest among males who play two or more sports. A high body mass index in those studies may represent a high muscle mass rather than fat.

The determination of the optimal exercise dose for preventing and treatment of obesity is of primary importance. How much, what modality, or what intensity of exercise is most efficacious in people with obesity of any age? The high intensity of the exercise, and the resulting increases in muscle tissue, may lead to favorable changes in body fat. It is reported that walking and high-intensity activity are significant negative predictors of weight gain, but group sports and job activity are not. Aerobic exercise significantly accelerates weight loss when combined with a reducing diet; however, the extra weight lost is small when compared to that caused by the diet. Rather, aerobic exercise improves cardiorespiratory endurance, blood lipid profile, psychological state, and decreases risk of obesity-related diseases.

Physical activity should be an integral part of weight-loss therapy and weight maintenance. Initially, moderate level of physical activity that causes only light sweating or slight-to-moderate increases in breathing or heart rate for 30–45 minutes, three to five days per week, should be encouraged. Physical activities involving vigorous intensity is generally not recommended for severely obese people because of existing comorbid conditions. Initial activities may be increasing small tasks of daily living such as taking the stairs or brisk walking or swimming or cycling at a slow pace.

With time, depending on the progress, the amount of weight lost, and functional capacity, the person may engage in more strenous activities. The person can start by walking 10 minutes, three days a week, and can build to 30–45 minutes of more intense walking at least three days a week and increase to most days. A regimen of daily walking is an attractive form of physical activity. Reducing sedentary time is also another approach to increasing activity. Strength training may increase fat oxidation and therefore be helpful in minimizing the increase in adiposity in the older obese adults because fat oxidation is principally determined by fat-free mass which decreases in older ages.

A primary goal of obesity treatment is to create a negative energy balance. Obese individuals are often not able to expend a significant number of calories in a given exercise session, particularly at the beginning of the program, because their level of fitness and excess weight limit both duration and intensity of activity. However, because obese individuals weigh more than lean individuals, and consequently have more muscle mass as well, there metabolic rate will naturally be higher. This essentially means that if an obese individual walks for 30 minutes at moderate intensity, they will expend more calories than if a lean person were to walk for 30 minutes. Thus, a small amount of movement, compared to a completely sedentary life, can make a large difference in overall energy balance if it is carried out long-term.

Heat intolerance, difficulty breathing, movement restriction, musculoskeletal pain and injury, local muscular weakness, and balance anxiety are exercise precautions for the obese, and their importance increases with the increasing degree of obesity.

Long-term maintenance of weight loss is a primary goal in the treatment of obesity. Thefore, education, developing good rapport, reviewing the expected course and outcomes, individual-provider alliance building, and group activities are very effective strategies for promoting adherence and addressing important psychosocial barriers to exercise for obese people.

SEE ALSO: Exercise; Obesity; Physical Activity.

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Obesity and the Brain

OBESITY, OR EXCESSIVE body fat, is the result of a chronic surplus in energy intake in relation to the body's energy requirements. This thermodynamic definition of obesity implies that obesity occurs when

eating behavior is not homeostatically regulated. The control of eating behavior, as with any other behavior, is complex and involves multimodal communication between various organs of the body and the central nervous system (CNS). This communication is mediated by neural and blood-borne signals.

More than a century ago, the role of the hypothalamus as the brain region fundamentally involved in the pathogenesis of obesity was suggested by two case reports on obesity associated with lesions of the hypothalamus. Over time, brain lesioning and stimulation studies pointed to the ventromedial hypothalamus as the "satiety center" and to the lateral hypothalamic area as the "hunger center." However, only with the discovery of leptin in 1994, the essence of the central control of eating behavior began to be understood.

Leptin is mainly secreted by adipocytes and circulates in the body in proportion to body fat. It is the primary homeostatic signal for the CNS, informing the brain on the amount of energy stored in the body as fat. In fact, leptin modulates neuronal activity in several regions of the CNS, primarily in the hypothalamus, which integrate responses to meal consumption, by enhancing the sensitivity to blood-borne and neurally mediated satiety signals, including cholecystokinin (CCK), glucagon-like peptide-1 (GLP-1), and peptide $YY_{3\text{-}36}$ ($PYY_{3\text{-}36}$). Within the CNS, leptin modulates the tone of peptidergic pathways by inhibiting orexigenic transmitters, including the neuropeptide Y (NPY) and agouti-related peptide (AgRP) and stimulating anorexigenic transmitters, including the proopiomelanocortin (POMC). In this way, the negative feedback of excessive food consumption is exerted.

For example, when an individual is in negative energy balance, the bioavailability of leptin decreases with the consequence of a reduced activation of POMC neurons and a reduced inhibition (= activation) of AgRP and NPY neurons in the arcuate nucleus (which is part of the ventromedial region) of the hypothalamus. In turn, POMC and AgRP/NPY neurons project to other hypothalamic regions, including the lateral hypothalamus where melanin-concentrating hormone (MCH) neurons, among others, are represented. MCH is thought to be an orexigenic signal, based on the phenotype of mice lacking or overexpressing MCH and the observation that food intake reduction and leptin deficiency stimulate its expression.

The above few examples of the peptidergic pathways involved in the control of energy intake and body weight in the hypothalamus of rodents are only intended to offer a perspective on how the classic topographic distinction of hunger and satiety regions of the hypothalamus has been revised in light of molecular scale information accrued in the last decades with animal models of obesity. The translation of these models to the understanding of human obesity and potential therapeutic applications is one of the challenges that clinical research currently faces.

It is known that in some monogenic forms of human obesity the absence or functional inability of anorexigenic signals are associated with extreme phenotypes of obesity. This confirms that learnings from animal models can be translated to humans and offered the possibility of very successful therapies. The example of leptin is enlightening. In a few human cases with severe hyperphagia and massive obesity, the absence of leptin has been documented and the phenotype of leptin-deficient individuals reversed by leptin replacement therapy. In these extreme cases, leptin shows its maximum efficacy as an anorexigenic signal.

Common forms of obesity, however, are characterized by high plasma concentrations of leptin, consistent with excess body fat. In these cases, leptin seems to be ineffective as an anorexigenic signal and the control of body weight seems to escape the principles of a negative feedback circuit. As a corollary to this, leptin resistance is now accepted as a phenotypic trait of common forms of obesity. The molecular mechanisms of leptin resistance are unclear, but candidate hypotheses include a defect in leptin transport across the blood brain barrier as well as the impairment of leptin-dependent signaling pathways. Put in terms of a concentration-efficacy relationship, the anorexigenic effect of leptin is maximal at low concentrations, as characteristic of leptin deficiency or starvation. It reaches a plateau (i.e., anorexigenic insensitivity) at the high concentrations characteristic of the obesity state, although across a wide span of interindividual variability.

Besides the homeostatic level of control of eating behavior, hedonic aspects of food consumption are regarded as primary determinants of overeating and consequently overweight and obesity. It is a common observation that the expectation and/or experience of pleasure and reward derived from food ingestion are

potent drivers of the search for food, the approach to it and the consumption of it. This is regarded today as the main motivational trigger of the world wide pandemics of obesity—the hedonic salience of food consumption often overrides its homeostatic value either as the initiating or the terminating factor of a meal.

The sensory experience of food consumption is the first contributor to the hedonic appreciation of food and usually involves sight, olfaction, taste, and tactile stimulation of the mouth. Sensory information is received by the brain in primary, secondary, and tertiary processing regions which integrate multimodal sensory signals and offer the combined sensory appreciation of food. Remarkably, several of these brain regions are also involved in processing the reward value of sensory and cognitive experiences, including the insula, anterior cingulate cortex, anteromedial temporal lobe-amygdala, and orbitofrontal cortex.

All these regions receive dopaminergic afferent fibers, consistent with the experimental evidence that indicates that dopamine is the main neuromediator of reward. However, it is still unclear whether dopamine is only involved in the anticipation of reward and motivation to eat ("wanting") or if it is also involved in the receipt of reward and the pleasure derived from the consumption of food ("liking"). The latter function has been repeatedly linked to opioid neurotransmission in animals, but a recent positron emission tomography study in humans reported dopamine release after consumption of a favorite meal in proportion to the self-reported pleasantness of the meal. Besides evidence linking dopamine release to dynamic contexts of motivation to eat as reported in response to the sensory experience of food or to the pleasure of an actual meal consumption, dopamine has also been related to overeating and obesity by a study, which reported a reduced dopamine type 2 receptor (DRD2) binding in obese compared to normal-weight individuals in resting conditions.

Functional neuroimaging has also shed some preliminary light on the regional cerebral differences between obese and normal weight individuals in response to the termination of a meal as compared to fasting. A wide array of limbic and paralimbic regions, including the orbitofrontal and insular cortices, anterior cingulate, hippocampus, and hypothalamic region reduce their neural activity after eating more in obese than in normal-weight individuals, whereas prefrontal areas increase their neural activity after eating more in obese than in normal-weight individuals.

Consistent with these findings, a model has been proposed where the prefrontal cortex signals satiation by sending inhibitory inputs to the limbic/paralimbic areas, thus suppressing hunger. It has been speculated that in obese individuals, this top-down control exercised by the prefrontal cortex over hyperactive orexigenic areas might require more neural activation to stop meal consumption. Cross-sectional comparisons among obese, normal weight and formerly obese individuals ("postobese") have also offered a preliminary insight on the existence of brain regions that might play a role in the predisposition to obesity. In fact, subregions of the insular cortex and hippocampus, which have also been involved in anticipation and reward (insula), learning/memory (hippocampus), and food craving (both), have been reported in postobese individuals to exhibit obese-like changes of neural activity in response to the sensory perception of food and consumption of a meal, respectively.

Far from offering a conclusive understanding of which neural abnormalities are associated with overeating and obesity, functional neuroimaging has played an instrumental role in documenting that several distinct regions of the brain, including cortical and subcortical areas, are involved in eating behavior and harbor possible neurobiological aberrations underlying weight gain and obesity. This is consistent with the notion that eating behavior is an idiosyncratic combination of decisions and actions which do not address only the homeostatic need to eat but also and mostly the hedonic and cognitive value of eating.

Therefore, not just the hypothalamus and brainstem, but also brain regions involved in processing reward and cognition are now gaining momentum in clinical research on the pathophysiology of obesity. It is not by chance that the most promising centrally acting antiobesity drugs act on neurotransmitter systems and receptors which are widely distributed across many different regions of the brain and are primarily involved in mood regulation, stress, emotional and reward processes, including the serotoninergic, dopaminergic, opioidergic, and cannabinoidergic systems.

SEE ALSO: Amphetamine; Anxiety; Appetite Signals; Cannabinoid System; Central Nervous System; Cholecysto-

kinin; CNS/Hypothalamic Energy Sensing; Depression; Dopamine; Drug Targets that Decrease Food Intake/Appetite; Fenfluramine; Flavor: Taste and Smell; Food Reward; Ghrelin; Gustatory System; Humoral Factors and Satiety; Liking vs. Wanting; Hypothalamus; Melanocortins; Monogenic Effects that Result in Obesity; Mood and Food; NPY (Neuropeptide Y); Nutrient Reward; Opioids; POMC Proopiomelanocortin.

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Obesity and the Immune System

OBESITY, THE STATE of having excess body fat relative to lean body mass, alters the immune system of the individual who suffers from this condition. Obesity leads to the chronic active stimulation of the immune system from within the adipose or fat tissue, in the lymphoid tissue (where adipose tissue and lymph nodes sit in proximity to one another), and through distant messaging systems. Adipocytes (fat cells) and the immune system are in frequent communication with each other, leading to inflammation and modulation of signaling that has ultimately been implicated in the pathogenesis of diseases related to obesity.

An effective immune response to injury is critical to species survival. Typically, this response is swift and strong, dealing quickly with insults. However, with obesity, the hypothesized insult is stress on the organism from excess fatty acid accumulation and chronic oxygen insufficiency as the expanding fat mass outstrips its blood vessels (by which the tissue gets oxygen and nutrients). The result of this chronic inflammatory state is diseases such as heart disease, diabetes, asthma, high blood pressure, and osteoarthritis.

The immune system is an intricate network of cells that communicate through cell-to-cell and protein-to-cell messaging systems that affect clearance of insults to an organism. Classically, injury such as a cut leads to recruitment of fighter cells and repair mechanisms whose calling cards are protein messengers sent out by the injured cells like SOS signals. Researchers looking at adipose tissue (an organ unto itself) have found that adipose or fat tissue is sending out the very same calling cards, eliciting the help of the immune system. Unlike in the case of a cut, however, the fat mass often continues to grow and therefore continues to send out these signals, resulting in a chronic stimulation of the immune system.

Lymph nodes located throughout the body act as filters and sites of concentrated immune activity where lymphocytes (white blood cells that are primed or already trained to recognize foreign invaders such as bacteria and viruses) reside, and respond as the organism's blood stream comes into contact with infectious organisms. Surrounding the lymph nodes are layers of fat cells or adipocytes that collectively are called adipose tissue. Lymphoid tissue and adipose tissue reside together in the bone marrow (the adult site of blood cell production), in the apron-like sheet of fat covering the abdomen called the omentum, and in other concentrated sites such as the axilla (under the underarm), groin, popliteal fossa (behind the knee), and cervical regions (near the neck).

There appear to be many functions of this spatial relationship, including the provision of energy for the lymph tissue in the form of fatty acids from the adipocytes. For example, a foreign invader such as the breakdown product of bacteria (specifically called lipopolysaccharide which is the outer membrane of certain bacteria) is recognized by the lymph cells in the lymph node. They make cytokines (chemical signals indicating they are responding to an insult). The adipocytes intercept these help signals and provide fatty acids as energy for the battle. Inflammation (the multiplication of cells that respond in a specific fashion to the insult and the further manufacture of cytokines to kindle the fire and signal other tissues to send in backup supplies) results. In response to mild inflammation, the adipocytes are depleted of their fatty acids stores and decrease in size.

However, there are signals to produce more fat cells and the number of layers of fat cells grows, increasing the number of fat cells surrounding the lymph node when there is chronic active inflammation.

The type of fatty acid that accumulates in the fat cells alters the immune response, providing one example of how nutritional composition in an organism modulates the immune system. Dendritic cells process the foreign invader and present it to the lymphoid tissue facilitating the activation of an immune response. In the example of bacteria—they present the lipopolysaccharide which is highly immunogenic (provides a high degree of immune stimulation and activation of immune cells). The number of dendritic cells increases in response to an increase in dietary lipids (sunflower oil, trans-fats, saturated fats), and decrease in response to fish oil. Interestingly, dendritic cells multiply to a greater degree in response to the fatty acids than they do to a low dose of lipopolysaccharide.

The number of dendritic cells also increases when the organism develops ongoing chronic active immune stimulation. Because the dendritic cells interact with many other immune cells including lymphocytes, their activation has the potential of modulating the immune response in as-yet unappreciated ways. While the extent of dietary nutrient composition's impact on the immune system has not been fully elucidated, the response of dendritic cells to dietary lipid content is one example of how the metabolic and immune systems impact one another.

Infectious agents themselves have previously been implicated in the development of obesity due to the elevation of inflammatory markers seen in virally infected organisms and in obese subjects. Certainly, as noted above, active inflammation can cause the replication and increase in number of adipocytes. However, viruses can also stimulate excess leptin production which is a proinflammatory hormone that causes an increased production of inflammatory cytokines and chemokines (small cytokines that send messages to nearby cells).

However, there is another recently discovered crossroads which could connect an infectious pathogen, excess lipid intake and or obesity, and the induction of an inflammatory response—the toll-like receptor. This receptor can recognize a variety of stimuli, including pathogens, certain lipids, and has even been implicated in autoimmune phenomenon (when an individual's immune system reacts against itself as if it were foreign). These primitive cell receptors trigger the immune system and provide key evidence for the intersection of metabolic and pathogenic pathways leading to the proliferation of common inflammatory molecules.

Adipocytes make a number of proinflammatory molecules including leptin (a hormone), tumor necrosis factor alpha, and interleukin-6 (cytokine) that are seen systemically in states of inflammation and injury. In addition, as the fat tissue increases, macrophages (cells that recruit more immune cells, plus engulf and digest cell debris, defending the organism as part of the immune system) infiltrate proportionately, and manufacture even more immune-stimulating signals.

Looking at just one of the cytokines produced, interleukin-6 (IL-6) provides some insight as to why its secretion in response to nutrient over intake was considered novel. Along with tumor necrosis factor alpha (TNF-alpha), its discovery led to looking at the implications of nutrient excess in stimulating the immune system and leading to a chronically active inflammatory state. IL-6 is classically made by T lymphocytes and macrophages in response to tissue damage, but also in the host response to foreign invaders such as a bacterium called *Streptococcus pneumonia*. It mediates fever and the acute phase response (in which proteins are made in response to inflammation). It supports increasing metabolism to generate energy to fight threats to the organism and, in so doing, increases body temperature, often leading to fever. While further investigating the origin of IL-6, researchers found that approximately 30 percent of the body's IL-6 is, in fact, made by the fat tissue. Clearly, the adipose tissue is a highly metabolically active tissue that interacts with the immune system.

The full extent to which obesity stimulates and modulates the immune system has yet to be fully elucidated. What we know is more than can be expounded above, but clearly the adipose or fat tissue and the immune system are interrelated, proliferate a similar set of inflammatory chemicals leading to a chronic inflammatory state in the setting of excess lipids and nutrition and are modulated by what we eat. There is much left to be investigated, but undoubtedly, the two systems have considerable interconnectedness.

SEE ALSO: Adipocytes; Obesity and Inflammation.

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Obesity and the Media

MEDIA IS A broad term that refers to forms of public communication designed to provide information to large groups of people. Common forms of media include newspapers, magazines, billboards, art, radio, television, and the internet.

The media not only conveys information reflective of cultural ideals, values, and current events, but it also dramatically influences public opinion and cultural norms. With regard to weight, mainstream media in Western cultures broadcasts pejorative messages about fat and obesity and laudatory messages about fitness, thinness, and muscularity. The purposes of this entry are (1) to review sociocultural messages conveyed by mainstream Western media about weight, and (2) to explore the influence of exposure to these messages on attitudes about fatness and obesity, psychological health, and eating behavior in media consumers.

MESSAGES ABOUT WEIGHT AND APPEARANCE IN WESTERN MEDIA

Mainstream Western media presents powerful information about weight and cultural ideals of appearance to the public at large. Western media refers to media originating from or reflective of the majority cultures of the United States, Canada, Australia, and Western Europe. A quick inspection of a fashion magazine, series of commercials, or billboard advertisement from Western media makes obvious dominant cultural attitudes about weight and ideals of appearance. With almost no exception, the ideal female is depicted with flawless skin, flowing hair, a thin waist, light-colored eyes, and long legs and the ideal man is muscular, fit, tall, and lean with broad shoulders and a well-developed chest.

Presentations of the ideal weight and appearance of men and women are omnipresent in Western media and have become increasingly extreme over the last few decades. For women, media representations have become exceedingly thin. Whereas Marilyn Monroe's voluptuous figure represented the classic ideal of beauty 50 years ago, emaciated, underweight models now dominate fashion media. *Playboy* and Miss America Pageant contestants are almost all underweight, with about 25 percent meeting weight criteria for anorexia nervosa (body mass index [BMI = weight (kg)/height $(m)^2$] < 17.5).

For men, models are increasingly more muscular and less fat. In a study of *Playgirl* centerfolds models from the 1970s to the 1990s, the average male model lost 12 pounds of fat and gained 27 pounds of muscle. Research documenting trends in action-figure toys marketed to boys (e.g., GI Joe) found that action figures grew significantly larger in size, more muscular, and less fat from the 1970s to the 1990s. When extrapolated to human size, the bodies of male action figures are as unattainable for boys as the Barbie doll is for girls.

There are few media representations of female and male models that do not promote and reflect the ultrathin, ultramuscular ideals. Overweight and obese individuals are extremely underrepresented in the media, particularly in fashion- and beauty-oriented outlets. In prime-time fictional television shows, for example, obese women only represent about three percent of characters, whereas about 33 percent of actors are underweight. This is in stark contrast to the current American population, in which almost one-third of individuals are obese and two-thirds are overweight.

When overweight individuals are presented in the media, they are generally presented with particular personality and demographic characteristics. Overweight and obese individuals are more likely to be the subject of ridicule and comic relief in television programs. Overweight women are less likely to have a romantic partner, have strong friendships, or be physically affectionate in mainstream media. Additionally, overweight actors are rarely presented in ways that do not draw attention to, mock, or utilize their bodily size and appearance in some specific way.

ATTITUDES TOWARDS OBESITY AND FATNESS

Media idealization of thin women and muscular men, combined with stigmatization of fatness, is believed to dramatically influence people's views about obesity. The media not only promotes an aesthetic preference, but it also ascribes morality and social value to individuals who meet the ideal. Those who meet the physical ideal for their gender are deemed desirable and socially valued, whereas the obese are reviled and socially rejected. In this way, the media promotes negative stereotyping and stigmatization of obese and overweight individuals.

Obesity stigma, defined as the social unacceptability of obesity and fatness, is prevalent to the degree that it has been called the last socially acceptable form of discrimination in the United States. Overweight and obese people are more frequently described as lazy, stupid, weak, sloppy, ugly, and less desirable as friends than their average- or underweight counterparts. They are more likely to be the target of interpersonal and institutional discrimination. Obese children are often shunned by their peers and regular targets of bullies. These prejudicial and discriminatory attitudes against overweight and obese children, adolescents, and adults have been demonstrated in diverse community samples and among nurses, psychologists, physicians, and social workers alike.

Media messages about the ideal appearance also suggest that individuals should obtain and maintain a slender, fit frame through whatever means are available and necessary. The media presents the ideal appearance as one that is readily attainable for anyone who works hard enough. Should one decide that one would like to attain the thin, muscular ideal, the media promotes numerous means of weight loss and muscle gain.

Products and services regularly marketed in the media include diet aids, exercise programs, drug use, special foods, over-the-counter pills, and plastic surgery. For example, diet foods, low-fat, and no-fat foods combined with weight-loss programs including the plethora of diets that emerge each year (e.g., South Beach Diet, Zone Diet, Atkins Diet, Weight Watchers) are increasingly more popular in the United States today. The dieting industry, promoted heavily in the media, has become a $50-billion-per-year business.

EFFECTS OF MEDIA MESSAGES ON PSYCHOLOGICAL HEALTH

Numerous survey, correlational, and experimental research studies have examined the influence of exposure to Western media messages of appearance on the psychological health of media consumers. Although undoubtedly a multidirectional, complex relationship influenced by numerous personal and environmental factors, the media can be viewed as one of many forms of socialization that influences psychological health and individual preferences, beliefs, attitudes, and identities.

One of the most profound ways that the media affects psychological health is by fostering body image disturbance in obese, normal-weight, and underweight individuals alike. Theoretically, when individuals are exposed to Western media images promoting a thin, fit ideal, they are likely to engage in an upward social comparison whereby they think about their physical appearance in comparison to the idealized models found in magazines, television, the internet, and movies. However, because few (if any) individuals view themselves as meeting the extremely rigid but highly valued cultural ideal, such comparisons often lead them to feel dissatisfied with their bodies and appearance. In turn, body dissatisfaction, defined as a negative subjective evaluation of one's physical appearance, is one of the strongest empirically supported risk factors for the development of disordered eating; it has been found to predict dieting, binge eating, purging, excessive laxative use, and cessation of all eating.

Numerous studies document the effects of exposure to idealized images of female models on women's body image. Experimental studies testing acute exposure to idealized media representations of female models indicate that viewing thin models increases body image disturbance and mood disturbance in adult women. Furthermore, women report significantly more body image disturbance after viewing thin models than after viewing average-size women, overweight women, or control objects.

Although much less studied to date, research also documents the influence of the media on body image in men. In line with ideals espoused by the media, men often strive for muscularity and low body fat. Men often report feeling undesirable, unattractive, and nonmasculine when they do not believe they have met the societal ideal. Emerging research suggests that males

may experience body image disturbance at comparable rates to those of women. Given that the goal of many media images is to capture one's attention and, through a process of social comparison, instill a desire to purchase a particular product or attain a given outcome, the fact that humans compare themselves to models within media images is not surprising.

Exposure to Western media messages of appearance can also have an influence on affect and self-esteem. Some individuals report higher levels of stress, guilt, shame, sadness, and insecurity after viewing idealized media images of models. Increased symptoms of anxiety and depression have been reported following media exposure.

Although the media is strongly implicated in perpetuating body image problems and mood disturbance, there are a number of mediating and moderating factors that will influence the degree to which exposure to media information influences body image. The degree to which an individual engages in social comparison with models, consumes media materials (e.g., the number of hours of television watched a day, number of magazine subscriptions), and internalizes the appearance of models as normative, readily attainable, and personally desirable will influence the effects of media exposure on body image and psychological health. For example, internalization is the extent to which an individual subscribes to the sociocultural ideals and participates in behaviors in pursuit of approximating these ideals. The more one is aware of mainstream ideals of appearance perpetuated by the media and aspires to attain them, the more one is likely to be affected by exposure to media messages.

Multicultural factors, such as gender, race, ethnicity, socioeconomic status, level of acculturation, and culture of origin, may also have a dramatic influence the degree to which one is influenced by media messages. Attitudes toward obesity and the centrality of appearance to one's self-worth and self-definition appear to vary significantly by ethnicity and gender. For example, African-American women are generally more tolerant toward obesity, idealize a larger physical ideal, have less rigid standards of physical appearance, and are less likely to ascribe negative stereotypes to obese individuals than white, Euro-American women. In traditional Mexican-American culture, larger, curvier women are viewed as more attractive and economically stable than thin women. Historically, appearance is less central to the social value of men because of traditional Western gender roles. It is critical to consider these multicultural factors when examining the role of the media on individuals.

INFLUENCE OF MEDIA ON EATING BEHAVIOR

In addition to influencing people's attitudes about obesity and psychological health, much research posits that mainstream Western media encourages unhealthy eating behaviors. One way this is evidenced is in the types of foods that are commonly advertised in the media. Most of the research on the effect of food advertising has been conducted with children. For example, more than 90 percent of all advertisements aimed at children feature candy, fast food, soft drinks, and sugar-filled cereal. Fast-food outlets alone spend an estimated $3 billion a year in television advertisements aimed at children. Comparatively, advertisements for healthy foods, such as fruits and vegetables, are rare.

Another way the media influences eating behavior is through the use of media icons, celebrities, and popular characters to promote certain brands and types of foods. For example, fast-food restaurants regularly market "kid's meals" to children that feature specific widely publicized cartoon or movie characters. This cross-marketing of food products with well-liked characters can lead individuals, particularly children, to want to buy and consume the advertised products. In fact, individuals may even erroneously believe that despite being high-calorie, low-nutrient foods, many advertised foods (e.g., fast foods) are healthier than home-cooked food because the models advertising the products are inevitably fit, thin, and attractive.

A third way the media is thought to influence eating behavior is by increasing food intake during media use. People are more likely to snack during media use. Additionally, increased television use has been correlated with higher energy intake, particularly for foods high in fat, sugar, and salt. Furthermore, when individuals who generally exhibit control over their eating view diet commercials and other thin-ideal promoting media, women are likely to overeat.

Finally, the media may influence eating behavior is by encouraging a sedentary lifestyle. Although watching television and an active lifestyle are not mutually exclusive, sitting in front of a television for hours a day is in and of itself is a sedentary activity that decreases energy expenditure and increases high density food

intake. Furthermore, television advertising increases preference for food types promoted in the advertisements—typically nonnutritive, high-density foods. Sitting and watching television or playing video games can lower one's metabolic rates.

SUMMARY AND CONCLUSIONS

In recent years, increased attention has been paid to sociocultural messages about weight and appearance promoted and perpetuated through mainstream Western media. With almost no exception, mainstream Western media idealizes thin women and muscular men. Overweight and obese individuals rarely appear in the media and, when they do, they are presented in a derogatory manner compared to their fit, thin counterparts. Should an individual not meet the extremely rigid, virtually unattainable Western appearance ideals, the media provides consumers with a barrage of diet plans and weight-loss products to help them better approximate the ideal.

Media messages about weight can detrimentally affect social attitudes toward obese individuals, thereby fueling obesity stigma. Although some subcultures in the United States and abroad do not view obesity in a negative light (e.g., African-American culture, various native cultures around the world), obesity stigma and weight discrimination are growing national and international problems.

For obese and overweight individuals living in Western culture, the effects of the media on self-concept, self-worth, and body image may be even more detrimental than for normal-weight individuals because of the overwhelming amount of weight prejudice and discrimination. Being overweight or obese is not only seen as a failure to be good looking and attractive, but is an indication of one's personality, social desirability, and cultural value.

SEE ALSO: Appearance; Disordered Eating; Eating Disorders and Obesity; Fast Food; Obese Children in the Media; Obese Women and Social Stigmatization; Weight Discrimination.

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Obesity and Viruses

SINCE 1980, THE prevalence of obesity among children and adults continues to increase worldwide. The implications for the health of obese or overweight individuals are tremendous. Some of the potential complications include hypertension, Type 2 diabetes, coronary heart disease, stroke, osteoarthritis, gallbladder disease, sleep apnea, and cancer (breast, uterine, colon). The economic burden caused by obesity and its complications is reaching alarming proportions. Therefore, it is important to evaluate all possible causes of such persistent obesity epidemic to offer effective treatments. Although viral causes of obesity remain a very controversial idea, the growing number of studies suggests a link between a common virus and obesity.

There are complex regulatory pathways in the human body that influence body weight. Certain hormones produced in the body can signal the brain to affect our appetite and energy expenditure. In addition, genes and environment are important causes of obesity. Heredity, including a number of inherited genetic disorders (such as Prader-Willi syndrome and Bardet-Biedl syndrome), and gene mutations may contribute to rising incidence of obese individuals. However, more popular view is that of the environmental factors being the most important causes of obesity. These

factors include diet, portion size, physical activity, cultural, social, economical, and educational influences.

Infectious agents, such as viruses and bacteria, are increasingly linked to many common chronic diseases. Bacteria are microscopic organisms (organisms that can be seen only through a microscope) that are composed from one cell and can cause disease or perform an important role as part of the human body (help with digestion). Viruses are infectious particles; they are not made of cells, but contain a shell with their DNA (hereditary information) inside. Viruses can reproduce and function only when they invade other cells. The treatment for bacterial infections often includes antibiotics. Vaccines offer a good protection against viruses.

Because obesity is such an important risk factor for many chronic conditions, studies have originated to explore a possibility of an infectious agent being associated with obesity. In the past two decades, it has been observed that certain viruses are associated with increased incidence of obesity in animals. These viruses were different strains of a common virus found in humans, called adenovirus. This virus is often associated with the common cold, flu, meningitis (inflammation of the membranes covering the brain), and diarrhea in some cases. Interestingly, the link between this virus and obesity in animals was discovered around the same time as the prevalence of obesity began to increase (1980).

Specifically, a strain of virus, called adenovirus-36 (Ad-36), has been shown to cause obesity in chickens, mice, and nonhuman primates. Researchers found that Ad-36 may increase the number of fat cells, which makes the animal store more fat. Recently, it was also discovered that obese or overweight individuals have a higher prevalence (20 to 30 percent) of the virus than slim or normal-weight individuals (11 percent). Infection with Ad-36 presents with only mild cold-like symptoms that may last one or two days. The presence of the virus grants some positive outcomes. It has been observed that in animals with Ad-36 virus, the levels of low-density lipoprotein cholesterol (LDL-C) (the "bad cholesterol" that accumulates deposit in the arteries) were decreased.

A clear causality between Ad-36 and human obesity has not yet been established. Many questions remain as to how these viruses function and how they may cause obesity in some animals/humans but not in others. However, if such discovery was to occur, it would provide a great potential for developing a vaccine against virally induced obesity. In addition, screening tests may be developed to determine the presence of the virus in general or selected population.

SEE ALSO: Appetite Control; Bardet-Biedl Syndrome; LDL Receptors; Prader-Willi Syndrome; Viral Causes.

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Obesity as a Public Health Crisis

THE UNITED STATES is not alone in the obesity epidemic. Worldwide, children and adults are chronically overweight and obese. The World Health Organization (WHO) estimates that in 2005, approximately 1.6 billion adults across the globe were overweight. In the WHO estimate, adults were considered to be people ages 15 years and above. Over 400 million adults worldwide were considered obese that same year. Every year, the number escalates.

In the United States, the Centers for Disease Control and Prevention (CDC) monitor annual obesity rates by state. Within the CDC, the National Center for Chronic Disease Prevention & Health Promotion maintains a Behavioral Risk Factor Surveillance System (BRFSS), a telephone survey system in operation since 1984. The BRFSS collects data on health issues such as diabetes and obesity. Obesity is determined by a person's Body Mass Index, or BMI. BMI takes into account an adult's height and weight. It is calcu-

lated by dividing weight in kilograms by the square of the height in meters. For most people, a BMI falling in the range of 18 to 25 is healthy. Between about 25 and 30 is considered overweight; BMI values exceeding 30 are in the obese range. Importantly, BMI values are valid for men and women, but are to be calculated for adults only. The WHO is currently working on a standard BMI scale for infants and children.

As of 2006, the state with the most obese population was Mississippi, with 31.4 percent of its adults in the obese range, while the least obese population was in Colorado, at 18.2 percent.

State	Percent Obese
Alabama	30.5
Alaska	26.2
Arizona	22.9
Arkansas	26.9
California	23.3
Colorado	18.2
Connecticut	20.6
Delaware	26.0
Florida	23.1
Georgia	27.1
Hawaii	20.6
Idaho	24.1
Illinois	25.1
Indiana	27.8
Iowa	25.7
Kansas	25.9
Kentucky	28.0
Louisiana	27.1
Maine	23.1
Maryland	24.9
Massachusetts	20.3
Michigan	28.8
Minnesota	24.7
Mississippi	31.4
Missouri	27.2
Montana	21.2
Nebraska	26.9
Nevada	25.0
New Hampshire	22.4
New Jersey	22.6
New Mexico	22.9
New York	22.9
North Carolina	26.6
North Dakota	25.4
Ohio	28.4
Oklahoma	28.8
Oregon	24.8
Pennsylvania	24.0
Puerto Rico	24.7
Rhode Island	21.4
South Carolina	29.4
South Dakota	25.4
Tennessee	28.8
Texas	26.1
Utah	21.9
Vermont	21.2
Virginia	25.1
Virgin Islands	26.1
Washington	24.2
Washington, D.C.	22.5
West Virginia	31.0
Wisconsin	26.6
Wyoming	23.3

There are many theories that account for the prevalence of overweight status and obesity in the United States and abroad. The fact is that an individual is overweight because he or she takes in more energy than he or she expends. The reasons for this energy imbalance are not always clear. Certainly some people eat too much and exercise too little. Some people have hormonal or other physiological imbalances that prevent their bodies from maintaining a proper weight. A popular theory tackles the rising obesity epidemic among youths—children growing up do not learn healthy eating and lifestyle habits.

Rising obesity levels have given cause to rising health costs, as well as to increased rates of morbidity and mortality. Many diseases are related to overweight status and obesity, including diabetes, cardiovascular problems such as cardiovascular disease, osteoarthritis and other skeletal problems, and even certain cancers. These health problems are directly related to obesity and being overweight; many more disorders, some doctors believe, could be resolved by a person's maintaining his or her weight.

SEE ALSO: Addictive Behaviors; Appetite Control; Body Mass Index; Centers for Disease Control.

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Obesity in Schools

PREVALENCE RATES OF overweight children have doubled in the last 20 years. In 1980, 7 percent of 6- to 11-year-olds were overweight compared with 18.8 percent in 2004. In the past 20 years, the prevalence rate for adolescents aged 12–19 years has tripled from 5 percent to 17.1 percent. Approximately 80 percent of obese adolescents will become obese adults. Children who are overweight by the age 8 are more likely to become morbidly obese in adulthood. Roughly 50 percent of all youth aged 12–21 years are physically active, and only 20 percent of adolescents in the United States eat the recommended daily servings of fruits and vegetables. These two factors are major contributors to the epidemic of obesity in the United States.

Obesity in childhood and adolescence contributes to many serious physical and emotional consequences. It is estimated that at least 61 percent of young people who are overweight have at least one additional risk factor with heart disease, high blood pressure, or high cholesterol. Obesity beginning in childhood is linked to many other diseases and ailments in adulthood, including bone and joint problems, sleep apnea, eating disorders, infertility, asthma, eye disorders, diabetes, certain types of cancers, gallbladder disease, and stroke. Many obese children suffer from hypertension, gallbladder disease, osteoarthritis, and menstrual irregularities in their youth. There has been a dramatic increase in pediatric Type 2 diabetes in the general population, with much of this increase being almost entirely attributed to the rise in childhood obesity. Children with obesity are nine times more likely to have persistent high blood pressure. High blood pressure in childhood is the strongest predictor of adult high blood pressure, significantly increasing the risk of adult cardiovascular disease.

Approximately 30 percent of morbidly obese children suffer from sleep apnea, a disorder where breathing during sleep becomes briefly interrupted. Sleep apnea is associated with depression, and memory, learning, and concentration problems. Although rare, some obese children are at risk for obesity hypoventilation syndrome. This is a life-threatening syndrome in which insufficient amounts of oxygen are breathed in while asleep and awake. Less common problems in obese individuals include slipped capital epiphyses and Blount's disease. These medical conditions may result in bowed bones, hip and knee pain, and altered gait. Thirty percent of women diagnosed with polycystic ovary disease, commonly associated with menstrual difficulties and infertility, are obese.

Being overweight brings about serious emotional and psychological consequences. Overweight children are much less likely to describe themselves as happy or confident. They are much more likely to describe themselves as lonely, sad, fearful, and different. Overweight children are five times more likely to say their self-confidence is poor compared to average-weight students. Overweight children, regardless of the topic, tend to have more worries than average-weight children, including worries about the future. Obesity is linked to a number of psychological difficulties. Regardless of age, individuals who are obese tend to be at higher risk for depression, eating disorders, distorted body image, low self-esteem, and anxiety. Obese adolescents report more sadness, loneliness, and anxiety. Childhood obesity is linked to oppositional defiant disorder in adolescence.

Many obese children are victims of bullying. Bullying may take the form of teasing, name-calling, threats, rejection, sexual harassment, and physical harm. Bullying behavior against girls may be more covert, through manipulation or control. Threats to withdraw friendships or rumor-spreading are common. Girls display bullying behavior through aggression in relationships compared to boys. As they reach adolescence, these obese children can become the perpetrators because of their size. Being victimized in the bullying process brings with it additional psychological and emotional consequences.

At an age where reliance on peers for social support, self-esteem, and identity is high, social development may be hindered. Both children and adults characterize obese children as having problems at school. For younger obese children, they are more likely to be the victims of bullying for their appearance. Older obese

boys tend to bully because of their size and physical dominance within their peer group. Being overweight and obese has a negative effect on high school performance and college entrance.

In 1995, annual healthcare costs related to obesity were about $52 billion. Today, approximately 300,000 people die prematurely from obesity and healthcare costs have dramatically increased to over $100 billion annually for treatment of obesity-related diseases. Including lost wages, this number reaches over $117 billion. In the last 20 years, the costs of obesity-related illnesses in children have risen from $35 million to $127 million. These numbers reflect a 228 percent increase in gallbladder disease and a 436 percent increase in sleep apnea related to childhood obesity. It is apparent that prevention of obesity needs to begin at an earlier age. Because children spend one-third of their waking lives in school, school-based obesity prevention programs can do well implemented in this setting. In the school environment, children are a captive audience. Children are receptive to school-based interventions and research demonstrates that these types of programs receive positive responses from students as long as the information is provided in a supportive manner and geared toward meeting the concerns of the obese child.

Successful school-based interventions targeting obesity have several common elements. Key to any program is the focus on promoting healthy weight rather than targeting a specific weight-loss goal or focusing on weight loss. Successful interventions focus on healthy eating habits and healthy lifestyle choices such as increasing physical activity. Successful programs utilize a combination approach to combat obesity that includes behavior management, nutrition education, physical activity, and family involvement.

The behavioral component focuses on the immediate surroundings of the obese child. This includes the physical and social environment as well as the child's own thoughts and behaviors. The purpose of this behavioral approach is to learn healthy behaviors that can replace unhealthy ones and can be continued into adulthood. Specific behavioral techniques utilized include self-monitoring, which helps the obese child monitor his or her eating and exercise habits; operant conditioning, in which goals can be reinforced; stimulus control, or changing the antecedents to eating and sedentary behaviors (e.g., parking the car farther away so that the family has to walk more); and eating management, instituting strategies during meals to reduce intake (e.g., putting smaller portions on the plate).

Nutrition education is vital for obese children. Daily caloric intake, nutritional values in foods, and the food pyramid are basic information that obese children need to understand to assist them in making healthy food choices. Interventions that focus on providing this basic information and teaching children how to prepare healthy snacks and meals can reinforce that healthy eating can taste good, be fun to make, and can get the entire family involved in meal planning.

Common school-based interventions for increasing physical activity typically focus on increasing time in physical education classes. This does not promote lifelong changes in physical activity. Additional behavioral interventions are needed to introduce activities that children find enjoyable and will continue participation in as adults. Interventions that introduce obese children to a variety of physical activities are important. Because of their excess weight, obese children do not have the capacity to engage in strenuous exercise; therefore, caution and close monitoring is advised when engaging obese children in any physical activity.

Parent involvement is the most crucial element to any successful obesity intervention with children. Parents can be educated to assist their children with implementing new behaviors. Parents can reinforce increased activity levels, engage in physical activity with their children, plan healthier meals for their entire family, and model good eating habits.

The Centers for Disease Control and Prevention (CDC) has published specific guidelines for school polices and procedures that promote physical activity and healthy eating. This well-thought-out strategy for promoting good health in the school enlists the support of parents and the community. Ten key strategies are addressed.

First, the CDC suggests having a Coordinated School Health Program (CSHP) to address physical activity and nutrition. CSHP is a systematic approach to promoting student health emphasizing the assessment of needs, planning, analysis of redundancies and gaps, and evaluation. CSHP integrates the efforts of the school community that affect student health: health and physical education; health and nutrition services; counseling, psychological and social ser-

vices; healthy school environment; promotion of staff health; and involvement of family and community. The focus of CSHP is to improve the quality of each of the above components and improve collaboration among the people working within each component.

Second, the CDC recommends a school health coordinator and active school health council (SHC) be designated. The school health coordinator is responsible for coordinating and managing policies, programs, and activities related to school health. The SHC would represent school staff and administration, parents, healthcare and social service professionals, and civic and religious community leaders. The SHC is responsible for providing guidance to the coordinator on activities to incorporate into the school health program and support for the programs initiated by the coordinator.

Third, it is recommended that each school's SHC examine his or her health policies and develop a plan for improvement. The CDC has developed *a School Health Index: A Self-Assessment and Planning Guide* (SHI) which assists schools in identifying both strengths and weaknesses in their current health policies. This instrument contains eight modules that assess the components of the CSHP, and assist schools in prioritizing changes, with focus on activities that promote nutrition and physical activity.

Fourth, schools are encouraged to strengthen their physical education and nutrition policies. It is recommended that these policies be distributed to the school's community for additional support. Research shows that not eating breakfast affects children's school performance, and as children get older, fewer eat breakfast. In terms of dietary intake, many children and adolescents do not eat enough fruits, vegetables, and fiber. They tend not to meet the dietary guidelines for saturated fat and calcium. Over the last 25 years, the consumption of soft drinks has doubled for adolescent females and tripled for adolescent males. Although physical activity is crucial in the maintenance of normal body weight, participation in physical activity steadily declines with age. Fifty-four percent of students in high school attend physical education classes. Of this number, 84 percent actually exercise or play some sort of sport for 20 minutes or longer in their physical education classes.

Fifth, it is suggested that a health promotion program be implemented for school staff that focuses on improving overall performance, attendance, and morale. This program can be geared toward giving staff the tools to use for becoming role models for students in becoming healthy adults.

Sixth, the CDC recommends establishing a high-standard health education curriculum aimed at improving health behaviors in students. Components of this curriculum include teaching students the skills needed to adopt new behaviors, examining the barriers that hinder adopting new behaviors, and providing numerous and varied opportunities to practice these new behaviors.

Seventh, a high-standard physical education curriculum is needed in schools. The CDC views physical education classes like other courses of study that are based upon strict national standards that dictate what students need to know. The CDC has outlined essential elements of this physical education curriculum. They include knowledge and skills for lifelong participation in physical activity; enjoyable activity that meets the needs of all students; class time that involves less inactivity; and instruction not only in motor, fitness, and sports skills, but also in self-management. It is proposed that elementary students engage in 150 minutes of physical education class per week and secondary school students engage in 225 minutes of physical education class per week.

Eighth, the CDC suggests providing students with additional opportunities outside physical education classes for physical activities. This may include unstructured play during recess at the elementary school level, or intramural sports, after-school sport clubs (e.g., ski club), and physical activity clubs (e.g., a running club at the secondary school level). Teachers now incorporate physical activity into daily lesson plans by using movement to teach concepts in many academic subjects.

Ninth, in terms of nutrition, the CDC proposes that schools have a sound meal program in which food service personnel are highly trained and have opportunities for professional development. In 1996, major changes were made in the federal school meal program that made school meals more nutritious. Schools can continue to make efforts to improve their own program by making the eating environment safe and clean and providing students with sufficient time to eat.

Last, it is recommended that schools provide healthy alternatives to junk food and soft drinks outside the

school meal program, including vending machines, at concession stands, at school parties, fund-raising campaigns, after-school programs, and school stores. Current federal regulations limit "minimal nutritional value" foods and beverages in cafeterias during meal times, including soft drinks, gum, and candy. Schools make their own regulations as to whether these foods and beverages will be allowed on school grounds.

The U.S. Department of Agriculture, the U.S. Department of Human Services, and the U.S. Department of Education have published *Making It Happen: School Nutrition Success Stories*. This publication outlines six strategies that schools can implement to help make a more nutritional environment. These strategies include: making more healthy foods and drinks available; influencing food and drink contracts to promote health choices; establishing standards of nutrition for the school to be followed; marketing healthy choices at school; limiting the amount of time students have access to non-meal program foods and drinks; and utilizing fund-raising activities to promote student health.

The CDC program is concise, with numerous support resources for schools to enlist for assistance. Most schools do not have the internal funds or state funding to implement such a program as they struggle to meet the pressures of standardized testing. Childhood obesity is at epidemic proportion in the United State, and prevention at the school is a logical treatment environment. Funding will need to become a national priority or schools will need to learn how to acquire their own outside funding to fight their own school's obesity battles.

SEE ALSO: Obesity and Academic Performance; Physical Activity in Children.

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Ob/Ob Mouse

THE OB/OB MOUSE is a genetically mutated mouse that is unable to produce the hormone leptin. The ob/ob mouse is different from the db/db mouse, which is able to produce leptin, but has an improperly working leptin receptor. The ob/ob mouse overeats and becomes severely obese during the course of its lifetime. There is a single gene that is mutated in this mouse which prevents the normal synthesis of leptin. Leptin is a hormone that is produced by fat cells, and it plays a role in eating and energy utilization. Leptin was first discovered in 1994 at Rockefeller University as the compound that was responsible for the obesity observed in the ob/ob mouse. The ob/ob mouse is one of several different animal models that are used to study obesity. Other mouse/rat models that are used to study obesity include the db/db mouse, Zucker rat, Zucker diabetic fatty rat, tubby mouse, and the agouti-yellow mouse.

There are many physiological and metabolic defects that are found with the ob/ob mouse. These include overeating, obesity, elevated insulin and glucose levels, hyperplasia (enlarged growth) of the cells inside the pancreas, insulin resistance, Type 2 diabetes mellitus, decreased growth hormone levels, impaired thyroid hormone levels, decreased body temperature, and infertility. The defective ob/ob gene can appear on several different strains (types) of mice and the

deleterious effects from the lack of leptin vary from strain to strain. For example, some strains of mice have moderately elevated insulin while other strains may have severely elevated insulin.

The ob/ob mouse has been used in numerous research studies to further understand how certain diseases develop. For example, the heart disease that results from Type 2 diabetes mellitus in the ob/ob mouse is very similar to the heart disease that develops from Type 2 diabetes mellitus in humans. This similarity has been useful in understanding the progression of the heart disease as well as in treatment development. Another area the ob/ob mouse has been incredibly useful in studying is the regulation of how/why people eat. Leptin is produced in fat cells and once these fat cells are filled to capacity, leptin sends a message to the brain to tell it that the body is no longer hungry. Leptin does this through the actions of some other neuropeptides (proteins in the brain). For example, leptin inhibits neuropeptide Y and agouti-related protein (which normally tells the body that it should eat) and by increasing the levels of alpha-melanocyte stimulating hormone (which normally tells the body to stop eating).

Treating ob/ob mice with leptin, improves all of the physiological and metabolic problems with which they are afflicted. For example, female ob/ob mice that are treated with leptin will have an increase in their follicle stimulating hormone and luteinizing hormone and thus have a return of their normal reproductive function. As long as these female ob/ob mice receive leptin, they will be able to get pregnant and have offsprings.

Several of the new treatments for Type 2 diabetes mellitus are the result of research from the ob/ob mouse. Incretins are hormones that are produced in the body's digestive system. They play a role in telling a person to stop eating as well as maintaining normal blood sugar and insulin levels. A new class of compounds known as incretin mimetics is being used as medicine in patients with Type 2 diabetes mellitus. A recent study found that use of incretin mimetics in ob/ob mice greatly alleviated some of the complications associated with diabetes. Thiazolidinediones (TZD) are another class of compounds that are used as diabetic medication. They function by activating peroxisome proliferators activated receptors (PPAR). Ob/ob mice that are treated with TZDs have had improvement in some of their diabetes complications.

While the ob/ob mouse is useful in studying how leptin deficiency relates to obesity, the data need to be interpreted with caution. Leptin deficiencies are very rare in humans. There are an incredibly small number of patients who are leptin deficient and they exhibit the symptoms described above. This deficiency can be identified early in a person's life and treatment with synthetic leptin will alleviate any complications. Paradoxically to what researchers hypothesized, most overweight and obese people actually have very high levels of leptin. It is now believed that these people may be leptin resistant and continue to eat despite their body telling them they are full. Thus, the db/db mouse might be a better mouse model to relate to human obesity because of normal leptin synthesis in the body despite impaired leptin functioning.

SEE ALSO: Db/Db Mouse; Leptin.

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Obsessive-Compulsive Disorder

OBSESSIVE-COMPULSIVE DISORDER (OCD) is an anxiety disorder characterized by intrusive thoughts and/or repetitive behavior that interfere with daily functioning. The obsessive thoughts and repetitive

behaviors in OCD are found in often overweight or obese individuals whose obsessive thoughts compel them to overeat or binge. Both OCD and obesity may be treated by increasing serotonin levels in the brain.

SYMPTOMS

The obsessions experienced in OCD are intrusive, negative thoughts that have no basis in reality and only serve to create anxiety. The obsessions are out of character for the individual even though the individual recognizes the obsessions as self-generated, coming from his or her own mind. Common obsessions include contamination, harming oneself or others, the need for symmetry, behaving unacceptably, making a mistake, and sexual or religious obsessions.

To relieve the anxiety caused by the obsessions, the individual performs compulsions, or repetitive behaviors, also called rituals. Rituals are not useful tasks and the preoccupation with performing the rituals interferes with daily life. Rituals can be physical behaviors such as washing, hoarding, checking, asking for reassurance, arranging, or organizing. A common example of a physical ritual is washing the hands until they are raw. Rituals can also be mental behaviors such as counting, repeating words in the mind, and attempting to neutralize the obsessive thoughts. A common example of a mental ritual is counting the number of steps between places. Performing the ritual only temporarily relieves the anxiety caused by the obsession, forcing the individual to perform the ritual multiple times a day or whenever anxiety from the obsessions becomes too much to handle.

CLINICAL DIAGNOSIS

OCD can develop at any time during the life span. In children, the symptoms are noticeable around age 6 or 7. Most cases of OCD develop by the age of 21. Women are only slightly more likely to develop OCD than men. Many individuals with OCD do not receive treatment because of the shame and humiliation of admitting they have a problem.

OCD is diagnosed using the following criteria from the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV):

- Having either obsessions or compulsions. Obsessions are defined as (1) recurrent and persistent thoughts that are intrusive and induce anxiety; (2) not excessive worries about real-life problems; (3) attempts are made to ignore or suppress the thoughts; and (4) the thoughts are recognized as a product of the person's own mind. Compulsions are defined as (1) repetitive behaviors or mental acts that the person feels driven to perform, and (2) aimed at reducing anxiety or preventing an event even though the thoughts and events are not connected in a realistic way.
- Recognizing that the obsessions or compulsions are unreasonable.
- Obsessions or compulsions are time consuming or interfere with normal functioning.
- If another disorder is present, the obsessions or compulsions are not restricted to it (e.g., preoccupation with food when the individual has an eating disorder).
- The obsessions or compulsions are not drug induced or caused by a medical condition.

CAUSES

The cause of OCD is not known; however, there are genetic, biological, and psychological factors linked to its development. Genetics may play a role in OCD. Prader-Willi syndrome, a genetic disorder involving chromosome 15, is characterized by food preoccupations, rituals, and compulsiveness, with the majority of affected individuals suffering from obesity by adolescence or early adulthood.

There are numerous biological factors linked to OCD. The condition may develop after brain damage or trauma, especially if injury occurs to the basal ganglia, which controls movement, or the prefrontal cortex, which is involved in planning and forming strategies. There have also been reported cases of OCD developing after a streptococcal infection such as strep throat. The antibodies that attack the strep may also affect brain function in some individuals, resulting in OCD. Finally, the most accepted biological factor is a lack of serotonin in the brain.

Serotonin is a neurotransmitter that helps regulate mood, eating behavior, sleep, arousal, and pain. Serotonin plays a major role in the medical treatment of OCD.

The psychological view of OCD is based on the idea that behaviors and thought processes are learned over the course of the lifetime. OCD may also develop after a traumatic experience such as a sexual assault. The

unlearning of the negative thoughts and behaviors play a major role in the psychological treatment of OCD.

TREATMENT

Two types of therapy are typically employed to treat OCD. The first is drug therapy. Selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants are commonly used to increase the amount of serotonin in the brain. Other drugs such as antipsychotics and anticonvulsants may be prescribed if the individual has an adverse reaction to serotonergic drugs. The second therapy employed is cognitive-behavioral therapy. This therapy uses various techniques to change thought and behavior patterns to impact emotions. For example, an individual with a hand-washing ritual may be told to touch something dirty, such as the floor, and refrain from washing their hands. During the process, the individual will retrain his or her own thought processes to get rid of the fear of contamination. Drug therapy and cognitive-behavioral therapy may be used independently or concurrently, with the greatest success seen using a combination of the therapies.

LINK TO OBESITY

Many overweight and obese individuals experience compulsions in the form of overeating and report eating to reduce anxiety. Although many obese individuals are not diagnosed with OCD, the similar thought and behavior patterns suggest a link between the two. In addition, drugs that act on serotonin may also act as appetite suppressants and have been suggested for use to treat obesity.

SEE ALSO: Binge Eating; Cognitive Behavior Therapy; Prader-Willi Syndrome; Serotonergic Medication.

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Office of Dietary Supplements

THE OFFICE OF Dietary Supplements (ODS) was created in 1994, as authorized by the Dietary Supplement Health and Education Act of 1994 (Public Law 103-417). It is located within the Office of Disease Prevention, Office of the Director, within the National Institutes of Health (NIH). The responsibilities of ODS are to explore the role of dietary supplements to improve healthcare, to promote scientific research on the benefits of supplements, to coordinate research within NIH regarding supplements, to collect and compile the results of scientific research concerning supplements, and to act as an adviser on issues concerning supplements.

ODS provides research funding largely through collaboration with NIH Institutes and Centers, because the ODS itself does not have granting authority. Program areas within ODS include evidence-based reviews of the efficacy and safety of supplements, dietary research on botanicals, training and career development for scientists interested in studying supplements, development of databases of dietary supplement ingredients, development of analytical methods and reference materials, and creation of educational and informational materials for the general public, including the Dietary Supplement Fact Sheets which are available for download from the ODS Web site.

ODS has developed several bibliographical tools to facilitate dissemination of information about supplements. The Computer Access to Research on Dietary Supplements (CARDS) database, available through the ODS Web site, contains information about federally funded research concerning dietary supplements. The International Bibliographic Information on Dietary Supplements (IBIDS), also available through the ODS Web site, contains bibliographic citations and abstracts from published international and scientific research concerning supplements. ODS is currently developing a database that will provide the composition of dietary supplements, as derived from analytical chemical data; this information will be combined with information from food composition databases to estimate nutrient intakes from foods and dietary supplements.

ODS has published an *Annual Bibliography of Significant Advances in Dietary Supplement Research* each year since 1999 (the most recent available year is 2005). These publications, which are available for download from the ODS Web site, include abstracts

of 25 scientific research papers selected as outstanding by a team of internationally recognized scientists, and citations of articles which appeared in previous annual bibliographies. ODS also maintains a listserv and publishes an electronic newsletter, *ODS Update*, which includes information about ODS activities and other projects and initiatives related to supplements.

The goals and initiatives of the ODS for the years 2004–09 are published in the document *Promoting Quality Science in Dietary Supplement Research, Education and Communication*, available for download from the ODS Web site. Goals cited in this document include evaluation of the role supplements play in disease prevention, fostering research evaluating the role of supplements in optimal performance, supporting research in the biochemical and cellular effects of supplements, promotion of the development of improved methodologies for studying supplements, and expanding educational and outreach activities to the general public, healthcare providers, and scientists.

SEE ALSO: Government Agencies; National Institutes of Health; Supplements and Obesity.

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Office of Minority Health

THE OFFICE OF Minority Health (OMH) was created in 1986 by the U.S. Department of Health and Human Services (DHHS). The mission of OMH is to develop health policies and programs to eliminate health disparities and thus improve the health of racial and ethnic minorities. OMH advises the Secretary of DHHS and the Office of Public Health and Science concerning public health activities that affect minorities, including American Indians and Alaska Natives, Asian Americans, African Americans, Hispanics, and Native Hawaiians and other Pacific Islanders.

Several committees and offices within OMH attend to specific tasks focused on improving minority health. The Advisory Committee on Mental Health was created in 1998 by the Health Professions Education Partnerships. It advises DHHS on the development of goals and specific program activities for OMH. The Regional Minority Health Consultants work for OMH within each of the 10 DHHS regional offices and focus on building networks of consumers and professionals who are working on minority health issues.

The OMH Resource Center, established in 1987, provides information and referrals on minority health issues for professionals, community groups, consumers, and students. The Resource Center also provides capacity development services to community-based organizations, including assistance in cultural competency, community outreach, communications, program design, and training. The Center for Cultural and Linguistic Competence in Health Care supports research, demonstrations and evaluations of innovative models to increase understanding of health risks and development of successful interventions for minority populations, and acts as a resource to address cultural and linguistic barriers to healthcare delivery and increase healthcare access for people with language barriers.

OMH has developed a number of campaigns to promote health prevention activities and draw attention to health disparities. Closing the Health Gap, which began in 2002, focused on increasing awareness of health issues among African Americans through a partnership with ABC Radio Networks. This campaign focused on providing health information to minority groups and stimulated interest in community health events including free health screenings and workshops.

Celebra la Vida con Salud (Celebrate a Healthy Life) is a program within Closing the Health Gap which helps link Latinos to health services and promotes prevention activities through a number of activities including placing public service announcements and programs focused on Latino health on radio programs and organizing a traveling health fair. Take a Loved One for a Checkup Day, also part of Closing the Health Gap, has been held on the third Tuesday in September since 2002, and encourages people to make an appointment for a health screening or help a friend or relative do the same. The Know What to Do for Life campaign focuses on reducing racial disparities in infant mortality through education, research coordination, and community risk reduction programs.

SEE ALSO: African Americans; Department of Health and Human Services; Ethnic Disparities among Obesity in Women; Ethnic Disparities in the Prevalence of Childhood Obesity; Ethnic Variations in Obesity-Related Health Risks; Hispanic Americans.

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Olfaction is involved with obesity in that smell and taste are closely linked and what drives hunger and taste.

Olfactory System

THE OLFACTORY SYSTEM is in charge of the sense of smell, aids in taste, and is involved with memory. Olfaction is the sense of smell, which is the detection of chemicals dissolved in the air. This body system involves the nose, nasal passages, and lining epithelial cells (some 50 million receptor cells in the human nose). These epithelial cells transfer odorant molecules in the air to the olfactory bulb, and eventually, to the brain, where they can be interpreted. The sense of smell is closely linked to the gustatory system, or sense of taste. In fact, the flavor in foods we consume is largely due to the sense of smell (about 95 percent), whereas a much smaller part of flavor in foods is conveyed by the sense of taste (about 5 percent). Because both sense of taste and smell are inextricably linked to the enjoyment of foods and beverages, they are also related to the development of obesity. Olfaction can drive hunger, particularly when one perceives a an aroma of a pleasurable food, such as fresh-baked cookies, or a meat on a barbeque grill. If either the biological systems that control smell or taste are damaged or function improperly, obesity can result.

The two main functions of the olfactory system are to distinguish one odor from another, and to make some judgment about the intensity or concentration of that odor. In addition to this function, the olfactory system also distinguishes "new odorants" that one comes across from background odors in the environment. Finally, an additional function of this system is to make connections between odors/aromas and memory, as the olfactory system innervates parts of the brain involved with this function.Indeed, olfaction and memory are linked—to what extent and in what chemical pathways are all still being elucidated in academic research. However, it is known that olfaction allows people to identify food, potential mates, predators, and provides both sensual pleasure, such as the smell of flowers, as well as warnings of danger such as spoiled food.

To accomplish all of these tasks, olfaction must first start with an odor. A chemical odorant must possess certain molecular properties to provide sensory information for the brain. The odorant must have water solubility, a sufficiently high vapor pressure, low polarity, and some ability to dissolve in fat and surface activity. These qualities are needed for the odorant to be dissolved in the air and for the olfactory system to detect it. The olfactory sense is able to distinguish among a potentially infinite number of chemical compounds.

The olfactory system involves the limbic system, amygdala, and cortex in the central nervous system (CNS). The amygdala is a small area in the brain that has two almond shaped sides with bundles of neurons located deep within the temporal lobes of the brain. Most research has shown that the amygdala processes memory and emotional reactions to life events and involve it with the limbic system. The limbic system is a set of structures throughout the brain that generate feelings, emotions, motivations, and help in learning and memory.

Lesions of the amygdala have long been known to produce overeating and obesity in some animals. There are possibilities that a sex difference in results can be

observed. For example, female rats with amygdala lesions may show a greater weight gain compared with male rats. However, the site of the lesion does play a role in how it affects eating habits. For example, some evidence reports that feeding behavioral changes can be due to a disruption of olfactory input altogether. Further research on the matter may be able to show more of a relation to the amygdale and obesity.

SEE ALSO: Appetite Control; Central Nervous System; Fat Taste; Flavor: Taste and Smell.

BIBLIOGRAPHY: Catherine Rouby, Benoist Schaal, and Daniele Dubois, eds., *Olfaction, Taste, and Cognition* (Cambridge University Press, 2005).

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Opioid Receptor

OPIOID RECEPTORS ARE a group of G protein coupled receptors which are the binding sites of opioids. There are three types of opioid receptors: mu (μ), kappa (κ), and delta (δ). Opioids act on the brain and body by attaching to specific opioid receptors, which are found in the brain, spinal cord, and gastrointestinal tract. When opioids attach to certain opioid receptors, they can block the perception of pain. Opioid receptors are also defined by their blockage with naloxane. Agonists for mu receptors include morphine; enkephalin is an endogenous opioid and is selective for delta receptors; and dynorphin is another endogenous opioid which is selective for kappa receptor.

Opioid receptors are found in high concentration throughout the brain; they are found in presynaptic terminals where binding with opioids inhibit release of neurotransmitters. Other areas in the brain with a high concentration of opioids and opioid receptors include periaqueductal gray, nucleus raphe magnus, nucleus reticularis, paragiganticellularis, and dorsal horn of the spinal cord especially lamina II known as substantia gelatinosa. Although the main action of opioids on opioid receptors is pain control, many other effects have been reported. One such effect is an increase in food intake upon stimulation. Morphine is associated with an increased food intake particularly stimulating fat consumption. Other opioid agonists, including mu, delta, and kappa agonists, also increase food intake while in the short term, opioid antagonists decrease food consumption. Mu receptors appear to modulate the efficiency of energy storage during high-fat diets through the regulation of energy partitioning. Mu receptors enhance intake of high fat via their stimulation within the nucleus accumbens in the brain.

Endogenous opioid peptides are also known to be involved in the modulation of feeding behavior. Endogenous opioids within the ventral striatum of the brain may participate in the mechanisms controlling preferences for highly palatable foods, especially those rich in fat. Opioid antagonists decrease the intake of foods, especially those rich in sugar and fat, without affecting hunger and satiety. It is thus suggested that the use of antagonists may result in a decrease in diet-induced obesity. Studies performed in rats suggest antagonists of the opioid receptors increase metabolic energy consumption, and reduce weight in obese rats while maintaining muscle mass. Thus, the opioid system could serve as target for the control of feeding behavior and management of obesity. However, the search for an effective antagonist that has an overall benefit to the patient and has minimal side effects continues.

SEE ALSO: Opioids; Palatability.

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Opioids

HUMANS CAN DEVELOP or be trained to like or dislike various foods by repeated exposure. The effect of this repeated exposure can lead to enhanced liking or even craving for a particular food. This is particularly true for sweet foods and fatty foods, the typical snack or junk foods, and appears to be centered in the nucleus

accumbens and ventreal pallidum where opioids appear to increase the liking or craving mechanism. It is also likely that interconnection with other brain regions enhances this effect. The hypothalamus regulates short-term and long-term dietary intake by the synthesis of neuropeptides that have an orexigenic, increased eating effect, or anorectic, decreased eating effect.

When food is consumed, peripheral neuropeptides such as cholecystokinin, ghrelin, peptide YY, amylin, and bombesin provide feedback to the central nervous system to control gastrointestinal motility, enzyme secretion, and nutrient absorption in the short term, while leptin and insulin regulate nutrient absorption and storage in the long term. This complicated mechanism may help in the selection of foods, for example, sweet or fatty foods to satisfy one's opioid receptors.

Animal research has found that in obese mice and rats that a diaryl ether derivative of nicotinamide, a mu-opioid receptor antagonist has an anorectic effect. This was particularly pronounced in animals with deficient mu-opioid receptors. Antagonism of kappa-opioid receptors in rats led to decreased food intake, decreased energy expended, decreased core temperature, and a slight reduction in body weight, but the weight loss was directly related to decreased intake not as increased activity or metabolic rate. Rat studies have demonstrated that a high-fat diet can induce obesity and is associated with increased hypothalamic mu-opioid receptors. When these rats were infused with a mu-opioid agonist, they had higher systolic blood pressure and heart rates, a common finding in obesity. When they were infused with a mu-opioid antagonist, there was no increase in blood pressure or heart rate. The same has been shown for kappa-opioid receptors.

Human research suggests that endogenous opioids have a role in glucoregulation and the pathogenesis of obesity. These may be linked to the metabolic changes seen in obesity, insulin resistance, and polycystic ovary syndrome. Altered opioid levels seen in hyperinsulinemia and the increased opioid activity in postmenopausal women combined with increased central body fat distribution further suggest a role for opioids and selected foods in the obesity epidemic.

SEE ALSO: Opioid Receptor; Palatability.

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Optifast

OPTIFAST IS A proprietary weight-loss program developed by the Novartis Nutritional Corporation, which is a member of the Partnership for Healthy Weight Management. Optifast is a comprehensive weight-loss and weight-maintenance program available only through licensed programs offered through hospitals and medical clinics in the United States and Canada. The Optifast program uses meal replacement formulas, medical monitoring, and lifestyle education from counselors and nutritionists to help people lose weight or manage their weight. Some Optifast clinics also offer bariatric surgery support, and although the Optifast system was developed for adults, some clinics also offer the Adolescent Weight Loss Education program.

The Optifast program is primarily intended for obese people, meaning those with a body mass index (BMI) over 30, and for overweight individuals with a BMI in the 25–29.9 range who have medical problems caused by their excessive weight. Individuals beginning the program receive an initial medical and lifestyle evaluation and ongoing medical monitoring. The dieting aspect of the Optifast system rests initially on the use of Optifast meal replacement materials, with the later reintroduction of normal food. For the initial phase of treatment, typically two to

four months, patients are assigned low-calorie diets, typically 800, 950, or 1,200 calories per day; the first category qualifies as a very-low-calorie diet. Dieters in the first phase consume Optifast liquid formula meal replacements and may also consume Optifast food bars, but not normal food, and attend a weekly group lifestyle modifications class. In the second phase of the program, which usually lasts four to six weeks, normal foods are reintroduced. The third phase of the program, weight maintenance, has no prescribed time period.

Novartis describes the use of its liquid formula diet as being like a "vacation from food" in which the dieter is provided with a nutritionally complete, calorically controlled diet which temporarily relieves them of the responsibility to make food choices. During this period, the individual is intended to lose weight and improve his or her health, both of which are self-reinforcing, and also to learn new activity and lifestyle habits which will be maintained when he or she resumes eating normal food. The maintenance diet followed by participants when food is reintroduced is typical of that recommended by many health experts, and is based on fruits and vegetables, grains, and low-fat protein.

The educational component of the Optifast system includes personal counseling from physicians, dieticians, and counselors, and small group presentations and discussions. Modules on many educational topics are provided, including stress management, shopping, maintaining health while traveling, and building support. Participants are also expected to increase their activity levels, and devising an appropriate exercise plan in conjunction with medical and behavioral counseling is part of the Optifast program.

In 2002, Novartis Nutrition began offering a Bariatric Surgery Support program which is available at some Optifast clinics. This program offers the Optifast meal replacement and education program to people scheduled for bariatric surgery for six to eight weeks prior to surgery. A major advantage of following this program is that it promotes presurgical weight loss, which can improve the patient's health, reduce risk of complications, and promote faster healing after surgery. In addition, use of a liquid diet before surgery may make it easier to adapt to the restricted diet necessary after bariatric surgery, and to identify problems patients may encounter in following such a regimen. In addition, Novartis produces a number of products designed for people who have had bariatric surgery, all sold in the Optisource product line; these include a vitamin and mineral supplement, a nutrition bar, and a high-protein drink.

Because Optifast was one of the first comprehensive, commercial weight-loss programs, it has been extensively studied since its inception in 1974. As with any diet program, results must be interpreted with caution because they typically represent only results from people who stuck with the program, who were probably more successful than those who dropped out.

Wadden, et al., evaluated 517 Optifast participants at 18 sites, and found that just over half completed the 26-week Optifast core program. Men who did complete the program lost an average of 70 pounds and women who completed it lost an average of 48 pounds. A one-year follow-up study of 118 patients found that they maintained on average 33 pounds of their original 54 pound average weight loss. A separate study by Wadden and Frey (1997) found that three years after completion of the Optifast Core Program, 73.3 percent of men and 55.1 percent of women maintained at least a five-percent reduction in their initial weight, which is the minimum proposed for a successful weight-loss program by the Institute of Medicine.

SEE ALSO: Body Mass Index; Cost of Medical Obesity Treatments; Liquid Diets; Low-Calorie Diets; Very-Low Calorie Diets.

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Orlistat (Xenical)

OBESITY POSES A serious threat for health, being a risk factor for the development of heart diseases, Type 2 diabetes mellitus and associated insulin resistance (metabolic syndrome), dyslipidemia, tumors, and reproductive function failure. The increasing prevalence of obesity and obesity-related disorders makes it a key discussion and research topic in several disciplines, including health and social sciences as well as in other disciplines such as physical education. Treatment of overweight or obese patients with drugs may be an important component of weight management, along with diet, exercise, and behavior modification, because many are unable to achieve a meaningful weight loss with lifestyle measures alone.

Orlistat (Xenical) is a drug used in the management of obesity, as an adjunct in weight management. It is indicated for obese patients with body mass index (BMI) of at least 30 and patients with BMI of at least 27 in the presence of other risk factors for arteriosclerosis such as high blood pressure, diabetes, and elevated blood cholesterol or triglycerides.

Orlistat promotes weight loss by preventing the digestion and absorption of dietary fat. It works by inhibiting the gastric and pancreatic enzyme known as lipase, which breaks down triglycerides in the intestine. Because the inactivated enzymes are unable to hydrolyze (split) the dietary fat in the form of triglycerides into absorbable free fatty acids and monoglycerides, the unabsorbed fat is then excreted as such in the stools.

Orlistat was approved for use in the United States by the Food and Drug Administration (FDA) in 1999. In most areas, orlistat is available by prescription only. Approval of an over-the-counter (OTC) formulation to be marketed under the name Alli™ was granted to the manufacturer in February 2007. This OTC formulation is now available only at half the dosage of the prescription medication.

Orlistat is taken by mouth three times daily, one hour after or during a meal containing fat. It is available in the form of a capsule at a dose of 120 milligrams. Doses greater than 120 milligrams, three times daily, have not been shown to have any beneficial effect or increase the weight loss. If a meal is missed occasionally or consumed without fat, the dose of orlistat can be omitted. Orlistat must be used with a nutritionally balanced, reduced-calorie diet that contains no more than 30 percent calories from fat.

Orlistat appears to be very effective when it comes to weight loss. Studies on orlistat have shown that individuals receiving orlistat lost significantly more weight compared to placebo. However, a significant number of subjects regained the weight after they stopped using orlistat. In addition to its well-established efficacy in achieving moderate weight loss, orlistat has been shown to improve blood sugars in obese individuals with Type 2 diabetes mellitus as well as some features of metabolic syndrome. Recent data have also suggested that orlistat has been shown to improve hormonal and metabolic profile in women with polycystic ovarian syndrome after six months of treatment, independently of BMI changes.

Orlistat is generally well tolerated. Most of the side effects seen with orlistat are related to the reduced absorption of fat from the diet. These include oily spotting on the underwear, abdominal pain or discomfort, flatulence, fecal urgency, fatty or oily stools, oily evacuation, and increased defecation and stool incontinence. To reduce the occurrence of these side effects, meals should contain no more than 30 percent fat. Nongastrointestinal side effects include headache, back pain, arthritis, upper respiratory infection, menstrual irregularities, fatigue, and anxiety. Patients with a history of calcium oxalate kidney stones may develop increased levels of oxalate in their urine, which may increase the risk of kidney stones when receiving orlistat. Individuals who should not take this drug include those who have binge eating disorder, bulimia nervosa, malabsorption syndromes and reduced gallbladder function, for example, after cholecystectomy, and in individuals with certain kidney problems.

Fat-soluble vitamins such as vitamins A, D, E, K, and beta-carotene are not absorbed and are eliminated in the stool in increased amounts along with the fat when orlistat is taken. Therefore, patients on orlistat should take a multivitamin containing fat-soluble vitamins at least two hours before or several hours after the orlistat to ensure adequate amount of the vitamins available for absorption. Besides the dietary supplements as mentioned above, orlistat may interact with other drugs such as warfarin, cyclosporine, pravastatin, and drugs used to treat diabetes. Patients on warfarin therapy who begin orlistat must have their blood clotting monitored closely because orlistat may cause levels of vitamin K to decline, which plays a role in the blood-thinning effect of warfarin.

Safe use of orlistat during pregnancy has not been established and hence it is not recommended during pregnancy or when trying to get pregnant.

CONCLUSIONS

Appropriate management of obesity and prevention of obesity-related diseases is of great importance for public health. Multiple cardiovascular and metabolic risk factors, including Type 2 diabetes mellitus, hypertension, dyslipidemia, and cardiovascular disease are increased by excess weight, particularly abdominal obesity. Orlistat may be a useful adjunct to lifestyle measures and has the potential to significantly contribute to weight reduction and risk factor improvement for overweight and obese individuals.

SEE ALSO: Fenfluramine; Pharmacological Treatment of Childhood Obesity; Rimonabant; Sibutramine (Meridia).

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Osteoarthritis

OSTEOARTHRITIS (OA) IS the most common type of arthritis. It can affect any joint in the body, but the hips, knees, hands, and spine are frequently involved. Among adults over 60 years of age, 17 percent of men and 30 percent of women suffer from the debilitating effects of the disease. It results from degeneration of the joint cartilage and changes in the surrounding bones, muscles, and soft tissue. Obesity and osteoarthritis coexist in a large segment of the population. Obesity plays an important role in the etiology and progress of osteoarthritis because of the impact on weight-bearing joints. Management of obesity significantly improves symptoms of osteoarthritis and is a key element in the treatment plan.

TYPES OF OSTEOARTHRITIS

Primary osteoarthritis is the result of age-related degeneration in the joint. This type is seen in the elderly and involves multiple joints. What makes the joint cartilage susceptible to faster breakdown is the progressive loss of water content along with changes in the protein composition. Secondary OA is seen in younger individuals who have previously had a trauma or surgery. This type may affect just one or two joints. Younger individuals with congenital or developmental disorders of the bone or joint can develop symptoms of OA before the age of 40.

RISK FACTORS

Several factors predispose to osteoarthritis. Genetic factors have been noted to be important in hip osteoarthritis and hand joint arthritis in women. Being obese or overweight is strongly related to the risk of having knee arthritis. Some studies have also linked obesity to hip and hand arthritis, although the association is not as strong. Having a higher body mass index (BMI greater than 25) before the age of 30 increases one's chance of developing arthritis of the knee by age 60. In fact, the risk is threefold higher among overweight and obese young men and nine times among women as compared to their normal weight peers. Other risk factors for developing OA are previous joint surgery, injury to a joint, diabetes, and excessive strain as a result of work.

HOW OBESITY CAUSES OSTEOARTHRITIS

Even though OA is often thought of as "wear and tear" arthritis, there is a complex interaction between mechanical and chemical factors within the joint. The joint cartilage in overweight individuals is subjected to greater mechanical stress and can undergo faster breakdown. This is especially true in the weight-bearing joints with a more marked effect on the knees than the hips.

Obese individuals have a higher incidence of OA in joints that are nonweight-bearing as well, for example, the hands. This has led to theories of chemical substances playing a role. These chemicals called cytokines are released from the fat cells and can alter

the synthesis and breakdown of the proteins that support the cartilage. High levels of insulin as seen in individuals with diabetes may also be important in the evolution of OA.

SYMPTOMS OF OSTEOARTHRITIS

The most common symptom of OA is pain. It is worse with activity and relieved with rest. Pain can lead to significant impairment in the ability to carry out daily activities. With long-standing disease, however, pain may be absent, although there may be significant deformity. Slight stiffness is another common complaint, often experienced in the morning or after a prolonged period of rest to the joint.

Long-standing disease resulting in severe malformation of the joint may require surgery. Often, there is a loss of stability and a feeling of the joint "giving way." Osteoarthritis of the hands can also lead to nodules over the knuckles.

DIAGNOSTIC TESTS

Doctors make the diagnosis of OA based on symptoms of pain along with certain findings on physical exam. X-rays help confirm the diagnosis. Occasionally, it may be necessary to aspirate fluid from the joint to differentiate OA from other types of arthritis when the diagnosis is unclear.

TREATMENT OF OSTEOARTHRITIS

The treatment of osteoarthritis depends on the severity of symptoms. For mild OA, topical analgesic creams that contain the compound capsaicin may be used. Oral medications for mild to moderate disease include acetaminophen, NSAIDs (nonsteroidal antiinflammatory drugs), and opioids. The popular NSAIDs are ibuprofen and naproxen. COX-2 inhibitors are a class of NSAIDs that are less likely to produce stomach ulcers. Celecoxib (Celebrex®) is available in the U.S. market, whereas rofecoxib and valdecoxib (Bextra®) have been withdrawn after reports of increased heart attacks and strokes among users.

Medications such as steroids and hyaluronic acid are often given as direct injections into the joint to provide pain relief. Physical therapy and exercises to strengthen the anterior thigh muscles are important. In overweight individuals, studies have shown that the progression of symptoms and deterioration of joint function can be remarkably slowed down by weight loss.

SURGICAL TREATMENT OF OA

Apart from relieving pain, treatment of OA involves correcting malformations and factors that affect joint stability. Surgery is done in patients who continue to suffer from symptoms despite maximal medical therapy. It may involve joint replacement, joint fusion (which is rarely done now), osteotomy (removing a part of the bone within the joint), or arthroscopic removal of debris.

Arthroscopy is a procedure by which a small fiberoptic telescopic device is inserted into the joint. This allows the surgeon to directly visualize the inside of the joint and remove broken pieces of bone or cartilage. Outcome after joint replacement surgery for osteoarthritis is worse in obese individuals.

WEIGHT LOSS IN OSTEOARTHRITIS

It is well documented that weight loss can lead to significant reduction in the pain and overall improvement in the quality of life. The impact of exercise may lead to further stress on the joint. Therefore, prior to an exercise program, weight loss can be achieved by dietary restrictions and medications for weight loss if prescribed by a doctor.

Strengthening exercises of the anterior thigh muscles (called the quadriceps muscles) help to improve knee function. Water therapy exercises, which are pool based, provide much of the benefits of regular exercise without as much impact on the spine. It provides a gentler environment for stretching and can eliminate the effect of gravity. Tai chi, an oriental form of exercise, and yoga, an ancient form of exercise that originated in the Indian subcontinent, are other exercise modalities that can provide relief in individuals with OA.

CONCLUSION

Obesity and osteoarthritis are common and often coexistent problems in a large number of people in the United States and worldwide. The treatment of obesity is key in the management of obese patients who suffer from OA. It not only helps ameliorate the symptoms of joint pain in OA, but can also slow the worsening of joint degeneration. Weight loss and exercise should always be part of the care plan of osteoarthritis.

SEE ALSO: Obesity as a Public Health Crisis; Prevalence of Childhood Obesity in the United States.

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Osteoporosis

OSTEOPOROSIS IS A systemic skeletal disorder characterized by a low bone mineral density (BMD) and microarchitectural deterioration of bone tissue, leading to enhanced bone fragility and susceptibility to fracture. Osteoporosis may be asymptomatic for many years and does not manifest until later in life. The first symptoms are usually pain due to a fracture, postural changes, and/or loss of height due to spinal compressions. These fractures typically occur in the hip, spine, and wrist. Fractures due to osteoporosis are a major public health hazard with high morbidity, mortality, and social costs.

The etiology of osteoporosis involves many factors. The role of the skeleton as a mineral storehouse is dependent on the proper functioning of a number of systemic or circulating hormones, such as parathyroid hormone and calcitonin that respond to changes in blood calcium and phosphorus. If serum calcium or phosphorus is low, the removal of these minerals from bone will ultimately weaken it. Many factors can interfere with the development and maintenance of a healthy skeleton including genetic abnormalities, which can produce small, weak bones, or bones that are too dense. Many hormonal disorders can affect the skeleton. Nutritional deficiencies can result in the formation of poorly mineralized bone or excessive loss. Lack of exercise, immobilization, and smoking are also be detrimental to bone.

Measurement of BMD is currently the single most important screening determinant for low bone mass, fracture risk, and to identify individuals who are candidates for therapeutic intervention. Bone densitometry studies show that for every decrease in BMD of 1 standard deviation (SD), the relative risk of fracture increases 1.5- to threefold. The World Health Organization has established BMD diagnostic guidelines for the interpretation of bone mass measurement in Caucasian postmenopausal women. Dual energy X-ray absorptiometery (DEXA) measures BMD and is expressed as "T scores"—the number of SDs above or below average in young normal adults (gender-matched).

- Normal bone mass: T score at least –1.0
- Low bone mass (or osteopenia): T score –2.5 to –1.0
- Osteoporosis: T score less than –2.5

However, it is recommended that low BMD should not be viewed as the only indicator of fracture risk and that other factors should be considered such as age, family or personal history of fracture, low body weight, race, lifestyle (i.e., history of low calcium intake or sedentary activity), medication use (i.e., thyroid hormone, glucocorticoids, etc.), and the presence of a previous fragility fracture (fracture that occurs spontaneously or following a minor trauma, such as a fall from standing height or sitting position). Central (hip and spine) measurements by DEXA should be used for both risk assessment and follow-up, as they provide the most accurate and precise measurements of BMD.

Patients should have an adequate total intake of calcium (1,200 mg per day for postmenopausal women) and of vitamin D (at least 400 to 800 IU per day) and participate in weight-bearing exercise—interventions that are safe and inexpensive. Osteoporosis drugs can substantially reduce the risk of fracture in patients at high risk for osteoporosis on the basis of BMD and other factors. Calcium and vitamin D are recommended as mandatory adjunct therapy to the main pharmacological interventions (antiresorptive and anabolic drugs).

EFFECT OF EXCESS WEIGHT ON BONE

Obesity is generally protective against osteoporosis, with a direct relationship between body weight and bone mass. A low body weight in older individuals is a major risk factor for fracture. Mechanisms that may be contributing to the increased BMD in obesity in-

Moderate weight reduction in men (middle aged) has not been shown to cause significant bone loss, yet weight loss in elderly men (around 70 years) is an important predictor of bone loss and increased incidence of osteoporosis.

clude greater or different mechanical stimulation and the hormonal/metabolic environment. The increase in bone mass with obesity may simply be due to increased mechanical loading. The loads on bone are derived from the forces imposed by the muscle tissue. Bones adapt to stronger or bigger muscles associated with obesity because there is increased weight bearing with daily activity.

In addition, the altered hormonal profile of the obese may also play an important role in regulating bone. In obesity, there is the secretion of bone-active hormones (i.e., estrogens, leptin, and adiponectin) from the adipocyte, the gut (i.e., ghrelin that stimulates growth hormone), and the pancreas (including insulin and amylin). In addition, obese individuals show lower levels of serum 25-hydroxyvitamin D. There is reported secondary hyperparathyroidism in the morbidly obese. Chronically altered levels of certain cytokines in obesity may also influence bone mass.

WEIGHT REDUCTION AND BONE

Weight loss increases bone turnover markers and has been shown to reduce bone mass. Calcium intake typically decreases with energy restriction and Calcium supplementation can suppress a rise in bone turnover during energy restriction in older women. In addition, a reduced Calcium absorption during caloric restriction may contribute to increased bone mobilization and loss. It is likely that multiple endocrine changes during weight reduction contribute to bone loss, but a decrease in estrogen levels appears as one of the most relevant candidates. In addition, a decrease in calcium absorption during energy restriction may play a role in reducing calcium availability, raising parathyroid hormone to ultimately result in bone loss. Finally, reduction in weight bearing due to weight loss or disuse are likely important regulators of bone metabolism during weight loss, and an increase in physical activity has been shown to be beneficial to bone.

Lean or overweight women who are close to menopause also respond to weight reduction by showing significant bone loss. Weight loss studies in premenopausal women show either a small decrease in bone mass, or no bone changes in controlled trials. However, more severe weight loss (at least 14 percent) in all age groups results in significant bone loss. Moderate

weight reduction in men (middle aged) has not been shown to cause significant bone loss, yet weight loss in elderly men (about 70 years) is an important predictor of bone loss and increased incidence of osteoporosis. In summary, while there seems to be agreement that bone loss occurs with weight loss in older women and in elderly men, it remains unclear whether there is any detriment to bone health in younger individuals. Hence, weight reduction should be encouraged to reduce the comorbid conditions associated with obesity in all individuals with advice to participate in physical activity and consume adequate calcium and vitamin D in order to minimize the risk of osteoporosis.

SEE ALSO: Adipocytes; Calcium and Dairy Products; Cytokines; Dual Energy X-Ray Absorptiometry; Ghrelin; Hormones; Leptin; Menopause.

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Ovarian Cancer

OVARIAN CANCER IS the second most common gynecological cancer among women. Because the tumor is not detected until late in its development, usually not until after it has spread beyond the ovary, it is difficult to treat. Thus, ovarian tumors are often fatal and account worldwide for almost half of the deaths from cancer in the female reproductive tract. Tumors of the ovary are classified by the cell type of origin—surface epithelium, germ cells, and stromal tissue. Metastatic ovarian cancer is also common. Most (around 90 percent) ovarian tumors arise from the ovarian surface epithelium (OSE) and produce relatively mild symptoms until the disease is far advanced. Symptoms include abdominal pain and distention, urinary and gastrointestinal tract symptoms resulting from compression by the tumor or invasion into adjacent organs, and vaginal bleeding. At late stages, tumors cause progressive weakness and unintended weight loss. Carcinomas extending beyond the ovary tend to seed the peritoneal cavity, covering the serosal surface diffusely with small (0.1–0.5 cm) surface implants and causing massive fluid accumulation (ascites). The five-year survival rates are poor (30 to 40 percent), thus, prevention and early detection are of high priority.

Family history of ovarian cancer is a major risk factor in 5 to 10 percent of cases. Risk increases from 1.4 percent in the general population to 8 percent in women with two affected first-degree relatives. Strong associations with familial breast cancer, and a lesser association with familial colon and endometrial cancer are known. Biomarkers for early detection include CA-125, a high molecular weight glycoprotein present on more than 80 percent of epithelial ovarian tumors, and osteopontin, expressed at high levels in ovarian cancer patients. Proteomic assays that examine the patterns of circulating proteins in patients compared with nonaffected controls are being conducted, but require validation before they can be used as screening modalities. Current screening strategies for at-risk women, assessed by positive family history, include testing for mutations in germ line tumor suppressor genes such as BRCA1 and BRCA2. Prophylactic removal of the ovaries is current practice for women at high risk, but the impact of these strategies on ovarian cancer death rate has not been determined.

The processes leading to ovarian tumor development are similar to processes for cancer initiation, promotion, and progression described in the "Obesity and Cancer" entry in this encyclopedia. The stationary ovarian surface epithelium (OSE) is a flat to cuboidal layer of epithelial cells with few distinguishing attributes. While on the ovarian surface, the OSE transports materials to and from the peritoneal cavity and takes part in cyclical follicle rupture and ovarian surface repair. Sometimes, in the process of follicle rupture, epithelial cells become entrapped in the underlying stroma. Through the years, after many cycles of rupture and repair, epithelial cells can become incorporated into the stroma. Normally, trapped epithelial cells differentiate and become incorporated into the ovarian stroma as stromal fibroblasts. Some epithelial

cells are unable to differentiate into fibroblasts, retain their epithelial phenotypes and aggregate within the stroma as inclusion cysts. While on the ovarian surface, epithelial cells do not express membrane-bound markers; however, epithelial cells in inclusion cysts are known to express a variety of markers including CA-125. It is these trapped epithelial cells that have a high potential to transform into cancer cells.

The complex processes that regulate cyclic follicle rupture and repair involve pituitary gonadotropins and sex hormones, primarily estrogens and androgens, as well as insulin and insulin-like growth factors and inflammatory mediators. Available research suggests that imbalance of regulatory mediators may increase risk for ovarian cancer. Epidemiological studies have shown that nulliparous women are at increased risk for ovarian cancer. The risk decreases in multiparous women, women 40–59 years of age who have taken oral contraceptive agents, and those who have undergone tubal ligation.

While the observed reduction in risk due to pregnancy and oral contraceptive use could relate to reduction in ovulation with subsequent reduced ovarian damage, observed risk suggests that other factors are involved. Dr. Harvey Risch conducted a comprehensive review of evidence from epidemiologic and experimental studies and suggested that pregnancy and interruption of ovulation alter exposure of the epithelial cells to sex hormones, especially androgens and progesterone, and that this altered hormonal exposure is implicated in the pathogenesis of ovarian cancer.

Risch presents evidence that ovarian epithelial cells, both on the surface and within stromal inclusion cysts in the premenopausal women, are normally exposed to higher plasma concentrations of androgens, including androstenedione, testosterone, and dehydroepiandosterone, than estrogens such as estradiol and estrone. Androgens are also the principle sex steroid synthesized in ovarian follicles. Only when a follicle enlarges and prepares for ovulation does it convert from androgen to estrogen synthesis. Thus, epithelial cells, especially those in stromal inclusion cysts, are exposed to high concentrations of paracrine and endocrine sources of androgens. Postmenopausal ovaries are thus highly androgenic. Epithelial cells are also known to express androgen receptors. Although androstenedione only weakly binds, epithelial cells are able to convert this steroid to testosterone, which does bind to receptors. Androgen binding has been shown to sustain growth in ovarian epithelial cells in culture, and in animal models, testosterone stimulates growth of ovarian epithelial tumors.

Epidemiologic evidence also supports the relationship of high androgen exposure and ovarian cancer. Of interest is the increased risk for ovarian cancer seen in patients diagnosed with polycystic ovary syndrome, known to result in elevated androstenedione and testosterone levels. Reduced risk during pregnancy may result from the massive progesterone production supplied first by the ovary, and later the placenta. During pregnancy, the placenta extracts adrenal androgens as substrate for estrogen conversion. Oral contraceptive agents appear to reduce risk relative to their ability to suppress ovulation and reduce androgen exposure.

The risk for ovarian cancer due to obesity is less well defined. While both insulin and IGF enhance normal ovarian progesterone production and stimulate ovarian aromatase to convert androgens to estrogen, obesity is associated with the development of insulin resistance, hyperinsulinemia, and decreased production of sex hormone-binding globulin with subsequent reduced capacity to bind plasma testosterone. Thus, even moderate obesity is associated with increased serum total and free testosterone. Central adiposity, assessed by waist–hip ratio and associated with visceral obesity, may confer greater risk due to its association with increased androgen exposure.

Studies relating body mass index as indicator of obesity are equivocal; however, few studies have controlled for parity, visceral obesity, and risk from both plasma androgen exposure and ovarian androgen synthesis. Investigations controlling for these factors, as well as known genetic mutations that increase risk for ovarian cancer, and the presence of biomarkers are urgently needed.

SEE ALSO: Obesity and Cancer; Polycystic Ovary Disease; Prevalence of Obesity in U.S. Women; Uterine Cancers.

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Ovarian Cysts

OVARIAN CYSTS ARE fluid-filled sacs usually found of the surface of an ovary. Ovarian cysts occur in 30 percent of females with regular menstrual cycle and in 50 percent of females with irregular menstrual cycles. There are many different types of cysts, each with a different underlying cause. The most common type is a functional cyst which forms during the normal menstrual cycle. Two types of functional ovarian cysts may develop; during the first two weeks of the cycle follicular, cysts occur and corpus luteal cysts occur in the later half of the cycle. The cysts usually disappear within 8–12 weeks and are common in women of childbearing age.

The other types of cysts include endometriomas, cystadenomas, dermoid cysts, and polycystic ovaries. Endometriomas are cysts formed when endometrial tissue grows in the ovaries. Cystadenomas develop from cells on the outer surface of ovary. Cystadenomas of the serous type are filled with watery fluid, while the mucinous is filled with sticky, thick fluid; both types are usually benign. If a neoplasm begins in a germ cell, a dermoid cyst may develop. Dermoid cysts are structures that are filled with pieces of bone, teeth, hair, and skin; they can become malignant. Polycystic ovaries are caused by hormone imbalances which causes multiple functional cysts to develop.

Ovarian cysts can be divided into three main classifications: functional, benign, and malignant. However, most ovarian cysts are benign in nature and are usually asymptomatic. When symptoms do occur, they include pain, particularly if it is associated with rupture (may cause peritonitis and shock), perforation into nearby structures, torsion of nearby structures, for example, to fallopian tube, and hemorrhage. Pain may radiate but is usually located within the pelvis. Strenuous activities, such as exercise or sexual intercourse (dyspareunia), may precede cyst rupture. The pain may become worse during bowel movements and during the course of the menstrual cycle. Other symptoms of ovarian cysts include abnormal uterine bleeding and abdominal enlargement, breast tenderness, change in frequency of urination due to pressure on bladder, nausea, vomiting, fatigue, increased levels of testosterone, hirsutism, and infertility. Risk factors for developing cysts include obesity, early menarche, hypothyroidism, and also the use of tamoxifen therapy.

Treatments for cysts depend on the size of cyst and symptoms experienced. Treatment for pain includes pain relievers such as nonsteroidal inflammatory drugs. Obesity is a significant risk factor for development of cysts particularly in women suffering from polycystic ovarian syndrome. These women have a slower metabolic rate than their normal counterparts and, hence, face difficulty in losing weight. Upper abdominal obesity in these patients can lead to insulin resistance and hence the resulting hyperinsulinemia leads to altered hormone metabolism and the eventual development of the disease.

In patients with polycystic ovaries who are obese, dietary restriction generally improves endocrine-metabolic parameters such as a decrease in free testosterone, hence decreasing the risk of hirsutism. Weight loss in patients with the disease who are obese is associated with a return of ovulatory cycles in 30 percent of women. A daily decrease in 500–1,000 calories deficit and 2.5 hours of exercise per week can cause ovulation in women.

The type of diet of an individual can influence the development of cysts. It is reported that women who have a high intake of beef and cheese in their diet experience a greater frequency of ovarian cysts. In contrast, the intake of green vegetables reduces the frequency and is also protective. Patients are advised to eliminate caffeine, alcohol, reduce carbohydrate intake, and increase intake of vitamins A and B. Oral contraceptive pills are reported to have some use in preventing formation of functional cysts. Limit of strenuous activity reduce risk of cyst rupture. Surgery is employed in serious cases to remove the cysts. The prognosis of ovarian cysts is variable and depends on

the type and size of tumor, associated complications and, patient's age.

SEE ALSO: Menstrual Problems; Polycystic Ovary Disease.

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Overall Diet Quality

THE INTRIGUING ASSOCIATIONS between diet and health have been the focus of research for many years. One way of looking into these associations is to examine the individual nutrients such as vitamins (e.g., vitamin A or C), minerals (sodium, calcium, etc.), fats, proteins, or carbohydrates and their relationships with various health conditions or diseases. However, people consume these nutrients in a number of different combinations rather than eating each nutrient separately. Interactions of various nutrients can have a different influence on the health status, and smaller, undetectable effects of various nutrients may add up to a much larger influence on health and disease. Further, taking into consideration as many potentially important aspects of a diet is likely to produce a more accurate profile regarding the dietary intake patterns and their associations with health status. To that end, various methods to examine the overall dietary patterns and quality have been developed.

Before looking into the measures of dietary quality, criteria to determine the dietary quality must be discussed. Interestingly, there is not a single criterion that is shared by various measures of dietary quality. Instead, a collection of factors such as diets meeting the specific nutrient needs, or dietary patterns that are likely to prevent certain diseases or that comply with generally recommended dietary guidelines are used to determine dietary quality. In some instances, these criteria are relatively subjective because researchers have to group a variety of foods into more manageable, lesser number of categories to be able to conduct statistical analyses. Indices to measure dietary quality are often calculated by using the predetermined nutrition-related criteria mentioned above. Alternatively, dietary intake patterns are identified by using statistical procedures based on the collected dietary intake data in a given population, and then further analyses are performed to determine the diet quality of each pattern. Recently, some researchers also started using a combination of these two approaches.

In general, as highlighted in a review by Ashima Kant, overall dietary quality measures are based on either nutrients, foods/food groups, or a combination of nutrients and foods/food groups. The following sections provide a brief overview of various measures of dietary quality.

MEASURES OF DIETARY QUALITY BASED ON NUTRIENTS

Most of the earlier indices were based on nutrient intakes. Some of these indices include nutrient adequacy ratio (NAR), mean adequacy ratio (MAR), dietary rating, nutritional score, diet quality scores, DINE score, food quality index, and nutritional quality index. The NAR uses the ratio of a nutrient intake in relationship to the recommended dietary allowances (RDAs, which are national recommendations for various nutrients), and the MAR is the average of a collection of NARs. Another approach similar to this one is the dietary rating, which rates the nutrients separately but by the same scale (excellent, good, fair, etc.) and comes up with an overall rating for the diet.

Several versions of nutritional or diet quality scores use a fixed cutoff point (e.g., two-thirds) of RDAs, and some indices incorporate additional factors such as carbohydrates, fiber, energy (e.g., DINE score), alcohol, and various types of fat intakes to determine the diet quality.

Food quality and nutritional quality indices take into account the nutrient density (amount of nutrients for a given level of energy) of the foods and diet, respectively.

MEASURES OF DIETARY QUALITY BASED ON FOODS OR FOOD GROUPS

Dietary diversity score (DDS) and recommended foods score (RFS) are examples of indices that use foods and/or food groups to determine the dietary quality. The scores are assigned depending on the

consumption of foods from five major food groups (fruits, vegetables, meat, dairy, and grains) at least at the nationally recommended amounts and the compliance with the recommended dietary guidelines such as consuming low-fat dairy, lean meats, and whole grains.

Aside from using the nationally recommended guidelines in determining a dietary quality score or index, another method of examining dietary quality involves using statistical methods such as factor analysis or cluster analysis to come up with specific dietary intake patterns. This method is distinctively different from the previously mentioned indices because dietary intake patterns are formed by using the dietary intake data collected in the studied population. In other words, data determines the intake patterns versus index scores are based on predetermined nutrition knowledge and guidelines. Hence, detected dietary patterns are specific to each population, and they may be quite different for another population.

A prudent approach to weight loss is characterized by greater intakes of fruits, vegetables, legumes, fish, poultry, and grains.

Factor analysis is one of the statistical techniques to determine dietary intake patterns. This method primarily identifies the foods that correlate well with one another (e.g., foods that are consumed together) and provides newly formed groups of foods. Because the number of food items that can be entered into such an analysis is limited, researchers may have to subjectively presort some of the foods such as combining similar vegetables in one category, but once they are entered into the statistical model, the food groupings are formed objectively based on their correlations with one another. After this point, it is up to the researchers to look at the foods in each group and to decide what that group represents. This approach enables researchers to determine the types of overall dietary intake patterns that may further be examined in relationship to disease risk or other health outcomes in determining what constitutes a higher quality diet.

One example of this approach is the "prudent" and "Western" dietary intake patterns that are found to be related to chronic disease risk. The prudent pattern was characterized by greater intakes of fruits, vegetables, legumes, fish, poultry, and whole grains, while the Western pattern was characterized by higher intakes of red and processed meats, sweets, desserts, fried foods, and refined grains. People who were in the top 25 percentile of the Western dietary pattern score were significantly more likely to develop Type 2 diabetes compared to those who were in the lowest 25 percentile of the scores for this type of a diet.

Another statistical approach that is used to determine the dietary intake patterns is cluster analysis. Differently than factor analysis, cluster analysis identifies groups of people, rather than foods, who are similar in their food intake patterns. Then, the dietary intakes of these distinctive groups of people can be analyzed to determine how the diet quality differs between groups, or whether the intakes of these groups fit into any of the established dietary guidelines for public recommendation purposes.

A newer statistical approach in this area is using reduced rank regression (RRR) or maximum redundancy analysis. This method allows for combining the prior knowledge about the disease-related nutrients with statistical prediction of food intake patterns, and

further research is needed to explore the potential of this technique.

MEASURES OF DIETARY QUALITY BASED ON NUTRIENTS AND FOODS

Indices such as Diet Quality Index (DQI), and Diet Quality Index-International (DQI-I), which is designed to be used internationally, utilize both nutrients and food groups to assign a score toward the overall quality of the diet. DQI-I takes into account the variety across and within protein sources, adequate intakes of fruits, vegetables, grains, and fiber, moderation of total fat, saturated fat, cholesterol, sodium, and foods with a low nutrient density (high in energy and low in nutrient content), and the overall balance of macronutrients (carbohydrates, proteins, and fats) in the diet.

Another index that uses both nutrients and foods is the Healthy Eating Index (HEI). The original HEI, which was developed by the United States Department of Agriculture, assigns equally weighted scores for consuming the five major food groups from the Food Guide Pyramid for meeting the intake recommendations for total fat, saturated fat, sodium, and cholesterol, and for consuming a variety of foods. The HEI was revised in 2005 to comply with the new Dietary Guidelines for Americans. This revised version uses a nutrient density approach by assigning scores based on calories.

For example, maximum score for fruit intake is assigned for eating 0.8 cup equivalent or more fruits per 1,000 kilocalories consumed. The 2005 HEI also incorporates the dietary variety into each food group by assigning separate scores for whole fruits in the fruit group, dark green and orange vegetables and legumes in the vegetable group, and whole grains in the grains group. In addition to the categories for fruits, vegetables, milk products, meats, oils, saturated fats, and sodium, a new category for solid fats, alcohol, and added sugars (SoFAAS) is also included in the 2005 HEI. The SoFAAS category carries a higher scoring weight relative to the individual weight of other categories within the index.

The alternate HEI (AHEI) is another diet quality index, which includes scoring for fruits and vegetables, cereal fiber, nuts and soy protein, trans and other types of fat intakes, the ratio of white to red meat, and multivitamin and alcohol use.

STRENGTHS AND LIMITATIONS

Using diet quality indices that are based on a single or a collection of nutrients have certain limitations. For example, if energy intake is used as the measure of quality, people who eat greater amounts of foods might end up having a better dietary quality. On the other hand, if a large proportion of energy intake is coming from alcohol and other drinks and foods with a low nutrient density, people would be misclassified as having a higher quality diet.

Usefulness of certain nutrient-based dietary quality measures such as the MAR is limited because nutrients with high intake levels and those with low intake levels can even out when averaged into one score for the overall diet. Also, nutrient-based indices usually include only a limited number of nutrients. Therefore, they may not completely reflect the influence of myriad of other nutrients and nonnutrient components of foods, and they usually are not powerful tools to reflect the overall diet quality. However, using overall dietary quality indices or scores usually produce more steady associations with disease-related outcomes in comparison to examining these relationships using single nutrients or foods.

One of the limitations of determining the dietary intake patterns using factor or cluster analyses comes from the fact that these techniques are data driven. Although some aspects of dietary intake may be the same across different populations, other dietary patterns are likely to differ between various populations depending on their demographic, socioeconomic, cultural, and ethnic characteristics. Therefore, generalizability of the detected dietary intake patterns are limited. Possibly due to these potential influences, dietary patterns determined by factor and cluster analyses usually do not explain a large proportion of the variation in dietary intake. Unlike statistically determined dietary patterns, dietary indices that are based on predetermined criteria are easier to be applied in a variety of populations.

Another limitation of using the statistical approaches is the fact that they only determine the distinctive food or food group patterns, and finding out about the effect of a single nutrient becomes problematic. Additionally, translating the statistically determined scores into practical food recommendations for the general public is rather complicated. On the other hand, dietary indices that utilize the current knowledge and

nutritional recommendations are easier to interpret because they are already based on certain levels of food and/or nutrient intakes.

It must also be noted that because dietary quality indices are based on the current knowledge and guidelines regarding the roles of nutrients and foods on health and disease, these indices cannot make up for what is not currently known or not included in the nutritional guidelines. Statistically determined patterns, on the other hand, do not depend on previous knowledge or guidelines as much, and they can detect the statistically significant correlations even if the potential effect of a certain food is not currently known or not recognized within the present guidelines.

DIETARY QUALITY AND OBESITY

The current literature supports the relationship between a healthier dietary intake pattern (e.g., high fiber, low fat, low soft drink, and low refined grains) and lower weight gain over the years or lower body mass index, which is a measure of overweight and obesity. Despite the fact that there are numerous measures of dietary quality, published studies show that a greater dietary quality, measured by several indices such as the DDS, RFS, DQI, HEI, and AHEI, is related to gaining less weight over the years or to having lower body mass index.

Although there has been some inconsistencies regarding the detected associations between dietary intake patterns and BMI or obesity, these may be partially due to the fact that dietary patterns are population specific and can be influenced by cultural, socioeconomic, gender, or age differences in the studied populations. Measurement errors must also be considered.

Possibly stimulated by the general desire of all people to have simple solutions to the health problems, at times, attention is given to one or two components of an overall diet. A typical example of this trend can be found in preference to eat low-fat foods while disregarding all the other dietary characteristics of these foods. Certain low-fat foods might be high in added sugars and calories, and obviously, a person who is trying to control his/her weight would not gain much benefit toward that goal by just paying attention to the fat content of the foods. The concept of dietary quality is a potential solution to help resolve this problem by providing guidance on the overall diet.

SEE ALSO: Food Intake Patterns; Healthy Eating Index; Variety of Foods and Obesity; Western Diet.

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Overweight Children and School Performance

WITH OBESITY BECOMING more prevalent among children, the potential impact of obesity on school performance has been receiving increased attention. A recent review by Howard Taras and William Potts-Datema described nine articles (three of which were conducted in the United States), which suggest that childhood obesity is negatively associated with school performance. In addition, overweight children have been found to score lower on measures of intelligence compared to normal weight counterparts. There is also evidence that overweight students are twice as likely to be placed in special education or remedial classes. Despite emerging evidence demonstrating an association between obesity and academic performance, few studies have examined the potential causes of this relationship.

It appears that several variables such as psychological distress, physical health consequences of obesity, and weight-related stigmatization may mediate the relationship between obesity and school performance. Psychological distress (e.g., depression, anxiety, lower self-esteem, and body image concerns), which may be

associated with being overweight, may interfere with children's ability to concentrate and learn in class. Physical health may be another important mediating variable, as physical health problems associated with obesity (e.g., sleep apnea, asthma) could affect school performance by leading to absenteeism or difficulty concentrating while in class.

Weight-based stigmatization likely also negatively effects school performance through several mechanisms. For instance, weight-related stigmatization is one potential cause of psychological distress, which negatively influences school performance. Marlene Schwartz and Rebecca Puhl reported in a recent review that children develop pervasive negative beliefs about obesity as early as 3 years of age, including beliefs about overweight children's school functioning and intellectual capacity. This bias appears to worsen as children get older and may lead to the teasing or bullying focused toward overweight children often observed in school.

Weight-related stigmatization might also influence school performance through a self-fulfilling prophecy. For example, there is evidence suggesting that overweight children are more likely to consider themselves "poor students" compared to average weight children. Stigmatization may also negatively affect teachers' expectations and behavior toward overweight students. Indeed, educators have been found to hold negative views about overweight children's academic abilities, which could lead to lower student performance. This would be consistent with the Pygmalion effect, which is a theory that posits that teacher's expectancies, either positive or negative, of children's academic abilities can influence the children's actual performance. Further, overweight children may internalize the low expectations that teachers have, which may also lead to poorer school performance.

Several studies have suggested that gender may moderate the relationship between obesity and school performance. For instance, Roland Datar and Ashlesha Sturm recently found that overweight girls appear to have greater school performance-related consequences than overweight boys. After controlling for demographic differences, they reported that gaining weight in elementary school for girls was related to lower math and reading test scores, more teacher-reported externalizing problems, and lower teacher-rated self-control, interpersonal skills, and learning behaviors (i.e., attentiveness, task persistence, eagerness to learn, learning independence, flexibility, and organization), but not for boys who gained weight during this time. It may be that girls are more affected by weight gain compared to boys, potentially related to a greater emphasis on physical appearance for females.

There is somewhat limited, but consistent, evidence indicating that obesity negatively impacts school performance in children. Research suggests that psychological factors and physical health may mediate the relationship between obesity and school performance and that weight-based stigmatization also may negatively affect school performance. Overweight girls appear to be particularly at risk for poor school performance. Further research is needed to better understand the causes of the obesity and school performance relationship.

SEE ALSO: Obesity and Academic Performance; Obesity in Schools.

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Overweight Children and the Media

CHILDHOOD OBESITY IS prevalent at epidemic proportions. According the Centers for Disease Control and Prevention, national rates of overweight in the United States have tripled in adolescents and more than doubled in children aged 6–11 since 1980. Simultaneously, media targeting children have dramatically increased, leading the average child living in the United States to consume up to six hours of media per day. Given the rise of childhood obesity coupled with the extensive amount of time children are exposed to

media messages, the purpose of this entry is (1) to examine the messages promoted by mainstream American media about overweight children and (2) to evaluate the influence of exposure to these messages on the psychological health of children.

MEDIA MESSAGES ABOUT OBESE CHILDREN

The term *media* describes the various types of public communication that convey information to large audiences through mediums such as newspapers, magazines, direct mail, billboards, bus signs, radio, television, and the Internet. Mainstream American society exposes children to endless amounts of media, particularly through television programs, video games, internet sites, and movies. For example, the average child in the United States views 10,000 advertisements a year. Of those aimed at children, more than 90 percent are for candy, fast food, soft drinks, and cereal.

These media present specific information about weight, food, and appearance. While children are bombarded with advertisements for unhealthy foods high in sugar and caloric density, they are simultaneously bombarded by images of idealized boys and girls. The ideal female has flawless skin, flowing hair, a thin waist, light-colored eyes, and long legs, whereas the ideal male is muscular, fit, tall, and lean. Given these physical ideals, it is not surprising that the media rarely contains images of overweight children.

Overall, the media broadcasts very negative messages about being overweight or obese.

Fatness is negatively stigmatizes to the degree that obesity stigma has been referred to by some scholars as the last socially acceptable form of discrimination in the United States. Negative stereotypes about overweight people are prevalent in children's television shows, movies, and literature. For example, in the *Harry Potter* series, the overweight Dudley is portrayed as greedy, mean, lazy, and piggish. In *Willie Wonka and the Chocolate Factory*, the only overweight child is depicted as greedy and is the first child disqualified in the con test because he is too preoccupied with food to stop from gorging himself and listen to the adults. Jake Harper from the television show *Two and a Half Men* is constantly preoccupied with food and the object of ridicule about his low intelligence.

Literature for children under age 6 also contains negative attributions about being overweight. In *The Hungry Pig*, the main character sneaks around and cheats to eat more food. In *Mr. Greedy*, children learn that the overweight main character loves to eat and learns not to be greedy as he loses weight. Some even critique Disney for its stereotypical cartoon renderings of overweight people as stupid and lazy.

INFLUENCE OF MEDIA PORTRAYALS ON OBESE CHILDREN

The messages about obese children perpetuated by Western media can detrimentally influence children's beliefs, feelings, and attitudes about overweight individuals and themselves. There has been an increase in negative stereotyping of overweight children by other children in the last few decades. For example, in 2003, children rated drawings of overweight children as being less likable than had children of the same age in the 1960s. Overweight children and adults are often described in negative terms and believed to have negative characteristics such as being lazy, sloppy, dirty, naughty, cheats, liars, argues, mean, ugly, stupid, and perpetually focused on food. Negative attributions about overweight people begin as young as age 3 and get stronger with age.

The stigma associated with being overweight also negatively influences children's emotional well-being. Peers are more likely to harass overweight children. Some research even suggests that being obese is positively associated with teen suicide. Furthermore, when overweight children internalize these negative attributions about people who are overweight, many experience low self-esteem, suffer from social isolation and teasing, and experience body dissatisfaction.

Internalization or personalization of mainstream media messages about weight can also lead normal-weight and overweight children to serious psychological problems. Researchers propose that children often compare their appearance to idealized images of models displayed prominently in the media. The fact that humans compare themselves to media images is not surprising given that the goal of many media images is to capture one's attention and, through a process of social comparison, instill a desire to purchase a particular product or attain a given outcome. Such comparisons most likely lead to negative self-evaluation because few, if any, kids view themselves as better than the rigid ideals portrayed in mainstream American media. Repeated negative self-evaluation

with regard to one's appearance, in turn, is likely to lead to increased body image disturbance, which is the most empirically supported risk factor for eating disorder development.

CONCLUSIONS AND SUMMARY

Recently, there has been considerable focus on the role of the media on children's weight problems. American media perpetuates negative attributions about overweight children, which can detrimentally influence children's beliefs, feelings, and attitudes about overweight individuals and themselves. Teaching children media literacy and encouraging them to actively evaluate media messages about appearance will not only promote tolerance of individuals of all sizes, shapes, and backgrounds, but also help protect them from potential negative psychological consequences of media exposure.

SEE ALSO: Disordered Eating; Eating Disorders and Obesity; Obesity Action Coalition; Obesity and the Media; Obese Women and Social Stigmatization.

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Oxytocin and Food Intake

OXYTOCIN IS A mammalian hormone that also behaves as a neurotransmitter in the brain. This neurohypophysial hormone was the first peptide hormone to have its structure identified and the first to be chemically synthesized in its biologically active form. Oxytocin has several functions in the body. It plays an important role in the expression of central functions, such as maternal behavior, sexual behavior, yawning, memory and learning, tolerance and dependence mechanisms, feeding patterns, grooming, trust formation, bonding, cardiovascular regulation, and thermoregulation. However, its roles in postpartum uterine contractions and feeding behaviors are the most relevant when discussing the hormone's relationship to obesity.

Oxytocin is a nine-amino acid peptide that is synthesized in neurons in the hypothalamus and is transported down axons to the posterior pituitary for secretion into the blood. In the pituitary gland, oxytocin is packaged in large, dense-core vesicles, where it is bound to neurophysin I. Neurophysin I is a large peptide fragment of the precursor molecule from which oxytocin is derived. Oxytocin and vasopressin are the only known hormones released by the human posterior pituitary gland to not act at a distance from its point of origin.

In pregnant women, oxytocin is released mainly after dilation of the cervix and the vagina during labor to facilitate birth, and after stimulation of the nipples to facilitate breastfeeding. Studies have shown that during pregnancy, body weight, and particularly adiposity, increase, due to increased food intake as opposed to decreased energy metabolism. This adaptation provides the growing fetus with adequate nutrition and prepares the mother for the metabolically demanding lactation period after birth. During feeding in nonpregnant rats, oxytocin neurons become strongly activated indicating their role in meal termination. However, in mid-pregnancy the excitability of these neurons is decreased, oxytocin release is inhibited, and patterns of oxytocin receptor binding in the brain alter. Thus, an increase in food intake occurs.

Oxytocin release during breastfeeding causes mild but painful uterine contractions during the first few weeks of lactation. These contractions facilitate in maternal postpartum weight loss. Studies have shown that the longer mothers breastfeed, the greater weight loss they experience and they are more likely to return to their prepregnancy weight. The findings from these studies and many others have been used to encourage women to practice optimum breastfeeding, not only to ensure adequate nutrition for their infants, but also to decrease postpartum weight retention. If adequate breastfeeding practices are maintained as well as physical activity levels, risk of obesity in later adulthood dramatically decreases.

Oxytocin has been studied in nonpregnant animals as well. It is considered to be a "satiety" hormone because it has been shown to reduce food consumption when administered peripherally and centrally in rats. Also, the inhibition of food intake is not directly associated with circulating oxytocin. When rats were administered with anorexigenic treatments such as cholecystokinin, hypertonic saline, and lithium chloride, pituitary secretion of oxytocin resulted and caused a greater increase in inhibition of food intake occurred. These data suggest that nausea and satiety induce a common hypothalamic oxytocin pathway that results in the inhibition of digestion. Another study showed that oxytocin reduced food consumption and the time spent consuming food, and it also increased the time before first meal in fasted rats. Conversely, when treating the rats with an oxytocin antagonist, their food intake increased.

How exactly does oxytocin mediate its influence on consumption behaviors? Some studies show that oxytocin enhances the effects of anorexigenic modulators as mentioned above and others show oxytocin may regulate the effect of leptin, a hormone that acts on the hypothalamus to suppress appetite. Data show that leptin secretion is inhibited by oxytocin in normal weight individuals, suggesting a likely role of oxytocin in the regulation of leptin. It was also discovered that this oxytocin-leptin regulatory system was disrupted in obese individuals, indicating a possible contribution to their weight gain. It has also been proposed that oxytocin could either act to modulate the activity of intrinsic brain stem reflex arcs or apply a direct control over vagal efferent nerves that regulate the gut and reduce the gastric motility. However, the exact mechanism is unclear.

SEE ALSO: Hormones; Leptin Supplements.

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Palatability

PALATABLE IS DEFINED by *Webster's Dictionary* as "agreeable to the taste, hence, acceptable, pleasing." Palatability is then the attribute of being palatable. This entry will consider various definitions and operations for measuring palatability, the neural mechanisms that underlie it, and its potential contribution to obesity.

Arguments among specialists abound in whether palatability inheres in the food or beverage being tasted, the reaction of the subject, or an interaction of the two. It has been stated that hunger widens the range of acceptability, thereby making foods more palatable. However, hunger may not change the actual pleasure from consuming the ingesta, but only the subject's motivation to consume. Technically, there may be more than one kind of palatability and several modulators of the word have been proposed to distinguish between the mechanisms by which changes in the subject or food result in changing intake. At least three modulators of palatability should be considered. First is intrinsic palatability, which attributes the responsiveness of subjects under constant conditions to changes inhering in the food or beverage. The property of tasting sweet is likely to be such an attribute because newborns show immediate acceptability responses to sweet tastes, although arguably, this could have been acquired in utero, because it is known that amnionic fluid is imbibed by the fetus. Organismic palatability is a label that has been appended to the change in acceptability that comes with changes in the state of the organism being tested. For example, reduction in blood glucose can make sweet-tasting foods taste sweeter. Palatability has also been described as either absolute or relative. Absolute refers to measures of palatability of a single item at a time, whereas relative palatability refers to a hierarchy among two or more items. Finally, palatability is probably largely learned as the result of pairing the stimuli in food with gastrointestinal, metabolic, or neurotransmitter system after-effects of consumption.

Palatability of foods and fluids is a quality which makes them particularly useful in reward paradigms in which effort to obtain food is used to assess the rewarding properties and is particularly useful in studying the neural basis of palatability. In the paradigms, an animal or person must perform some task for which it periodically receives a small piece of food or drop of fluid. In such a situation under unchanged deprivation or physiological state, reward value or palatability is greater for more effort expended, faster response rate, or toleration of impediment.

Two major neurotransmitter systems, opioid and dopamine participate in the control of palatability and it has been proposed that they control separate aspects of reward. "Liking" indexed by the opioid system is the label for the hedonic or pleasure-inducing effects of rewards, whereas "wanting," indexed by the

dopamine system is the label for "incentive salience" or the quality of the reward that impels the individual to pursue it. Alternatively, dopamine may function as neurotransmitter that stamps in the memory of the association of an action with the stimulus.

Palatability is measured by differences in intake, differences in response rates or facial expressions, or differences in ratings made with appropriate scaling metrics between foods or individuals. Measurement of intake leads to circularity when it is argued that what is palatable is what is eaten in greater quantity, so that the confounding of various controls of intake with palatability cannot be ruled out. Hence, measures that are independent of intake would be preferred. Such measures as rate of eating, running down alleys, choices in T-mazes, lick bursts and interlick intervals, pressing levers on various schedules of reinforcement have all been used as alternatives to amount consumed in animals. However, no measure is without its own problems. In animal studies, drugs presumed to affect the rewarding value may also affect motor responses, which could interfere with performance.

Palatability of foods has been proposed as both a cause of obesity and a method for controlling it. Obese individuals may consume more because they like certain nutrient combinations more than do nonobese individuals; hence, reducing palatability by providing standard amounts of formula diets whose palatability has been reduced to help reduce intake is often effective in reducing weight. For individuals who can tolerate reduced palatability in foods, this could be an effective long-term solution to keeping weight off. Problems in the scales used for palatability have confounded efforts to determine whether palatability contributes to weight differences among groups.

SEE ALSO: Food Preferences; Food Reward; Neurotransmitters; Nutrient Reward.

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Parental and Home Environments

A NUMBER OF factors related to home and parental environment may have an effect on children's eating habits and consequent weight status. Many studies have demonstrated a significant heritable component of eating habits and body weight. However, changes in population genetics could not occur rapidly enough to fully explain the dramatic increases in rates of obesity in recent years. Thus, environment seems to play a critical role in the development of obesity. Home and parental environment have received much attention by researchers investigating factors related to development of obesity, because it is in the home that most early experiences with food and physical activity occur.

Some demographic variables of the home have been found to relate to children's eating, level of physical activity, and weight status. While evidence on the impact of socioeconomic status is mixed, many studies suggest an association between low income and increased rates of obesity. This may be due to a number of factors. In lower-income households, higher weights in early childhood may be considered a mark of good health, foods such as fruits and vegetables may be less available or considered to be too expensive, and the environment may be less stimulating and conducive to physical activity. Parental education may also play a role. Lower levels of parental education have been linked with childhood obesity. Further, children of parents who are lacking in nutritional education tend to have greater caloric and fat intake and less fruit and vegetable consumption.

Also related to childhood obesity is a single-parent home environment. Children from single-parent homes

tend to eat fewer meals but more snacks. This erratic eating schedule may be result from the single parent's need to work long hours as the sole household provider and may be responsible for the increased obesity risk. Parents' work schedules appear to impact children's risk of obesity. Increases in the number of mothers in the workforce have often been blamed for poorer household eating habits; however, there is no evidence that maternal employment affects dietary quality. That said, the number of hours that parents work may affect a child, such that children of parents who work longer hours are at increased risk for obesity. This is likely due to decreased availability of these parents to provide healthful food choices and set meal schedules for children.

The quality of the food and drinks available in the home environment can have an impact on a child's weight outcome. Consumption of certain foods, such as fruits and vegetables, foods high in fiber, and dairy products has been linked to favorable health outcomes, while other foods (e.g., high-fat foods) and drinks (e.g., soft drinks) have been linked with increased risk of obesity. There is evidence to suggest that the availability of and familiarity with healthy and unhealthy foods may influence a child's weight. Fruit and vegetable intake, as well as soft drink and snack consumption has often been linked with the availability of these foods within the home environment.

These findings may simply result from convenience factors. Children often report eating whatever is available and accessible in the home. However, especially in younger children, familiarity is strongly related to food preference and increased exposure to a food leads to increased acceptance of the food. This indicates children may eat more of what is available at home not just because it is easier, but also because they come to prefer the foods that are commonly available to them. Therefore, by making healthier options available for children in the home, parents may positively affect long-term eating habits.

Evidence also suggests that children are able to regulate dietary intake with a good deal of accuracy

Meal consumption at home is associated with increased fruit, vegetable, dairy, and vitamin and mineral intake and decreased intake of soft drinks and fried foods; it allows parents a forum to provide children with healthier food choices and to model healthy eating behaviors.

if a number of nutritious options are available. However, in the presence of many highly-palatable, high energy-density foods, desire to consume such foods can override natural regulatory abilities, thus resulting in overeating. Therefore, by providing a number of nutritious foods with lower energy-density (e.g., vegetables) in addition to nutritious foods of higher caloric value, parents may help children to regulate their energy intake more efficiently.

Simply consuming more meals at home can affect children's weight. Children and adolescents who eat more meals with the family at home tend to have healthier eating habits. Meal consumption at home is associated with increased fruit, vegetable, dairy, and vitamin and mineral intake and decreased intake of soft drinks and fried foods. Meals allow parents a forum to provide children with healthier food choices and to model healthy eating behaviors.

However, within recent years, eating has been taking place outside of the home environment more frequently, and children and adolescents have been making more decisions regarding family food selection and meal preparation, perhaps due to increases in parent time spent at work. Meals eaten outside of the home, such as in fast-food venues or restaurants, tend to be higher in fat and energy content and serve larger portions. Additionally, when adolescents are responsible for their own food decisions, they are more likely to engage in erratic eating patterns (e.g., skipping meals) and to eat more junk foods. Family meals provide a protective environment against such factors.

Another variable related to the home environment that has been found to relate to obesity is the availability and utilization of technology that promotes sedentary behavior (e.g., television, video games, etc.). In particular, television viewing has been linked with the development of obesity. Research has consistently found an association between the amounts of time spent watching television and rates of obesity. This relationship is likely to be determined through a number of causes. If individuals are spending many hours watching television, less opportunity for physical activity is available. Thus, by opting for sedentary, rather than active pastimes, an individual may not be allowing sufficient energy output to counterbalance caloric intake.

Additionally, television watching is associated with poorer eating habits. Television viewing has been found to promote greater caloric intake, as well as greater intakes of fat and nonmilk caloric beverages. People tend to snack more while watching television. Research has found that those who watch television during meals consume less fruit, vegetables, and juice than those who abstain from mealtime viewing. Television also allows a greater opportunity for exposure to food and beverage advertisements. The foods advertised on television can often be high in fat and sugar and low in nutritive value. Studies demonstrate that children tend to request the foods that they see on television, which may therefore lower the quality of their diet. The last several decades have shown a dramatic increase in television viewing and the number of televisions typically available in the household. This may be one factor that has contributed to the increasing rates of obesity.

Parents' attitudes and behaviors toward eating and physical activity can have a significant effect on those of their children. Parents' energy, fat, fruit and vegetable, snack, and soft drink intake and level of physical activity have been consistently positively associated with that of their children. Parental disordered eating behaviors, such as food restriction and disinhibition while eating, are often mirrored in their children's eating habits. While much of this may be related to similarity of genetic makeup and resource availability, research demonstrates that parent modeling of eating and exercise behaviors impacts children's habits.

For instance, children are more likely to sample a novel food item if they see their mother consume this object. They are more likely to accept a food if they are offered the food by their mother, as opposed to an adult stranger. Additionally, parental involvement in physical activity often predicts the child's engagement in such activities. This effect can be long lasting. Some research indicates that parents' level of activity is associated with level of activity in children even into early adulthood. In this way, parents may promote healthy eating and physical activity habits in their children by engaging in such healthy practices themselves.

Much evidence suggests that parental approaches to feeding their children can have a direct or indirect effect on a child's eating and weight. The parent feeding variable most consistently related to childhood overweight is that of dietary restriction. In other words, parents who restrict their child's access to foods or otherwise impose strict limits on their child's

eating may be inadvertently encouraging habits that lead to increased risk of obesity.

There are a number of explanations for this connection. Studies have demonstrated that restricting a child's access to a palatable food can increase the desirability of and preference for the food. Children will therefore consume more of the food when restrictions are removed. Thus, by restricting a child's access to certain foods, parents may actually be causing the child to want the food more. Additionally, by placing a high level of restriction around feeding, parents may be encouraging children to pay more attention to external cues, rather than their internal sense of hunger and satiety. As previously noted, children generally have an innate ability to accurately regulate their energy intake; however, those whose parents demonstrate higher levels of restriction have poorer regulatory abilities and are more likely to eat in the absence of hunger. These behaviors put the child at risk for becoming overweight.

The effect of parental restrictive feeding practices may be most profound in samples of children who are already overweight. Parents are more likely to place restrictions on their child's eating if the child is overweight, presumably in an effort to help the child to lose weight or curtail continued weight gain. However, this practice may be especially detrimental. Studies have shown that overweight girls whose mothers place restrictions on their eating eat more in the absence of hunger than leaner counterparts whose mothers place restrictions on eating, as well as peers whose mothers do not place restrictions on eating (whether overweight or lean). Parents are therefore encouraged to promote overall healthy eating practices, geared toward consuming nutritious foods and focusing on internal hunger/satiety cues, instead of being overly restrictive of child's intake or placing the child on a diet.

Other variables relating to parental feeding style have been found to relate to children's eating habits. Pressuring a child to eat in response to the mere presence of food (e.g., requiring a child to "clean the plate"), rather than in response to internal hunger and satiety cues is associated with greater caloric intake, as well as greater energy intake from fat, snack foods, and high-calorie beverages. This parent feeding technique has also been found to lead to disinhibited eating behaviors in children. Children's food preferences may be influenced by parents' presentation of the foods. For instance, if a food is given as a reward or paired with attention, preference for the food tends to increase. On the other hand, if a food is presented as a contingency for another behavior (e.g., "If you eat your vegetables, you may go out and play") or if a parent otherwise uses coercion to get a child to eat a food, preference for the food generally decreases.

Parent behavior can also have a significant affect on children's level of physical activity. Parents can reinforce the value of physical activity through encouragement, support, and praise of children's engagement in sport or exercise. Children are more likely to be physically active if parents sign them up for sports team and provide transportation to venues for physical activity. However, when parents place too much pressure on a child's participation in a sport, children tend to experience burn out. Therefore, encouragement, not coercion is advised. Additionally, children are more likely to be physically active if parents take time to engage in physical activity with them and if parents generally discourage sedentary behaviors, such as television watching, at home.

The relationship between general parenting style and variables relating to child's eating, physical activity, and weight has received less attention. However, there is suggestion that a parenting style characterized by support, communication, and responsiveness coupled with clear limit setting may lead to healthier eating and increased physical activity in children. Additionally, extreme disorganization, neglect, and abuse in some cases have been linked with the development of severe obesity and eating disorders.

Parents' attitudes toward their child's weight may also have an impact on the child's eating behaviors and weight. Children who receive negative comments regarding their weight at home or who perceive their weight as being very important to their parents tend to engage more in dieting and unhealthy weight control behaviors. These behaviors, in turn, increase the child's risk for development of obesity and eating disorders. Additionally, overweight children report benefiting from parental support, rather than dieting advice. Researchers suggest that parents focus more on encouraging healthy eating in children, rather than promoting a particular body weight or shape.

SEE ALSO: Accessibility of Foods; Family Behavioral Interventions; Family Therapy in Treatment of Overweight Children; Genetics; Maternal Influence on Child Feeding.

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Patient Sensitivity

IN DEALING WITH the overweight, obese, or morbidly obese patient, practitioners must be fully aware of the patient's mind-set and the patient's lived experience. They need to be fully cognizant of the many dimensions of this experience, and the depth to which it permeates every fiber, experience, and aspect of the patient's life. Patients with weight issues suffer daily from the culturally and socially integrated biases, stereotypes, and perceptions that label them as unworthy, invaluable, substandard, and defective.

It is imperative that practitioners treat the "whole person" and not just a particular problem or symptom.

Practitioners should recognize some of the more common stereotypes related to people with weight issues: lowered intelligence; socially awkward; less desirable in love relationships; lazy; stupid; no self-control; bookish; jolly; good cooks; food obsessed; available to pick up slack for others; uninvolved in social activities; women are fat; men are "big guys"; and laugh easily at themselves and are not bothered by it

Related to stereotypes are labels, title, generalizations, and assumptions that are made or applied by the general populace to those with weight issues.

Labels (from others or society as a whole) include: hypochondriac; whiner; wimp; unworthy; troublemaker; burden; defensive; and defective.

Titles (from self or internalized from others) include: Rock of Gibraltar; Therapist and Fixer; Everyone's Backup; Mrs. (assume woman is married); Mom; and Tough guy.

Generalizations (from society as a whole) may include: lack self-discipline; lack self-respect; lack ability and desire for perfection; weak; pitiful; unfriendly; moody or sullen; uncooperative; and not credible.

Assumptions (from society as a whole) include: are a parent; married; had sex; had relationships; dated; having nothing else to do but meet your needs; noncompliant; have held a job; do not have much education; and have not tried very hard to lose the weight before now.

Practitioners should be mindful not to perpetuate any of these stereotypes, labels, titles, generalizations, or assumptions, and should take the time for personal introspection about personally held beliefs as they relate to those with weight issues. Practitioners who identify and confront their own biases and perceptions of persons with weight issues will be less likely to engage in or promote pigeonholing roles, situations, or behaviors as well. *Pigeonholing* is defined as roles and situations into which obese and morbidly obese people are thrust and trapped by others with whom they interact.

Examples of "pigeonholing" obese patients.

- Standardized charts, tests, measures

- Others' expectations and perceptions
- Others' values and morals
- "You're just a weight issue to me and nothing more"
- Jobs that do not go anywhere
- Particular types of jobs
- Parent roles
- "You're just additional work for me"
- "You're just a number or a file"

Related to all of the foregoing is prejudice and discrimination. Prejudice is the prejudging of a person or situation based on attitudes, not perceptions. Discrimination is behavior for or against a person or situation. Practitioners should be extremely mindful of their internal prejudices, and do not engage in discriminatory practices. This includes the conscious or unconscious use of particular words or language when addressing obese patients.

Many professionals working with obese patients have come to this field from other areas of healthcare or social service. They have brought with them their experiences, knowledge base, and vocabulary. Where these have all served them well in other aspects of their professional lives, they can now become a liability instead of an asset.

Obese patients are very, very sensitive to the specifics of language. Words have been used against them all their lives. What was once a label for a member of the bovine species became a derogatory term for a fat woman—a *cow*. What was created as a simple term for defining the state of having fat cells over a certain amount has become a term slung at people as more of an accusation and condemnation of their character than simply as an actual description of their status—you are *obese*. It is not even a word that sounds nice. *Cancer*, a factual statement of a medical condition, has the connotation that something has happened *to you*. *Obesity* has the connotation that you did something to yourself, and thus you are at fault.

It is difficult for patients who have been mistreated, dismissed, and abused by society and the medical community all their lives to suddenly grow a callous to the previously hurtful nature of the terms used to describe them. Although most bariatric professionals use terms such as *obesity, morbid obesity, supermorbid obesity, body mass index,* and *fat* in their clinical terms, patients hear them and feel them like hurtful little barbs of condemnation. Language selection can cause huge resistance issues. The goal is to overcome resistance, reduce fears, and facilitate people's journey toward a healthier lifestyle, not to win a war of words so the practitioner can claim victory for the use of their terms.

In addition to what has been outlined above, practitioners should employ patient sensitivity measures that include creating effective trust relationships, acting with integrity and competence, consistency, loyalty, and openness. It is imperative that practitioners also treat the whole person and not just a particular problem or symptom. The obese population is a highly complex population that is unlike any other patient population at all. Emotional scars run deep and affect every aspect of the patient's personality, behaviors, perceptions, and choices. Practitioners are encouraged to continue their research and understanding about the nature and needs of obese individuals, and to respond with appropriateness, kindness, understanding, and empathy.

SEE ALSO: Anxiety; Appearance; Body Dysmorphic Disorder; Body Image; Depression; Fat Acceptance; Loneliness; Mood and Food; Obese Women and Social Stigmatization; Quality of Life; Self-Esteem and Obesity; Self-Esteem in Obese Women; Stereotypes and Obesity; Stigmas against Overweight Children; Weight Discrimination.

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Peer Influences on Obesity in Children

PEER INFLUENCES MAY impact the development of obesity in childhood. Peers can exert influence through their attitudes and behaviors toward food, exercise, and weight status in ways which may promote or, alternatively, discourage healthy behaviors related to eating and physical activity. Peer influence is of appreciable importance in childhood and adolescence. Most children spend as much or more time in school and engaged in after-school activities with their peers as they do at home with their family. Peer group has been found to significantly influence many health-related behaviors throughout childhood, such as smoking and sexual behavior. Additionally, children share at least one meal, in addition to potential snacks, with peers during the average weekday and typically engage in physical education together through school curriculum. Therefore, it seems likely that eating and physical activity habits would be affected by peer group to some extent.

One way in which peers may affect the development of obesity in childhood is by affecting children's eating habits. Little research exists regarding social influence on infant and toddler populations. This is presumably due to the fact that eating is largely controlled by parents during this developmental stage. While in very young children (2 years of age and under), peer influences on food preference might not be strong, for children in preschool environments with ample interaction with peers are strongly influenced by their classmates choice of foods.

Studies have demonstrated that food preference, selection, and intake can be affected by that of one's peers at this age. Witnessing another child selecting, eating, and enjoying a food has been found to make the food more desirable and acceptable to a young child. This finding is corroborated by parental report. Parents tend to report that children of this age begin requesting foods based on what they report others are eating at school. This influence of peer modeling on the food preferences of young children appears to be maintained over time and, thus, may permanently affect children's dietary selection.

Some peers may be more influential than others in altering food choice. According to existing research, peers who are prestigious, well-liked, and nonaggressive are the most likely to affect the eating behavior of other children. Older children appear to exert considerable influence on the food choices of younger children, likely because younger children look up to them as role models. Additionally, females may be more influential models of eating behavior than males. Some argue that this is because children are used to looking to female figures within their families, namely their mothers, for cues on how to eat. This effect may also be a reflection of a temperament, as girls tend to be less aggressive than their male peers, which may in turn make them more desirable models of behavior.

There is less evidence to suggest that peers significantly impact on the eating behavior in middle childhood and preadolescence (ages 6–12). Several studies demonstrate that peer modeling effects on food choice are stronger in younger subsets of children. This is likely because food preferences have become more stable by this time and, therefore, less susceptible to outside influence.

Individual friends do not appear to influence choice, preference, or overall fat or energy intake of middle school children. However, general normative attitudes of a peer group may still be influential. In addition, even if peers' food choices do not directly affect children's eating habits, peers may still have an indirect effect at this time. For instance, middle school children report that fruit and vegetable intake is generally discouraged by peers. While children may not report that this directly affects intake of these foods, it may affect children's attitudes toward these food items, which may then affect their likelihood of incorporating them into their diet.

As in middle childhood, in adolescence, friends do not appear to directly affect overall dietary preference, choice, or intake. However, peers do seem to influence snack food intake at this time. This seems logical because adolescents are more likely to eat snacks with friends than meals. This is also significant because during adolescence, food is increasingly obtained from outside the home, often from sources such as fast-food restaurants, convenience stores, and vending machines. Accordingly, snack food selections tend to be high in calories and lacking in nutritive value. Therefore, peer effects on snacking may impact the likelihood of unhealthy food selections and thereby weight gain. Additionally, research demonstrates that friend selection and social status may

affect adolescents' eating attitudes and habits. Adolescents who aggregate toward more academically focused social groups report healthier food choice selections and more dieting, while more popularity-focused adolescents report making poorer food choices but exercising more. Adolescent groups that tend to get into more trouble and engage in riskier behavior report making poorer food choices and engaging in more eating disordered behavior.

Overweight children, in particular, may be sensitive to peer influences on eating habits. Research demonstrates that overweight children eat greater quantities with other overweight children than with leaner peers. This may indicate a tendency to restrict themselves in the presence of thinner peers and/or a tendency to overeat as a display of camaraderie with peers of a similar size. This finding could also demonstrate a greater level of comfort with similarly overweight peers. In fact, there is some suggestion that overweight children are drawn to friendship with peers who share similar risk for obesity.

Additionally, peer perceptions of and attitudes toward overweight peers can have a profound effect on eating behavior. Overweight children may receive stronger social pressure to alter their eating. Overweight girls are less likely to be talked out of dieting by friends and consequently report more attempts to engage in dieting behaviors. This tendency places overweight children at risk as dieting in childhood and adolescence is associated with the emergence of eating disordered behavior and continued weight gain. Further, overweight children (and underweight peers to a lesser degree) are more likely to be teased about their weight and appearance. Weight-related teasing by peers is associated with poor body image, dieting, binge eating, purging, and other extreme weight-control behaviors. Thus, the effects of peer influence may place overweight children at risk for development of eating disorders and obesity in adulthood.

Another way in which peers significantly affect the development of obesity is through their influence on children's level of physical activity. Results are more consistent regarding peer influence on exercise and physical activity than on eating habits. Research has consistently demonstrated that social support strongly influences children's engagement in physical activity. Friends' praise of and encouragement for participation in physical activity are related to increased engagement

During the average weekday, children share at least one meal, and potentially snacks, with peers.

in such activities. Children are more likely to participate in a sport or a fitness class if a friend also joins or to engage in physical activity if a friend invites them to. Additionally, perceptions of peers' attitudes toward exercise can affect children's attitudes toward exercise, consequently affecting their activity levels.

This may also put overweight children at a disadvantage. Research indicates that overweight children are more socially isolated, are more often excluded from exercise- and sport-related activities, and less likely to receive support of physical activity than normal-weight peers. This can lead to decreased opportunity for shared physical activity and less interest in engaging in exercise. Further, social isolation and exclusion can lead to depression and loneliness, which are associated with decreases in activity level.

Bullying and teasing about weight are also associated with increases in depression, anxiety, and somatic complaints, and decreases in physical activity. Further, overweight children are more likely to be criticized about their weight by peers while engaging in physically activity (e.g., during gym class). This decreases the reinforcing aspect of the exercise and can cause embarrassment, thus increasing desire to avoid such activities. However, research indicates that overweight children can overcome the deleterious effects of teasing on physical activity level by using problem-focused and avoidant coping techniques.

Research on the impact of peer groups on the development of obesity-promoting behaviors can be

informative to the development of new interventions for the prevention and treatment of obesity in childhood, particularly those applied to school settings.

SEE ALSO: Morbid Obesity in Children; Obesity in Schools; Physical Activity in Children; School Based Interventions to Prevent Obesity in Children; Stigmas against Overweight Children.

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Perilipins

PERILIPIN IS A protein involved in the formation and stabilization of lipid droplets in mammalian adipose cells. Perilipins are expressed only in adipocytes and steroidogenic cells and they are centrally involved in both lipid storage and degradation. The regulation of lipid droplets is currently poorly understood, and future studies in this area are critical in order to realize its implications in weight gain, obesity, and associated potential treatments.

Lipid droplets are the main energy depots in the body and they contain primarily triacylglycerols. These stores are formed during periods of energy surplus and the release of fatty acids from these droplets occurs when energy is in need through lypolysis (hydrolysis of lipids). Phospholipids form a layer which surrounds and protects the lipid droplet from hydrolysis. Perilipin is the most abundant protein associated with this phospholipid layer.

During periods of stress or low blood sugar, the hormone catecholamine is released from the adrenal gland. When cells are exposed to catecholamine, a lipolytic response is initiated. This response begins with G protein stimulation and through a series of reactions activates protein kinase A (PKA). PKA can then phosphorylate and activate both hormone-sensitive lipase (HSL) and perilipin. Phosphorylated perilipin is necessary for normal lipolytic response because it facilitates the interaction of the HSL with lipid droplets. When HSL interacts with the lipid droplet, it begins hydrolysis of triacylglycerol, releasing fatty acids which can then be used as energy.

In addition to assisting in lipid degradation, perilipin regulates the storage of triacylglycerols by providing a barrier against lipolysis and thereby preventing the interaction of HSL with triacylglycerol.

There are two forms of perilipin in the body: perilipin A and B. These isoforms are transcribed from the same gene and are alternatively spliced to form functionally different proteins. The most common isoform, and the one mainly responsible for the lipid regulation, is perilipin A. Perilipin B does not protect against HSL hydrolysis and may act only to permit a basal level of lipolysis to provide a constant level of fatty acids.

There have been recent studies showing the relationship between perilipin expression and body weight maintenance. A perilipin knockout mouse model had 60 to 70 percent less adipose tissue compared to the control mouse. This dramatic difference in adiposity is likely because of an increase in hydrolysis of triacylglycerol in the adipocytes due to the lack of perilipin protection.

SEE ALSO: Hormone Sensitive Lipase; Protein Kinase.

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Peripheral Nervous System

THE PERIPHERAL NERVOUS system (PNS) connects the central nervous system (CNS) to sensory organs (such as the eye and ear), other visceral organs of the body, muscles, blood vessels, and glands. The peripheral nerves include the 12 cranial nerves, the spinal nerves and roots, and autonomic nerves that are concerned specifically with the regulation of heart muscle, the muscles in blood vessel walls, and other glands. The PNS sends nerve impulses for the periphery and trunk to the CNS in relation to hormonal and homeostatic controls. Indeed, the PNS is crucial in the regulation of energy balance in the body.

Obesity concerns the PNS in many facets such as cases of atrophy and necrotic tissue in extremities to complete loss of sensory and motor function when diseases such as diabetes mellitus are not properly treated or diagnosed. The PNS serves as the main conduit for all messages coming to and from the extremities to the CNS for processing.

The PNS regulates many hormones for body function. For example, the endocrine pancreas relies on the PNS to help control some metabolic processes. The function of the pancreas is to make insulin so that the body can use free glucose in the blood from food for energy. The pancreas also makes enzymes that help the body digest food. Spread all over the pancreas are areas called the islets of Langerhans. With these islets are cells that make chemical compounds such as glucagon, which raises the level of glucose in the blood while others make insulin. A third type of cell produces somatostatin which is a hormone that controls the rate of nutrient absorption into the bloodstream. These cells receive information from the PNS and CNS via neuronal connections to the pancreas that originate in the ventral hypothalamus, an area of the brain important to glucose regulation and body energy balance with metabolism.

For example, some such studies include animals and look at the genetic propensity to develop diet-induced obesity (DIO) by observing a reduced responsiveness to signals such as leptin and insulin. Obesity and Type 2 diabetes are on the rise in the United States and are major health concerns. Leptin and insulin are hormones that circulate in the blood in proportion to body fat stores and interact with their respective receptors expressed in key brain areas such as the hypothalamic arcuate nucleus (ARC) that regulate food intake and glucose metabolism.

The arcuate nucleus is a collection of neurons present in the hypothalamus, the center of and main CNS operator with masses of neurons that can send messages to other areas of the brain and body. When activated, these masses of neurons can produce ravenous eating desires and may be regulated by glucose, insulin, and leptin. The PNS plays a role in this signaling. Thus, arcuate neurons in the CNS are responding to information on whether the body has sufficient calories and nutrients and acting on needs of the body. Any malfunctions with the above homeostatic mechanism can lead to obesity.

The concept of feedback from the pancreas to the hypothalamus via insulin for the regulation of food intake and energy expenditure. This idea of a peripheral metabolic signal for the brain has been given a big boost recently with the discovery of the ob gene and its circulating adipose tissue hormonal gene product, leptin. Interestingly, leptin and insulin both appear to regulate the same neural circuit, the synthesis and release of neuronal NPY in the arcuate nucleus.

SEE ALSO: Autonomic Nervous System; Central Nervous System; Sympathetic Nervous System.

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Personal Relationships and Obesity

DECADES OF RESEARCH have documented the ways in which personal relationships, including family ties, friendships, and romantic relationships, can be beneficial to health and well-being. Social networks

can provide companionship, foster self-acceptance and esteem, give help and support in times of need, and motivate people to be their best. However, personal relationships can also be sources of negativity through strain, conflict, increased obligations, criticism, and rejection. One may question whether obesity makes any difference in personal relationships. That is, whether obese people have as many personal relationships as others; whether their relationships serve the same functions; whether these relationships are likely to be of higher, lower, or similar quality; and whether any of these factors change from childhood and adulthood.

RELATIONSHIP QUANTITY AND FORMATION

There is some evidence that obese people have fewer personal relationships compared to people who are not obese. This difference tends to emerge in childhood when obese children have greater difficulty making friends and are likely to be less popular, are lonelier, and even endure victimization by other children through teasing and bullying. Negative treatment from peers often continues through adolescence, a time when self-consciousness increases along with the perceived importance of conformity and peer acceptance. While many teenagers develop resilience and strategies to buffer themselves, teasing and rejection by peers can contribute to body dissatisfaction and unhealthy weight control practices can occur, such as taking diet pills and symptoms of eating disorders. There is some evidence that the relative dearth in friendships improves as adolescents age.

Overweight adolescents are less likely to participate in romantic relationships and dating activities in part because of the large emphasis on physical attractiveness. This difference is most pronounced for obese young women, who often begin dating at an older age and date less frequently. Obese individuals are somewhat less likely to marry and tend to do so later in life. There is also evidence that obese adults are more likely to marry other adults who are heavy.

Family relationships, including bonds with parents, siblings, and extended family members, vary less with weight status. There is great social pressure to maintain family ties out of obligation, even if they can be a source of conflict and negativity. However, because there is a strong genetic component to obesity, and because family members typically live in the same environment, family members are often obese themselves, and may be especially sympathetic and supportive. Even so, family members also have the widest latitude and may be the sources of damaging negativity. Family ties, and especially parents, influence psychological development, shape patterns of interaction with others, and contribute to the development of exercise and eating habits.

RELATIONSHIP FUNCTIONING AND QUALITY

There is some evidence that obese children and adults may have weaker, antagonistic, lower quality friendships. Stereotypes about obese people may contribute to negative interactions with peers, but peers' negative expectations can actually lead obese individuals to communicate less effectively, thus inadvertently confirming the negative expectation. Internalizing criticism, teasing, and negativity from peers helps to explain why overweight is associated with poorer psychological well-being. Stigmatizing experiences early in life can lead obese youth and young adults to distance themselves from social interactions. Obese individuals may come to expect rejection and become less willing to take risks on new relationships. Even so, most obese people find sympathetic friends who can provide companionship and help buffer against negative interactions, although the overall numbers may be smaller.

Although obese adults are slightly less likely to marry, there is little evidence that their marital relationships are of lower quality compared to the relationships of others. A few studies report increased levels of dissatisfaction, conflict, and marital problems, but many others find no differences. Indeed, obese couples do not have an increased risk of marital separation or divorce.

Childhood obesity can be a symptom of family strain and conflict as well as a cause. Parents may see their children's weight status as a sign of personal failure and a source of embarrassment, in addition to being concerned about children's long-term health and well-being. Arguments over food choices, physical activity, and weight control may undermine the quality of the parent–child relationship. Family members tend to be the most frequent sources of weight-related stigma because they have such prolonged, extensive contact, but they can also be sources of support, companionship, and motivation.

Weight status, eating habits, and exercise habits can be targets for negative exchanges among family mem-

bers across the life span. The emotional issues and physical limitations associated with obesity and may also pose challenges that evoke can more negative interactions with loved ones. Relationship partners may resent being caretakers and providing concrete and psychological support through these challenges.

PERSONAL RELATIONSHIPS AND WEIGHT CHANGES

Personal relationships can influence health behavior, including diet, exercise, and weight loss efforts in a number of ways. Close connections can become supporters and saboteurs of healthy habits, and sometimes both simultaneously.

Weight gain over the first years of marriage is typical and may even be an indicator of relationship stability. Marked weight loss, in contrast, can be evidence of marital problems, although it can have positive ramifications for the relationship in terms of increased social activity and sexual function. It may be that modest weight gain reflects stability and harmony, and pronounced weight loss or gain can signal problems.

Friends, romantic partners, and family members may feel an obligation to try to directly change their obese loved ones' habits, either for altruistic or selfish reasons. These attempts may take the form of monitoring, criticizing, and nagging, but they can also entail more constructive behaviors such as modeling healthy habits, working together, and encouraging. Spouses', supportive friends', and family members' involvement in weight-control efforts can be helpful. Working together can be superior to individual-focused interventions, especially for women who are trying to lose weight.

RELATIONSHIP MAINTENANCE

People enter into friendships and romantic relationships voluntarily, whereas family relationships tend to be sustained to a greater extent by obligation. Body weight may have a stronger influence on the continuation of voluntary relationships because there is an expectation that family ties will be maintained regardless of quality. If obese people are being treated badly, they may dissolve problematic friendships and romantic relationships. However, if obesity leads people to discount their worth and value themselves less, they may persevere in a lower quality relationship because of the perception that there are no higher quality alternatives.

It is more likely, however, that obese individuals choose to selectively maintain relationships. Supportive, fulfilling relationships with sympathetic, nonjudgmental others may endure as more conflict-ridden, problematic relationships end.

SEE ALSO: Appearance; Obese Women and Social Stigmatization; Parental and Home Environments.

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Pharmacological Treatment of Childhood Obesity

OBESITY IS THE most prevalent nutritional disorder among children in the United States. The number of obese children has risen significantly over the past two decades, likely due to inactive lifestyles and poor eating habits. Obesity in children is defined using body mass index (BMI), which is a calculation that uses a patient's height and weight to estimate the amount of body fat. After the BMI is calculated, it is plotted on the appropriate BMI-for-age growth chart (chart used differs based on sex and age). Children with a BMI above the 95th percentile are considered overweight. Patients with a BMI above the 95th percentile are considered obese. BMI is used as a screening tool for obesity, but other factors must be taken into consideration. These factors include diet, physical activity, family history, and other appropriate health screenings. Childhood obesity increases the patient's risk for several future problems including adult obesity, diabetes, high blood pressure, disease of the heart and blood vessels, lung diseases, and cancer. Therefore, it is very important that childhood obesity is managed appropriately.

The best treatment option for children with obesity is an age-appropriate behavior modification program consisting of exercise and dietary counseling. If behavioral modification fails, pharmacological treatment may be indicated for select patients based on the magnitude and extent of other medical conditions. However, research involving the management of childhood obesity with medications is very limited and the patient should be referred to a specialist before beginning therapy. Available medications for the treatment of obesity work by reducing food intake and increasing energy use or by preventing fat absorption.

Most appetite suppressants reduce food intake due to their effects on the brain. The brain triggers the release of norepinephrine, a hormone released in the blood stream. The body's response to norepinephrine is to increase blood pressure and heart rate, release glucose from storage centers, and break down fat stores. Most appetite suppressants including phentermine, diethylpropion, benzphetamine, and methamphetamine are only approved for short-term use and most of these drugs are limited to patients 16 years old or older (benzphetamine is approved in children 12 years old or older). Additionally, these drugs increase blood pressure and heart rate, increasing the risk of developing heart disease.

Sibutramine (Meridia®) is the only appetite suppressant approved for longer use (up to two years use per the manufacturer). Sibutramine works differently than the other appetite suppressants by blocking the reabsorption of norepinephrine, serotonin, and dopamine. However, similar problems limit its use including side effects of increased blood pressure and heart rate and it is only approved for children 16 years old or older. Other limitations for sibutramine's use include risk of serotonin syndrome, a rare but life-threatening adverse event that results from the presence of too much serotonin in patients on other drugs (including some antidepressants) which increase serotonin, weight loss levels off after approximately four to six months' use, and long-term continuation of the medication is needed to keep off the weight.

The drug orlistat (Xenical®) works by preventing the absorption of fat. Specifically, orlistat prevents the actions of chemicals in the stomach, thus preventing the fat from entering the body and increasing the amount that is removed. Orlistat has been effective in adults at decreasing body weight as well as decreasing cholesterol and risk of developing diabetes. However, in the United States, orlistat is only approved for children 12 years old or older. The use of orlistat in adolescents resulted in similar weight loss as adults, but adolescents had difficulty taking the medication due to known side effects. If dietary fat is not reduced, gas and diarrhea can be very bothersome for the patient. Additional issues with this medication include the risk of vitamin A, D, and E deficiencies even with daily supplementation, weight loss levels off over time, and long-term continuation of the medication is needed to keep off the weight.

Other agents that have shown some benefits include metformin and leptin. Metformin is an oral agent used in the treatment of diabetes to decrease the production of glucose by the liver, decrease absorption of dietary sugar, and improve the body's ability to use sugar. In obese adolescents, metformin decreased food intake, increased weight loss, decreased fat stores, improved cholesterol levels, and decreased the risk of developing diabetes. Although studies have shown metformin to be beneficial in childhood obesity, it is not approved for this use at this time. Leptin is a hormone in our bodies which has several important roles. Leptin helps our bodies maintain body weight and break down and utilize food for energy. Leptin deficiency is a rare genetic disorder, but in patients with this disorder, treatment with leptin leads to drastic weight reduction.

In conclusion, the role of medications for childhood obesity is very limited. Diet and exercise should be first-line therapy for childhood obesity and medications should only be used after failure of behavioral modification in patients at high risk for developing weight-related medical conditions. Very few antiobesity medications are approved in children due to lack of research. However, orlistat is appropriate in children as young as 12 years old and sibutramine can be considered in adolescents at least 16 years old. The side effects and long-term effectiveness of these medications also limit their clinical use. If pharmacological treatment of childhood obesity is started, it must still be combined with behavioral modifications including diet and exercise. Further research is needed to determine if any additional medications are safe and beneficial for childhood obesity.

SEE ALSO: Leptin Supplements; Metformin; Obesity and Drug Use; Orlistat (Xenical); Sibutramine (Meridia).

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Physical Activity and Obesity

PHYSICAL ACTIVITY IS defined as bodily movement (any form) produced by the contraction of skeletal muscles that increases energy expenditure above the basal level, and can be categorized in various ways, including type, intensity or strenuousness and purpose. Obesity is a condition describing excess body weight in the form of fat, with a body mass index (BMI) of 30 or greater. Obesity is often multifactorial, based on both genetic and behavioral factors. Physical inactivity has been found to associate with obesity because obesity arises from a mismatch between energy intake and energy expenditure that results in a net accumulation of energy stores, mainly as fat, in the body. Persuasive evidence indicates that both increased adiposity and reduced physical activity are strong and independent predictors of death.

PHYSICAL ACTIVITY AND ENERGY EXPENDITURE

Humans oxidize (metabolize) carbohydrate, protein, and fat (and alcohol) to produce energy that can be consumed in various forms of mechanical and chemical work and heat. The main component of the daily energy turnover in a typical subject is the energy expenditure for maintenance processes (resting energy expenditure). The remaining components are diet-induced energy expenditure and activity-induced energy expenditure. Diet-induced energy expenditure is less than 10 percent of total energy expenditure, depending on the macronutrient composition of the food consumed.

Activity-induced energy expenditure is the most variable component of the daily energy turnover, ranging between an average of 25 to 35 percent, up to 75 percent in extreme situations during heavy, sustained exercise as physical activity levels in free-living humans vary from day to day, seasonally, and in response to environmental factors.

The physical activity involves mechanical work, which must be performed by the muscles to move the center of mass of the segment. During the movement, energy expenditure will increase to propel body segments from one position to another in which mechanical work will be produced proportionally. Thus, work efficiency (the amount of work-/activity-induced energy expenditure) is one of major factors to determine activity-induced energy expenditure. The total amount of energy required by individuals depends on the level of activity, on work efficiency and on their body weight. The more active, heavier, and less efficient they are, the more energy they require. The rates of activity-induced energy expenditure vary depending on intensity, duration, and frequency of the activity and on the body mass and fitness of the person performing the activity.

There is currently a secular trend toward decreased physical activity in work time, which means that activity-induced energy expenditure during leisure time is becoming more dominant in determining total energy expenditure. Thus, physiology, behavior, and lifestyle play major roles in determining energy expended in activity.

PHYSICAL INACTIVITY AND MORBIDITY

Physical activity is a fundamental part of human life. It is a primary link with the environment around us. We can explore, interact with, and change our environment through physical activity. This influences our mental and physical health, which in turn influences our capacity to move.

Regular physical activity is associated with a decreased risk of all-cause mortality. Cross-sectional epidemiological studies and controlled experimental investigations have suggested that physically active persons, in contrast to their sedentary counterparts, tend to develop and maintain a higher level of metabolic fitness.

Metabolic syndrome is defined as comprising three of five lipid and nonlipid risk factors of metabolic origin (large abdominal circumference, hypertriglycidemia, low high-density lipoprotein (HDL), high blood pressure, and high fast blood glucose). Physical activity diminishes the magnitude of all five risk factors.

Existing data from multiple studies also have demonstrated the clear significance between physical inactivity and risk of several chronic diseases, including coronary heart disease, hypertension, noninsulin-dependent diabetes mellitus, osteoporosis, cancers, and anxiety and depression. As many as 250,000 deaths per year, approximately 13 percent of total deaths, are attributable to lack of regular physical activity.

A low level of physical activity is a major risk factor for excess fat gain, and increased participation in physical activity is associated with a reduced risk of obesity. Obese persons engage in low levels of habitual physical activity compared to their nonobese counterparts. The available data seem to indicate a secular decline in overall physical activity that has occurred at the same time, or possibly before, the temporal increase in obesity. It is likely that physical inactivity contributes significantly to the development of obesity.

PHYSICAL ACTIVITY MEASUREMENT

Determination of the amount of physical activity needed to derive specific health benefits requires accurate and reliable methods of measuring energy expenditure in free-living individuals. Currently a number of methods have been utilized to assess physical activity and its contribution to energy expenditure during physical activities. These vary significantly in their approaches, accuracy, and usefulness.

Direct observations provide a comprehensive and accurate physical activity assessment tool that allows characterization of habitual physical activity either directly or after review of film or videotape and diverse dimensions related to physical activity can be quantified, and involve little interference with the participant's routine. This technique can be used as a criterion method for validating other measures of physical activity, and can be used for short-to-moderate time periods in a variety of settings. However, it is time consuming, is not practical for large-scale investigation, and may lead to alterations in normal behavior. Results are also difficult to quantify and to compare between subjects.

Physical activity index (PAI) composite score is calculated by summing the number of hours spent in each activity intensity level and multiplying by a respective weight factor derived from the estimated oxygen consumption requirement for each intensity level. PAI is frequently used to analyze data in large-scale epidemiologic studies. PAI is useful in energy balance studies and can be used as a measure for validating physical activity survey questionnaires. However, individual variability in physical activity and energy expenditure patterns reduces the accuracy of physical activity index. Accuracy of physical activity index in estimating daily energy expenditure also depends on recorded details and the reference energy cost of each activity used to develop the composite physical activity scores.

Survey questionnaires and interviews are designed to assess physical activity by self-reported responses or interviewer-completed assessments. Survey questionnaires or interviews provide a large volume of information on a variety of physical activity relative to their cost and time invested. Survey instruments are easy to administer and are unobtrusive. For these reasons, they are perhaps the most popular types of physical activity assessment tools for epidemiological study. One universal drawback of surveys is the changeable nature of individual physical activity.

Recall questionnaires assess "usual" or actual physical activity participation during the previous one to four weeks. Many such questionnaires have been validated against measures of aerobic fitness, total and HDL cholesterol levels, body composition, physical activity and food records, and motion detector readings. The principal advantages of recall questionnaires are that they provide information about specific types of physical activity performed, are easy to complete, and allow quantification of physical activity during the period assessed. However, an important limitation is that physical activity recalled from a previous week or month may not accurately represent an individual's true year-round activity pattern.

There are mechanical and electronic devices for objective recording of physical activity. A pedometer is a small, simple, and noninvasive mechanical movement counter that is clipped to a belt at the waist or worn on the ankle. Pedometers tend to underestimate distances walked at slower speeds and overestimate distances during fast walking or running. The main shortcoming of pedometers is that they are insensitive to gait difference such as stride length, which can vary significantly among activities and from person to person. The large-scale integrated motor activity monitor (LSI) is a simple device that uses a mercury

switch sensitive to a 30-degree tilt in a single axis. The LSI was used to distinguish between groups of adults who differ significantly in physical activity status. However, LSI readings correlate poorly with estimated energy expenditure levels during walking, running, and bicycle riding. Similar devices such as wrist/ankle watches and actometers also exist. Because most of these devices used switches as sensors, their "on" or "off" categories of information can only give qualitative type of results with limited information on the intensities of physical activity. Electronic load transducers and foot contact monitors were reported which could be inserted into the heels of shoes to monitor loads held, lifted, or carried, and walking activity. However, due to technical and practical limitations of these measuring techniques, these devices have not been used widely in epidemiological research, and little information is currently available on their accuracy in assessing habitual physical activity status and daily energy expenditure.

Modern accelerometers use piezoelectric or piezoresistive rods to sense accelerations in three-dimensional space. Because the electric signal output from the sensor(s) is proportional to its acceleration, it is possible to quantify the intensity of movement. Most devices operate by embedding one set of sensor(s) (one to three axes) in an enclosure together with circuitry, and attaching the enclosure to a single location on the body, usually on the waist (most common because it is closest to the center of body mass) or on an ankle or wrist. In several validation studies using these monitors, correlation values of *r* ranged from 0.65–0.92 between energy expenditure measured by indirect calorimeter and accelerometer readings during various activities, where level walking showed the highest correlation with the waist-worn triaxial accelerometers after individual calibration (r = 0.99). This indicates the excellent reproducibility of the device. The advantages of this class of devices include small size, noninvasiveness, low cost, and minimal intrusion on normal subject movements during daily activities. The duration, frequency, and to some extent, intensity are also measurable.

The major problem is that the device only detects moving or shaking of the sensor that is attached to a single part of the body. It is not "smart" enough to know what type of physical activity is performed. Therefore, these devices cannot determine related mechanical work, power, and energy expenditure of the activity. The single sensor location does not allow the device to detect movement coordination of the limbs or to determine postures. Sensor output could be higher for a person driving a car than that of the same person riding a bike.

Heart rate monitors present a simple and objective method for the estimation of energy expenditure during certain levels of physical activity/exercise and can provide minute-by-minute data for up to several days. Results from treadmill and bike tests showed a close-to-linear relation between energy expenditure and heart rate over the range from moderate to relatively intensive physical activity levels. However, various factors can influence heart rate in the absence of physical activity but not proportionally change energy expenditure. Fatigue, state of hydration, body temperature, substances such as caffeine and ephedrine, and emotional state (startle, panic) all affect the heart rate–energy expenditure relationship. The heart rate–energy expenditure correlation values range between 0.7 and 0.8. In addition, heart rate monitors cannot distinguish accurately between light and moderate intensity activities,

Doubly labeled water (DLW) is an alternative approach for estimating energy expenditure in humans and is applicable for use in both laboratory and field studies. DLW estimates energy expenditure by measuring CO_2 output in the free-living state and has been frequently used as a gold standard under free-living conditions. The validity of the DLW technique for measurement of energy expenditure under controlled conditions appears satisfactory. However, it is expensive and takes at least three days to get only average daily energy expenditure without detection of type, onset, duration, and intensity of components of physical activity and their energy cost.

Pressure insoles were designed to provide a plantar pressure distribution, which is used to decide the physical activity. Plantar pressure measurement systems offer the researchers a high degree of portability, permitting utilization among multiple clinic sites. Although in-shoe data collection would appear to be a good choice for the clinician, there are several problems associated with this technique. Because the number of sensors that can be incorporated into the pressure sensor insole is less than the possible number of sensors used in a force platform system,

the resolution is usually diminished. The sensor insoles are more susceptible to mechanical breakdown because transducer cables connecting the sensors to the computer can be bent or stretched as they exit the shoe. Individual sensors can also be damaged by continuous repetitive loading. In addition, the hot, humid, and usually contoured environment within the shoe can affect the reliability and validity of measurements of the sensor's performance. As the sensor insole can only measure "normal" force because of the position of the insole sensor within the shoe relative to the supporting surface, the measurement of "true" vertical does not occur during the initial and late portions of the walking cycle.

Multiple sensors system use multiple sensors placed on different locations of body. The multiple sensors system is able to integrate information from multiple sensors and provide direct results—the type, frequency, duration, and intensity of physical activity and the estimation of energy expenditure, which are easier to understand and use. The inconvenience of wearing multiple sensors is a major limitation for the application. In addition, the high cost and less reliable nature of the system has prevented its use in the large population study.

OBESITY AND MORBIDITY

Since the 1980s, there has been a doubling of the prevalence of obesity among adults, and a tripling of the number of children who are overweight. At present, 31 percent of adults are obese and 16 percent of children are overweight.

Morbidity-associated obesity is striking. Obesity is a risk factor for Type 2 diabetes mellitus, gallbladder disease, hypertension, dyslipidemia, insulin resistance, breathlessness, sleep apnea, coronary heart disease, osteoarthritis, hyperuricemia, gout, cancer, and several other medical conditions.

The risk of diabetes increases as BMI increases, with the relative risk of diabetes increasing by about 25 percent for each unit of BMI over 22. Overweight and obesity are also associated with increased morbidity and mortality from coronary heart disease (CHD). There is a threefold increase in rate of coronary heart disease in women with BMIs of 29 or greater compared with women with BMIs less than 21, and a 10 percent increase in coronary events in men with each BMI unit above 22.

Hypertension prevalence increases from 16 to 18 percent to 32 to 38 percent as BMI goes from less than 25 to greater than 30, and a similar relationship, although not as dramatic, is seen between BMI and high blood cholesterol. These problems can be ameliorated, or sometimes reversed, through weight loss.

PHYSICAL ACTIVITY IN THE PREVENTION AND TREATMENT OF OBESITY

Physical activity is the only component of total energy expenditure that can be voluntarily modified. Therefore, it is a reasonable behavioral target for the prevention and treatment of obesity. Chronic changes in physical activity can produce changes in energy requirements, which if not compensated for, can lead to changes in the levels at which body weight and body composition are maintained over time. This is especially true for understanding the etiology of obesity because physical activity can significantly affect energy intake, energy expenditure, and body composition.

Combining changes in diet and exercise can lead to greater short-term weight loss than changes with restriction in energy intake alone. Continued physical activity/exercise after a weight-loss program is probably the most effective way of controlling the rapid postprogram weight regain.

As one of three principal components of lifestyle modification (diet, physical activity, and behavior therapy), the physical activity has been promoted in all prevention and treatment programs of obesity. However, the optimal level of physical activity and dose-response effect of physical activity is still controversial as a higher level of physical activity is not necessary to negate the risk associated with adiposity.

It is comprehensive task to adopt physical activity in the prevention and treatment of obesity. The wiliness and persistence over long period of time for participants to follow the physical activity guideline is first key factor for success. The method for effective and accurate assessment and measurement of physical activity is another challenge. The most difficult part of prevention and treatment of obesity is to develop an effective activity plan, which includes frequency, intensity, duration, and type of activity. The CDC and the American College of Sports Medicine recommend at least 30 minutes of moderate-intensity physical activity on most, preferably all, days of the week, while compelling evidence that

prevention of weight regain in formerly obese individuals requires 60–90 minutes of moderate intensity activity or lesser amounts of vigorous activity.

Although it is abundantly clear that public health recommendations must focus on the benefits of both a healthy weight and regular physical activity, when it comes to a simple question—"How much physical activity do we need to prevent obesity and how much physical activity do we need to treat obesity?"—definitive data are lacking.

The 1996 Surgeon General's Report on Physical Activity and Health concludes that physical activity is important for weight control, primarily because of the positive findings from studies testing the effects of physical activity on weight loss. However, the report also stated, "It is commonly believed that physically active people are less likely to gain weight over the course of their lives and are thus more likely to have a lower prevalence of obesity than inactive people: accordingly, it is also commonly believed that low levels of physical activity are a cause of obesity. Few data, however, exist to evaluate the truth of these suppositions." The research to determine the most important features of physical activity that confer specific health benefits is in urgent need.

Education about the long-term health consequences and risks associated with obesity and how to achieve and maintain a preferred weight is necessary. While many individuals attempt to lose weight, studies show that within five years, a majority of them regain the weight. To maintain weight loss, good dietary habits must be coupled with increased physical activity, and these must become permanent lifestyle changes. It is still not clear, however, which behavioral approaches are best for achieving these changes, particularly long term. A 1998 National Heart, Lung, and Blood Institute workshop on Maintenance of Behavior Change in Cardiorespiratory Risk Reduction concludes that additional research is needed to examine factors associated with long-term maintenance of weight loss, long-term maintenance of increased physical activity levels, and the relationship between the two.

The question of whether physical activity enhances long-term maintenance of weight loss has not been formally examined in randomized trials. The relationship between physical activity and obesity appears to be complex and requires further study.

SEE ALSO: Physical Activity in Children; Physical Activity Patterns in the Obese.

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Physical Activity in Children

THE PREVALENCE OF childhood obesity is rising dramatically in the United States as well as in many other countries. Compounding the problem is the fact that children are not as physically active as they were in decades past. Although physical activity has been shown to decline throughout the lifespan, the decline in physical activity is greatest during adolescence. In addition, girls are less active than boys at all ages. Children can benefit from regular physical activity, structured exercise, and specific exercise training regimens. However, the volume and intensity of exercise in which children participate needs to be monitored, because children are at greater risk for musculoskeletal injury, heat disorders, and emotional distress than adults. Injury prevention can best be achieved by encouraging children to participate in exercise for fun, at moderate intensities, and in moderate environments.

Recent research suggests that as children grow and mature, their level of daily physical activity decreases.

Boys and girls between 9–13 years old show a dramatic decline in physical activity. When comparing chronological ages, boys' decline in physical activity is less pronounced than girls. However, when comparing the genders by biological age, the gender differences in physical activity disappear. However, both genders show a similar decline in physical activity with biological maturity. It is not difficult to surmise that as these young people continue on through high school, their physical activity levels will continue to decline, when considering only 33 percent of high school students participate in physical education.

There has been a recent upsurge in the number of children who are contracting Type 2 diabetes, previously know as adult-onset diabetes. Type 2 diabetes is a disease that previously was only rarely seen in adults under 40 years old. More children are being diagnosed with Type 2 diabetes, most between 10–19 years old, the same age where physical activity and participation in physical education declines. Because regular physical activity increases insulin sensitivity, the declining number of physically active children is thought to be a major contributor to this trend in the prevalence of Type 2 diabetes in children.

Recent research suggests that as children grow and mature, their level of daily physical activity decreases.

Because the rise in the prevalence of obesity in children is a relatively new phenomenon, the body of research that deals with pediatric exercise prescription is relatively scant. Up until now, the need for deliberate exercise programming for children has been minimal, because historically, children have been quite physically active. While it is generally accepted that conventional aerobic exercises such as running, swimming, and cycling are beneficial for children, misconceptions surrounding the efficacy and safety of strength training for children unfortunately persist.

The most common concern regarding strength training or resistance training and children involves the risk of damage to underdeveloped or immature tissues. It is generally agreed that incorporating resistance training exercises that do not use maximal loads will not result in trauma to these tissues. In fact, according to the National Strength and Conditioning Association, there are no justifiable safety reasons to preclude prepubescent individuals or adolescents from participating in a properly designed and supervised resistance training program. Resistance training in children has been shown to increase strength beyond what is expected during normal growth and development. In adults, resistance training increases the strength of ligaments, tendons, and bones, which decreases the risks of injury. It is likely that these same protective effects occur in children as well. The benefits of resistance training for obese adolescents have also been documented.

Obese adolescents who undergo several weeks of circuit-type resistance training program show a reduced body fat and normalized vascular blood flow. Reductions in body fat following resistance training are likely brought about due to an increase in muscle mass which leads to an increase in one's metabolic rate. Resistance training has also been shown to improve insulin sensitivity and glucose tolerance in patients with Type 2 diabetes.

Properly designed resistance training programs for children should target all the major large muscle groups, and should incorporate multijoint exercises such as the barbell squat, dead lift, and bench press.

Training intensity should initially be light but progress toward loads that allow for the completion of six to 15 repetitions. Training should occur two to three times per week on nonconsecutive days, with one to three sets per exercise.

The current position of the National Strength and Conditioning Association is that a properly designed and supervised resistance training program

1. is safe for children;
2. can increase the strength of children;
3. can help to enhance the motor fitness skills and sports performance of children;
4. can help to prevent injuries in youth sports and recreational activities;
5. can help to improve the psychosocial well-being of children; and
6. can enhance the overall health of children.

Many children and adolescents participate in organized sports and personal fitness programs designed to develop athletic skills and increase fitness. However, new injury patterns are developing as the focus of these programs shifts from free play to regimented competition. An estimated 50 percent of all injuries or harm sustained by children and adolescents while playing organized sports are preventable. The major portion of responsibility for safe participation in exercise and sport for youth lies not with the youth, but with adults. To provide the safest environment for children participating in exercise and sport, adults need to understand how and when children are most susceptible to exercise-induced harm.

Exercise harm for youth can be classified into two categories: physical injuries and emotional distress. Children are most susceptible to physical injuries during growth periods, and most of these injuries are related to the shearing forces that act upon the musculoskeletal system during intense exercise. The growing bones of children are at greater risk for mechanical injury than mature bones of adults because the ends of the growing bones are less dense and relatively weak. In addition, the ends of the bones are more prone to joint injuries because of undeveloped cartilage and tendons, which are connective tissues that connect bone to bone and muscles to bone. This risk can be overcome by not having children participate in heavy strength training and intense prolonged aerobic training.

Another physical problem that children face is their inability to dissipate heat, which puts them at greater risk for heat disorders than the exercising adult. This problem can be avoided by not allowing children to participate in prolonged intense exercise in hot, humid environments. Children should also be encouraged to drink plenty of fluids during physical activity, even when they are not thirsty.

Many children are specializing in sports at an early age and train year-round to compete at a higher level. The ever-increasing requirements for success create a constant pressure for athletes to train longer and harder. Pressure to win or perform at a high standard is probably the greatest stressor inducing psychiatric illness in young athletes. The focus of sport for children should not be competition and children should not specialize in sports until after adolescence.

SEE ALSO: Physical Activity and Obesity; Physical Activity Patterns in the Obese.

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Physical Activity Patterns in the Obese

THE PREVALENCE OF obesity in the United States is rising at the same time that participation in physical activity is declining. Although exercise can reduce obesity, most Americans cannot or will not exercise enough to achieve a normal weight. However, the health benefits of exercise can be achieved without weight loss.

The Centers for Disease Control and Prevention reports that over 50 percent of U.S. adults do not get the

Obese individuals can receive the benefits of regular exercise, even if their body weight does not normalize.

recommended amount of physical activity, while 24 percent get no physical activity at all. It is reported that from 1980 to 2004, obesity increased in adults aged 20–74 years from 15 to 33 percent. It seems the rise in obesity is simply a reflection of our sedentary lifestyle. In 2005, the three states with the highest rates of obesity—Mississippi, West Virginia, and Louisiana—were also among the highest for rates of physical inactivity. When considering the recommended 30 minutes or more of moderate physical activity per day, these three states report that about 60 percent of their adult population fall below the recommendation.

The news is just as disheartening for children and adolescents. Since 1980, the percentage of children who are classified as overweight has tripled. Today, 16 percent of children between ages 6–19 are considered overweight. More than one-third of adolescents between grades 9 and 12 get no regular vigorous physical activity. Participation in high school physical education classes has dropped from 42 to 33 percent between 1991 and 2005. These numbers are worrying because research has shown that obese adolescents have a 70-percent chance of becoming obese adults. Adult obesity increases the risk of high blood pressure, diabetes, heart disease, and some cancers. Clearly, it is imperative that the message of the role of daily physical activity in the lives of the obese, as well as normal-weight population, be made a priority by the American health and medical professions today.

Little information exists that focuses on the factors that influence the exercise behaviors of obese individuals. Intuitively, one might expect that increasing body fatness might be a motivating factor to increase one's physical activity. However, an equally plausible opposing view might be that an increase in body fat might cause one to be more self-conscious or embarrassed about one's appearance, resulting in a decreased desire to exercise, especially in public.

Still another argument might be made that obesity per se may not influence exercise behavior at all, and that the exercise behaviors of obese individuals are no different than those of normal-weight individuals. With regard to exercising in public, research shows that one's intention to exercise at a health club is significantly reduced as one's body mass index (BMI) increases. That is, as people become fatter, they have lower intentions of exercising at a health club. Interestingly, this significant negative relationship between body fatness and intention to exercise at a club disappears when one's perception of one's health is factored into the equation along with BMI.

In short, obese people who equated being overweight with poor health are more likely to report a lower intention to exercise at a health club, while obese people who perceive themselves as being in good health are just as likely to exercise in a club as nonobese people. Similarly, normal-weight people who perceive themselves as being unhealthy also report lower intentions to exercise at a health club than those seeing themselves as healthy.

On the other hand, it is unknown whether people who currently exercise in a health club are more likely to perceive themselves as being in good health, or whether perceiving oneself as being in good health is what motivated one to exercise in a club. It is likely that both factors influence each other in a positive-feedback cycle; exercising at a club improves one's perceived and actual health, which in turn increases one's intention to exercise. Thus, an obese person's

self-perception may be the dominant factor in his or her exercise participation.

Previously, obese individuals who maintain a reduced body weight report that regular exercise is the greatest factor responsible for their prolonged weight maintenance. Research has determined that for a previously obese person to maintain a reduced body weight, he or she needs to perform moderate intensity exercise for 80 to 90 minutes a day. This translates into burning approximately 700 kilocalories each day in exercise. Unfortunately, to expend 700 kilocalories in exercise, a person needs to walk or jog about seven miles. This amount of exercise is beyond the reach of most obese people, and presents a tremendous barrier to long-term weight control. Moreover, many obese individuals do not exercise at all, or may have medical conditions that preclude or limit exercise participation. Thus, the expectation of obese people exercising enough to obtain and maintain a normal body weight seems unrealistic.

On the other hand, obese individuals can receive the benefits of regular exercise, even if their body weight does not normalize. Exercise lowers blood pressure, lowers cholesterol, and improves blood glucose control in the obese, even without weight loss. Furthermore, these health benefits of exercise are manifest in as few as seven days with only 30 to 60 minutes a day of moderate intensity exercise.

Although the obese individual may have more health problems than his or her normal-weight counterpart, obesity per se does not alter the physiologic response to exercise. At the same relative intensity—meaning the same percent of maximal capacity—the obese person's body responds the same as a normal-weight person. Thus, in most situations, the healthy obese person can follow the same exercise guidelines as the nonobese.

Research also shows that the obese person can improve his or her fitness level without weight loss. When obese individuals participate in a fitness training program, they improve at the same rate and degree as previously sedentary nonobese individuals. This includes all indicators of fitness (except weight): aerobic capacity, muscular strength and endurance, and flexibility.

SEE ALSO: Physical Activity and Obesity; Physical Activity in Children.

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Physician-Assisted Weight Loss

THE PREVALENCE OF obesity has reached mammoth proportions with over 60 percent of the U.S. population being overweight or obese. It is well known that being obese or overweight increases the chances of developing heart disease, cancer, gallstones, high blood pressure, diabetes, obstructive sleep apnea, and arthritis, to name a few. In fact, more than 400,000 annual deaths can be attributed to obesity-related medical conditions. Obese and overweight people are thus more likely to seek medical care and be hospitalized. Physicians who take care of these patients are best suited to motivate, advise, and follow up on weight-loss efforts of their patients. Several weight-loss therapies also require physicians to monitor patients because of the risk of complications and even death during rapid weight loss.

HISTORICAL ASPECTS OF MEDICAL WEIGHT LOSS

Historically, several methods have been tried for weight loss. Thyroid hormones and amphetamines both induce weight loss, but there was a tendency to regain weight after these medications were stopped. Moreover, the potential for abuse was high with amphetamines. Thyroid hormones, act by increasing the body's metabolic rate (rate at which body burns calories to provide energy). However, these hormones have side effects and can precipitate arrhythmias (abnormally fast or irregular heartbeat) and heart failure. Phen-fen or phentermine and fenfluramine were two

drugs approved by the Food and Drug Administration in 1996. This combination was withdrawn from the market after a number of alarming reports on the incidence of cardiac valve and lung problems among users. Phenteremine alone is still prescribed. Phenylpropanolamine hydrochloride was also withdrawn after reports of bleeding in the brain (cerebral hemorrhage). Gastric vagotomy in which the vagus nerve supply to the stomach was cut, failed to produce appealing results and was abandoned.

PATIENT AND PHYSICIAN BARRIERS TO WEIGHT LOSS

It is a well-known fact that weight loss can have far-reaching benefits. Despite this, overweight and obese patients do not always receive advice on healthy eating and exercise from their doctors. At least 40 percent of obese patients are not told by their physicians to lose weight. Among those patients who are already on treatment for various disorders such as high blood pressure, diabetes, and heart disease, less than half are never counseled on weight loss. It has been shown that being advised by one's physician is a potent stimulus to attempt weight loss. In fact, obese subjects are up to three times more likely to lose weight if advised by their healthcare team.

The reluctance of physicians to counsel and assist in weight loss could be due to several reasons. Obesity was originally considered a social problem resulting from overeating and a lack of willpower. It was not recognized for what it is now—a medical problem with a genetic basis that can be modified by lifestyle factors such as stress, exercise, and eating habits.

Even today, despite the magnitude of the problem, doctors do not receive adequate training in weight-loss management in medical schools or residency. Doctors often feel that patients may not act upon their advice to lose weight. In addition, most insurance companies do not reimburse physicians for time spent on patient counseling.

Very often, patients who consult physicians for weight loss or for medical conditions associated with obesity are frustrated from previous failed attempts. Individuals who are successful at losing weight initially may be discouraged when they cannot lose more weight or the rate of weight loss slows down. This is usually referred to as a plateau phase. A significant proportion regains weight within a year of initial weight loss. A good discussion between patients and their doctors about these common problems can help patients better tackle them and not quit their efforts.

Depression exists in 20 to 30 percent of obese patients and is often a key factor that may be overlooked. Such patients are unlikely to benefit from the usual weight-loss advice and programs if depression is not treated.

DIAGNOSING OBESITY

Recognizing and diagnosing obesity is the first step. The current definition of obesity is based on body mass index (BMI). Calculation of BMI can easily be done in a physician's office from a person's height and weight. BMI above 25 is overweight, above 30 is obese, over 40 is morbidly obese, and over 50 is super-obese.

An alternative way of defining obesity is by directly estimating body fat. There are several instruments available on the market called bioimpedance analyzers (BIA) that can directly calculate body fat. BIA machines cause a small amount of electricity to pass through the body and directly display the percent body fat. In men, a fat percent above 25 percent and in women over 30 percent is considered obese.

These portable or handheld devices can have a margin of error but are suitable for use in health clubs, gyms, and doctor's offices. Dual X-ray absorptiometry (DEXA), which is popularly used to check bone density, is another method to accurately determine the body's fat percentage.

Once a weight problem has been identified, the physician should find out the patient's readiness to start a weight-loss program. In addition, specific medical problems such as depression, medications that contribute to weight gain, unhealthy eating patterns, and previously failed weight-loss attempts should be identified and addressed. Patients undergo physical examination and blood tests to help identify conditions such as abnormal hormone production, and liver, kidney, and heart disease that can lead to weight gain. Such patients require management of the underlying medical problem and will not benefit from diet or exercise alone.

FORMULATING A WEIGHT-LOSS PLAN

During the initial weight-loss planning and also during follow-up visits, physicians can ensure that a balanced diet with all essential vitamins and minerals is being consumed. The percent intake of carbohydrates,

fats, and proteins in the diet should be tailored to the specific patient's need. Referral to a dietitian is particularly helpful for people with diabetes, hypertension, and heart failure. Another important role that doctors can play is to constantly encourage and reinforce patient efforts to lose weight and adopt healthy lifestyle changes. Continued encouragement has been shown to be a key factor in successful weight-loss programs.

The National Institutes of Health recommends treatment for any patient with a BMI over 30 or for those with a BMI between 25 and 29.9 and two or more risk factors such as diabetes, coronary artery disease, hypertension, and so forth. Currently, the treatment modalities offered to patients for weight loss are lifestyle changes, which include diet and exercise; weight-loss medications; and weight-loss surgeries. The advantages and disadvantages of each of these are discussed below.

POPULAR DIETS

Physicians are often sought out by their patients to give advice when choosing a diet or deciding on a commercial weight-loss program. There are several weight-loss diets and programs available. Head-to-head comparisons of the different programs have not been conducted.

The National Cholesterol Education Program, the American Heart Association, and the National Institutes of Health in 1984 promoted a low-fat, high-carbohydrate diet. There is controversy on whether this has inadvertently contributed to the current epidemic of obesity.

Some of the popular diets are Atkins, South Beach, and Weight Watchers. The Atkins Diet is the most popular diet in the United States. Over 40 million people around the world have tried this diet. It permits fat and protein intake, but discourages high carbohydrates in the diet. A lifetime approach to weight loss is the cornerstone of the weight-loss philosophy. When the Atkins Diet was introduced as a low-carbohydrate, high-protein diet, medical literature to support it was scarce. There is, however, mounting evidence that higher protein diets help in weight loss and improve muscle mass, blood glucose control, and blood cholesterol.

The South Beach Diet is similar to the Atkins Diet and is based on the premise of eliminating simple carbohydrates. It is not a low-carbohydrate or low-fat diet. A balanced meal plan incorporating vegetables, grains, fruits, and lean proteins can induce up to 8–14 pounds of weight loss in two weeks.

The Mayo Clinic Diet (not endorsed by the Mayo Clinic) is a low-carbohydrate diet that encourages consumption of fatty foods and grapefruit. It is based on the premise that fatty foods cause early feeling of fullness. This would help restrict the total calories consumed.

The Mediterranean diet is flavorful and rich in fruits, breads, cereals, and olive oil. It has a low amount of saturated fatty acids and is high in monounsaturated fats, primarily thought to be due to the use of olive oil.

Overeater Anonymous is a free, 12-step program that provides support to members especially those with binge eating and overeating related to stress. No diet plans are given and members usually follow their doctor's advice.

VERY LOW-CALORIE AND HIGH-PROTEIN DIETS

Very-low-calorie diets can induce rapid short-term weight loss in individuals with a BMI above 30. It utilizes commercial bars or meals that have the recommended dietary quantities of micronutrient and vitamins. These diets require physician supervision and can cause side effects which include gallstone formation, nausea, or in rare instances, even death. Medical supervision is mandatory in the United States, but regulations are less stringent in Europe and other countries. Very-low-calorie diets should not be started in pregnant women, cancer patients, psychiatric patients, and those who have recently recovered from a stroke or a heart attack. Information on very-low-calorie diets is available from the National Institute of Diabetes and Digestive and Kidney Diseases Web site at http://win.niddk.nih.gov/publications/low_calorie.htm.

High-protein diets provide 30 to 50 percent of daily caloric requirements from protein. While these diets are useful for weight loss, there is also a higher chance of developing kidney stones and osteopenia (thinning of bones).

EXERCISE

The American College of Sports Medicine (ACSM) has specified exercise guidelines for weight management. The goal of exercise should be to attain a 10

percent weight loss over six months with a 1–2 pound loss per week. Incorporating a regular physical activity program is the best predictor of long-term weight loss and maintenance. Aerobic and resistance exercises can both help in achieving weight loss. Before starting a regular planned exercise, it is sometimes necessary to estimate a person's baseline exercise capability. Healthy individuals can start an exercise regimen without any formal testing, but those with medical disorders, for example, heart disease, lung disease, and so forth, should be tested before they can safely start exercising. If your physician deems it necessary, you can be referred to an exercise physiologist or exercise specialist. These are professionals who can evaluate people with medical conditions and determine their fitness level before commencing exercise. They can assist in designing a program that can fit individual needs and goals.

Aerobic exercises such as running, swimming, brisk walking, and playing tennis improve your heart's ability to deliver oxygen to the body cells. It also helps burn fat and induce weight loss. Resistance training, which involves improving muscle strength by subjecting muscles to resistance or force, for example, lifting weights, are also recommended. These exercises decrease the body's overall fat percentage and increase muscle mass. Building muscle increases the amount of energy burned by the body even at rest, thus helping long-term weight loss and maintenance. Weight training is also proven to improve bone strength and may prevent the development of osteoporosis in both men and women.

MEDICATIONS FOR WEIGHT LOSS

Use of medications should be considered only after nondrug interventions have been attempted for at least six months. The total expenditure in 2002 on weight-loss medications amounted to $362 million, with patients paying 26 percent out of pocket and insurance plans covering the remaining 74 percent.

Medications for weight loss act in three different ways. They may act on the brain to suppress appetite, act on the intestines to reduce absorption of fat, or act to increase the energy burned by the body.

Anorexiants are medications that suppress the appetite by stimulating the release of the chemicals dopamine and norepinephrine in the hypothalamus (part of the brain that controls feeding). Benzphetamine, diethylpropion, mazindol, phendimetrazine, and phentermine are approved for weight reduction in the United States. However, phentermine (Adipex®, Lonamin®) is most often prescribed and is for short-term use only. As tolerance to the drug develops, efficacy is lost. It has amphetamine-like properties, but has low addictive potential. Side effects include dry mouth, nervousness, constipation, and high blood pressure.

Sibutramine (Meridia), on the other hand, is indicated in the long-term treatment of obesity as its effect can last at least one year. It also inhibits the appetite but by a different mechanism in the brain. It produces the best results when used in conjunction with low-calorie diets. It should not be used in those who have recently suffered a heart attack or stroke, or suffer from abnormal or irregular heart rhythms.

Orlistat (Xenical®) acts in the intestine to activate the enzymes that help in fat absorption. It can also be used for the long-term drug treatment of obesity. Weight loss with orlistat and diet has been shown to improve diabetes control. Because orlistat is not absorbed into the body, it does not cause side effects similar to the others mentioned. The main complaints are bloating, abdominal pain, and decreased absorption of fat-soluble vitamins A, D, E, and K. Caution is advised in those taking cholesterol-lowering medications called statins. Vitamin supplements are usually prescribed along with orlistat.

Orlistat, sibutramine, and phentermine cost $120, $100, and $40 per month, respectively [figures from 2007]. Rimonabant is a new drug that has been approved by the FDA for treatment of obesity but is not yet available on the U.S. market. It is available in the United Kingdom for treatment of obesity and smoking cessation. It blocks a specific type of receptor, CB-1 or type 1 cannabinoid receptors. These receptors are found in the brain and fat cells. These receptors send signals that stimulate the body to overeat. Trials have shown that when used in obese individuals, rimonabant decreases appetite, leads to weight loss, helps in smoking cessation, and improves the high-density lipoprotein (HDL) cholesterol (aka "good" cholesterol). All these can reduce the chance of developing heart disease.

SURGICAL APPROACHES

Bariatric surgery is the term used to describe weight-loss surgery. The method by which surgery works in weight loss is by decreasing the capacity of the stomach to accept and store food. The two most common

methods are gastric laparoscopic (gastric lap) banding and gastric bypass. Surgery as a treatment modality for obesity is recommended when BMI is greater than 40 or for those with a BMI over 35 if they have one or more medical conditions. These conditions include diabetes, obstructive sleep apnea, severe hypertension, severe degenerative arthritis, and severe interference in the ability to carry out regular activities of living.

Gastric bypass produces excellent weight-loss results. Parts of the stomach and small intestines are removed. This reduces the appetite and can result in up to 50 to 75 percent weight loss. Gastric lap surgery is a reversible procedure that involves putting a restrictive band around the upper part of the stomach. Both procedures are equally effective. Gastric lap band requires a shorter time to complete.

NONMEDICAL COMMERCIAL WEIGHT-LOSS PROGRAMS

Three of the largest nonmedical commercial programs are LA Weight Loss, Jenny Craig, and Weight Watchers. These programs include dietary advice, behavior counseling, and exercise recommendations. Weight Watchers-sponsored research trials show that people can lose and keep off pounds lost for up to two years after being on a Weight Watchers program. The estimated three-month cost of Jenny Craig is approximately $1,200 and Weight Watchers is approximately $167.

MEDICALLY SUPERVISED COMMERCIAL WEIGHT-LOSS PROGRAMS

Medically supervised weight-loss programs offer a comprehensive team approach to weight loss and includes physicians, registered dietitians, and counselors. Optifast, Medifast, and Health Management Resources (HMR) are three programs that offer physician supervision for weight loss. HMR and Optifast offer a three-phase weight-loss program: an initial 12–18 weeks of rapid weight loss, a three to eight week transition period, and long-term maintenance. All three programs offer low-calorie (800–1,800 kilocalories/day) and very-low-calorie diets (<800 kilocalories/day) and meal replacement plans. Meal replacement plans include food products, protein bars, and energy drinks which participants can buy directly from the companies. They are expensive with the average initial three-month cost anywhere between $840 for Medifast and $2,000 for both Optifast and HMR [figures from 2007].

With Optifast and HMR, those who complete the program lose between 15 to 25 percent of their initial weight in the first three to six months. In the maintenance phase, 8 to 9 percent of weight loss is sustained at the end of the first year, and 5 percent at four years of follow-up.

SUMMARY

Obesity is a chronic medical condition that has contributed to inflating healthcare costs. Physicians along with exercise specialists, exercise physiologists, and dietitians have a vital role in assessing patients and devising weight-loss plans. Overcoming physician and patient barriers can help forge a team approach to the diagnosis and treatment of obesity. Diagnosis of coexisting medical problems as well as follow-up visits are essential to provide continued support to patients. Commercial physician-supervised programs are available, and although expensive, can be quite effective. Weight-loss medications and surgery are reserved for patients who meet specific criteria. The key to successful weight loss and prevention of weight regain is a sustained lifestyle change that incorporates exercise and dietary modifications.

SEE ALSO: Atkins Diet; Body Mass Index; DEXA (Dual Energy X-Ray Absorptiometry); Dopamine; Exercise; High Protein Diets; Jenny Craig; Optifast; South Beach Diet; Very Low Calorie Diets; Weight Watchers.

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Physiological Aspects of Anorexia Nervosa

ACCORDING TO THE American Psychiatric Association, the following criteria are used to diagnose anorexia nervosa: body weight under 85 percent of that expected for height (BMI of less than 17.5) accompanied by an intense fear of gaining weight, denial of the dangers and implication of the low body weight, extreme shape concerns, and loss of three consecutive menstrual periods, if menstruation has begun. There is a further breakdown into restricting type (characterized by food restriction) and purging type (characterized by some caloric restriction along with compensatory behaviors such as overexercise or purging, for example, vomiting or laxative abuse). The physiological consequence of the latter type may include the physiological consequences of both anorexia nervosa as well as those of bulimia nervosa.

Anorexia nervosa is a serious psychological disorder with important and devastating physiological consequences. These consequences are known to include infertility, low bone density, cardiac complications, and truncal fat deposition in refeeding. In addition, low blood pressure, low heart rate, increased heart rate variability, and arrhythmias can lead to cardiac failure both during and following anorexia nervosa. Dehydration and low blood pressure along with increased strain resulting from increased ketone bodies can lead to kidney failure. Liver failure can also result from malnutrition and dehydration. Furthermore, anorexia nervosa can be associated with numerous hormonal changes including decreased levels of estrogen, estradiol, lutenizing hormone, L-3,5,3'-tri-iodothyronine (T3), thyroxine (T4), thyroid stimulating hormone (TSH), leptin, and increased levels of cortisol.

Anorexia nervosa affects approximately 1 percent of the female population and a small percentage of the male population. Despite the relatively low prevalence, the physiological consequences of anorexia nervosa make it one of the most costly and deadly psychological disorders. Partly due to the dire physiological consequences, inpatient treatment for care can exceed $1,000 per day and months of inpatient treatment may be required to achieve medical stability. The vast and potentially devastating physiological consequences along with high suicide rate among persons with anorexia nervosa make this the deadliest psychological disorder.

Physiological consequences of anorexia nervosa can impact a variety of bodily systems and have lasting effects even if full weight recovery is achieved. Persons with anorexia nervosa often suffer from malnutrition, vitamin deficiencies, and mineral deficiencies. The consequences of malnutrition manifest in muscle, bone, and organs. Persons with anorexia nervosa are at risk for osteoporosis, infertility, and cardiac, renal, and liver failure.

Changes in both the skeletal and muscular systems occur. Low body weight and the lack of nutrients seen in anorexia nervosa is associated with the premature development of osteopenia (low bone mineral density leading to increased likelihood of developing osteoporosis over time) and osteoporosis (thin and brittle bones associated with an increased risk breaking bones). Muscular changes can also occur. As the body enters into prolonged starvation, protein from dietary intake and muscle may be broken down as an energy source to supply the brain and other organs with the energy to keep functioning. As a result, muscle loss and weakness are likely to occur. The loss of lean body mass (LBM) may be accompanied by a decrease in resting energy expenditure (REE). The metabolic rate is likely to slow to conserve energy, especially if the anorexia nervosa is present for a prolonged period of time.

Organs try to conserve energy in anorexia nervosa. As mentioned before, anorexia nervosa is the deadliest psychological disorder. Many of the deaths from anorexia nervosa are a result of heart failure. Bradycardia, or very low heart rates, are commonly seen in persons suffering from anorexia nervosa. Tremendous strain is put on the body and heart as the heart rate drops below 60 beats per minute. In addition to the decreased heart rate, ejection fraction, stroke volume, and cardiac output are also lower, putting additional strain on the heart and other organs.

Along with low heart rate and heart rate variability, heart arrhythmias are also present in persons with anorexia nervosa. The most notable arrhythmia is likely QTc prolongations. Prolongations in the QTc interval are most likely to be found in persons who have hypokalemia (low blood potassium) and may not be present in persons with anorexia nervosa who are able to maintain normal potassium levels. In addition to QTc

interval prolongations, R-R abnormalities have also been observed. Heart arrhythmias may cause many of the cardiovascular episodes and deaths in anorexia nervosa especially as heart rate drops lower in prolonged anorexia nervosa.

Additional cardiovascular problems result from changes in the heart muscle itself. Similar to decreased large muscle mass seen in anorexia nervosa, the heart muscle also experiences atrophy. As atrophy occurs, the heart weakens and increases the risk for heart problems and cardiac failure. Decreased left ventricular mass becomes problematic in anorexia nervosa especially when systolic function becomes affected.

Refeeding can pose additional strain to the heart, so care must be taken during the refeeding process. In the refeeding process, problems such as congestive heart failure and sudden cardiac death have been observed. Thus, when a person who has suffered from anorexia nervosa is entering into recovery, healthcare professionals must be careful to ensure that cardiac function is not further compromised.

Hypotension or low blood pressure and low core temperature are also a consequence of anorexia nervosa. Lower systolic blood pressure is often seen in persons with anorexia nervosa. Orthostatic hypotension (low blood pressure upon standing) often occurs. Low blood pressure can contribute to some of the cardiac abnormalities seen in anorexia nervosa. Several factors including low fat mass and low thyroid hormone could contribute to low core temperature. In attempts to counteract the low core temperature, persons with anorexia nervosa tend to grow fine hair (lanugo) to keep the body warm; this is one of the hallmark physical manifestations of anorexia nervosa.

Along with cardiac failure, liver and kidney failure are seen in anorexia nervosa. Persons with anorexia nervosa often suffer from dehydration. Dehydration has adverse effects on both the liver and kidney. Low blood pressure and hypokalemia are damaging to the kidneys. Hypokalemia may result in nephropathy and eventual renal failure. In addition, decreased glomerular filtration rates, high creatinine (likely due to increased muscle breakdown), and high blood urea all cause kidney damage. Many persons with a history of anorexia nervosa will suffer from kidney diseases and renal failure. Liver damage may result from dehydration and malnutrition. Malnutrition seems to be the main cause of liver damage in anorexia nervosa. If liver failure does not occur, liver damage seems to be reversible with refeeding.

Hypoglycemia is common in anorexia nervosa. As blood sugar decreases, people may become shaky and irritable; fainting may occur. In the prolonged starvation seen in anorexia nervosa, the body will begin to produce ketone bodies as an alternate fuel source for muscle and organs, including the brain. These ketone bodies are made by the liver and used systematically. Ketone bodies are quite acidic and ketoacidosis often result from prolonged increased ketone body production. Consequences of ketoacidosis include acidic blood and dehydration, both of which cause further harm to the liver and kidneys. In addition, "cloudy" thinking or lack of concentration and focus is also seen in people in a ketonic state. This could pose additional difficulties for psychological treatment of anorexia nervosa.

Numerous hormonal abnormalities have been consistently observed in persons with anorexia nervosa. One of the diagnostic criteria of anorexia nervosa is amenorrhea. Low estrogen, estradiol, and luteinizing hormone levels are present in persons with anorexia nervosa. Changes in reproductive hormones, mainly decreased production, can lead to the amenorrhea characteristic of anorexia nervosa. Infertility is often a long-term consequence of anorexia nervosa.

Thyroid hormones, T3, T4 (both total and free), TSH response to thyroid releasing hormone, and thyroid binding hormone levels may all be decreased in anorexia nervosa. Hypothyroidism is associated with low REE, feeling cold, and muscle weakness which people with anorexia nervosa often experience. One of the most devastating consequences of low thyroid hormone is heart failure. As mentioned previously, cardiac concerns are great in this population.

Cortisol and leptin levels are altered in anorexia. These can have both short- and long-term affects. Increases in cortisol are often seen in anorexia nervosa. Initial increases in cortisol levels can help with fat mobilization and the sustainment of life; however, increased cortisol levels can be associated with increased truncal fat deposition and associated health risks. Leptin level also decrease in anorexia nervosa presumably to try signal hunger.

Hence, anorexia nervosa has systemic physiological impact on hormone levels, various organs including bone, heart, kidney, liver, and reproductive organs

that may have both immediate and long-term health consequences.

SEE ALSO: Anorexia Nervosa; Caloric Restriction; Childhood Onset Eating Disorders; Genetic Influences on Eating Disorders; Physiological Aspects of Bulimia Nervosa.

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Physiological Aspects of Bulimia Nervosa

BULIMIA NERVOSA IS characterized by continual binge/purge episodes in attempts to control weight. The American Psychiatric Association outlines the diagnostic criteria for bulimia nervosa as follows: binge eating with inappropriate compensatory behaviors (vomiting, laxative abuse, overexercise, fasting, with holding of insulin, etc.) occurring at least twice a month for three months. The person must be overconcerned with body weight and shape and have a body mass index (BMI) above 17.5.

While a binge is extremely hard to define and may come in the objective (most would agree that the current episode qualified as a binge) or subjective (the person could have eaten a few bites of something, possibly a forbidden food, feels out of control, considers the current eating occurrence a binge and compensates by engaging in purging behaviors), the diagnostic criteria for bulimia nervosa specifies eating at one sitting what most would consider much larger than average in that period of time. The person must also experience a loss of control accompanying this episode.

Persons with purging type anorexia nervosa may also engage in some of the aforementioned purging episodes. Physiological consequences are likely to be seen systemwide. Initial signs of bulimia nervosa may include erosion of tooth enamel or enlargement of salivary glands. Gastrointestinal changes including esophageal narrowing or erosion, stomach or esophagus ulcers or tears, and gastric reflux may occur. In the colon nerve, damage and dependency on laxative may be long-term problems. Slowed gastric emptying, cholecystokinin, ghrelin, and peptide yy release in response to food may encourage overeating. Electrolyte imbalances, which can result from purging along with fluid shifts, carry severe consequences which could include heart attack and death.

Bulimia nervosa has been a growing problem since the mid-1900s. Estimates of the prevalence of bulimia nervosa are 1 to 3 percent of the female population, but higher in the female college population. A small amount of males suffer from bulimia nervosa comprising 5 to 10 percent of the total population with this disorder. Bulimia nervosa is costly to treat and can have long-term devastating physiological consequences.

Despite often being normal weight, people with bulimia nervosa still suffer devastating physiological complications and death. Tooth decay and loss of tooth enamel are some of the early complications of bulimia nervosa and may actually alert health professions to what is occurring with the individual. The acidic content of the vomit is able to wear away the enamel on teeth, especially the back of the teeth. The continual exposure to this stomach acid causes long-term tooth damage and dental staining. Gingivitis and cavities may also result from bulimic episodes. Cuts in the mouth may result from purging episodes. Damage to oral mucosa can also occur from purging, dry mouth following purging episodes, and from poor dental hygiene often seen in persons with bulimia nervosa.

Along with tooth damage and decay, persons with bulimia nervosa often suffer from swollen glands (especially the parotid or salivary glands) and puffy cheeks. Parotid hypertrophy may result due to the increased demands for saliva production following purging episodes. Additionally, high amounts of amalyase enzyme are present in persons with bulimia nervosa. Furthermore, increased production of amalyase does not come from pancreatic secretion. Therefore, it is possible that increased need for amalyase to digest the large amounts of food being consumed is met by the parotid gland. This increased strain would help contribute to the hypertrophy noted in the parotid glands. Stones may form in the salivary ducts as a result of constant purging. In addition, over time, the

salivary glands may tire and begin to produce less saliva, resulting in dry mouth. In most cases, the parotid glands return to normal size when the bingeing and purging episodes have ceased.

The esophagus is often damaged in bulimia nervosa. This occurs from the continual binging and purging episodes. The content of the vomit being purged is extremely acidic which can have several consequences to the esophagus. These can be painful, permanent, and in some cases, require surgical intervention. Esophagitis has been shown in persons with bulimia nervosa. This may lead to esophageal ulcers or erosion. While generally it is not as common as other consequences of bulimia nervosa, esophagitis has been associated with Barrett esophagus and eventual adenocarcinoma, which can result in death, making this a severe consequence yet unlikely of bulimia nervosa. Esophageal ulcers can result from continual exposure to acidic vomit.

In addition, ulcers or sores of the mucosa may occur in the duodenum due to binge/purge cycles. Small tears or ruptures in the esophagus and/or stomach often occur with repeated purging episodes. Esophageal statures, or narrowing of the esophagus, has been noted along with erosive gastritis, inflammation, and deterioration of the stomach lining. Erosive gastritis is caused as a result of the increased stomach acid. While each of these problems has been described in the literature, it is important to note that studies have not consistently found many of these problems in populations studied. It may be the case that these occur in only some cases of bulimia nervosa, yet the significant consequences of these esophageal problems make them important to mention and important for healthcare professionals to consider when treating people with bulimia nervosa. Overall, repeated exposure of the esophagus to stomach acid can lead to increased pain and heartburn.

Alterations in gastric functioning and emptying have been observed in bulimia nervosa. Binge/purge cycles can result in delayed gastric emptying. Along with delayed gastric emptying, increased stomach capacity and less ability for gastric relaxation often occur. Furthermore, levels of cholecystokinin tend to be lower in persons with bulimia nervosa. Increased gastric capacity and delayed gastric emptying, in conjunction with lower and slower release of cholecystokinin, are likely to result in decreased ability to feel satiated, which would lead to increased food consumption. Because these changes persist at least into initial recovery from bulimia nervosa, this could lead to increased weight gain or people must learn to cease eating before satiated. In addition, slower gastric transit time could lead to feeling bloated, rectal distention, stomach pain, and gastric reflux.

Short-term and especially long-term laxative abuse can cause irregular bowel movements and constipation. In the acute phase of bulimia nervosa, laxative abuse can lead to uncontrollable bowel movements. Often, people who suffer from bulimia nervosa will plan daily activities around laxative consumption. Of more physiologic consequence are the detrimental effects that laxative abuse can have on the body. Abuse of laxatives and diuretics can lead to extreme loss of water, leading to dehydration and severe electrolyte imbalances. These electrolyte imbalances place people at increased risk for fainting and cardiac episodes, and dehydration places strain on the liver and kidneys.

In the long-term, resumption of normal bowel movements can take weeks or months to restore. Constipation is likely to occur as people stop taking laxatives. Due to the body's grown dependence on laxatives, it is often suggested that people be gradually weaned off laxative to give the body, mainly the colon, time to adjust. Still, irregular bowel movements may be a chronic problem in many cases. In some dire instances, surgery may be necessary to repair colon damage. Nerve damage to the colon may result from long-term laxative abuse.

Along with decreased release of cholecystokinin, other hormonal changes are associated with bulimia nervosa. These include higher fasting ghrelin levels, lower inhibition on ghrelin following a meal, and lower increase of peptide YY following a meal. Each of these could encourage higher food consumption, especially when coupled with the aforementioned gastrointestional abnormalities. In addition, increased cortisol levels in bulimia nervosa, especially during a rise in binge/purge episodes, may lead to health problems due to increased central fat deposition.

Electrolyte imbalance can occur from various forms of purging including vomiting and laxative and/or diuretic abuse. Electrolyte imbalance can include the following, low potassium, low magnesium, and altered sodium levels. Low potassium levels could have a devastating effect on the heart, causing arrhythmias and even death.

Typically, purging in bulimia nervosa is thought of as vomiting, laxative or diuretic abuse, or overexercise. While these may be the most common, other forms of purges exist and can carry severe consequences. For example, persons with type 1 diabetes may misuse insulin by failing to take or underadministering insulin. This will result in weight loss because of the body's inability to process glucose. As a consequence of insulin misuse, diabetic complications including nephropathy, nerve damage, and blindness often occur more quickly. Furthermore, cardiovascular disease may also result more quickly from the increased glucose levels.

Thus, bulimia nervosa has systemic physiological impact on various organs including the esophagus, stomach, colon, heart, and kidneys that may have both immediate and long-term health consequences.

SEE ALSO: Bulimia Nervosa; Caloric Restriction; Childhood Onset Eating Disorders; Genetic Influences on Eating Disorders; Physiological Aspects of Anorexia Nervosa.

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Pima Indians

THE PIMA INDIANS of the Gila River Indian Community in Arizona have very high rates of obesity and Type 2 diabetes. Both genetic and environmental risk factors contribute to the high rates of diabetes in this community. One-half of adult Pima Indians over the age of 35 years have Type 2 diabetes and 95 percent of those with Type 2 diabetes are overweight. Collaboration with the community in understanding the extent and cause of Type 2 diabetes and obesity in this population has been ongoing since 1965. The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) has established a branch in the southwest (the Phoenix Epidemiology and Clinical Research Branch) specifically to work with the community to investigate the causes and complications of obesity and Type 2 diabetes.

From this collaboration, risk factors for development of weight gain and development of Type 2 diabetes have become clearer. Higher body weight itself is an important risk factor for Type 2 diabetes. Other important risk factors for diabetes include the decreased ability of insulin to metabolize blood sugar (insulin resistance), family history of diabetes, exposure to diabetes in utero, and mild elevation of blood sugars.

Risk factors for weight gain in this population include lower insulin resistance (or higher insulin sensitivity), lower metabolic rate measured at rest or over 24 hours, lower activity of the sympathetic nervous system (thought to play an important role in the rate of body metabolism), and lower concentrations of thyroid hormone.

Complications from Type 2 diabetes, including eye, kidney, and vascular disease, cause a substantial amount of morbidity for this population. In fact, mortality from cardiovascular and kidney disease are much higher in Pima Indians with diabetes compared to those without diabetes. Therefore, treatment and prevention have also been main targets of research for the NIDDK. Members of the Gila River Indian Community participated as volunteers in a large clinical trial (the Diabetes Prevention Program) that has shown that lifestyle modification (modest weight loss of about 15 pounds and habitual physical activity of 150 minutes per week) can prevent or delay the development of Type 2 diabetes. Members of this community are also participating in a clinical trial to test the effect of weight loss on development of cardiovascular disease in individuals with Type 2 diabetes and a clinical trial to investigate the impact of medication to delay worsening of diabetes-related kidney disease.

Collaboration with the Pima Indians of the Gila River Indian Community has improved our understanding of the causes of Type 2 diabetes and obesity, and has led to progress in diabetes prevention. These advances have both directly, via participation in these intervention studies and subsequent programs modeled on such studies, and indirectly, via advances in treatments based on findings from these studies, improved care for Gila River Indian Community members at risk for and with Type 2 diabetes.

SEE ALSO: Insulin; Native Americans; NIDDK; Type 2 Diabetes.

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Pituitary Gland

THE PITUITARY GLAND is a small pea-sized protrusion from the base of the brain and sits on a bony pouch called the Turkish saddle (or sella turcica) on the sphenoid bone near the base of the brain. Also called the hypophysis, the pituitary gland is responsible for endocrine function from two major regions, an anterior and posterior, which secrete hormones regulating homeostasis and which act on other endocrine glands throughout the body. It is directly connected to the hypothalamus which is the main central nervous system (CNS) center and regulator. The hormones that the pituitary secretes help to regulate many body functions and an abnormality in this system can result in uncontrollable weight gain and lead to morbid obesity.

POSTERIOR PITUITARY

The posterior, or neurohypophysis, has a direct connection with the hypothalamus via a stalk that receives chemical messengers directly which gives rise to a tuberoinfundibular pathway. Hormones are made in the actual nerve cell body that are positioned in the hypothalamus which travel down to the posterior lobe which fire into the blood supply of the pituitary gland. The two main hormones secreted by the posterior pituitary are oxytocin and antidiuretic hormone (ADH), or vasopressin.

In women, oxytocin is released mainly after distension of the cervix and vagina during labor, and after stimulation of the nipples, facilitating birth and breastfeeding. Oxytocin is released during orgasm in both sexes. In the brain, oxytocin is involved in social recognition and bonding. Antidiuretic hormones' (ADHs') main action is to conserve body water by reducing water loss in urine.

ANTERIOR PITUITARY

The anterior, or adenohypophysis, is also loosely connected with the hypothalamus via the hypophysial-portal blood vessels. This vascular connection allows signals to be transmitted and either stimulate or inhibit certain body functions. The anterior lobe is genuinely glandular and is a major component of the endocrine systems. The main hormones released by the anterior pituitary are human growth hormone (hGH), prolactin, follicle-stimulating hormone (FSH), luteinizing hormone (LH), thyroid-stimulating hormone (TSH), adrenocorticotropic hormone (ACTH), and endorphins.

The sum of the calculated release of these hormones are responsible for human growth, blood pressure regulation, in women for pregnancy and childbirth, breast milk production, sexual organ function, thyroid gland function, overall metabolism, and water regulation. If any of these mechanisms does not work properly, obesity and even excessive weight loss gain result. The pituitary regulates and controls cortisol production by the adrenal glands.

An overproduction can be caused by a tumor in the pituitary gland. When a pituitary tumor secretes too much adrenal cortical tropic hormone (ACTH), it simply causes the otherwise-normal adrenal glands to produce too much cortisol. This type of Cushing syndrome is termed *Cushing disease* and it is diagnosed like other endocrine disorders by measuring the appropriateness of hormone production. It is a disease that results from an increase in corticosteroid secretion from the adrenal gland and leads to obesity.

SEE ALSO: CNS/Hypothalamus Energy Sensing; Cushing Syndrome; Corticotropin-Releasing Hormone; Ghrelin; Growth Hormone; Hormones; Hypothalamus; Oxytocin and Food Intake; POMC Proopiomelanocortin.

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Policy to Prevent Obesity

POLICIES DESIGNED TO prevent obesity are based in a public health prevention model of disease prevention. Unlike public policies aimed at treating obesity (i.e., by reducing body mass index [BMI]), obesity prevention policies seek to stop people from becoming obese in the first place and to prevent people who are already overweight or obese from gaining more weight. Obesity prevention policies tend to focus on changing the "toxic" food and physical activity environment that contributes to obesity, such as making healthy foods less expensive and making it easier for people to engage in physical activity (e.g., installing bike paths for commuters).

Awareness of obesity as a public health problem has increased along with public support for obesity prevention policies.

A prevention, rather than treatment, model for obesity is supported by data that suggest that long-term weight loss is unrealistic and highly improbable for most people. Furthermore, there are significant hazards in using drugs or surgery to treat obesity. Given that obesity results in high social costs, associated diseases (e.g., diabetes), and early mortality, and that obesity will account for increasingly larger healthcare costs over time, many public health experts believe it may be the best use of public resources to focus on policies that will prevent obesity.

Obesity prevention policies exist in the private sector and at all levels of government. In the private sector, many workplaces have sponsored healthy living initiatives that incorporate policies to prevent obesity in their employees, such as reducing the prices of healthy foods in their cafeterias, building exercise facilities, and subsidizing gym memberships. It is not known how many private corporations support wellness and obesity prevention programs. However, there are data that support the fact that corporations only receive a return on their investment (in the form of lower healthcare costs) only over the long term. Therefore, companies with high employee turnover and businesses with fewer employees are less likely to implement workplace wellness and obesity prevention policies.

The federal government's obesity policies tend to focus more on weight reduction than prevention of future weight gain. It is estimated that there are over 300 programs related to obesity within the purview of the federal government, many of which are concerned with public education, such as the MyPyramid program through the U. S. Department of Agriculture (USDA), and the Centers for Disease Control and Prevention's (CDC's) VERB youth media campaign designed to encourage physical activity in adolescents.

Although at the federal level, the obesity objectives outlined by the U. S. Public Health Service were given to the Department of Health and Human Services (DHSS), the actual implementation of these programs were scattered across at least nine federal agencies, such as the CDC, USDA, and the Food and Drug Administration (FDA). Critics of the federal government's approach to obesity prevention have highlighted the lack of concentrated and consistent leadership on this critical public health issue. An additional barrier regarding implementation of obesity prevention policies at the federal level concerns the lack of funding. Some

estimate that the United States spends less than an average of $3.00 per citizen on obesity prevention [figure is from 2007], which some argue is inadequate given the increasing enormity and gravity of the problem.

State obesity prevention policies vary widely. Some states, such as California and Arkansas, have enacted policies to prevent obesity in adults and children to a greater extent than the federal government. As of 2006, 17 states and the District of Columbia have passed taxes on unhealthy foods and/or soda. Twenty-eight states have also received funds from the CDC to develop state "obesity plans" designed to prevent (and treat) obesity and promote wellness. Seven of these 28 states were also funded at the "basic implementation" level, meaning that they were given funds to implement their nutritional and physical activity plans, and evaluate the progress and impact of their state plans.

However, much of the obesity-prevention efforts at the state level have focused on school-based policies, such as setting nutritional standards for school meals that go beyond the federal standards, setting nutritional standards for foods sold outside the cafeteria (i.e., "competitive" foods—which tend to be unhealthy beverages and snacks), limiting access to these competitive foods, informing parents of the BMI of their children, and screening for diabetes. In general, state-based obesity prevention policies, particularly those aimed at children, tend to be more progressive than federal policies.

Recent data suggest that awareness of obesity as a public health problem has increased along with public support for obesity prevention policies. As of 2007, 85 percent of adults supported tax breaks for employers who provided exercise space to employees and 72 percent of Americans reported that they would support government-based policies that required health insurance companies to cover obesity prevention and treatment programs.

SEE ALSO: Built Environments; Center for Science in the Public Interest; Centers for Disease Control; Federal Initiatives to Prevent Obesity; Food Stamp Nutrition Education Program; Governmental Policy and Obesity.

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Polycystic Ovary Disease

POLYCYSTIC OVARY syndrome (PCOS), also known as Stein-Leventhal syndrome or functional ovarian hyperandogenism, is a reproductive-age endocrine metabolic disorder of women. The prevalence of PCOS is 4 to 12 percent in the United States and 6 to 18 percent worldwide. PCOS is associated with peripheral insulin resistance, hyperinsulinemia, dyslipidemia, and hyperglycemia. PCOS with obesity enhances other complications. The principal features of PCOS are lack of regular ovulation and excessive amounts or effects of androgenic hormones such as testosterone, androstenedione, and dehydroepiandrosterone sulfate (DHEA-S).

In 1990, the National Institutes of Health (NIH)/National Institute of Child Health and Huma Development (NICHD) defined PCOS as women with the signs of androgen excess hormones and oligoovulation. In 2003, the European Society for Human Reproduction & Embryology (ESHRE)/American Society of Reproductive Medicine (ASRM) defined PCOS as women having at least two out of three factors: oligoovulation and/or anovulation, excess androgen activity, polycystic ovaries. The contributing risk in PCOS are acne; weight gain and have trouble losing weight; male pattern baldness or thinning hair on the scalp; hair growth on the face, back, or chest; high blood sugar (hyperglycemia); and endometrial hyperplasia and endometrial cancer (cancer of the uterine lining) are possible due to overaccumulation of uterine lining, and also lack of progesterone resulting in prolonged stimulation of uterine cells by estrogen, high blood pressure, dyslipidemia (disorders of lipid metabolism—cholesterol and triglycerides), and increasing inflammatory mediators. The symptoms vary with each individual woman.

The development steps of PCOS are increase of GnRH pulse frequency, LH over FSH dominance,

increase in ovarian androgen production, decreased follicular maturation, and decreased SHBG binding. Women with PCOS have a higher risk of miscarriage, irregular cycles and lack of ovulation. The major diagnostic tool is pelvic ultrasound with biochemical screening including two-hour glucose tolerance tests, and hormone levels and lipid panel will help to diagnose PCOS in reproductive women. They can become normal with early treatment regimen (diet and drugs such as insulin-lowering medications, e.g., metformin hydrochloride [Glucophage®], pioglitazone hydrochloride [Actos®], and rosiglitazone maleate [Avandia®]).

An alternative nutritional adjunctive therapy, chromium supplementation, may be effective for PCOS women with insulin resistance. Chromium is an essential trace mineral required to maintain insulin health. Low-chromium status is associated with a number of metabolic syndrome risk factors. Recently, two pilot studies on chromium picolinate supplementation to PCOS women have shown significant improvements in insulin sensitivity. Long-term studies are required to show their effect on other risk factors in the long run. Surgical treatments include laparoscopy electrocauterization or laser cauterization, old procedure of ovarian wedge resection, and ovarian drilling.

SEE ALSO: Fertility; Pregnancy.

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POMC (Proopiomelanocortin)

THE SENSATIONS OF hunger and satiety, which is the feeling of fullness after a meal, are regulated in the hypothalamus and pituitary of the brain. Proteins called neuropeptides are expressed by specific groups of neurons in these regions of the brain. Each of these neurons interacts with each other to control the response to hunger or satiety. Neurological or genetic defects in these neuropeptides or their respective precursors can lead to obesity, as was seen in knockout mice who were genetically manipulated such that they do not express the gene called proopiomelanocortin (POMC).

POMC is a protein expressed in the arcuate nucleus of the hypothalamus and is the precursor to nine biologically active peptides. Several of these are neuropeptides that play a vital role in regulating the response to hunger and satiety. This protein is a precursor to neuropeptides called melanocortins, specifically alpha-melanocortin (alpha-MSH) secreting hormone, beta-melanocortin (beta-MSH) secreting hormone, and gamma-melanocortin (gamma-MSH) secreting hormone. These neuropeptides decrease energy intake and increase energy expenditure, that is, prevent the body from gaining weight by increasing its metabolism. They do so by binding to five receptors called G-protein coupled receptors where an effect is initiated and completed within the cell using secondary signaling proteins.

Melanocortin-coupled receptor 3 (MCR-3) and melanocortin-coupled receptor 4 (MCR-4) are highly expressed in the central nervous system and are the most important in maintaining the balance between energy intake and expenditure. More focus has been placed on the melanocortin-coupled receptor 4 being that it has been previously shown to play a larger role in energy homeostasis. Mice and humans who lack this receptor are obese and hyperphagic, eating

frequently compared to people who have this receptor. Alpha- and beta-MSH bind to the MCR-4 receptor with higher affinity than gamma-MSH, thus this pathway has been investigated in more detail.

In addition, mutations in the MCR-4 receptor and POMC results in severe obesity due to increased energy intake and reduced energy expenditure, the opposite of what they normally do to the body. Recently, a specific genetic mutation in the region of POMC that expresses beta-MSH was found to be more common in obese subjects in a small clinical study. Mutations in the POMC gene were screened in 538 Caucasian subjects from the United Kingdom with severe early-onset obesity. The mutation was a change in the amino acid tyrosine to cysteine that alters the three-dimensional structure of the protein, reducing the ability of the protein to bind to its receptor and facilitate its action in the body. As a result of this study, the POMC gene expresses neuropeptides that maintain energy intake and expenditure to prevent the onset of obesity.

SEE ALSO: Genetic Influences on Eating Disorders; Genetic Mapping of Obesity Related Genes; Melanocortins.

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Portion Control

PORTION CONTROL IS regulating the amount of food that is eaten. Controlling the number of calories ingested during one meal, within a wide and varying range of foods, along with providing an adequate amount of nutrition, can hold the body in a healthy and satisfied state. Eating beyond the amount of calories or portions of food can contribute to weight gain and possible obesity. Unfortunately, the answer to correcting obesity does not lie in simply eating a variety of healthy foods and exercising. For most individuals struggling with weight, the difficulty lies in the inability to accurately know how much food is enough. A closer look at managing the amount of foods eaten can make the difference between normal body weight versus high body weight.

A serving size is the specific amount of food defined by common measurements, such as cups, ounces, or pieces.

OBESITY

Research indicates that 66 percent of adults are overweight or obese. Studies suggest controlling the portion of food eaten is more likely to control weight gain and assist in weight loss, particularly in cases of obesity. Maintaining a normal body weight not only boosts self-image, but it can also help in controlling cholesterol, blood pressure, and blood sugar. Weight-related

diseases, such as heart disease, diabetes, arthritis, and some cancers, may also be kept at bay by keeping the weight in a healthy range.

CONTROLLING PORTIONS OF FOOD

Meals eaten in moderation can lead to a normal weight that can be maintained. This may sound like a simple solution to obesity; however, it is not. Most individuals who eat large portions of food do not know how to gauge the amount of food needed for each meal. In fact, individuals often rely on what is seen on the plate rather than a full signal from the stomach.

Understanding serving size is the key to losing weight. A serving size is the specific amount of food defined by common measurements, such as cups, ounces, or pieces. Often, one portion of food is compared with one serving of food, which is not always correct information. A standard serving of food contains a specific amount of calories and nutrients. A portion of food many times is larger than one serving size. For starters, to understand portion control is to visualize serving sizes. Below is a list of foods to assist in visualizing this process:

- A medium apple or orange is the size of your fist.
- 3 ounces of meat is the size of a deck of cards.
- 1 tablespoon of peanut butter is the size of your thumb.
- 1 teaspoon of butter is the size of the tip of your forefinger.
- 3 ounces of grilled fish is the size of two fists together side by side.
- A standard bagel is the size of a hockey puck.

The serving sizes listed on food labels are the amount of food based on the product's nutritional analysis, not necessarily the recommended amount of food that equals a portion. The servings listed on the label are determined by the amount of a particular food people normally eat based on standard household measurements: cups, ounces, and pieces. Reading food labels to determine serving sizes can aid in reducing the sizes of portions consumed. Restaurants often serve a larger portion than the serving size recommended. To meet the appropriate portion standards, eat half the portions served and take the remaining part of the meal home in a takeout container for another meal.

PORTION CONTROL AND BINGE EATING

Breaking free of diet mentality and food rituals is a first step in recovering from overeating and binge eating. It is not uncommon for overweight binge eaters to have difficulty determining when they feel full or have had enough to eat, either during eating their meals or a binge. Binge eaters are often overweight. There is a strong association between binge-eating disorder and obesity.

Binge-eating disorder is a relatively recently recognized disorder, sometimes referred to as compulsive overeating. Some researchers believe it is the most common of the eating disorders affecting millions of Americans. Binge-eating disorder is a condition, not yet fully recognized as an eating disorder, involving consumption of large amounts of food without inappropriate compensatory behavior. Patients with BED do not use inappropriate compensatory behavior to control their weight such as self-induced vomiting, misuse of laxatives, or fasting and excessive exercise. Binge-eating disorder has been proposed for further study in the appendix of the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders*. Research indicates more than 30 percent of patients who receive obesity treatment meet the criteria for binge-eating disorder.

Whether a person is suffering from obesity or binge-eating disorder, the simple solution to the weight aspect is to cut back on the intake of food. One of the reasons obesity is becoming more of an issue today rests in the inability to gauge the adequate amount of food needed. In addition, meals outside the home tend to be double the needed amount. In the last 20 years, the average American meal eaten out of the home has more than doubled in size. Case in point, an average serving size of soda today is 20 ounces compared to less than 8 ounces a few decades past. The Big Gulp-sized 64-ounce soda contains close to 800 empty calories. People are conditioned to expect large portions in restaurants and may find it difficult to figure out what normal portions of food are. A small order of French fries today was at one time the only size available.

Portion control is about controlling portions not about dieting. Unfortunately, weight loss through dieting is often regained. Calorie counting or measuring foods can be a useful tool for determining the framework of normal eating. The normal amount of

calories per individual is dependent on many factors such as height, weight, and gender. It is projected an average 200 pound man or woman eats approximately 2,400 calories a day to maintain his or her current weight. Temporarily monitoring calories taken in on a given day can give a clear picture on whether too much or too little food has been eaten. It is suggested that a range between 1,500 and 2,500 calories a day would avoid over or under eating.

MINDFUL EATING

Understanding the basic principles to good nutrition can lead to healthy lifestyle change and often a natural healthy weight loss while controlling portions of foods eaten. Eating a wide variety of fruits and vegetables, along with lean proteins and increasing complex carbohydrates high in fiber, low-fat dairy foods and decreasing total fat intake can not only lead to weight loss, but also promote health and reduce risks for such illnesses as diabetes and cardiovascular disease. It is suggested meals are eaten at regular intervals every four to five hours.

Combining mindful eating with portion control through a measuring system may be one strategy to maintain a healthy body weight and avoid obesity. For example, outlining breakfast, lunch, dinner, and a snack with controlled portions of specific food groups eaten in four to five hour intervals could provide the nutrients needed along with enough food to offset hunger. The six food groups are protein, fruit, fats, vegetables, starch, and dairy. A suggested meal plan is as follows:

Breakfast:

- 2 poached eggs
- 1 cup of cooked oatmeal
- 1 teaspoon of coconut oil
- 1 medium orange
- 1 cup of skim milk

Lunch:

- 2 cups mixed raw or cooked vegetables
- 4 ounces of salmon
- 1 cup of brown rice
- 1 teaspoon of olive oil

Dinner:

- 4 ounces of lean steak
- 2 cups of raw spinach and tomatoes
- 6 ounce of sweet potato
- 1 teaspoon of olive oil

Snack:

- 1 cup of plain low-fat yogurt
- 1 cup of frozen blueberries

CONCLUSION

A healthy weight can be maintained by controlling the portion of food ingested. Attention to the sizes of portions and types of foods eaten directly affects the amount of nutrients and calories taken in. Avoid large portions of high-calorie foods and focus on low-calorie natural foods such as lean protein, fresh vegetables and fruit, low-fat dairy, whole grains to naturally reduce the number of calories consumed, obtain the right nutrients the body needs, and to feel satisfied.

SEE ALSO: Binge Eating; Caloric Restriction.

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PPAR (Peroxisome Proliferator-Activated Receptors)

PPAR ARE TYPES of nuclear receptors. This means that PPAR can regulate the expression of different genes in the body. PPAR work through dietary fatty acids. After a person has ingested food, some of the fatty acids may travel to the peroxisome within the cell and then bind to the PPAR. Once a fatty acid has attached itself to the PPAR, the fatty acid-PPAR complex will travel to the nucleus and attach itself to

part of the DNA within the nucleus. Depending on the type of fatty acids or PPAR, different genes will be affected and subsequently the expression of different compounds will either increase or decrease.

There are several different types of PPAR. These include PPAR-alpha, PPAR-gamma, and PPAR-delta. PPAR-alpha is mainly expressed in the liver, but has also been found in the kidney, heart, skeletal muscle, and brown adipose tissue. PPAR-alpha plays a role in activating genes to increase fatty acid oxidation. It increases the expression of many of the enzymes that are necessary for fatty acid oxidation to occur. Additionally, it increases the levels of transport proteins which help deliver fatty acids into the cell and into the mitochondria for fatty acid oxidation.

There are two types of PPAR-gamma. The first is known as PPAR-gamma-1 and is found in very low levels throughout the body. Its function is not currently known. The second is known as PPAR-gamma-2. PPAR-gamma-2 is found in adipose tissue (fat tissue) only. This entry will refer to PPAR-gamma-2 as PPAR-gamma. PPAR-gamma will activate genes that will help synthesize new adipocytes (fat cells) in the body. It will also increase the expression of genes to increase fat storage in the body (i.e., lipoprotein lipase), fatty acid synthesis (i.e., acyl-CoA synthase), and glucose (sugar) transport (i.e., GLUT4). PPAR-gamma may also decrease the levels of leptin, a hormone that enhances fat use. The third PPAR, PPAR-delta, is sometimes referred to as PPAR-beta. This PPAR is found in most of the tissues and organs in the body. Its function is not completely understood, but it is believed to help increase HDL levels (good cholesterol).

Different types of fatty acids will activate PPAR at varying rates. Saturated fatty acids, such as fatty acids that you may find in meat or dairy products, are not very effective at activating PPAR. Polyunsaturated fatty acids, such as the fatty acids that you find in plants and fish, are very good at activating PPAR. Two types of polyunsaturated fatty acids that exist are omega-3s and omega-6 fatty acids. Omega-3 fatty acids, which can be found in both plants (i.e., flaxseed) and animals (i.e., fatty fish), are even better than the omega-6 fatty acids at activating PPAR.

Because of their ability to affect the expression of many genes that pertain to diseases such as heart disease and diabetes mellitus, PPAR have been studied to be used as pharmaceuticals. The rationale for this is that a medicine could bind to PPAR and then travel to the nucleus to either increase or decrease certain compounds in the body. The first class of pharmaceuticals to be discovered was the fibrates. Fibrates are drugs that play a role in lowering triglycerides and cholesterol. Fibrates work by binding to PPAR-alpha, which in turn promotes fatty acid utilization. If more fatty acids are being used in the body, then triglycerides and cholesterol levels in the blood will decrease.

Another exciting use of PPAR as a pharmaceutical is the treatment of diabetes mellitus. Scientists were trying to discover other cholesterol-lowering medications (like fibrates). During this time, they discovered a class of medicines known as thiazolidinediones (TZDs). These drugs were shown to lower blood glucose (sugar) levels and improve insulin sensitivity. TZDs activate PPAR-gamma. PPAR-gamma was responsible for activating genes that make new fat cells. It is hypothesized that TZDs function by increasing the body's ability to store fat, which in turn would lower the levels of fatty acids in the blood.

With lower fatty acids in the blood, glucose (sugar) can be used more effectively by the body and thus the symptoms of Type 2 diabetes mellitus become lessened. One side effect of TZD is an increase in body weight, despite an improvement in a person's diabetes mellitus.

SEE ALSO: Db/Db Mouse; Metabolic Disorders and Childhood Obesity.

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Prader-Willi Syndrome

THE PRADER-WILLI SYNDROME (PWS), caused by the absence of certain paternal genes, is the most common genetic cause of obesity. It is estimated that 375,000 people worldwide suffer from PWS and it is seen one in every 125,000 live births. It is a complex of cognitive disabilities, short stature, incomplete sexual development, and chronic hunger that can lead to overeating and obesity. There are no risk factors for developing PWS and PWS affects men and women equally.

PWS is attributed to genomic imprinting disorder. In 70 percent of PWS cases, there is a paternal deletion of genes in the long arm of chromosome 15. In 28 percent of cases, two maternal copies of the genes are expressed. In less than 1 percent of cases, there is a mutation in the imprinting control center. While PWS usually arises sporadically, the type of mutation present determines the recurrence risk in future generations. In paternal deletion or maternal disomy, there is less than a 1 percent risk of recurrence. However, if PWS arises from chromosomal translocation, there is a 25 percent risk of recurrence. An imprinting control center mutation predicts a 50 percent risk of recurrence.

Indications for screening for PWS include decreased muscle tone with feeding difficulties as a toddler, developmental delay and overeating as a child and obesity, behavior problems, and sexual immaturity as an adolescent. Major criteria for diagnosing PWS include decreased muscle tone, neonatal feeding problems, characteristic facial features, sexual immaturity, developmental delay, and overeating. Minor criteria for diagnosis include infantile lethargy, characteristic behavior problems, sleep apnea, short stature, and hypopigmentation. To be diagnosed with PWS, children must fulfill several major and minor criteria. Characteristics facial features include narrow face, almond-shaped eyes, and down-turned corners of the mouth. Characteristic behavioral problems include temper tantrums, violent outbursts, and obsessive-compulsiveness. Diagnosis and etiology is confirmed by molecular testing.

In infancy, decreased muscle tone may lead to difficulty breathing and poor suck, which creates feeding difficulties and a failure to thrive. However, in early childhood, overeating results in obesity. Compared to other obese children, PWS children have increased fat mass and reduce muscle mass. Menarche and testicular descent can be significantly delayed. Adult complications are related to obesity and include sleep apnea, heart failure, diabetes, and atherosclerosis. Osteoporosis results from incomplete sexual development. People with PWS demonstrate a decreased ability to vomit and an increased pain tolerance that can promote bingeing.

Treatment of PWS involves growth and sex hormones to treat short stature, sexual immaturity, obesity, and osteoporosis. Calcium and vitamin D are used to prevent osteoporosis. Management also includes treating complications. Food intake is carefully limited to prevent obesity while preventing cognitive delays due to lack of nutrition.

SEE ALSO: Behavioral Treatment of Child Obesity; Binge Eating; Childhood Onset Eating Disorders; Genetic Mapping of Obesity Related Genes; Implications of Restriction of Foods on Child Feeding Habits.

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Pregnancy

THE PANDEMIC OF obesity affects women of all age groups in the United States. The impact of overweight and obesity on women in the reproductive age group is particularly alarming because of the health risks to the pregnant mother and the baby. Complications such as gestational diabetes (onset of diabetes for the first time during pregnancy), high blood pressure, higher number of cesarean sections, and birth defects in the newborn add to the rising healthcare costs.

According to the National Health and Nutrition Examination Survey (NHANES) data for 2003–04,

61.8 percent of U.S. women are overweight (defined as body mass index [BMI] 25–29.9), 33.2 percent are obese (BMI 30–39.9), and 6.9 percent are extremely obese (BMI more than 40). In women of the childbearing age, between 20–39 years, 51.7 percent are overweight and 28.9 percent obese. The BMI criteria to diagnose overweight and obesity are the same among pregnant women and nonpregnant women.

GUIDELINES FOR WEIGHT GAIN DURING PREGNANCY

A normal pregnancy in humans is on an average 280 days or 40 weeks. It starts with fertilization and implantation and ends with the successful delivery of the baby. The entire duration of pregnancy is divided in three trimesters, each lasting 12 weeks. The changes that occur in a woman's body and her nutritional requirements depend on the trimester.

The Institute of Medicine in 1990 issued guidelines on how much weight women should gain during pregnancy. Underweight women, with a BMI of less than 18 kg/m2 should gain between 28 to 40 pounds; normal weight women should gain 25 to 35 pounds; overweight 15 to 25 pounds; and obese women should restrict their weight gain to 15 pounds. During a normal pregnancy, the recommended weight gain is three to five pounds in the first trimester and one to two pounds per week thereafter. When carrying twins, a gain of 35 to 45 pounds is expected. Despite these guidelines, approximately over 40 percent of women gain more than the recommended weight.

So what constitutes the weight gain of pregnancy? On an average, the baby weighs six to eight pounds, uterus gains two to five pounds, breast tissue two to three pounds, and maternal blood volume four pounds.

OBESITY AND FERTILITY

Obese women may experience problems when trying to conceive. They are much more likely to have irregular menstrual cycles and face anovulation (ovaries not releasing eggs during the menstrual cycle). Those who become pregnant are at increased risk of having a miscarriage early on. Doctors encourage obese women who are undergoing infertility treatment to lose weight. Unfortunately, those undergoing infertility treatment are also more likely to have twin or multiple pregnancies. This by itself leads to excess pregnancy weight gain.

RISKS TO THE MOTHER

The uncomfortable symptoms of pregnancy such as back pain, leg swelling, bloating, shortness of breath, and fatigue can all be exaggerated in women who are overweight and those who gain excess weight during the course of pregnancy. Other complications that can affect the mother during pregnancy include high blood pressure, onset of diabetes during pregnancy (also called gestational diabetes), increased number of urinary infections, preeclampsia (leg swelling, protein in the urine, and liver problems), and eclampsia (preeclampsia associated with seizures). Women who develop gestational diabetes often have normal blood glucose levels after delivery. A small proportion, however, will go on to manifest Type 2 diabetes within a few years of delivery. There is also a 60-percent chance that future pregnancies will be complicated by gestational diabetes.

The risk of forming clots in the legs (called venous thrombosis) is higher among obese compared to nonobese pregnant women. Epidural analgesia (a method by which anesthetic medications may be injected into the spinal canal) is commonly employed to reduce labor pain. This technique has been shown to be not as effective in obese women.

RISKS TO THE BABY

Pregnancy in the overweight mother poses increased risks to the baby as well. Macrosomia or increased birth weight of the baby in excess of 4,000 grams, and birth malformations can be the result of the mother developing gestational diabetes. This can lead to fractures, birth injuries, and increased need for cesarean delivery. The usual detection of these abnormalities in the pregnant woman is by ultrasound. However, the quality of ultrasound images in the obese woman may not be adequate to detect malformations early enough.

Research studies have led to concerns that maternal obesity may increase the child's risk of developing heart disease in adult life. Other birth defects include spina bifida ("split spine" in Latin, a developmental abnormality of the spinal cord and vertebrae) and anencephaly (poorly developed fetal brain). Anencephaly is incompatible with survival and can lead to death or death soon after delivery, whereas spina bifida in its very mild form may not affect survival. Babies born to women who develop gestational diabetes are also at a higher risk of being obese in their adolescence and adult life.

INCREASED MEDICAL EXPENSES

Maternal and fetal complications can lead to more tests, need for early hospital monitoring, cesarean deliveries, and more admissions to the neonatal intensive care unit. Together, they contribute to a substantial increase in medical costs.

FACTORS THAT PREDICT EXCESS WEIGHT GAIN DURING PREGNANCY

Being overweight or obese before conceiving increases a woman's risk of gaining more than the recommended weight during pregnancy. Those who had gained excessively during previous pregnancies are also at a higher risk. Lower socioeconomic status, lack of awareness of the nutritional recommendations, and increased stress can lead to overeating, unbalanced diets, and excess consumption of fatty and high carbohydrate meals.

EATING DISORDERS AND PREGNANCY

Eating disorders are common in young women and adolescent girls. Two of the commonly known ones are anorexia nervosa and bulimia. Women with anorexia are malnourished and have very poor dietary intake. Women with bulimia tend to binge eat followed by self-induced vomiting or purging with laxatives. They are often overweight. Binge eating during pregnancy can easily lead to excess weight. Night eating syndrome, though not formally recognized as an eating disorder, is another condition more common in obese individuals. In this condition, more than one-fourth of the daily calorie requirements are consumed at night. These conditions are frequently seen in obese and young women in the reproductive age group, but it is not known how common they are in pregnant women.

STRATEGIES TO PREVENT EXCESS WEIGHT GAIN

Women should not change their caloric intake during the first three months of pregnancy. Those who are obese or overweight when they conceive should talk to their doctor and get advice from a dietitian. Often, getting the correct nutritional advice can help women avoid overeating. Prenatal exercises, if done regularly, can be continued during the course of pregnancy and after delivery. However, attempting weight loss during pregnancy may not be safe and cannot be recommended.

PREGNANCY AS A CAUSE OF OBESITY

Research studies have attempted to find out the factors that contribute to weight gain and weight retention after pregnancy. An interesting study conducted in Stockholm, the SPAWN (Stockholm Pregnancy and Weight Development) study followed up women after their pregnancy for over 15 years. Changes in the body weight at different time intervals were measured.

The single best predictor of keeping the weight gained during pregnancy was being overweight at the end of the first year after delivery. Being overweight or obese before conceiving does not by itself increase one's long-term chance of overweight. In other words, whatever one's weight before being pregnant, losing weight within one year after delivery is a good way to prevent pregnancy-associated obesity.

WEIGHT LOSS DURING PREGNANCY

Dietary restriction and attempts to lose weight during pregnancy may affect the development of the fetus. There have been no studies to indicate that this may be safe. Current recommendations suggest that weight loss efforts should be made before or after, but not during pregnancy.

EXERCISE DURING PREGNANCY

The American College of Obstetricians and Gynecologists have issued recommendations regarding exercise during pregnancy. In general, women who are

Being overweight or obese before conceiving increases a woman's risk of gaining more than the recommended weight during pregnancy.

not on a regular exercise program should not initiate one after they conceive without approval from their healthcare provider. Walking and swimming are considered safe. The benefits of prenatal exercises are numerous. These include less pregnancy weight gain and back pain, lower stress and anxiety during pregnancy and labor, and less "postpartum belly." The chances of surgical interventions are lower; progression through labor is faster; better digestion; and improved muscular and aerobic fitness are all well-known benefits.

After the first three months, avoid exercises lying on the back. Excess rise in body heat during a workout can increase the risk of spontaneous miscarriage, particularly in the first trimester. Exercise should not be done if a woman has been advised against exercising because she or her baby has a complication (e.g., high blood pressure, growth retardation in the baby).

Women who exercise should also know warning signs that require them to stop exercise and consult their doctor. These include unexplained belly pain, poor weight gain (less than two pounds/month in the last two trimesters), vaginal bleeding, severe or persistent headaches, excess chest pain or palpitations (sensation of fluttering in the chest), and swelling of hands, face, or legs in excess of expected.

The onset of belly contractions that last continuously for more than six hours is worrisome and may indicate premature labor (onset of labor before 37 weeks).

BARRIERS TO WEIGHT LOSS AFTER DELIVERY

One of the most important reasons why women are unable to lose weight after delivery is lack of sleep. Inadequate sleep and fragmented sleep patterns are associated with hormonal changes that contribute to increased appetite. Leptin, a hormone that inhibits appetite, is reduced in the blood, while ghrelin, a hormone secreted by the stomach, is increased. The effects of both hormones combine to create a craving for high carbohydrate, calorie-rich foods.

Another factor is the level of physical activity. Women who exercise and make a conscious effort to lose weight are more successful at reducing their weight than those who do not increase their physical activity levels. With the added responsibility of taking care of the newborn, most women are unable to find the time for a planned exercise program. Yet others feel self-conscious when exercising with normal-weight women.

Stress, postpartum depression (depression that is seen during anytime after delivery usually in the first few weeks), smoking, and age can all have complex interactions on the hormonal and appetite regulatory system to promote weight gain.

BREAST-FEEDING AND POSTPARTUM WEIGHT LOSS

A woman burns more calories (approximately an extra 500 kilocalories/day) when she is breast-feeding than during her pregnancy (extra 200–300 kilocalories/day). Because of the added energy output during breastfeeding, it has been shown to promote maternal weight loss in the first few months, unless it is continued for over a year. Obese women have been noted in several studies to be less likely to breastfeed their babies. This unfortunately increases the baby's risk of developing obesity in adolescence and adulthood. It is thought that the hormone leptin present in the mother's milk protects against obesity in the infant.

CONCLUSIONS

The majority of the women in the reproductive age group in the United States are overweight or obese. The health-related complications to the mother and baby can be disastrous and lead to escalating medical expenses. Nutritional advice on eating a balanced diet may help women to prevent excess weight gain. Healthy eating habits and regular exercise before conceiving should be continued during pregnancy. A conscious effort to attain normal weight within a year of delivery helps prevent long-term weight gain as a result of the pregnancy alone.

SEE ALSO: Body Mass Index (BMI); Breastfeeding; Women and Dieting.

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President's Council on Physical Fitness and Sports

THE PRESIDENT'S COUNCIL on Youth Fitness was founded on July 16, 1956 by U.S. President Dwight D. Eisenhower with the aim of encouraging children to be healthy and active, and reducing the increasing prevalence of childhood obesity. The impetus for the council came from a report which indicated that young people in the United States were less physically fit than their European counterparts. Some people had suggested that compulsory military service might encourage more young people to be fit, but Eisenhower felt that the schemes to be promoted should be voluntary and should include girls. As a result, on June 19, 1956, Vice-President Richard M. Nixon announced that the council would be established at the end of the two-day Conference on Fitness of American Youth, held at the Naval Academy in Annapolis, Maryland.

The initial aims were that all facets of the U.S. community were to have a role. These would include schools, churches, social organizations, sporting groups, and government agencies, with girls being given the same attention as boys. Eisenhower constituted the council which consisted of members of cabinet and also a Citizens Advisory Committee "to alert America on what can and should be done to reach the much-desired goal of a happier, healthier and more totally fit youth in America."

The initial chairman of the first meeting, held on July 16, 1956, was Vice-President Nixon, with Shane McCarthy appointed as the Executive Director, serving until 1961. In 1961, Charles "Bud" Wilkinson, a prominent American football player from Minnesota, was appointed as a Consultant to the President on Physical Fitness.

On January 9, 1963, President John F. Kennedy issued an executive order by which he changed the name of the council to the President's Council on Physical Fitness, with the aim of changing its focus from solely dealing with the youth to serving all Americans, placing the new council under the chairmanship of the Secretary of Welfare. Three years later, President Lyndon B. Johnson created the Presidential Physical Fitness Award, which was later changed to the President's Challenge Youth Physical Fitness Awards Program, and this resulted in the council being changed to the President's Council on Physical Fitness and Sports, the name it still operates under.

Under Johnson, Stan Musial chaired meetings from 1964 until 1967, as Consultant to the President on Physical Fitness, with James A. Lovell serving as the chairman from 1967 until 1978. Carson Conrad served as Executive Director from 1970 until 1984. After Lovell, the council was chaired by Governor Jerry Apodaca (1978–80), George Allen (1981–88), Arnold Schwarzenegger (1990–92), Florence Griffith Joyner and Tom McMillen (cochairs from 1993 until 1998 and 1997, respectively), and Lee Haney (1999–2002).

The major changes since 1968 involved the creation, by President Richard M. Nixon, of the Presidential Sports Award Program, and in 1983, the U.S. Congress declared that May each year would be the National Physical Fitness and Sports Month. Thirteen years later, in 1996, Audrey F. Manley, the acting Surgeon General, issued his *Report on Physical Activity and Health*, with the President's Council, in the following year, publishing its report on *Physical Activity and Sport in the Lives of Girls*. The President's Council now maintains a Web site providing information on physical activity, and also promoting community leadership awards to promote physical fitness as a way of combating obesity.

As now constituted, the President's Council on Physical Fitness and Sports operates as an advisory committee consisting of volunteer citizens who advise the President through the Secretary of Health and Human Services about ways of improving physical activity, fitness, and the promotion of sports. The council has certainly been active in promoting physical fitness and can take some credit for assisting with the introduction of programs to reduce the increasing prevalence of obesity in U.S. society. Further impetus has also been given to the council by President George W. Bush when he issued Executive Order 13265 on June 6, 2002. This appointed Lynn C. Swann, a former professional football

player, a sports broadcaster, and a Republican, as chairman, a position she held until 2005; and Dorothy "Dot' Richardson, the softball player who won a Gold Medal at the 1996 Olympics, and later an orthopedic surgeon, as the vice-chairman.

The programs suggested by the President's Council include a plan to encourage people to eat a nutritious diet, to take part in physical activity each day, to make healthier choices in their lifestyle, and to get preventive screenings on a regular basis. Known as Healthier US, the program was initially launched by President Bush on June 20, 2002, with a new initiative to help promote it further on April 15, 2003. This has led to a new national health promotion program known as Healthy People 2010. This focuses on 28 areas that have been identified as ways of improving the health and fitness of Americans. In 2005, John P. Burke, a businessman strongly associated with cycling, was appointed chairman of the President's Council, with Melissa Johnson as the council's executive director.

SEE ALSO: Federal Initiatives to Prevent Obesity.

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Prevalence of Childhood Obesity in Developing Countries

THE PREVALENCE OF childhood obesity is increasing worldwide. In fact, some argue that rates of childhood obesity are increasing most dramatically in developing countries. The term *developing country* is defined by the World Bank as low- and middle-income economies, technically those with a Gross National Product (GNP) per capita of less than $3,465. Chronic undernutrition and infectious disease have long been associated with developing countries; however, in recent years many developing countries have shown a concurrent increase in both obesity and undernutrition often referred to as the "dual burden" of malnutrition.

Globalization, economic development, and other factors such as commercialization of agriculture and urbanization have led to changing patterns of living, which can be viewed as part of the nutrition transition. Nutrition transition is generally defined as the shift away from diets high in fiber and complex carbohydrates toward more energy-dense diets that are high in sugars, refined foods, and saturated animal fats, coupled with increasingly sedentary lifestyles. These dietary and lifestyle shifts have been attributed to changes such as the low cost of highly refined oils and carbohydrates, the move toward motorized transportation, the increase in sedentary occupations as well as ownership of a television.

While children are exposed to these influences, it seems the impact of the nutrition transition increases with age, meaning that older individuals are more likely to become obese. In addition, the pattern of obesity within developing economies varies greatly according to class, gender, age, and region (urban–rural). For example, while certain countries, such as Brazil, Mexico, and Chile, show an inverted relationship between social class and obesity, this pattern is not yet observed in India. Regardless of the distribution of obesity within country, in developing countries it has been recognized that rates of obesity are increasing rapidly in children.

It must be acknowledged that these prevalence statistics should be read with awareness of their limitation. Given the importance of monitoring overweight and obesity prevalence, it is remarkable that there is a general lack of precise data by country. Part of the explanation for this is that obesity rates are increasing so fast that prevalence statistics become quickly outdated. In addition, it is difficult to make comparisons between studies because of a lack of consensus on the most appropriate way to define obesity in childhood. For example, before 2000, there was no internationally accepted definition of childhood obesity. In adult populations, a body mass index (BMI) cutoff has been selected to reflect functional impairment and health risk; however, many of the detrimental health effects of childhood obesity appear in adult life, not necessarily in childhood. Prior to 2000, some studies

defined obesity in children as weight-for-height Z-scores over two standard deviations above the World Health Organization (WHO)/National Center for Health Statistics (NCHS), while others used this cutoff to define overweight.

In 2000, the International Obesity Task Force devised a set of guidelines for defining overweight and obesity in childhood based on nationally representative cross-sectional surveys on growth from six countries (Brazil, Great Britain, Hong Kong, the Netherlands, Singapore, and the United States). These standards provide cutoff points for BMI in childhood using data set-specific centiles linked to adult cutoff points of a BMI of 30 at age 18 years. However, given that functional impairments occur at different BMIs for different populations, some epidemiologists have argued that a flexible BMI standard should be used.

In addition, there has been criticism that the six selected countries are not applicable worldwide, which has meant that many countries have created their own definitions and cutoff points. Finally, BMI does not distinguish between fat mass and nonfat mass or give an indication of body fat distribution which both affect health risk. Other than defining childhood obesity, another challenge with data reliability is that different studies use different age groups, which makes comparison difficult. Studies also vary in the time period they were taken as well as in how obesity was assessed (externally measured versus self-reported anthropometric indicators).

Regional trend estimates both quantitatively and qualitatively on the scale of childhood obesity can be illuminating. It is generally recognized that the highest rates of obesity occur in the Pacific region while the lowest rates are found in South Asia, closely followed by East Asia. Rates are also quite high in Middle Eastern and Latin American and Caribbean countries although both regions display considerable heterogeneity. Rates are not quite as high in the African region, but like the Middle East, Africa has variable rates. While it was argued in 2000 that obesity would not be a public health problem in children in developing countries, this assessment did not take into account the rapid lifestyle changes that have occurred over the past seven years in all sectors of society in developing countries.

The accurate monitoring of childhood obesity prevalence is important for several reasons. First, this information can be used to identify populations at risk of becoming obese along with the associated health factors in adult life, such as cardiovascular disease and cancer. Second, reliable data can assist international agencies, government officials, and policymakers with both the mobilization and adequate allocation of resources to address obesity. Third, knowledge of the extent to which obesity is affecting all regions of the world, albeit to varying degrees, can help build consensus on global strategies.

Nutrition transition is related to the global capitalist system and associated neoliberal ideology, thus strategies to counter childhood obesity cannot only be addressed at the national level. International strategies are necessary. Finally, as an increasing number of developing countries embark on national programs for the control of overnutrition, prevalence estimates provide baseline data for the monitoring of effectiveness and impact.

SEE ALSO: Ethnic Disparities in the Prevalence of Childhood Obesity; Prevalence of Childhood Obesity Worldwide; World Patterns.

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Prevalence of Childhood Obesity in the United States

MUCH LIKE ADULTS, children have experienced a rapid and dramatic increase in the prevalence of overweight during the last 30 years. This change has occurred among children of different ages, genders,

socioeconomic classes, and ethnic groups, although certain populations are at higher risk than others. Overweight children have an increased risk of becoming overweight adults, raising concerns about an increased burden of diseases such as diabetes, hyperlipidemia, and hypertension which are associated with obesity in adults. In addition, some of these complications of obesity are being seen more frequently even during childhood.

DEFINITIONS

Before discussing national trends, it is important to define some terms. Obesity refers to a condition of excess body fat, or adiposity, usually expressed as a percentage of body fat. Body fat may be determined by measuring energy density via water displacement or by dual energy X-ray absorptiometry (DEXA) or estimated indirectly by measuring waist circumference or triceps skin-fold thickness. By contrast, overweight refers to excess body weight. In the United States, overweight in adults is defined as a body mass index (BMI) greater than 25, and obesity is defined as a BMI greater than 30. Overweight in children, on the other hand, is usually determined by comparing the child to an existing set of standard growth charts created by the Centers for Disease Control and Prevention. By convention, a child whose BMI falls above the 85th percentile on the chart is described as at risk for overweight, and a child whose BMI falls above the 95th percentile is described as overweight.

Some critics have raised concern in the media and elsewhere about using BMI to determine risk for future health problems in children. Because BMI is a measure of overweight (it is defined as weight in kilograms divided by height in meters squared), it does not account for bone density or lean muscle mass and therefore might theoretically overestimate the number of children who are at risk of becoming obese. However, other measures of obesity discussed above are either difficult to perform in clinical settings or vary widely depending on the measurer. BMI, on the other hand, is simple, easily duplicated, and has been shown in several studies to have a high specificity but a relatively low sensitivity. In other words, someone who is not obese is unlikely to be classified as obese using this measurement, although it is possible that someone who is truly obese may be classified as normal weight by mistake. Furthermore, an elevated BMI in adulthood is associated with a number of complications of obesity.

TRENDS IN PREVALENCE OF OVERWEIGHT IN CHILDREN

Data regarding the prevalence of overweight in childhood in the United States is available through the National Health and Nutrition Examination Surveys (NHANES), a series of national surveys of representative samples of the entire U.S. population that have occurred periodically since the early 1970s. These surveys replaced the National Health Examination Surveys (NHES), which date back to the 1960s. Thus, a representative sample of weight in children in the United States can be followed for more than 40 years.

These data show that the prevalence of overweight in children and adolescents was relatively stable during the 1960s and 1970s and began to increase steadily during the 1980s and 1990s. In boys, the prevalence of overweight for adolescents aged 12–17 remained between 5.2 and 5.4 percent from 1963 until 1980, increased to 12.8 percent in 1988–91, 14.8 percent in 1999–2000, and 18.3 percent in 2003–04. In girls aged 12–17, the prevalence of overweight varied between 5.2 and 7.2 percent until 1980, increased to 8.8 percent in 1988–1991, 14.8 percent in 1999–2000, and 16.4 percent in 2003–04. Similar trends occurred for children of other age groups. The overall prevalence of overweight in children aged 2–19 was 17.1 percent in 2003–04.

These trends represent more than just a shift in the normal distribution of weight among children. Not only are the percentages of children in the overweight category increasing, but also the entire population of children is skewing toward overweight, and those who are overweight have a higher BMI than ever before. In other words, those prone to overweight are becoming more overweight than in previous years and at higher rates.

This epidemic preferentially affects minority children and those of low socioeconomic status. Mexican-American children and adolescents are more likely to be overweight than white children, and black girls are more likely to be overweight than white girls. Twenty-two percent of Mexican-American boys aged 2–19 were overweight in 2003–04 along with 23.8 percent of non-Hispanic black girls. Unfortunately, because the survey includes only small numbers of children in each

of these subgroups, it is difficult to assess trends over time with statistical confidence.

Children of low socioeconomic status have a two to three times higher risk of obesity. While poverty increases the risk of overweight overall, it may not do so equally among different ethnic groups. Among White children, a higher income is associated with a lower prevalence of overweight. No such relationship occurs for black or Hispanic children. This finding is particularly disturbing because it suggests that race and ethnicity carry an increased risk of adverse health outcomes regardless of socioeconomic status.

POTENTIAL CONTRIBUTORS

The rapid change in the prevalence of overweight during the last 20 to 30 years is more than can be explained by a change in genetic factors alone. Recent studies suggest that between 25 and 40 percent of the risk of obesity can be attributed to genetic risk. A family history of obesity also confers a two to three times greater risk of obesity in a child. It seems logical that interplay between genetic susceptibility and environmental changes have combined to produce the current epidemic.

Because overweight can ultimately be related to an imbalance between energy intake (i.e., the number of calories eaten) and energy expenditure (i.e., the number of calories used either through metabolism or physical activity), changes in the environment can be analyzed in much the same fashion.

A number of trends in production and consumption of food are possible contributors to the change in prevalence of overweight in children. Overall, more food is available in the United States at cheaper prices than ever before. The actual cost of food production has decreased compared to the cost of packaging, marketing, and distribution. Perhaps as a result, portion sizes of commonly available convenience foods have increased, as have the portion sizes of soft drinks. Some evidence suggests that most adults and children given larger portion sizes will consume more calories over time. Some evidence also correlates consumption of sugared drinks (soda and juice) with prevalence of overweight.

Along with an increase in rates of obesity, the last 30 years have seen an increase in the number of meals eaten outside of the home. Meals consumed away from home tend to have more calories. Over the same time, vending machines have made soft drinks and high-calorie snacks more available to children in schools. Although no direct evidence ties these changes to increases in the number of overweight children, these factors may contribute to an environment that encourages increases in caloric consumption.

Others have pointed to a more sedentary lifestyle in parents and children as a major culprit in the obesity epidemic. Several studies have focused on television watching as a major risk factor for overweight. Television watching may displace more active pursuits and thus decrease energy expenditure, or it may encourage more energy intake either via food advertising or directly via food consumed while watching. Evidence has been mixed in connecting overweight to television time, although therapies aimed at decreasing television time have shown changes in rates of overweight.

Changes in the built environment have led to more automobile travel during the 1980s and 1990s. The percentage of children walking or biking to school has decreased over the last three decades. When asked why, most parents report that the school is too far or that there is no safe walking route. At the same time, schools have decreased the time allotted for recess and physical education. Although no evidence ties these trends directly to the prevalence of overweight, the fact that these changes have occurred at the same time as the occurrence of the obesity epidemic makes them possible contributors.

CONSEQUENCES

Given the increase in the prevalence of overweight in children, the question becomes, what is the significance of these changes for the long-term health of children? First, we must understand how well these patterns track over time. Do overweight children become overweight adults?

The vast majority of adults who are overweight were not overweight as children. Nonetheless, it is clear that being an overweight child increases the risk of being an overweight adult somewhat. For the youngest children (aged 1–2), there is little correlation, and in these children, parental obesity is a better predictor of future risk than the weight of the child. After age 6, the risk of adult overweight increases, especially with rapid weight gain. During adolescence, the risk of adult obesity becomes much higher. According to one study, overweight 15- to 17-yearolds

are 17.5 times more likely to become overweight than those who are not already overweight.

Overweight adolescents have an increased risk of loss of socioeconomic status, physical illness, and even death as a result of their weight. Adult women who had been overweight adolescents have been shown to have higher rates of poverty, are less likely to be married, and have completed fewer years of school, even after adjusting for their baseline socioeconomic status and aptitude scores. Over the long term, overweight in adolescence has been shown to increase the risk of all-cause mortality and cardiovascular mortality in particular, although this result has not been confirmed in all settings.

Overweight adolescents face health risks not only in the future, but also in the present. Recent studies have shown a dramatic increase in the prevalence of Type 2 diabetes in children and adolescence, and in this age group, the disease is almost exclusively seen in the overweight. Furthermore, 28.7 percent of adolescents meet the definition for metabolic syndrome, a condition defined by a combination of high blood pressure, alterations in cholesterol and lipid levels, and elevated fasting blood sugar.

Obesity in adulthood is notoriously difficult to treat once it occurs. For this reason, much of the focus in health policy and elsewhere has shifted to obesity prevention, especially in childhood. Given the dramatic increases in the prevalence of childhood obesity in the United States, policy makers may have to start earlier in childhood than they anticipated. The complex interplay of genetic factors and changes in the environment may require a coordinated approach involving individuals as well as larger institutions. Given the potential health risks of obesity, such an investment would likely be well worth it in the long run.

SEE ALSO: Accessibility of Foods; Assessment of Obesity and Health Risks; Body Mass Index; Breastfeeding; Built Environments; Childhood Obesity as a Risk Factor for Adult Overweight; Prevalence of Childhood Obesity Worldwide.

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Prevalence of Childhood Obesity Worldwide

CHILDHOOD OBESITY IS a particular public health concern because excess weight in childhood is likely to lead to excess weight in adulthood and raise the risk of obesity-related chronic disease from an earlier age. Overweight children themselves may show the early signs of chronic disease and also suffer psychosocial problems due to their obesity. Virtually all countries with reliable data are showing rising trends in child obesity prevalence, and that in many economically developed countries, excess bodyweight is affecting more than a quarter of all schoolchildren.

AGREEING STANDARD DEFINITIONS

An ideal measure of body fat should be accurate in its estimate of body fat; easy to obtain in terms of time, cost, and acceptable to the child; and well-documented with published reference values. Sadly, no existing measure satisfies all these criteria. Measurement of fatness (adiposity) in children and adolescents occurs in a range of settings, using a range of methods, and compared against a range of different reference charts and tables.

Direct measures of body composition, such as underwater weighing, magnetic resonance imaging, computed axial tomography scans, and dual energy X-ray absorptiometry are impractical for everyday purposes such as checking children in a family doctor's clinic or in a school survey. Much easier to measure, but less accurate, are waist, hip, and other girth measurements, skin-fold thickness, and height and weight. Height and weight taken together provide body mass index (BMI). Although it has its shortcomings, BMI is by far the most commonly used method for assessing adiposity in children and in adults, and in this entry on obesity prevalence, the figures are based on BMI.

Even with BMI, care has to be taken about how the information is collected. If a trained, experienced person is measuring children, then the BMI will be as good as can be expected, but if children or their parents do the measuring, or if children are asked just to say what weight and height they believe they are, then the BMI will probably be rather inaccurate. Self-reported heights and weights are notoriously bad, with girls tending to underestimate their true weight, and boys tending to overestimate their true height, so that in both genders the BMI will be inaccurate. Typically, this can mean that quite a few children who would be classified as overweight using measured BMI slip below the threshold and are classified as normal weight based on their self-reported BMI. In this entry, only measured BMI is reported unless no other data are available.

With adults, it is generally accepted that the BMI thresholds of 25 and 30 [2] are the thresholds for being overweight (BMI greater than 25) and obese (BMI greater than 30), although there is some discussion that lower values are more appropriate for some population groups. For children, the situation is different. The BMI for a normal child changes as the child grows through infancy, childhood, and adolescence, and therefore, the thresholds of BMI 25 and 30 need to be adjusted to take into account these growth patterns. There are several approaches that have led to a range of different reference charts and tables.

The first approach is to use a survey from the national population taken before obesity was widespread, and define the top 10 percent, say, as being overweight and the top 3 percent, say, as being obese. This gives a set of values representing the top-ranking children at every stage. Separate sets can be defined for girls and boys, as growth patterns differ between the two sexes. There are two problems. The first problem is that the thresholds of 10 and 3 percent, or any other pair of thresholds (some charts use 15 and 5 percent, for example), are arbitrary, whereas the adult BMI 25 and 30 are based on a link to the raised health risks associated with being above each of those thresholds. The second problem is that the set of data used to define the top percentages is also arbitrary—should they be based on the national population 10, 20, or more years ago, or perhaps on another country's data, or on some internationally agreed set of data?

A second approach is fairly similar to the first, but instead of using a standard percentage as a threshold, it uses a statistical definition in terms of the distribution of a reference set of data, for example, two standard deviations above the mean, or the mean plus 20 percent of the mean. The resulting threshold values are just as arbitrary in terms of their relationship to actual health outcomes. They also suffer the same questions about the original data set from which the thresholds are taken.

In many economically developed countries, excess bodyweight is affecting more than a quarter of all schoolchildren.

The third approach, and the one used here, is to create a large data set from across several different countries to set the BMI thresholds of 25 and 30 as applying at age 18, and then to plot backward from age 18 keeping the same proportion of children above the thresholds. Thus, if, for example, the large combined data set showed 9 percent of children above BMI 25 and 4 percent above BMI 30 at age 18, these same percentages are tracked back through to a young age and the resulting set of BMIs at every age is used instead of the adult 25 and 30 figure. This calculation has been done and a set of cutoff BMIs for children aged 2–18 years, for boys and girls separately, were published in 2000, based on large national data set for six different countries. The cutoffs are known as the Cole cutoffs (after the main author) or the International cutoffs (IOTF, after the International Obesity Task Force, which promoted the idea). In this entry, the figures are all defined according to the Cole-IOTF cutoffs.

Readers should note that the United States has its own definitions, based on a national set of figures, and its own terminology, sometimes referring to "at risk of overweight" and "overweight" for the top two tiers of adiposity, and sometimes to "overweight" and "obese."

The World Health Organization (WHO) has for many years recommended using a "2 standard deviations" set of cutoffs, based on reference data obtained from the United States, but more recently the WHO has been reviewing its recommendations. The WHO is concerned that the U.S. data included large numbers of formula-fed infants, which changes their growth patterns and underestimates the true extent of overweight in younger children, and the WHO is now in the process of producing a new "gold standard" set of reference data from healthy breast-fed babies. In principle, these could be used in conjunction with the Cole-IOTF methodology to produce a "gold standard" set of BMI cutoffs similar to those already produced by Cole-IOTF but based on a more robust set of data.

Last, care should be taken when looking at prevalence figures for overweight and obesity. Some authors use "overweight" to define all people with BMI above 25, and some to define all people with a BMI between 25 and 30. Thus, in some reports the prevalence of "overweight" people includes obese people and in other reports it does not. In the present entry, "overweight" includes obese, so the term should properly be understood to mean "overweight including obese."

Note that for young children, it has been common practice to use a different measure of fatness, namely "weight for height." This came from the days when most researchers were concerned with underweight and stunting, and so "weight for age," "height for age," and 'weight for height" were used to assess infant growth. This is still used in young children, usually with a threshold of 2 standard deviations above a reference mean as the criteria for excess weight for that child's age. This approach is used in this entry only for children under age 5 where shown.

It should also be noted that the definitions are very helpful for making comparisons between different groups of people, or comparing a group of people over time. Using these threshold definitions is not recommended for the clinical assessment of individual children, where more careful examination of the child is needed to be sure that, for example, a high BMI is not due to extra muscle.

CHILD OBESITY PREVALENCE AROUND THE WORLD

Two problems have hindered a complete understanding of the global circumstances surrounding obesity in children and adolescents: first, the lack of comparable representative data from different countries, and second, the use of varying criteria for defining obesity among different countries and researchers, referred to previously.

Even where some data are available, they need to be treated carefully. First, were they collected using proper measurement procedures or are they self-reported (or parent reported)? Are they nationally representative surveys or are they smaller surveys that do not represent national populations? And when comparing two surveys across a period of time, are the surveys properly comparable in terms of the children's ages and locations, and so forth? The trend figures presented here are those based on the most objective, representative, and comparable surveys available.

Taking an estimate for the world as a whole, in 2004 some 10 percent of school-age children (aged 5–17) were defined as overweight (including obese) (equivalent to BMI >25) of which some 2 to 3 percent were

Table 1. Estimated Prevalence of Excess Bodyweight in School-Age Children by 2010

REGION*	OBESE	OVERWEIGHT (INCLUDING OBESE)
Americas	15%	46%
Middle East and North Africa	12%	42%
Europe and former USSR	10%	38%
West Pacific	7%	27%
Southeast Asia	5%	23%
Africa	>1%	>5%

** Countries in each region are according to the World Health Organization.*

obese (equivalent to BMI >30). This global average reflects a wide range of prevalence levels in different regions and countries, with the prevalence of overweight in Africa and Asia averaging well below 5 percent and in the Americas and Europe above 20 percent.

THE AMERICAS

The most comprehensive and comparable nationally representative data on trends in the prevalence of obesity are from the United State, from the National Health Examination Survey (NHES) I and II surveys undertaken in the 1960s and the series National Health and Nutrition Examination Surveys (NHANES) from 1971 onward. The most recent data (for 2003–2004) show that 36 percent of children aged 6–17 were overweight, including 13 percent obese. These figures are based on the international (Cole-IOTF) definitions; using U.S. definitions, the equivalent figures are 36 percent and 18 percent, respectively.

In Canada, the available data (from self-reported information) for 1996 show 33 percent of boys and 27 percent of girls were overweight. In Brazil, the prevalence of overweight among school-aged children was 14 percent in 1997. In Chile, in 2000, the prevalence of overweight among school children was 26 percent.

There are few data available for schoolchildren in most other South and Central American countries, but some data have been collected for preschool children. In Bolivia, the prevalence of overweight (defined as 1 standard deviation above a reference mean) was 23 percent in 1997, and in the Dominican Republic, it was 15 percent in 1996. In a few countries in the region, obesity prevalence has fallen—in Colombia, it fell from 5 to 3 percent between 1986 and 1995.

EUROPE

A number of studies have examined childhood overweight and obesity prevalence in European countries. The highest prevalence levels are observed in southern European countries. A survey in 2001 found that 36 percent of 9-year-olds in central Italy were overweight, including 12 percent obese. In northern Greece in 2000, 26 percent of boys and 19 percent of girls aged 6–17 years were overweight, while data from Crete in 2002 show 44 percent of boys aged 15 to be overweight or obese. In Spain, 35 percent of boys and 32 percent of girls aged 13–14 years were overweight in a survey in 2000.

Northern European countries tend to have lower prevalence. In Sweden in 2000–01, the prevalence was 18 percent for children aged 10 years. In the Netherlands, the figures are remarkably low, with only 10 percent of children aged 5–17 overweight, including only 2 percent obese, in a 1997 survey. In France, the figures are a bit higher, at 15 percent overweight and 3 percent obese in a northern French survey in 2000, and in England higher still: with 29 percent overweight, including 10 percent obese, in a 2004 survey.

The reasons for a north–south gradient are not clear. Genetic factors are unlikely to be the explanation, as the gradient can be shown even within a single country, such as Italy and all countries are showing a marked increase in prevalence. The child's household or family income may be a relevant variable, possibly mediated through income-related dietary factors such as maternal nutrition during pregnancy, or breast- or bottle-feeding in infancy, as well as the quality of the diet during childhood.

NORTH AFRICA, EASTERN MEDITERRANEAN, AND MIDDLE EAST

Several countries in this region appear to be showing high levels of childhood obesity. In Egypt, for example, the prevalence of overweight (based on standard deviation greater than 1) was over 25 percent in preschool children and 14 percent in adolescents. Similar figures are found in other parts of the region. A fifth of adolescents aged 15–16 years in Saudi Arabia were defined as overweight (based on BMI greater than 120 percent of reference median value). In Bahrain, in 2002, 30 percent of boys and 42 percent of girls aged 12–17 were overweight, including over 15 percent obese in both groups.

ASIA AND PACIFIC

The prevalence of obesity among preschool children is around 1 percent or less in many countries in the region, for example, Bangladesh (1.1 percent), the Philippines (0.8 percent), Vietnam (0.7 percent), and Nepal (0.3 percent), but it should be noted that no data are available for some countries in the region (e.g., the Pacific islands) where adult obesity prevalence rates are known to be high.

In more economically developed countries the prevalence figures for preschool and school-age children are considerably higher. Among Australian children and adolescents aged 2–18, the prevalence of

overweight and obesity was over 20 percent in 1995 and was likely to be around 28 percent by 2005.

In mainland China, whose population accounts for one-fifth of the global population, the prevalence of obesity has been rising quickly in both adults and children during the past two decades. A survey in 1997 showed the prevalence of overweight, including obesity, among schoolchildren to be 7.7 percent, but this represented an average of urban and rural populations: in urban areas the level was 12.4 percent, and showing how quickly the figures may soon change, among preschool children, the level was 29 percent.

It is worth noting that although the epidemic of obesity seems to have affected a wide range of countries in this region, undernutrition is still a major problem. In China, the prevalence of underweight (less than 5th percentile BMI of the U.S. reference) was 9 percent among children aged 6–9 years, and 15 percent among children aged 10–18, in 1997. In Indonesia, over 25 percent and in Bangladesh and India over 45 percent of children under 5 are underweight. Thus, several of the most populous countries in this region are facing a double burden of continued undernutrition and rising overnutrition.

REGION: SUB-SAHARAN AFRICA

The burden of undernutrition remains very high in this region, with continuing poverty, war, famine, disease (especially human immunodeficiency virus [HIV]/acquired immunodeficiency syndrome [AIDS]), and very high rates of child mortality. There are very few surveys from African countries that can provide prevalence figures for childhood obesity, as most public health nutrition programs have been focused on undernutrition and food safety problems. In general, the prevalence of childhood obesity remains very low in this region, except for countries such as South Africa where obesity has become prevalent in adults, particularly among women, and where childhood obesity is also rising. Data from South Africa show the prevalence of overweight (including obesity) among young people aged 13–19 years to be over 17 percent with boys generally less at risk (7 percent) than girls (25 percent). Prevalence was highest (over 20 percent for both boys and girls) in White and Indian population groups.

TRENDS OVER TIME

The prevalence of excess weight among children is increasing in both developed and developing countries, but at very different speeds and in different patterns. North America and some European countries have the highest prevalence levels, and have shown high year-on-year increases in prevalence. As can be seen from the examples in the table below, the prevalence of overweight is increasing rapidly in a number of countries.

Data from Brazil and Chile are good examples to show that the rate of increase of obesity among children in some developing countries is similar to or even faster than that in the United States or Europe. In Brazil, three nationwide, large-scale surveys were conducted in 1974–75, 1989, and 1997. The prevalence of overweight (including obesity) tripled between the 1970s and the late 1990s, increasing from 4.1 to 13.9 percent among children and adolescents aged 6–18. In Chile, two large surveys conducted on 6-year-old children showed a remarkable increase in childhood overweight (including obesity) between 1987 and 2000, from 12 to 26 percent in boys and from 14 percent to 27 percent in girls.

The rapid rise in the prevalence of overweight is shown in most developed economies, but an interesting exception is Russia, where the economic downturn in the early 1990s may explain the decline in the prevalence of overweight children during the period.

INTERACTION OF NATIONAL WEALTH AND PERSONAL WEALTH

Examination of differences in the distribution of overweight and obesity among children coming from different social classes (defined by family income levels) shows an interaction. In the more economically developed, industrialized countries, it is children in lower socioeconomic groups who tend to show higher prevalence levels of overweight and obesity. In contrast, in countries that are not economically developed or are undergoing economic development, overweight and obesity levels are higher among the higher income sectors of the population and among urban populations rather than rural ones.

In Brazil in 1997, 20 percent of children in higher income families were overweight or obese, compared with 13 percent of children in middle-income families and only 6 percent of children in lower income families. In countries in Western Europe and North America, the trend is the other way, with children in lower income families more likely to be overweight or obese than those in higher income families.

The effect is likely to be closely linked to urban versus rural location. Children in urban areas in less developed countries tend to show higher prevalence levels of overweight and obesity, and to have higher levels of family income, compared with their rural counterparts. In North America and Western Europe, children in rural areas tend to have higher levels of overweight and obesity, and lower levels of family income, compared with their urban counterparts.

Underlying these interactions are several changes in the "obesogenic" environment experienced by children which is closely linked to economic development and urban–rural differences. One is the change in physical activity levels, from hard agricultural labor and domestic labor to television watching, and from active transport (walking, cycling) to motorized transport (especially the use of cars) even for short journeys such as getting from home to school or to shops. Another is the change in diet from traditional staples to a modern, processed diet with high levels of soft drinks, fast foods, snacks, and confectionery. As a country's economy develops, these processed foods are initially relatively expensive and available only in urban areas. In a developed, industrialized economy, these foods are cheap sources of food energy and are easily found even in rural areas.

THE NEED FOR SURVEILLANCE

The data shown in this entry give a picture of what is happening and the trends over time. They show the extraordinarily high levels of excess bodyweight among children in many regions of the world, and that these levels are rising rapidly. However, for many regions and large numbers of countries, there is very little information and no way of telling whether the trends show a similar, slower, or even faster increase in child obesity.

Nationally representative data in developing countries are particularly needed, especially for older children (over 5 years old) and adolescents, and these data will be especially valuable for monitoring trends in obesity during periods of economic change and urbanization.

Surveillance is not only useful to identify trends and to compare different countries, but it also provides essential information to policy makers on what they should do. Without adequate surveillance, it would be very difficult to tell if an intervention, such as a program to improve school food services or to restrict advertising to children, is having an effect on obesity levels.

To improve the situation, it is important for governments to devote sufficient resources to ensure that they can survey the status of their children and to repeat this surveillance at regular intervals to build up a pattern showing what is happening.

Furthermore, there needs to be an accepted international definition of child overweight and obesity, allowing the trends and changes in different countries to be compared. Revised standards from the WHO are expected to be able to provide this, and some means of converting previous data to the new standards will need to be devised.

SEE ALSO: Prevalence of Childhood Obesity in Developing Countries; Prevalence of Childhood Obesity in the United States.

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Prevalence of Disordered Eating

DISORDERED EATING IS a general term that describes the numerous types of abnormal or unhealthy eating behaviors experienced by people with and without an eating disorder. Some of the most common disordered eating behaviors include binge eating, chronic dieting and restrictive eating, emotional eating, general overeating, and night eating.

Disordered eating is common in community and weight-loss seeking samples of overweight or obese individuals. The purpose of this entry is to provide a general overview of the types and prevalence of

disordered eating common to overweight and obese individuals. A brief review of disordered eating in children, ethnic minorities, and individuals of low socioeconomic status is also presented.

BINGE EATING

According to the *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed., Text Revision (DSM-IV-TR), binge eating is defined as eating an amount of food that is definitely larger than most individuals would eat under similar circumstances. The eating must occur in a discrete period of time which, although controversial, is generally operationalized as eating that occurs within a two-hour period. The individual must also feel a loss of control or inability to stop eating.

In addition to being a central diagnostic criterion for both bulimia nervosa and binge-eating disorder, binge eating is a common and serious problem in noneating disordered overweight and obese men and women. According to the National Comorbidity Survey conducted between 2001 and 2003, about 4.5 percent of Americans binge eat. In overweight and obese individuals, binge eating tends to be more common among those in weight-loss programs; an estimated one-third of obese individuals engaging in some type of weight-loss treatment binge eat on a regular basis.

Although an individual must both feel a loss of control and consume a large amount of food in a short amount of time to technically have an objective binge, many obese individuals report subjective binge-eating episodes in which they overeat without a loss of control or feel a loss of control but do not eat an objectively large amount of food. These subjective binge-eating episodes are often as distressing as objective binges and are likely extremely common in overweight and obese individuals.

Food consumption patterns and nutrient composition differ in obese individuals who binge eat from obese individuals who do not binge eat. Individuals who binge eat consume more kilocalories, a higher percentage of calories from fat, and less energy from protein than obese individuals who do not binge eat. Additionally, obese binge eaters often report eating continuously over time or "grazing" in which they feel out of control of their eating over an entire day.

Given that many obese individuals who binge eat do not engage in healthy compensatory behaviors to reduce the calories ingested during a binge (e.g., through exercise), it is likely that binge eating contributes to increased weight gain and worsening comorbid physical and psychological conditions. Consequently, decreasing binge-eating behavior in obese individuals is important to effective weight-loss treatment.

CHRONIC DIETING AND RESTRICTIVE EATING

Chronic dieting and restrictive eating are terms that refer to altering one's food consumption patterns to reduce one's overall energy intake. The aim of those who chronically diet is generally to lose weight or body fat.

The American public is obsessed with weight-loss programs and dieting products. Diet aids, exercise programs, drugs, special foods, and over-the-counter pills dominate grocery store aisles, pharmacy shelves, and media advertisements. Popular diets of today range from severe caloric restriction to myriad fad diets that promote specific dietary regimes, such as eating foods very low in carbohydrates, high in protein, or low in fat. Other types of diets include eating only foods grown under specific conditions, such as macrobiotic diets, or eating only nonanimal products (e.g., vegan). Dieting is so popular and prevalent in the United States that it is now a $50 billion-per-year business.

Dieting is normative among obese, overweight, normal-weight, and underweight individuals alike. In a national survey of high school students, more than 40 percent of girls reported that they were on a diet, many of whom did not believe they were overweight. The overwhelming majority of obese individuals, particularly those seeking weight-loss assistance, report they have tried every method of weight loss possible with no long-term success. Unfortunately, research suggests that although obese individuals can lose weight while dieting, long-term, sustained weight-loss through calorie restricting dieting is not typical. More than 90 percent of individuals who lose weight will regain it eventually and within five years of being on a very-low-calorie diet, about 40 percent of individuals actually weigh more than they did before they started dieting.

In fact, prolonged dietary restriction and chronic dieting may actually lead to unhealthy food consumption patterns, lowered metabolic rates, difficulty experiencing feelings of satiety, and increased disordered eating behavior. Strict dietary restriction can actually place one at higher risk for increased future weight gain because resting metabolic rates decrease when energy intake is reduced. If an individual repeatedly restricts

his or her energy intake by dieting, weight gain may occur at a faster pace when regular eating resumes. Many individuals describe this commonplace dieting experience as "yo-yo" dieting or "weight cycling" because dieters will lose and gain weight in a patterned format characterized by periods of severe dietary restriction followed by attempts at healthy, normal eating that lead to weight regain. Dieting and restrictive caloric intake also often precede binge-eating episodes.

In the case of obese individuals, finding interventions and strategies that successfully facilitate long-term weight loss have received considerable attention for obvious reasons. In general, individuals who engage in chronic dieting and severe dietary restriction should consult health professionals to ensure that their dietary schedule contains adequate nutrients to maintain health. The most effective, long-term weight-loss diets are generally characterized by moderate, gradual weight loss through reduced portion sizes and limited consumption of high-calorie, low-nutrient foods.

EMOTIONAL OVEREATING

Emotional overeating is simply eating to soothe emotion. Most often, emotional overeating occurs when individuals feel angry, sad, lonely, tense, nervous, bored, or anxious. Research suggests that up to 75 percent of overeating may occur in the presence of strong emotional states. For individuals who engage in emotional overeating, food often becomes a tool to cope with stress and daily life problems. Overeating can become a substitute for emotional connection, such as love and attachment. In fact, some obese individuals will report that although they would like to lose weight, being overweight serves as an important metaphorical physical and psychological barrier to emotions that would arise if they lost weight and confronted painful experiences in their past and current lives.

While overeating, many obese individuals report that food is comforting and soothing, often acting as a sedative and escape from negative emotion. However, the temporary soothing of adverse emotion comes at a cost because the emotional relief gleaned from overeating is temporary and often leads to feelings of guilt and shame. In the long term, emotional overeating can lead to decreased self-efficacy, lowered self-esteem, and emotional instability.

Overweight individuals who chronically diet are at increased risk for emotional overeating. Experimental and correlational research suggests that chronic dieters who would ordinarily sustain control over their eating are much more likely to overeat when emotional.

Eating to regulate emotion is commonplace in obese individuals. It is likely that all individuals engage in emotional overeating on occasion. For those who regularly overeat in the presence of strong emotional states, however, it can be a barrier to weight loss and psychological health. People who emotionally overeat must learn to experience their feelings and cope with emotional discomfort in healthier, nonappetitive ways.

SIMPLE OVEREATING

Simple overeating is defined as eating more calories than is recommended given one's height and age. Most, if not all, overweight and obese individuals consume more food than would be dietarily recommended on a regular basis. In general, obese individuals are more likely to engage in overeating during mealtimes, periods of low arousal at which time they are not actively engaging in an activity, and when emotionally upset.

One reason for the prevalence of overeating in the United States is environmental. Regular portion sizes have increased substantially over time. By the late 1990s, the average American ate about 500 more calories a day than he or she did in the 1950s. Restaurants regularly provide much larger portions of foods than recommended. Additionally, restaurant foods tend to be higher in fat, sugar, salt, and preservatives than home-cooked meals.

What is clear is that consuming more calories than one expends will lead to weight gain. For obese and overweight individuals, successful weight loss will necessitate eating smaller portions of healthy food and avoidance of overeating.

BINGE-EATING DISORDER

Binge-eating disorder is a psychological disorder particularly relevant to obese and overweight individuals because the overwhelming majority of individuals suffering with binge-eating disorder are overweight or morbidly obese. According to DSM-IV-TR, binge-eating disorder is not a recognized psychiatric disorder per se, but rather, a proposed psychological disorder in need of further study.

According to the proposed diagnostic criteria, binge-eating disorder is characterized by binge-eat-

ing episodes associated with three or more of the following: eating more rapidly than normal, eating until uncomfortably full, eating when not hungry, eating in secret due to embarrassment over the amount and types of food being consumed, and feelings of distress or disgust after eating. Unlike individuals diagnosed with other eating disorders such as bulimia nervosa, there are no compensatory behaviors (e.g., excessive exercise, purging, laxative use) to rid oneself of the calories ingested during binge-eating episodes.

Prevalence rates for binge-eating disorder vary. According to the National Comorbidity Survey, lifetime prevalence of binge-eating disorder is about 3 percent in the general American population. In obese individuals, prevalence rates in the United States are estimated to be about 2 to 4 percent. However, in treatment centers and weight-loss programs, an estimated 15 to 50 percent of obese individuals meet diagnostic criteria for binge-eating disorder.

Individuals with binge-eating disorder differ from those with uncomplicated obesity in various ways. They are more likely to suffer from other psychiatric disorders including major depressive disorder, anxiety disorders, and personality disorders (particularly borderline and avoidant personality). They are also more likely to seek treatment for obesity, but are significantly more likely to regain weight after weight-loss treatment than obese individuals without binge-eating disorder. Finally, there is a higher prevalence of individuals with binge-eating disorder who are severely obese (body mass index [BMI = weight (kg)/height $(m)^2$] = 40) than those without an eating disorder. That said, individuals with binge-eating disorder are more likely to be morbidly obese and are, therefore, at risk for medical complications of obesity.

NIGHT EATING AND NIGHT EATING SYNDROME

Night eating is a term used to describe individuals who maintain relatively healthy eating patterns during the day but are unable to stop themselves from eating at night. Night eating syndrome is a severe form of night eating. First described by Dr. Albert J. Strunkard, night eating syndrome describes individuals who severely restrict their eating during the morning hours to the degree that they may eat no food or negligible amounts of food early in the day. This period of restriction is followed by evening hyperphagia, defined as the consumption of more than 25 percent of one's daily caloric intake after the evening meal.

Individuals who engage in night eating generally wake with little or no appetite in the morning. Often, they eat more than half of their daily food intake between dinner and breakfast. Many individuals who engage in night eating report significant sleep impairment and, in fact, may not have an awareness of their eating as it occurs or memory of their eating. When aware of their eating, however, night eating is associated with feelings of shame and guilt, not enjoyment.

Prevalence estimates of night eating and night eating syndrome are preliminary because few outcome studies have been conducted. In community samples, an estimated 1 to 2 percent of the adult population in the United States engage in night eating. In obese individuals seeking weight-loss treatment, an estimated 6 percent struggle with night eating. In bariatric surgery patients, preliminary estimates vary widely and range from 8 percent to greater than 40 percent. Some researchers believe that more than 25 percent of people 100 pounds or more overweight have some night eating problems.

Night eating and night eating syndrome are different from other forms of disordered eating and eating disorders because they are considered to represent a unique combination of disordered eating behavior, mood dysregulation, and disordered sleeping patterns. A thorough assessment and, when needed, intervention that modifies eating and sleeping behaviors is important to curb night eating.

SPECIAL POPULATIONS

The prevalence of overweight and obesity is increasing in all major age, socioeconomic, and ethnic groups. However, prevalence rates of disordered eating in children, ethnic minorities, and low-income groups deserve mention.

The prevalence of obesity in children is noteworthy because childhood obesity is a risk factor for disordered eating and a wide range of psychosocial difficulties. In the United States today, approximately 15 percent of American teenage girls and 14 percent of teenage boys are obese. An additional 30 percent of girls and 28 percent of boys are somewhat overweight.

With regard to disordered eating, recent research suggests that up to 4 percent of obese children meet diagnostic criteria for binge-eating disorder. Approximately 20 to 60 percent for girls and 30 to 50 percent for boys aged 6–11 report that they are currently on a diet. Additionally, adults with binge-eating disorder are more than three times as likely to have been overweight as children when compared to individuals with other, noneating-related psychiatric diagnoses. When working with children who engage in disordered eating, interventions should begin as soon as problematic eating is discovered to reduce the risk of psychiatric problems and obesity in adulthood.

Although there is little written about issues related to obesity in ethnic minority and low-income groups, the prevalence of disordered eating and obesity in these populations is staggering. It is estimated that rates of obesity are 10 to 300 percent higher in ethnic minority individuals than in Euro-American Whites in the United States. The highest rates of obesity are observed in Native Hawaiians, Pacific Islanders, African Americans, and Hispanics. In weight-loss seeking individuals, there may be issues related to ethnic ideals of appearance and cultural eating patterns, such as food preparation techniques, traditional recipes, and typical food groups, that warrant attention when working with obese individuals from different cultural and ethnic group.

Low-income communities tend to have higher rates of obesity than higher income areas in the United States. Healthy foods such as fresh fruits, vegetables, meats, and dairy products are more expensive than prepackaged meals and fast food. They also take more time to prepare before eating. Issues related to the availability of financial resources to purchase diet aids and healthier food, time to devote to preparing healthier food and exercise, health priorities, and resources to promote weight-loss and physical health must be considered to adequately assist weight-loss–seeking individuals from low-income communities in losing weight.

SUMMARY AND CONCLUSIONS

Disordered eating behaviors including binge eating, chronic dieting and restrictive eating, emotional eating, general overeating, and night eating are common in obese and overweight individuals. Mainstream American culture, teeming with fast-food restaurants, snack foods high in sugar and fat, and stigmatization of fatness, is not conducive to weight loss for the millions of overweight and obese individuals living in the United States. Organizations such as Overeaters Anonymous have been created to help individuals decrease disordered eating in all its forms and aid obese and overweight individuals lose weight in healthy, sustainable ways.

SEE ALSO: Anorexia Nervosa; Binge Eating; Bulimia Nervosa; Compulsive Overeating; Dieting: Good or Bad?; Disordered Eating; Eating Disorders and Obesity; Food "Addictions"; Low Calorie Diets; Mood and Food; Night Eating Syndrome.

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Prevalence of Obesity in U.S. Women

OBESITY HAS BEEN identified as a public health issue of great concern in the United States with more nearly two-thirds of the population considered overweight or obese. Although obesity rates overall have been climbing at an alarming rate for the last 30 years, they appear to have leveled in the female population. However, over 30 percent of all women in the United States are obese, and factors such as socioeconomic status and race/ethnicity are related to disparities in prevalence, particularly for poor and minority women. Obesity is associated with health risks such as cardiovascular disease, diabetes, and cancer for both men and women, but women also experience additional health risks and complications from obesity related to reproduction, childbirth, and greater social stigma. Several initiatives and public health programs target women, and particular subsets of women, in the effort to reduce, prevent, and treat obesity.

OVERALL PREVALENCE AND MEASUREMENTS OF OBESITY

Obesity has been labeled as a public health crisis in the United States. It is estimated that one-third of all adults in the United States are overweight and an additional one-third are obese. Since the 1960s, the number of individuals who are classified as overweight or obese has increased at rapid and alarming rates. The prevalence of adult obesity (aged 20–74), for example, rose from 13.3 to 30.5 percent from 1960 through 2002. The prevalence of extreme or severe obesity rose from 0.8 to 4.9 percent during the same time period. In fact, the fastest-growing group of obese individuals in the United States is the severely obese group (those with a BMI over 40). From 2000 to 2005, for example, the prevalence of severe obesity increased by 50 percent.

Body mass index (BMI) is the most commonly accepted measure used for categorizing individuals as under- or overweight, obese, or severely obese. BMI is calculated by dividing a person's weight in kilograms by his or her height in meters (weight [kg]/height [m^2]). BMI is not gender specific and does not differentiate the amount of weight from muscle mass versus fat, so very muscular individuals could be misclassified by using BMI alone. A person with a BMI under 18.5 is considered underweight, while a healthy weight individual has a BMI in the range 18.5 to 25. A BMI of 25 to 30 indicates that a person is overweight. Individuals with a BMI of 30 or higher are considered obese. A person with a BMI of at least 40 (or at least 35 with a significant comorbid condition) is classified as morbidly or severely obese. Waist circumference or waist-to-hip ratio and percent body fat are also sometimes used to classify the degree of overweight and obesity. Women with a waist circumference of over 35 inches are considered at risk for complications of obesity. A body fat range of 25 to 30 percent in women is considered a normal range; however, women who have over 30% body fat would be classified as obese. Alternatively, an individual may be considered severely obese when he or she is 100 pounds or more over ideal body weight, as specified by the Metropolitan Life Insurance Company standard height and weight tables for men and women. The tables were originally developed to indicate a range of desirable weights associated with the lowest mortality rates.

PREVALENCE OF OBESITY IN U.S. WOMEN

Overall, approximately 62 percent of adult women (aged 20–74) in the United States are overweight, and over 30 percent of all women in the United States are considered obese. The prevalence of obesity in women increased during the late 1980s and 1990s; however, more recent data (1999–2004) indicate that the increase in rates in women may be leveling off. Additional data will need to be collected, however, to confirm whether these rates have truly stabilized. Women overall are more likely to obese than men, 33 percent versus 28 percent, respectively; however, men are more likely than women to be classified as overweight.

The prevalence of obesity in women can also be categorized according to factors such as socioeconomic status, race/ethnicity, age, and marital status. For women overall, individuals with a lower socioeconomic status (income below 130 percent of the poverty threshold) are more likely to be obese than those with a higher socioeconomic status. Minority women in the United States are more likely to be overweight or obese that white women. The prevalence of overweight and obesity combined (BMI of at least 25) is almost 82 percent for non-Hispanic black women, over 75 percent for Hispanic women, and approximately 58

percent for non-Hispanic White women. When looking at obesity (BMI at least 30) by itself, 38 percent of non-Hispanic Black women, 27 percent of Hispanic women, and 21 percent of non-Hispanic White women are obese. Furthermore, approximately 29 percent of American Indian or Alaska Native women are classified as obese. In terms of age, the prevalence of obesity tends to increase as a person gets older and then levels off around age 60. For women aged 20–64, the prevalence of obesity is approximately 64 percent versus 47 percent for women aged 65 or older. Women who have never been married are the least likely to be overweight or obese.

RISKS ASSOCIATED WITH OBESITY

Obesity is associated with a multitude of health risks and increased mortality in women. Although women are at risk for some of the same obesity-related conditions as men, such as cardiovascular disease, diabetes, and esophagus, colon, liver, pancreas, and kidney cancers, there are also many medical conditions linked to obesity that affect only women or impact them to a greater extent. For example, women who gain anywhere from 20–45 pounds or more from their late teens into midlife are thought to be at twice the risk for breast cancer than women who maintain a stable weight. For postmenopausal women, obesity also appears to be linked with higher rates of breast cancer. The risk of endometrial cancer is estimated to be three to four times higher for obese women.

Women with obesity tend to report receiving less financial support for higher education and in turn, attending fewer years of college.

Obesity is also highly correlated with obstetrical and gynecological disorders such as menstrual abnormalities, infertility, and complications of pregnancy and birth defects. Obese women are three times more likely than women of healthy weight to experience abnormal menstrual periods or to have none at all. In addition, polycystic ovarian syndrome (PCOS) is also associated with obesity, given that about 50 percent of women with PCOS are overweight or obese. PCOS is an endocrine disorder, characterized by enlarged ovaries, which disrupts hormone cycles. It is hypothesized that PCOS may be related to the visceral or abdominal fat rather than gluteal fat distribution, although this is not conclusive. Studies have demonstrated variability as to where fat accumulates (regional fat distribution) and its associated risks between males and females. Women tend to accumulate larger amounts of gluteal fat, resulting in a larger hip circumference (creating an "apple shape"), while men tend to store fat in the abdomen, resulting in a larger waist circumference ("pear shape").

Obese women who are pregnant are at higher risk for preeclampsia (pregnancy-induced hypertension that may result in death), gestational diabetes, prolonged pregnancies, and cesarean delivery. Obese women are also more likely to experience wound and urinary tract infections and endometritis (inflammation of the inner lining of the uterus) after pregnancy. Maternal obesity is also thought to be associated with birth defects, particularly neural tube defects (NTDs). NTD is a condition where the neural tube in the developing embryo fails to close properly, most commonly resulting in anencephaly (absence of part of the brain, skull, and scalp) or spina bifida (an incompletely formed spinal cord). In addition, obesity can reduce fertility levels in women, although this may be reversible with weight

loss. Research has demonstrated that infertile obese women are often able to become pregnant after weight loss and carry their pregnancies to full term with no negative outcomes to mother or child.

Obese women are also at greater risk than obese men and healthy-weight women for other obesity-related conditions such as arthritis, gall bladder disease, and urinary stress incontinence (involuntary loss of urine). Obese women are more likely than obese men and healthy-weight women to develop osteoarthritis (also referred to as degenerative joint disease). Osteoarthritis is a painful condition affecting the ability to walk or even stand, resulting from the erosion of cartilage that cushions the joints. It is exacerbated by increased weight, and often muscles around the joints will begin to atrophy as a person becomes less physically active because of the pain. Obese women are at twice the risk of healthy-weight women for developing gall bladder disease, and are more likely to experience urinary stress incontinence.

Research also demonstrates a correlation between body weight and deaths from all causes in women aged 30–55. In addition, for women with a BMI of at least 30, the risk of obesity-related mortality is increased by 50 percent. Mortality rates from cancer may be over 60 percent higher for obese women than for women who maintain a healthy weight.

Finally, individuals with obesity also face discrimination and stigmatization. Obese women are more likely than obese men to face stigmatization and discrimination related to weight. Women with obesity tend to report receiving less financial support for higher education and in turn, attending fewer years of college than women at a healthy weight. In addition, research has demonstrated that for obese women who undergo bariatric surgery as a weight-loss treatment, their likelihood of becoming employed or being selected for a better job increases dramatically.

PROGRAMS AND INITIATIVES TO REDUCE AND PREVENT OBESITY IN WOMEN

Several national initiatives and public health campaigns have been implemented to address obesity, some of them focused specifically on women as a group, or segments of the female population, such as African-American women. The National Institute of Diabetes and Digestive and Kidney Disorders (NIDDK) is one of 27 institutes and centers within the National Institutes of Health (NIH) focused on reducing the burden of disease and improving public health through prevention and treatment of obesity and associated conditions. The NIDDK has developed national initiatives as a part of the Weight-control Information Network (WIN), such as Sisters Together: Move More, Eat Better, which encourages African-American women to maintain a healthy weight via diet and exercise.

Healthy People 2010, issued by the U.S. Department of Health and Human Services, is a set of 10-year evidence-based national health objectives focused on increasing quality and years of happy life and eliminating health disparities. The initiative is supported by partnerships with other federal agencies, businesses, communities, tribal organizations, and state and local governments. The Healthy People 2010 plan identifies 10 leading health indicators, including the prevalence of overweight and obesity and physical activity levels, which will be used to measure the nation's health over time.

Specific goals in Healthy People 2010 include increasing the proportion of adults at a healthy weight and reducing the proportion of children and adolescents who are overweight or obese. In terms of women, the goals include reducing the prevalence of obesity to a target of 15 percent over the 10-year program period. Healthy People 2010 objectives related to nutrition and weight will also be used to measure progress in implementing recommendations from the Dietary Guidelines for Americans, which contain specific recommendations for nutrition and caloric intake for women.

SEE ALSO: Eating Disorders and Gender; Ethnic Disparities among Obesity in Women; Healthy People 2010; NIDDK; Polycystic Ovary Disease; Sisters Together; Women and Dieting.

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Prevention

PREVENTION OF OBESITY and overweight has become a major concern of public health officials and policy makers throughout the United States as statistics about the health and financial cost of the obesity epidemic have emerged. State and local panels have convened across the country to devise strategies and develop initiatives to reduce the unhealthy eating patterns and sedentary lifestyles that are widely believed to be the cause of the unprecedented weight gain of the past three decades among American children and adults. In addition to those health initiatives aimed at individual behavior change, policy makers are intervening in both the public and private sectors to address factors in the community, in schools, and in the food industry that may contribute to the rise in obesity. Legislation has been passed and laws have been put in place that will make it easier for the public to engage in healthful behaviors, such as limiting the public's exposure to harmful trans fats, and prohibiting the dissemination of free baby formula in public hospitals to increase the chances that mothers breast-feed their infants.

While there is general agreement that the public has to eat healthier foods and get more exercise, there is some disagreement in the health community over how to best accomplish this. Important new research is helping professionals understand the complexities of weight gain and weight loss, and the emotional consequences of societal pressure to change such a fundamental aspects of human beings: their bodies. New insights and understanding are being used to evaluate prevention initiatives so that the intense pressure that is heard in the media and elsewhere about the importance of being thin does not inadvertently result in unwanted consequences such as increased rates of depression, isolation, low self-esteem, and suicidal thoughts and ideation. The focus is shifting from an emphasis on weight loss to an emphasis on increasing healthy behaviors in an attempt to ensure that adoption of healthy lifestyles is supported. It is generally agreed that increasing blame and bias toward obese people will not help improve health outcomes or the quality of life of the targeted population.

TRADITIONAL PUBLIC HEALTH APPROACH

The traditional public health approach to overweight and obesity aims at changing individual health behaviors, such as eating more nutritious, low-calorie foods, instead of sugary, high-fat, and fast foods, and in choosing active behaviors instead of sedentary ones. These initiatives might consist of public health announcements in the form of television, print, and radio campaigns, as well as asking pediatricians and schools to focus on the problem with both children and parents.

There has been some concern in the health community that the zeal with which some sectors are tackling the problem may cause new problems. Requiring school districts to send notes home to parents indicating their child's body mass index (BMI) is highly controversial because of the emotional distress it can cause. These letters, which are becoming more frequent in locales around the country and have become known as "weight report cards" or "fat report cards," can further stigmatize children who may already be teased and excluded by peers because of their body size. Furthermore, they have the effect of making parents feel shamed and stigmatized. School districts argue that this is a legitimate way of improving health outcomes because it calls attention to the problem and it pressures parents to act.

Others have pointed to research that demonstrates that this approach might inadvertently backfire and, for example, cause obese children to overeat to self-soothe, after the embarrassment of having failed their weight report cards. Some school districts seem ill-informed about the complexities of weight gain and weight loss, and appear to be promoting the notion that the child and the family are to blame for the overweight or obese condition. A large segment of our society has been shown to hold these beliefs; hence, the expectation that warnings and even stigma might make the offending parents and children feel

uncomfortable enough to act to remove the offensive condition (obesity) and the subsequent stigma. Health professionals fear that societal pressure may not actually help in reducing obesity, but rather, have the unintended consequence of weight gain instead.

Some studies have shown that children whose parents try to restrict their intake of unhealthy snack foods the most, will eat more of this type of food when it becomes available or if the restricting adult is not present. This framework sees these children as "rebounding" from the strict control over their food intake by overcompensating when that control is not in effect. Other studies show that children tend to do the opposite of what their parents want when it comes to healthy choices such as eating certain foods.

When young children whose food intake and food choices are restricted are left alone in a room with these restricted snack foods, these children will consume more of the high-fat and high-calorie food than will children who have not been restricted. Adults who remember more food rules in their families that restricted the intake of many foods may tend to be more frequent binge eaters than those who were not exposed to these food rules. Studies have also shown that some people who develop anorexia were initially frequent dieters, demonstrating a possible relationship between frequent dieting and eating disorders. Balancing the messages about healthful eating with the possible damaging consequences of restricting food intake as well as the potential harmful consequences of stigma and blame is a complicated, tricky endeavor, but one that is needed when implementing programs aimed at stemming the rise of obesity.

Hypothetically, messages about healthier eating could lead to poor health outcomes and less healthy eating. This information needs to be conveyed to parents and considered as parents try to improve the nutrition of their children. Stigma and blame that is intended to induce healthier behaviors might also lead to poor health outcomes in other areas, such as depression, or attempts to self-soothe with harmful substances such as tobacco, alcohol, and/or drugs. More careful research is needed in this area, and policy makers should be made aware of the sometimes delicate balance interventions require, given the complexity of human behaviors. Perhaps, too, this is an area where heightened sensitivity and compassion toward the obese is required.

THE ROLE OF STIGMA AND BIAS IN PREVENTION

Stigma against obesity and overweight is quite prevalent in American society, and it has been linked to the public's beliefs about the causes of obesity. People who hold strong beliefs that obesity is the "fault" of the overweight, and who also blame the obese for being overweight, have been found to more frequently and intensely stigmatize the obese.

When subjects, both children and adults, are shown pictures of obese people and average-weight people, they tend to assign negative characteristics to the obese. The obesity is viewed as a function of the person's laziness, lack of willpower, and overall poor character. Obese children and adults have been characterized by children as mean, untrustworthy, and stupid. Adults have been shown to stigmatize and stereotype obese people as well. One study showed that teachers believed obese students to be less tidy, more emotionally unstable, less likely to succeed, and more likely to come from troubled families.

A majority of teachers surveyed believed that obesity stemmed from lack of love in the home, and that overeating was a compensation for this lack. When school principals were questioned about the causes of obesity, they overwhelmingly blamed lack of control and psychological problems. Research has shown that obese applicants to college are more likely to be rejected than normal-weight applicants with equivalent scores. Stigma and blame have been shown to be present even among health practitioners who work with obese patients. Subjects show more compassion toward the obese when told that their obesity is due to an illness or medical condition.

It is believed that stigma is highly correlated to blame of the obese person for his or her condition. This becomes problematic because stigma and ostracizing can paradoxically lead to an increase in unhealthy eating behaviors and an increase in weight gain. Research suggests that obese girls may be at increased risk for emotional distress, low self-esteem, depression, and suicidal thoughts and attempts. The more an obese girl feels that the obesity is her own fault, the greater her chances of suffering from low self-esteem. An informal review of the literature suggests that more and more research about the negative consequences of stigmatizing obesity is being done, as well as research into the harmful impact of a national obsession with being thin.

The media's focus on the "epidemic" and "crisis" of the state of obesity and overweight today has been cited as contributing to dissatisfaction with body size and appearance, regardless of the reality of one's actual weight; distorted body image among a significant number of American girls and women (and increasingly men as well); and the adoption of unhealthy fad diets and nonnutritious attempts at weight loss. Experts have pointed out that a fad diet might result in eventual health problems; therefore, even if a person achieves their goal of weight loss, he or she may be endangering his or her health rather than improving it. It is felt that the health community, and others who are in the business of promoting wellness such as schools and educators, need to factor in all these developments when exploring prevention strategies so that the antioverweight message does not result in negative health outcomes. If it can be shown that healthy markers can be reached and maintained, even at a larger size, then health professionals might convey this to the public as well.

SOCIAL-ECOLOGICAL MODEL

The Centers for Disease Control and Prevention (CDC) utilize a social-ecological model when it explores the problem of obesity, and when it designs and implements programs to prevent and reverse the ever-increasing obesity problem. It recommends that other agencies use this approach as well, and it requires that it be done when a state receives certain types of funding for obesity-prevention programs.

One important feature of this model is that it examines each and every population and community anew, because each one has different needs, problems, and challenges. The model looks at multiple levels of influence on eating and exercise behaviors, such as the individual, interpersonal groups, organizations, communities, and society, and addresses ways of intervening in all of these levels in order to improve health outcomes and the health behaviors of individuals. Using this model, it might be determined that poor people, who are disproportionately affected by the problem of obesity, have limited access to a variety of affordable, fresh produce.

Policy makers might look at ways of getting greenmarkets or farmers markets closer to these communities, on days and during hours that would best meet the needs of the community. This model contains the notion that unhealthy behaviors are not just a function of individual choice, but of social factors and influences as well. It encourages an exploration of the many factors that are at work in any given community that might decrease the healthful behaviors of individuals. It might result in legislative change, changes in school policy, improvement in parks and recreation, and in asking or requiring the food industry to alter or eliminate unhealthful trends such as the proliferation of "supersize meals" in fast-food establishments. It might address the fact that in many schools, after-school physical education opportunities are usually limited to competitive sports. Students who are unable to participate competitively due to skill or desire may be unintentionally excluded from much-needed physical activities.

According to this model, a change or intervention in any one of these levels will effect change in other levels of the paradigm. This model might effect individual behavior change by intervening on a societal or community level, such as improving access to parks or biking trails. Because human behavior is such a complex phenomenon and it is often resistant to change, this model often seeks and effects change in other ways. Improved and increased park facilities will most likely have an impact on the activity level of local children. Intervening in this way might even be less costly in the long run and more effective than programs aimed at changing one human being at a time.

INNOVATIVE PREVENTION PROGRAMS

An innovative program in a Kentucky community used scorecards for children to record their physical activity over the summer. Businesses, public organizations, and churches were asked to provide free or reduced-price physical activities for children. Summer dances were organized, reduced-price rollerskating was available, and incentives were offered so that children could swim, play volleyball, and in general, partake in age-appropriate individual and group activities to increase physical activity. Sixty-five percent of the parents reported that their children were more active as a result of this program, and as a result of its success, other communities in Kentucky are adopting the program.

One elementary school in Colorado set up a fruit and vegetable market during lunch where children could work as well as purchase fresh produce. It was reported that 20 percent of the students became regular customers of the market, and between 150 and 200 pieces of produce were being sold each week.

Workplace changes, such as providing private rooms for nursing mothers, providing workplace gyms, or reimbursement for yoga classes, are ways for employers to intervene and help employees become more physically active or choose options such as nursing over formula feeding, which has been shown to help prevent obesity in children. Recently in New York City, policy was initiated whereby public hospitals are prohibited from dispensing free formula to new mothers upon leaving the hospital. This measure is aimed at increasing the number of women who choose breastfeeding after the birth of a child. Often, when new mothers accept free formula, they embark on a course whereby their milk production stops and they are forced to dilute formula when they cannot afford to buy adequate supplies of it. This further puts the infants' health at risk, and may contribute to childhood obesity.

PREVENTION AND THE LARGER SOCIETY

Some health policy analysts have explored the need to reframe the issue of obesity in America. They invoke the analogy of the health problem of tobacco use or childhood injuries wherein manufacturers and producers become the major focus of prevention campaigns. Over the years, we have seen cigarette advertisements removed from television and manufacturers of children's toys held responsible for parts that might harm children.

These campaigns do not exclude personal responsibility, such as the importance of parental vigilance when it comes to child safety; they also do not exclude societal responsibility or industry responsibility. Some cases in point are the fact that some of the very schools that are sending home BMI reports to parents to alert them that their children are overweight are serving high-fat, high-caloric foods in their cafeterias. Or they may enter into contracts with soft drink manufacturers to solely offer their products in vending machines, and as a result are contributing to the problem of obesity. Targeting individuals and families without looking at the larger influences is like suggesting that the only way to prevent accidental drowning among children is to have parents keep an eye on their kids at all times, instead of investigating ways of making swimming pools more resistant to unsupervised use.

THE BUILT SOCIETY

Many American communities are designed so that there are few or no safe places to walk. In suburbs, there are often no sidewalks, and attempts to build walking into daily routines are thwarted by the fact that families and individuals are faced with the choice of walking on unsafe, high-trafficked roads, or choosing the car. Americans often choose to move away from cities, and developers often build on former farmland, wooded areas, or undeveloped land far from the center of commerce. When efforts have been made to build communities that incorporate some of the features of cities, such as public places to sit, gather, and walk, with close proximity to neighbors, schools, and businesses, buyers and renters respond overwhelmingly favorably.

These "New Urbanist" communities often sell out quickly, and it is dissatisfied suburbanites who often flock to them. People seem to enjoy pushing their children in strollers on the way to retrieve siblings from school. These communities enable errands to be done using bicycles, where adults and even children can safely ride on bike paths that lead to a town square that houses the post office, shops, and restaurants. Teenagers are not forced to look to the mall as a gathering place, but rather have parks and paths where they can walk, rollerblade, or ride their bikes. Elderly residents can walk safely, and gather and sit on benches. They might walk to yoga class at the community center. They can walk to the market to buy provisions for dinner. They can walk to a local restaurant and meet friends for a meal.

In traditional suburbia, these same residents are often at the mercy of someone who offers to drive them to the supermarket or the pharmacy. Furthermore, the elderly are simply less isolated when they can sit on a bench and smile at passersby, or watch out when young children play while a mother may need to tend to a crying baby.

These types of communities decrease isolation, increase physical activity levels, and help people pool resources, such as shared supervision of children. All of these factors appear to improve health outcomes. Often, these communities build in mixed-use housing, such as apartment rentals above shops, or multifamily dwellings. This form of housing provides opportunities for the retired to live in or near the center of activity, and for the low income to avoid

endlessly long commutes that encourage them to turn to fast food when they arrive home at the end of the day. The structure and nature of these communities encourages residents to spend less time in their homes and more time in public gathering places and places of recreation. They can contribute to big changes in individual behaviors that lead to improved health, decreases in obesity, and the adoption of new healthy habits.

THE FOOD INDUSTRY

Prevention efforts are increasingly aimed at the food industry, with the expectation that there is a need for accountability when it comes to consequences in human health. In 2002, there was a lawsuit against McDonald's in New York City on behalf of a group of obese children who claimed that their obesity was the fault of the fast-food restaurant for failing to fully disclose the unhealthy contents of their food. This legal action was derided in the press and among the public, who poked fun at it by dubbing it "McLawsuit." The attorney was accused of being unscrupulous, the lawsuit frivolous, and the children (teenagers) just plain clueless for not knowing that a Big Mac would make them fat. The case was thrown out of court to the sounds of a cheering media and public.

It is noteworthy that attitudes seemed to have changed in the ensuing years, with the public more prepared to expect food industry accountability, and the industry seemingly scrambling to make changes. Shortly after the lawsuit, McDonald's announced a Happy Meal it planned to introduce that would offer the option of fresh fruit instead of french fries. Frito-Lay announced its plan to remove all trans-fats from its products.

These actions seemed to usher in a new era of asking the food industry to be more responsible for the health of consumers. More recently, New York City banned the use of trans fat in all restaurants, and while there was a minor outcry about how silly it is to ask McDonald's to make french fries taste good without using trans fat, the public and private sectors now seem ready to expect accountability from the people who sell them the food they eat.

SEE ALSO: Eating Out in the United States; Governmental Policy and Obesity; Obesity and the Media; Overweight Children in the Media; Recreational Facilities in the U.S.; School Based Interventions to Prevent Obesity; State and Local Initiatives to Prevent Obesity.

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Prostate Cancer

PROSTATE CANCER IS the most common form and the second leading cause of cancer death in men, with an age-adjusted incidence of 69 per 100,000 in the United States. In addition to symptomatic tumors, latent, microscopic foci of prostate cancer are frequent incidental findings that also increase with age and are found in over 70 percent of men in their seventh decade of life. Wide racial and ethnic variations in incidence are known—Asian men have very low incidence of prostate cancer, while African-American men have the highest rates of the disease. Interestingly, while incidence of clinical disease varies widely, incidence of latent disease is almost identical across populations, suggesting that environmental factors, including diet, may increase the probability that latent cancer foci will progress to high-grade tumors.

The normal prostate encircles the neck of the bladder and urethra and consists of variously sized glandular spaces lined by two layers of epithelium and separated from the fibromuscular stroma by a distinct basement membrane. Both epithelial and stromal cells have receptors for testosterone and its metabolite, dihydrotestosterone (DHT), produced largely by

the stromal cells, and respond to these growth factors with cell proliferation. While the initial causes of prostate cancer are not known, it is likely that the initial neoplastic change involves cumulative damage to deoxyribonucleic acid (DNA). Evidence that obesity plays a major role in the pathogenesis of prostate cancer risk is inconsistent. Possible reasons for inability to detect an association include the difficulty in screening obese patients and the confounding influence of diabetes mellitus, known to increase risk for prostate cancer. However, many lines of evidence suggest that abdominal obesity, even in lean patients, is associated with increased risk for cancer progression, and that obesity facilitates conversion to androgen-independent growth patterns. Such tumors tend be high grade, aggressive and resistant to treatment, and contribute to high mortality from prostate cancer.

Abdominal adipose tissue actively secretes endocrine hormones or adipokines. Leptin regulates body weight by central actions on food intake and energy expenditure and also known to be important in events such as reproduction, hematopoiesis, angiogenesis, and insulin secretion through receptor binding and downstream signaling pathways, known to be involved in cell proliferation and survival in androgen-independent prostate cancer cells. Other adipokines, including interleukin-6 (IL-6) and insulin-like growth factor (IGF), have also been implicated in prostate cancer risk.

Obesity in both men and women is associated with increased conversion of adrenal androgens to estrogens via an aromatase enzyme located in adipose tissue. Increased local or paracrine production of estrogens by breast adipose tissue has been associated with increased risk for breast cancer. Thus, while circulating adipokines may facilitate prostate cancer progression, it is also likely that transformed prostate cells may be exposed to paracrine secretions from adjacent retropubic adipose tissue. This possibility would explain the clinical observation that obesity contributes more to prostate cancer progression than to its initiation.

Further, consumption of a high-fat, low-fiber, nutrient-poor "Western" diet increases risk for both obesity and prostate cancer. Epidemiological and laboratory studies have shown that diets containing large amounts of green vegetables, fruits, grains, and legumes reduce risk for many cancers, including prostate cancer. Specific components in these foods, such as lycopene, the red isoprenoid pigment in tomatoes, and inositol hexaphosphate (phytate) in grains are under investigation; however, it is likely that multiple bioactive components in whole foods interact to reduce prostate cancer risk. Research is under way to determine whether diets high in bioactive components modify adipocyte secretions. For example, IGF known to stimulate cell proliferation and inhibit apoptosis is lowered, while its binding protein (IGFBP), shown to stimulate apoptotic cell death is raised. It is also possible that adipokines and their binding proteins can be used as biomarkers to assess risk for prostate cancer initiation and/or progression.

SEE ALSO: National Cancer Institute; Obesity and Cancer.

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Protein Kinase

A KINASE IS an enzyme that transfers phosphate groups from high-energy donor molecules to specific target molecules in a process known as phosphorylation. More specifically, protein kinases are a family of enzymes that phosphorylate other proteins. These protein kinases catalyze the transfer of a phosphoryl group from ATP to the OH group of a

serine, threonine, and/or tyrosine residue. The purpose of phosphorylation is to alter the function of the target molecule in one of several different ways. These changes include activating or upregulating activity of the molecule, inhibition of molecular activity, or binding to other molecules to initiate or inhibit a specific signaling system.

The human genome contains over 500 genes meant to encode protein kinase enzymes, which is nearly 2 percent of all human genes. These genes are essential for production of the protein kinases, which are the primary agonists for signal transduction in human cells. Signal transduction is the process of converting one type of stimulus to another, generally through biochemical reactions involving "second messenger" systems in which a cascade occurs with few proteins being involved at the beginning, and numerous proteins becoming involved as the reaction occurs. Protein kinases also control many other cellular processes, including metabolism, transcription, cell movement, apoptosis, and differentiation. Protein phosphorylation also plays a critical role in intercellular communication during development, homeostasis, and in the functioning of the nervous and immune systems.

Two primary sources of protein kinase activity of interest to those interested in the obesity/diabetes epidemics are insulin-like growth factor-1 receptor (IGF-1R) and AMP-activated kinase (AMPK). IGF-1R is a transmembrane protein that displays intrinsic tyrosine kinase activity. Insulin-like growth factor-1 (IGF-1) is a polypeptide stimulated by growth hormone that acts similarly to insulin and affects cells throughout the body. IGF-1R binds to IGF-1 at a significantly higher rate than does the insulin receptor.

As for AMPK, it is a fuel-sensing enzyme that responds to decreases in cellular energy state by activating processes that generate ATP (such as fatty acid oxidation) and inhibiting nonessential functions that consume ATP. A decrease or improper functioning of AMPK has been implicated in the metabolic syndrome. It is expressed in a number of tissues, including the liver, brain, and skeletal muscle. The effects of AMPK activation include stimulation of hepatic fatty acid oxidation and ketogenesis, inhibition of cholesterol synthesis, lipogenesis, and triglyceride synthesis, stimulation of skeletal muscle fatty acid oxidation and muscle glucose uptake, and modulation of insulin secretion by pancreatic beta-cells. Protein kinase activity, such as those of IGF-1R and AMPK, warrant further research and targeting from drug therapies in the fight against obesity and Type 2 diabetes.

SEE ALSO: G-Protein Coupled Receptor; Growth Hormone; Insulin-Like Growth Factors.

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Psychiatric Medicine and Obesity

THE RELATIONSHIP OF psychiatric medicine and obesity has become an important topic that goes beyond the implications for physical health in psychiatric patients. Understanding the connection between psychiatric medicine and its metabolic effects may eventually provide important clues about the nature of mental illness itself.

The risk for obesity in patients with psychosis has increased significantly with the advent of the "atypical," or second-generation, antipsychotic drugs. Examples of these atypical antipsychotics include clozapine, olanzapine, quetiapine, and risperidone. Weight gain is most notable in women, and appears to be caused primarily by hyperphagia (increased consumption of food), but changes in metabolic rate are also likely. Significant metabolic changes occur at a relatively young age in this patient population.

It has become apparent that although the most acute side effects of the atypical antipsychotics (agranolocytosis, a blood disorder; myopericarditis, inflammation of heart muscle; and QT-elongation, an abnormal electrocardiogram [EKG] feature) are rare and limited predominantly to clozapine, the chronic

metabolic side effects and associated weight gain are not. There are serious consequences of the chronic metabolic changes. The mortality rates in individuals with schizophrenia are reported to be approximately double that of the general population and cardiovascular disease is thought to be responsible for 50 percent of this excess mortality. The chronic metabolic changes can be severe enough to lead to outright ketoacidosis (when energy usage shifts from a balanced utilization of glucose and fat to predominantly fat) and death attributable to the ketoacidotic state.

Understanding the basis for these side effects is important as the promise of these second-generation antipsychotics to improve psychiatric symptoms has been largely realized, particularly for those with symptoms refractory to treatment with the first-generation neuroleptics. Although the basis for the increased antipsychotic efficacy of the second-generation antipsychotics was originally presumed to occur via the spectrum of effects on the extended family of dopamine receptors, particularly the D4 receptor, that theory has not been validated. Effects on the histamine and glutamaterigic systems have been proposed as relating to their mechanism of action. In addition, increasing attention has been given to the observation that the cardiac risk factors of the atypical neuroleptics cannot be easily separated from the antipsychotic benefit.

Much as the "Seeman plot" revealed a striking correlation between efficacy of the first-generation antipsychotics and D2-receptor binding affinity, the efficacy of the second-generation antipsychotics (in addition to D2-receptor binding affinity) show a remarkable correlation with weight gain, particularly in women. This perception has been borne in other, smaller studies carried out to investigate this correlation. The degree of risk for weight gain generally occurs in the following order: clozapine, olanzapine, quetiapine, risperidone. In addition, a prospective study of the effect of body mass index (BMI) on the risk of hospitalization for psychosis, depression, or suicide revealed that a greater BMI was significantly protective for those outcomes.

What component of the metabolic change is relevant to antipsychotic effects, or is a downstream marker of these effects, is not clear. When patients were matched for weight in risperidone-treated and in clozapine-plus-risperidone–treated groups, the improved psychiatric ratings in the clozapine-treated individuals correlated with the blood triglyceride level. Many other components of the metabolic change associated with weight have not been measured, however, including the key indicator of metabolic ketosis, B-hydroxybutyrate. B-hydroxybutyrate is particularly of interest because it is an agonist of the HM74-A niacin receptor that shows evidence of dysfunction in unmedicated schizophrenia.

It is through stimulation of the HM74-A receptor that lipolysis is suppressed. Thus, stimulation of the HM74-A receptor would serve to normalize lipid and cholesterol profiles that are perturbed by the medication, but may also correct a basic underlying defect common in this patient population. Of the metabolic markers elevated in the "metabolic syndrome," B-hydroxybutyrate is arguably the most benign. Thus there is reason to hope that if this is one mediator of the additional antipsychotic benefit realized with the atypical neuroleptics, then more targeted neuroleptics can be designed. There are quite a few studies illustrating the important role that B-hydroxybutyrate plays in cardiac function during periods of caloric restriction. However, one negative side effect has been reported in animal studies: Development of insulin resistance in heart muscle tissue is attributed to beta-hydroxybutyrate.

Clearly, it is going to be a top research priority to separate the weight gain from antipsychotic efficacy for the long-term health of this patient population.

MEANS OF REDUCING WEIGHT GAIN

The toxicological burden placed on a patient taking antipsychotic drugs is not minor. Adding more pharmaceuticals to this mix must be done with great care and other options should be pursued with vigor. Interventions that dovetail with antipsychotic efficacy should be the first priority.

Increased exercise in psychiatric patients may not only lead to better physical health, but to better mental health as well. Based on a small pilot study, Faulkner and Sparkes (1999) have proposed that increased exercise has physical benefits for patients with schizophrenia and also improves their self-reported perception of auditory hallucinations, self-esteem, and improved sleep patterns and general behavior. In a somewhat larger study by Beebe et al. (2005), exercise in schizophrenia patients lead to lower body mass indexes and fewer psychiatric symptoms, as measured

by the PANNS testing procedure. A systematic review of studies that evaluated psychiatric outcome supports the concept that psychiatric benefit does occur with exercise. However, due to a lack of goal-directed behavior, which is a core feature of schizophrenia, the adherence to a program that is not part of a structured setting can pose a problem for this group of patients. Archie, et al., studied the adherence to a voluntary exercise program among a group of olanzapine-treated patients given a free membership to the YMCA. The result was that at the six-month time point, 90 percent of the subject participants dropped out. As expected, the patients who adhered to the program experienced the most weight loss. A comparison of the effectiveness of pharmaceutical interventions to exercise and other lifestyle changes (diet) in this population supports the premise that the latter approach is currently more successful.

In obesity-related research, the potential for human-derived pharmaceuticals is enormous. Of the many mediators of obesity, however, only a select few have yet been shown to also affect psychiatric symptoms. Leading the field in this regard are the melanotropin family of peptides. Numerous studies have demonstrated the effect of endogenous melanotropins to regulate feeding and metabolism. Animal studies have shown that for at least one endophenotype (characteristic feature) found in schizophrenia (processing of sensory input), the effect of administering a specific melanotropin, alpha-MSH or its analogues, would be expected to be beneficial for psychological state. Intranasal administration of a potent alpha-MSH-like agonist has been shown to be effective in normal-weight individuals for inducing weight loss, although the effectiveness in patients who were already obese was not significant. Thus, it is possible that these endogenous peptides would hold potential as prophylactic therapy, as an adjunct to initiation of atypical neuroleptic therapy. The potential for a negative interaction with the antipsychotic would need to be addressed first in an animal model.

MEANS OF REDUCING SPECIFIC CARDIOVASCULAR RISK FACTORS

The reduction in the cardiovascular risk with the statin class of drugs has been somewhat disappointing. Although the importance of the statins in non-mentally ill patients has been well established, in the schizophrenia population whose level of physical activity is already quite low, the statins may only further decrease the ability or desire of patients to exert themselves physically. The only component of neuroleptic-induced metabolic syndrome improved by rosuvastatin therapy was the serum triglyceride level. Nutraceuticals for cardiac health may hold more promise in this group, and some may be of benefit to mental state as well.

Niacin remains a very effective treatment for increasing high-density lipoprotein (HDL), although there is report of a possible negative interaction with clozapine, relating to a skin rash. Niacin can cause an elevation in blood glucose in the short term, which would potentially exacerbate blood glucose changes due to the atypical antipsychotic drugs. Drug-to-drug interactions that affect the liver are a concern with niacin. In particular, patients who might be on valproate may have an increased risk of liver injury.

Historically, niacin has been used as a treatment (with mixed success) for psychosis in otherwise unmedicated schizophrenia patients and is the basis for reversing the depression and/or psychosis with adequate nutrition in the niacin-deficiency disease, pellagra. There are also case reports of sudden cessation of niacin therapy bringing on episodes of major depression, illustrating both the psychological benefits of niacin and the need to monitor its withdrawal.

In addition, the expression of the niacin receptor in adipose tissue of individuals with schizophrenia has not been studied, and it is not known if the niacin-receptor deficit, which has been shown for the brain and is evident in the epidermis, is also present in adipose tissue. If so, overcoming the effects of decreased expression of the niacin receptor in schizophrenia may not be possible with niacin alone and other supplements should continue to be explored to mitigate cardiovascular risk. Acipimox, a niacin analogue, has been widely available in countries other than the United States for several years. Studies have shown that it has a mixed effect on hormones mediating appetite, increasing leptin and but decreasing adiponectin.

Schizophrenics developing the metabolic syndrome show elevated homocysteine. Folate intake in control subjects substantially decreases the markers for cardiac risk, including homocysteine levels, yet the risk for cardiac events is not decreased by increased

folate. Folic acid with vitamin B6 plus B12 therapy has paradoxically resulted in an increased risk. These reports would indicate that homocysteine serves as a marker for cardiac risk, but does not by itself mediate that risk. Nevertheless, psychological symptoms are reported to be improved when homocysteine is reduced in schizophrenic patients.

Omega-3 fatty acids have also been reported to have positive mental effects and have clear benefits for cardiovascular health. Patients taking clozapine have achieved a decrease in triglycerides with omega-3 fatty acids, but they also experienced an increase in low-density lipoprotein (LDL) and total cholesterol.

Lipoic acid is another supplement that holds some promise for treating the metabolic syndrome, preventing lipotoxicity-induced cardiomyopathy in mice, reversing streptozotocin-induced endothelial changes, and has been found to be safe in a two-year course of treatment in humans. The effects on glucose levels may also be positive.

Coenzyme Q10 has long been proposed as a beneficial adjuvant therapy for cardiomyopathy and congestive heart failure, but its use for that purpose or for metabolic syndrome–induced cardiovascular disease has not been well studied. It is reported to be beneficial for heart muscle in reperfusion models of myocardial ischemia and in postmyocardial infarction, which may be due to its antioxidant capabilities.

ANIMAL MODELS

Obviously, it would be advantageous to study the prevention or correction of obesity and adverse cardiovascular changes in an animal model. To date, the greatest success has been achieved with olanzapine treatment in female rodents, who show a greater weight gain when treated with olanzapine than untreated controls. Studies have shown that olanzapine must be administered at a much higher dose in rats to reach equivalent occupancy of dopamine D2 receptors seen in humans, because the half-life of the drug is so much shorter in these animals than in humans. Even with the high doses, the degree of weight gain and the full spectrum of metabolic changes seen in humans have not yet been captured in the animal experiments. In other words, it has been somewhat difficult to completely replicate the human condition. The regulation of food intake in animals appears to be more tightly controlled than in humans and animals are less likely to ingest the plethora of high-fat foods that are available to the human patient. Modeling the human condition will likely require a diet that is unusual for lower species of animals.

SEE ALSO: Body Mass Index; Depression; Medications that Increase Body Weight.

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Puerto Rican Americans

PUERTO RICAN AMERICANS are people residing in the United States who emigrated from or who have ancestry from Puerto Rico, an island in the Caribbean which is a territory of the United States; therefore, those born in Puerto Rico are American citizens. The largest concentration of Puerto Rican Americans in the United States is in New York City and Florida, although people self-identifying as having Puerto Rican ancestry reside in all states in the United States. According to the U.S. Census Bureau, in 2003, over 3.8 million Puerto Ricans lived in the United States. Puerto Rican Americans are the second largest Hispanic group in the United States, after Mexican Americans.

Information about obesity among Puerto Rican Americans must be gleaned from several sources—research focusing specifically on Puerto Rican Americans or on several Hispanic subgroups, research about Hispanic Americans in general, and research about Puerto Ricans living in Puerto Rico. Application of studies about Hispanics in general to Puerto Ricans in particular must be tempered by the knowledge that

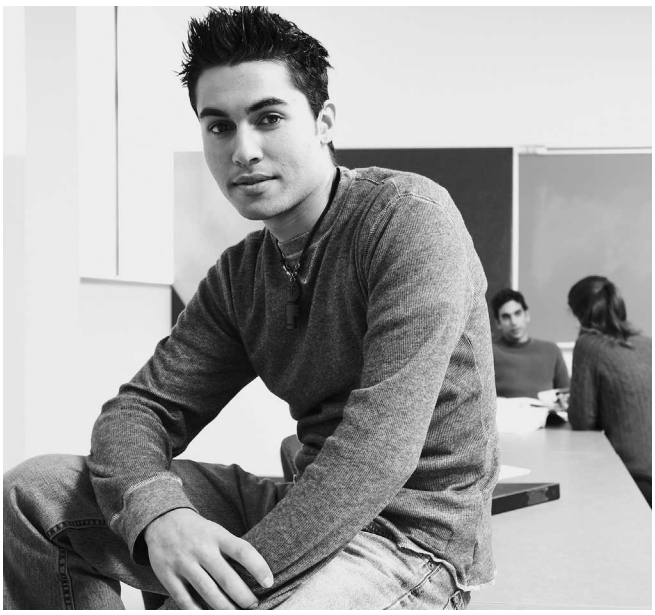
in the 2000 U.S. Census, the predominant Hispanic groups was Mexican American (58.5 percent of the total), with Puerto Rican Americans making up only 9.6 percent of the total).

For the purposes of collecting information on health and other population surveys, the Untied States government defines Hispanics or Latinos as persons of Cuban, Mexican, Puerto Rican, South or Central American, or other Spanish culture or origin, without regard to race. This adds a complication to interpreting information collected about Hispanics as a group, because race has been identified as a significant health risk factor in many studies. Most Puerto Ricans identify themselves as white, with about 8 percent self-identifying as black, and the remainder a combination of Asian, American Indian, or other races.

In the 2000 U.S. Census, Hispanics represented about 37.4 million people, about 13.3 percent of the total U.S. population. The Census Bureau projects that Hispanic Americans will total about 87.5 million by 2040, representing 22.3 percent of the U.S. population. Hispanics share some aspects of a common cultural heritage and social experience but also vary widely on characteristics such as racial background, time and conditions of immigration to the United States, degree of acculturation, rates of education and poverty, and many cultural factors such as food preferences, attitudes toward physical activity, and tolerance of and preference for particular body types.

Rates of obesity (defined as body mass index over 30) among U.S. adults (aged 18 and older), as calculated from self-reported height and weight on the Behavioral Risk Factor Surveillance Survey (BRFSS), have been increasing among all racial/ethnic groups since 1995. Hispanic rates of obesity are intermediate: lower than those of non-Hispanic white Americans, higher than those of non-Hispanic black Americans. In 1995, the BRFSS found that 16.8 percent of Hispanic adults were obese, versus 14.5 percent of non-Hispanic white Americans and 22.7 percent of non-Hispanic black Americans. In 2000, the rates were 23.4 percent for Hispanics, 18.5 percent for non-Hispanic whites, and 29.3 percent for non-Hispanic African Americans. In 2005, the rates were 26.5 percent for Hispanics, 22.6 percent for non-Hispanic whites, and 33.9 percent for non-Hispanic blacks. The 1999–2002 National Health and Nutritional Examination Survey (NHANES), an ongoing nationally representative survey, found similar results for children and teenagers aged 2–19. The NHANES found that 37.3 percent of Hispanics in this age group, versus 34.7 percent of non-Hispanic whites and 43.4 percent of non-Hispanic blacks in this age group had ever been told by a doctor or health professional that they were overweight.

One of the federal health surveys which collects sufficient information on Hispanics to allow estimation of health status for different Hispanic subgroups, and to compare Hispanic with non-Hispanic black and white populations, is the National Health Interview Survey (NHIS). This study is conducted annually by the National Center for Health Statistics of the Centers for Disease Control and Prevention. In 2000, Hajat and colleagues aggregated 4 years of NHIS data to study health indicators for four Hispanic subgroups: Mexican Americans, Cuban Americans, Puerto Rican Americans, and all other Hispanics. Their results underlined the diversity of experiences among the subgroups classified as Hispanic. For instance, most of the Puerto Rican population lived in the Northeast United States, while most Cubans lived in the South, and most Mexicans in the South and West. Ninety-seven percent of Puerto Rican Americans reported being born in the United States, much higher than any other Hispanic subgroup and comparable to white and black Americans. In most cases, Anjit found that the health



Puerto Rican Americans are the second largest Hispanic group in the United States, after Mexican Americans.

indicators for Puerto Rican Americans were generally worse than those for other Hispanic subgroups, while those for Cuban Americans were generally better than the other subgroups.

SEE ALSO: Central America and Caribbean; Hispanic Americans; Immigration and Obesity.

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Qualifications for Gastric Surgery

OBESITY PROFOUNDLY AFFECTS quality of life, overall health, and life expectancy. Healthcare providers have sought safe and effective nonsurgical treatment strategies, but have had little reproducible long-term success. Surgical treatment for obesity, on the other hand, is very effective for the majority of patients; however, it is associated with significant risk. To determine whether a patient is a candidate for surgery requires a thoughtful calculation of the risk-to-benefit ratio of the procedure relative to no intervention. Several organizations have created criteria for patient selection including various medical societies, the National Institutes for Health, Medicare and Medicaid Services, and, perhaps most important, individual insurers. Qualifications for surgery thus are not uniform and differ slightly depending on prospective.

Most guidelines use body mass index (BMI) as the primary determinant for bariatric surgery candidacy. BMI is a person's weight in kilograms divided by the square of their height in meter. BMI is an accurate representation of adiposity in the morbidly obese and allows for comparisons between individuals by correcting for differences in patient height. Surgery is generally offered to patients whose BMI is 40 or greater or patients who have a BMI 35 or greater with serious weight-related medical comorbidities. Serious comorbidities may include the presence of diabetes, hypertension, and sleep apnea. A few insurers do not use BMI as a criterion. Some require a weight that is at least double one's ideal body weight, and others, a weight 100 pounds greater than ideal weight.

BMI is not the only criterion used to determine whether a patient is a candidate for bariatric surgery. Many other factors are equally important. A thorough psychosocial evaluation is often recommended as part of the preoperative workup of a morbidly obese patient. Surgery for obesity is significantly life altering and patients must be able to cope with these drastic changes. As such, important criteria for surgery includes the patient's ability to reasonably understand and assess the risks and benefits of surgery, and the patient's ability to maintain long-term follow-up with the bariatric surgery program. Most guidelines exclude a patient with severe mental retardation or unstable psychiatric disorders.

For example, depression, bipolar disease, and even schizophrenia are not absolute contraindications; however, someone who is suicidal, manic, or actively delusional should be in the care of a behavioral health specialist and stabilized before considering bariatric surgery. Patients with Prader-Willi syndrome would be considered an absolute contraindication by most surgeons. Last, the patient must have a reasonably supportive social network.

Obesity surgery at the extremes of age has been long controversial. Many bariatric centers restrict

their practice to an age range between 18 and 65. Although numerous single institution series document the safety of obesity surgery in adolescent and elderly patients, the data are quite limited. Examination of large national databases suggests a significantly higher mortality rate in Medicare patients who are greater than 65 relative to younger Medicare patients. However, it is unclear if these patients who may be at highest risk from surgical complications are also the patients who may benefit the most. Medicare and Medicaid services currently offer bariatric surgery benefits to all of their members, regardless of age.

Bariatric surgery in teenagers is becoming more common despite the lack of high-quality long-term studies. Teenagers who are obese suffer from poor social acceptance, often perform poorly in school, and have significant health consequences. Atherosclerotic changes to major vessels can be seen young children with poor dietary habits. The incidence of Type 2 diabetes mellitus increases with weight in teens, and obstructive sleep apnea is widely seen in obese children. Only a few studies with small numbers of adolescent patients have been published. Most document excellent weight loss, acceptable complication rates and resolution of medical comorbidities. As these patients are too young to give informed consent for bariatric surgery, parents and surgeons must balance the benefits of surgical weight loss to the inherent morbidity and mortality rates these procedures entail. Patient assent is required. Children and adolescents arguably are more successful with caloric restriction through lifestyle modification and extensive efforts at nonsurgical management are warranted. Bariatric surgery should only be performed in a setting where adolescent-specific resources are available. Some have suggested using different weight criteria for those under 18 years old. For instance, a BMI of greater than 50 with significantly life-altering medical comorbidities or a BMI of greater than 40 with two life-threatening comorbidities was recently suggested by a panel of pediatric specialists. Others use the same criteria as in adults. Furthermore, adolescent patients should have achieved 95 percent of their predicted adult height and a Tanner stage of IV.

The extent of previous diet attempts is another controversial criteria used to determine eligibility for bariatric surgery. Patients are often required to demonstrate prior attempts at weight loss with conservative medical and dietary management before considering bariatric surgery. Some insurers require six-month (or longer) physician-supervised diets with documentation of nutritional education, behavioral modification, and exercise therapy. These requirements are controversial because multiple studies have demonstrated that the long-term success rate of medical weight loss is dismally low. Many patients will transiently respond to medical therapy for 12 to 24 months, but the vast majority return to their pre-diet weight, and some even weigh more after a dietary program. Proving failure of medical weight-loss therapy may simply delay an inevitable surgery for one to two years, and subsequently delay the medical and social benefits of weight loss to patients.

The environment in which surgery is performed is critically important to the safety and success of the procedures. Although not specifically patient criteria, there are important guidelines as to who should be performing these complex procedures. Outcome-based performance has led to the concept of "Center of Excellence" designations in bariatric surgery. Only specially trained centers devoted to the care and management of obese patients should offer bariatric surgery. For instance, Medicare patients can obtain bariatric surgery services only through accredited centers that have met detailed safety requirements. Many private insurers also have accreditation for bariatric surgery centers based on outcomes. Studies have also demonstrated that hospitals that perform more than 100 annual cases report lower morbidity and mortality rates and shorter hospital length of stay. Published mortality rates by Centers of Excellence are 0.3 percent.

In summary, qualifications for bariatric surgery are complex. While they depend on many objective factors such as BMI and age, there are many subjective factors that may qualify patients for surgery at some programs, while disqualifying them at others. Insurers may use different criteria than surgeons. Controversial qualifications such as adolescent bariatric surgery and extent of prior nonsurgical weight-loss attempts must be assessed on an individual basis for each bariatric program. Finally, many qualifications depend on the surgeon experience and the hospital's ability to effectively manage these complex patients. The hope is that through further research, preoperative screening evaluations will more accurately de-

termine which patients would benefit the most from obesity surgery.

SEE ALSO: Bariatric Surgery in Children; Bariatric Surgery in Women; Lap Band; Laparoscopy; Roux-en-Y Gastric Bypass.

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Quality of Life

SCIENTISTS HAVE CONDUCTED a review of studies to examine the effects of obesity on health-related quality of life and summarize that obese persons report significant impairments in quality of life that worsen with increasing body mass index (BMI), and that obesity has a greater impact on physical functioning than mental health. In addition, this review showed that pain is a particularly important factor in obesity that impacts quality of life independently from weight. Several studies have found that obese people in the general population and clinical samples report impairments in physical functioning (e.g. ease of walking, climbing stairs, physical work, and participation in sports) more so than impairments in other domains.

Researchers have observed that within morbidly obese, treatment-seeking populations, often the most significant detriments to quality of life occur in relation to physical functioning, vitality and bodily pain (e.g. lower back pain and joint pain). Compared to other chronic medical conditions such as congestive heart failure, obese patients report higher levels of bodily pain. Specifically, their level of reported impairment related to pain was similar to that of patients who suffer from chronic migraine headaches. Furthermore, compared to obese patients who did not report pain, those who did report pain showed significantly lower impairments on all dimensions of quality of life. These findings suggest that the low energy levels and pain associated with obesity may interfere with an individual's ability to lead an active lifestyle.

Studies regarding the impact of obesity on psychological functioning yield mixed results. While some studies have found that obese people report impaired mental and emotional functioning on both generic and obesity-specific quality of life measures, other studies have found little significant differences between obese and nonobese participants in psychological domains. In general, studies have found that obese women display more depressive symptoms than obese men, and those who seek treatment are more likely to report depression than those who do not.

It is important to note that most quality of life studies are conducted with participants who are seeking treatment for obesity, making it difficult to draw conclusions about quality of life for obese people in the general population. However, several recent studies conducted in both the United States and Europe have assessed the association between obesity and quality of life using large samples of the general population. These studies uniformly report that obese individuals have significantly lower quality of life scores than non-obese individuals, especially in physical functioning. Quality of life scores worsened with increasing obesity, so that the severely obese (BMI > 35 kg/m2) had the poorest reported quality of life. Poorer quality of life was seen even in obese people who reported no other chronic diseases that are known to be linked to obesity.

However, obesity in the general population does not appear to be associated with significant impairments in mental, emotional and social functioning, except in those who are severely obese. On the other hand, those who reported at least one or more chronic medical conditions in addition to obesity did show significant deterioration in emotional well-being.

Given the adverse consequences associated with obesity, it is important to determine how weight loss affects quality of life. A review conducted by Maciejewski concluded that weight loss interventions did not have a consistently positive impact on quality

of life. Self-reported depression, in particular, did not improve in the majority of weight loss trials. Surgically-induced weight loss (e.g. gastric bypass, gastric banding, etc.) yielded the most significant improvements in quality of life for morbidly obese patients, especially in physical domains. Other research has demonstrated that significant weight loss is associated with improved physical function, vitality, and decreased bodily pain, and is in general more strongly associated with domains of physical health, rather than mental health. Some research suggests that complications or side-effects from weight loss surgery can actually worsen quality of life, although this research is limited. Studies also show that subsequent weight regain after surgery is associated with worsening quality of life, although perceptions of well-being still remain slightly improved compared to pre-surgical levels.

A parallel body of research examining the psychosocial consequences of negative attitudes towards obese persons illustrates that the pervasive nature of weight stigma in our society has harmful affects on obese individual's life experiences and well-being. This research suggests that decreasing societal-level stigmatization and discrimination towards obese persons can improve their quality of life in meaningful ways. Furthermore, research also suggests that negative experiences resulting from weight stigma may perpetuate the problem of obesity by increasing unhealthy eating behaviors and avoidance of physical activity.

SEE ALSO: Depression; Self-Esteem and Children's Weight; Self-Esteem and Obesity.

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Quantitative Trait Locus Mapping

QUANTITATIVE TRAIT LOCUS (QTL) mapping is a statistical tool utilizing the science of quantitative trait loci (QTLs). A QTL is a section of deoxyribonucleic acid (DNA) on a chromosome that holds two or more genes which all play a role in a particular trait. QTL mapping has been used to identify genes involved in skin color, body mass, diabetes, cancer, and more.

DNA is organized in the cell into chromosomes. Humans, for example, have 22 pairs of chromosomes along with the two sex chromosomes, or 46 chromosomes all together. Roughly speaking, the chromosomes are organized into noncoding DNA and coding DNA, which encodes ribonucleic acid (RNA) that is usually translated into proteins. The region of a chromosome which encodes a particular RNA or group of RNAs is called a gene. A hypothesis that is the base of QTL theory is that groups of genes near to each other on a chromosome may have a multigene effect on certain polygenic traits. Using sophisticated mathematics and genetic experiments, investigators can examine these gene regions and their relationships to traits. This is the process for QTL mapping. In other words, scientists look at how often particular genes are expressed together in relation (linkage) to specific traits.

With advanced modern technology, scientists can use QTL mapping as a tool for determining the genes involved in a particular trait. For example, if an animal model such as a mouse exhibited a trait similar to a human polygenic trait, researchers could determine which region of DNA is responsible for the trait. This region could then be sequenced and the genes analyzed. The region is found using analysis which determines the regions of DNA that are more likely than chance to associate with a particular trait.

To narrow down from an animal's entire genome to a DNA region of interest, scientists use the technique of markers. Known single-gene characteristics, such as coat color, that are peppered throughout the genome are used to track DNA regions along the trait of interest. If, for a simplified example, in an inbred (genetically identical) colony of obese mice, more obese mice were black than brown, and the difference were greater than chance alone, scientists could statistically determine that the QTL related to that mouse strain's obesity were near to the black coat gene.

Another use of QTL mapping is determining genetics of food sources which might provide more or less nutrition, or might make a consumer more or less susceptible to overweight or obese status. For example, plants or meat animals might be analyzed via QTL mapping, as might dairy cows for milk production.

Because more than one gene is involved, standard rules of Mendelian genetics do not apply to polygenic traits; rather, they are inherited along a spectrum. In other words, the phenotype (usually the physical appearance) of a trait will vary along a gradient—where it lies in the gradient depends on all the individual genes and their interactions. Most polygenic traits are quantifiable phenotypes, hence the "quantitative trait" in QTL. Locus refers to the characteristic of having a position on the chromosome. An example of a polygenic trait that can be quantified is human body weight. Mendelian law predicts every grown child to have an intermediate weight between the weights of the two grown parents. Thus, two children of the same parents should have the same weight. This would be the case if only one gene controlled weight; however, because multiple genes have an effect on body weight, offspring have weights on a gradient, not necessarily falling between the two parental values.

Other examples of polygenic traits are diabetes, obesity, cancer, Parkinson's disease, and other disorders with multiple genetic components. Many, if not all, polygenic traits have some degree of environmental component as well. Some theorists ascribe to the idea that a person's genetic makeup may make him or her more or less susceptible to environmental threats, stresses, or toxins. This theory explains why some people in a particular environment develop a particular disorder such as depression while others do not. Furthermore, those people from the same environment who develop the same disorder will develop different clinical levels of that disorder.

With the knowledge that body mass and obesity have QTL components, a vast market has sprouted, catering to peoples' desires to take control of their seemingly uncontrollable weight. While some of these products are genuine and may be of help, many are unfortunately shams merely preying on those desires. Numerous marketing schemes have developed around the idea of "feeding your genes"; it is difficult to tell which product is beneficial and which has negligible, or worse, negative effects.

This idea of genetically caused obesity is beneficial in that it inspires funding in scientific research on obesity; however, it can be dangerous because it may allow a passive, resigned attitude toward obesity. On the other hand, a genetic theory may provide hope for someone who has fought a lifelong battle with obesity and prevent that person from sliding into depression or another emotional disorder.

SEE ALSO: Animal QTLs; Genetics; Genomics; Human QTLs.

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Respiratory Problems

BECAUSE OF THE size of some people who suffer from obesity, many have developed respiratory problems. This has been evident throughout history with much of it thought to come from the extra pressure that the weight puts on the person's heart, although it is now clear that it is because of the increased work that has to be done by the lungs of someone suffering from obesity.

Medically, this is often because people who are overweight or obese develop a much heavier chest wall, which is more difficult for the lungs to lift, putting extra strain on them. This has often meant that people with severe cases of obesity have trouble undertaking even simple tasks such as walking medium-to-longer distances, shopping, climbing stairs, or exercise. This lack of activity further contributes to putting on more weight and becoming more overweight or obese. This is evident by obese people who wheezing as they climb stairs or undertake what is to many other people a relatively simple manual task.

Obesity by itself does not cause asthma or bronchitis, but because obesity can interfere with breathing, it is possible for it to aggravate an attack. For this reason, a significant number of obese people do suffer from asthma or other breathing problems, and are more susceptible to complications if they contract bronchitis.

Another respiratory problem concerning obesity is sleep apnea, a condition in which someone may stop breathing while he or she is asleep. This occurs when soft tissue in a person's throat collapses around the airway to form a complete blockage, and is then prevented from breathing. The blockage causes the person to wake up and reposition himself or herself in the bed. If this happens on a regular basis, it will lead to interrupted sleep patterns, which in turn leads to drowsiness, daytime sleepiness, and even headaches.

People who suffer from obesity are more likely to have sleep apnea, as the problem is more likely to occur when people have fat deposits in the tongue and the neck. It has also been found to be more likely to occur when a person sleeps on his or her back rather than on his or her side. Although an odd occurrence is not a major problem, in the long term, if a person regularly suffers from sleep apnea, it may lead to his or her being more likely to have high blood pressure, heart rhythm disturbances, heart disease, or a stroke, and in cases where the brain is deprived of oxygen for a longer period, it may affect mental function. In the most extreme cases, it can even lead to sudden death.

For people who suffer from severe obesity, especially those weighing over 350 pounds, obesity hypoventilation can be a major concern. It is more common with people who suffer from sleep apnea, and is an abnormal breathing condition that can result in the accumulation of significantly high levels of carbon

dioxide in the blood, and in extreme cases, these levels can become toxic.

SEE ALSO: Asthma; Sleep Apnea.

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Rimonabant

RIMONABANT IS THE first inverse agonist discovered for the cannabinoid-1 receptor (CB1R) and is being developed for the treatment of obesity. Although rimonabant binds to the CB1R, it is not a cannabinoid-like compound and bears no structural resemblance to cannabinoids.

In cell-based in vitro assays, rimonabant inhibits the intrinsic activity of CB1R and produces an effect in the opposite direction compared to an agonist, hence rimonabant is classified as an inverse agonist. It also inhibits the binding of cannabinoid agonists and endocannabinoids to the CB1R, hence it is sometimes referred to as an antagonist. In experimental animal studies, rimonabant does not exhibit cannabimimetic properties and it inhibits many agonist-induced effects. In rodent studies, rimonabant can cause significant weight loss or reduction of weight gain.

In clinical trials involving obese patients, rimonabant has been shown to cause weight loss and reduction of waist circumference. Beneficial secondary endpoints have also been demonstrated, including increased high-density lipoprotein cholesterol (HDLC) and improved glycemic control. Rimonabant has been approved in the European Union (EU), Argentina, and Mexico for the treatment of obesity. As of March 2007, it is under review by the U.S. Food and Drug Administration and it is not yet available in the United States.

Rimonabant at 20 milligrams is generally well tolerated. As described in the Summary of Product Characteristics approved by the EU regulatory agency, the most common adverse reactions resulting in discontinuation include nausea, mood alteration with depressive symptoms, anxiety, and dizziness.

SEE ALSO: Acomplia; Cannabinoid System; G-Protein Coupled Receptor.

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Roux-en-Y Gastric Bypass

ROUX-EN-Y GASTRIC BYPASS (RYGBP) is an operation that can be used to cause significant weight loss in morbidly obese individuals. This operation is not new and has been performed for weight loss since 1967. However, the last decade has seen explosive growth in all types of weight-loss surgeries going from 20,000 to 30,000 yearly in the early 1990s to over 120,000 in 2006. Most of the growth is attributed to the development, maturation, and application of minimally invasive surgical techniques to the field of bariatric surgery. Numerous outcome-based studies have now clearly shown this operation to be safe and effective for long-term control of obesity along with resolving associated life-threatening conditions such as diabetes, high blood pressure, and sleep apnea.

The RYGBP should be considered in any patient who is morbidly obese and has tried and failed to lose weight with diet and exercise programs. The procedure is currently restricted to obese individuals who have a body mass index (BMI) of 35 or more with medical problems related to obesity or any patient with a BMI greater than 40.

The first step in the surgical procedure is to make the stomach smaller. The stomach is divided into a small upper section commonly referred to as a *pouch* and a larger bottom section referred to as the *remenant*. The pouch receives food, while the remenant stomach pro-

duces gastric juice and is not connected to the pouch in any way. The second step is to divide the small intestine. One end is connected to the pouch. This limb is referred to as the Roux limb. When patients eat, the food will now travel from the pouch through the Roux limb, bypassing (hence the term *gastric bypass*) the remenant portion of the stomach, the duodenum, and a portion of the jejunum. The last step is to reconnect the base of the jejunum with the remaining portion of the small intestines from the bottom of the stomach, forming a y-shape. This "y-connection" allows food to mix with pancreatic fluid and bile, aiding the absorption of important vitamins and minerals.

RYGBP is a procedure that combines restricted intake and reduced absorption of calories ingested to be successful. The restriction in food intake is secondary to the small pouch size. The malabsorption of calories occurs from bypassing the remnant, the whole duodenum, and a portion of the jejunum. This results in approximately 20 percent of all fat calories consumed to pass through the body undigested. However, the body may not absorb vitamins and minerals properly and lifelong supplementation of these vitamins and minerals are essential to prevent defiency. Additionally, patients also experience anorexia between six and 18 months after surgery. Anorexia following RYGBP is thought to result from changes in the bodies hormonal enviroment. This greatly assists in the weight-loss patients' experience.

Recently, minimal invasive laparoscopic techniques have been used to perform this complex operation. In the laparoscopic approach, aproximately five to six small incisions (1 centimeter or less) are made in the abdominal wall instead of one big incision. This results in significantly less pain with fewer wound complications such as infection and hernias. The other advantages of the laparoscopic approach include faster recovery, shorter hospital stay, and less perioperative morbidity.

Mortality rate in centers where more than 120 cases a year are done averages 0.3 percent. This is less than lung, heart, and most joint replacement surgery. Most deaths are from pulmonary embolism or gastrointestinal leaks. Later complications include anemia from B12 or iron deficiencies (almost always in premenopausal women) and intestinal obstruction from hernias. A condition known as dumping syndrome can occur from eating too much sugar or large amounts of salty food. While it is not considered a serious health risk, the results can be very unpleasant. Symptoms can include vomiting, nausea, weakness, sweating, faintness, and on occasion, diarrhea. Some patients are never able to eat sugary foods after surgery. Approximately 60 percent of patients get some form of dumping syndrome. Most patients do consider themselves fortunate because it prevents them from eating foods that would make them regain the weight.

Most patients lose an average of 10 pounds per month and reach a stable weight between 18 and 24 months after surgery. A typical patient would lose 70 percent of their excess body weight by year three and keep it off. Most studies have found that exercise, nutritional counseling, and the support of other patients who have undergone weight-loss surgery are extremely important to help patients lose weight and maintain weight loss following gastric bypass. Typically, weight regain is related to snacking or drinking high caloric drinks.

The greatest strength of this surgery is its ability to cure a majority of the life-threatening conditions associated with obesity. Improvement in Type 2 diabetes is seen a few days after the operation and eventually more than 90 percent of of type 2 diabetics are cured of their disease or no longer have to take medication. Eighty percent of patients with hypertension no longer have to take medication, 80 percent of patients with high cholesterol or triglycerides no longer have to take medications, 80 percent of patients with joint pain resolve, the rate of cancer deaths drops by half, and very often fertility and urinary continence return after RYGBP. These changes are mediated by weight loss and hormonal changes from the surgery itself.

SEE ALSO: Body Mass Index; Gastric Bypass; Laparoscopy; Multidisciplinary Bariatric Surgery Programs; Qualifications for Bariatric Surgery.

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Safe Play Opportunities for Children

OBESITY IS THE second leading cause of preventable death in the United States. According to the Centers for Diseases Control and Prevention, obesity rates have doubled among children and tripled among adolescents since 1980, and currently, about one in six children and adolescents are estimated to be overweight or at risk for overweight. Overweight during childhood and adolescence is likely to continue into the adulthood, and these earlier years in life are very important times to establish a healthy lifestyle to prevent obesity and other related health problems.

Causes of obesity are multifactorial and complex, ranging from genetic susceptibility to high energy consumption, increasing sedentary lifestyles, and socioeconomic and environmental factors. Many of the minority populations and people with low socioeconomic status have higher rates of obesity. People with low socioeconomic status, as measured by education and income levels, are also more physically sedentary during leisure-time compared to the individuals with a higher socioeconomic status.

Physical activity can be a strong preventive strategy against obesity, and national guidelines recommend that children and youth should perform at least moderate intensity physical activities for 60 minutes per day on most days of the week. However, large proportions of children lack this level of physical activity, and furthermore, participation in physical activities decline as children get older. Nationally, it is estimated that more than one-third of high school students does not get recommended levels of physical activity.

Physical activity levels can be influenced by several environmental factors such as availability of school and after-school physical activity programs, and access to safe walking, bicycling and play areas. Because children spend most of their time in school, availability, type, and amount of physical activity programs in schools or after-school settings are very important in increasing physical activity. Unfortunately, only a very small portion of schools offer daily physical education. Research shows that physical education classes can be effective in improving children's physical fitness. Additionally, simple methods such as marking the school grounds for active play and providing balls and other equipment during recess also help increase the physical activity level among children.

Transportation to and from school is another opportunity for children to engage in physical activities such as walking or bicycling. However, the built environment and lifestyles in general have changed in a way that made driving almost a necessity for families; walking or bicycling to and from school is no longer a common practice among students. The distance to school, availability of sidewalks, and traffic patterns

influence parents' decisions to let their children walk to and from school. In addition to the physical availability and access issues, safety of the route between home and the school is also a likely factor affecting whether children walk to and from school.

Overall, children have been reported to be less physically active and to be less likely to walk or bicycle to and from school in unsafe neighborhoods compared to safer environments. Street traffic can also be a safety barrier against walking. However, studies show that street improvements and decreasing the driving speed rather than the traffic volume can be effective ways to overcome this barrier.

Children are more physically active when they spend time in outdoor activities. For example, availability of parks is an important predictor of physical activity during leisure-time. Research shows that living within one mile of a park versus living farther away is associated with greater physical activity levels during leisure-time and outside the school hours. However, safety of the environment is a determinant of outdoor activities especially for girls, and concerns about crime and safety in the neighborhood may lead to increases in sedentary activities indoors such as watching television or video games. Furthermore, increased levels of concern about neighborhood safety may lead to psychosocial stress or anxiety and result in social isolation, decreased leisure-time physical activity and increased food intake.

Having safe environments for play, sports, or transportation activities is a large part of encouraging children and youth to remain active. Examples of such opportunities include easy access to parks, walking trails, sidewalks, sports facilities, and promotion of transportation activities like walking or bicycling to and from school. Minorities and people who live in socioeconomically compromised neighborhoods appear to be in a greater need to improve the safety of the environment and the access to facilities and equipment as they are even at a higher risk for sedentary behaviors and greater obesity rates.

SEE ALSO: Built Environments; Community Programs to Prevent Obesity; Overall Diet Quality; School Initiatives to Prevent Obesity.

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Safety of Urban Environments

PERSONAL SAFETY IN urban areas is an important concern as people consider whether their neighborhood is conducive to leisure activities. Such leisure activities can include walking, jogging, and bicycling as well as utilizing public recreational areas. Safety in the study of the urban environment and obesity is usually centered on physical activity, namely walking within the neighborhood.

There are certain physical and social characteristics of the urban environment that contribute to the safety and thus the walkability (i.e., the ease and convenience of walking) of the environment. Physical characteristics such as well-maintained and continuous sidewalks, accessibility of crosswalks, and traffic patterns that allow for pedestrian travel help to promote walking in neighborhoods. Similarly, social characteristics such as the perception of crime also contributes to the likelihood that people will walk within their communities.

It is important that the areas around an individual's home offer numerous safe walking routes to a variety of different locations. People who live in areas that have a diverse combination of commercial space (e.g., convenience stores and grocery stores) and green spaces (e.g., parks and gardens) among residential properties are more likely to walk within these areas.

In contrast, communities that have low-density land use areas consisting of separate residential, shopping and business areas are considered not to be conducive to walking. This type of community tends to have long distances between homes and destinations (i.e., workplaces, stores) and low street connectivity making pedestrian travel difficult and increasing the dependence on driving. By increasing the dependence on driving, there is a subsequent

increase in the amount of cars on residential streets. This creates a more hazardous environment for pedestrians and bicyclists due to the increased probability of traffic accidents. Long block lengths and low street connectivity also means that there are fewer crosswalks for pedestrians to safely cross intersections, potentially increasing the risk of an accident among vehicles and pedestrians.

Although actual crime can be objectively measured, it may be more important to assess subjective measures of an individual's fear of crime. People are less willing to walk in their neighborhoods if they perceive threats to their physical well-being. Similarly, parents are less likely to allow their children to play outdoors if they perceive a threat to their child's safety. Criminal behavior can induce fear in several ways. Direct victimization, for example, having been the victim of a criminal offense, can create fear of a second criminal act. Indirect victimization, either through the media or personal communication, can also generate fear in people who were never actually victims of a crime.

Aside from criminal offenses, the physical and social environments also have the ability to induce fear of crime. Social incivilities are minor forms of misbehavior such as loitering, drug use, and panhandling. Physical incivilities are attributes of a neighborhood that create a feeling of disorder and may include graffiti, vandalism, or abandoned cars. Both physical and social incivilities act as warning signs to residents that they are at risk of victimization, potentially causing feelings of fear even to residents who have never been victims of crime. In an effort to deter crime and engender a feeling of safety, the design characteristics of the neighborhood can prove effective in creating a safe environment. For example, a well-lit neighborhood with homes where doors and windows look onto streets and parking areas will increase feelings of security because residents know that others can see and intervene if a crime does occur.

The U.S. Bureau of Census defines an urbanized area as a place, including the adjacently surrounding territory, with a population of at least 50,000 people. In the research literature on urban environments and obesity, the definition of neighborhood or community is not consistent between studies. When assessments of walkable areas are made, boundaries are drawn around an individual's home. Quite often, neighborhoods are comprised from census track boundaries or some larger geographic area that has a political significance such as voting districts or historical community districts.

An increased dependence on driving creates a more hazardous environment for pedestrians and bicyclists.

In other reports, the neighborhood could be defined as the one-mile circumference around an individual's home. These areas are analyzed and interpreted as the space most likely to be relevant when people determine the walkability of their living area. The health implications of densely populated areas pose unique public health challenges especially as it relates to obesity and obesity promoting behaviors. These behaviors are influenced by access to healthy food choices and places conducive to physical activity. As such, the use of the term *urban environment* in the health literature implies the inclusion of health-related issues that are of most concern in areas with a dense population of often various racial/ethnic and socioeconomic peoples in a defined physical space sharing a number of resources.

SEE ALSO: Built Environments; Physical Activity and Obesity.

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Satietin

SATIETIN IS A 50,000 dalton glycoprotein, or a protein that has been conjugated with a carbohydrate. Produced within the body and isolated from the plasma of blood, it is a highly potent anorectic substance. In other words, it has the activity to suppress the appetite of an individual. Also, its use has shown to be effective in reducing the body weight when utilized in rat studies. With regard to the highly complex system which mediates satiety and its hyperfunction in terms of obesity, much is still unknown about the exact chemical nature, pathways of synthesis, and metabolism of satietin.

Originally discovered in 1977, satietin was isolated from plasma in the human blood. Since then, it has been isolated in several different species of mammals including horse, rat, guinea pig, rabbit, steer, cat, dog, and goose. The glycoprotein itself is composed of 70 to 75 percent carbohydrate, and 14 to 15 percent amino acid by weight. Specifically, the 5-carbon sugar rhamnose and the 6-carbon sugar glucose have been associated as carbohydrate constituents of satietin.

Satietin is a part of a very complex system which regulates feeding. It was hypothesized that its natural function in the human body was to induce satiety. For example, ingestion of food would liberate and convert satietin to an active anorectic agent which would also fully activate the satiety system. Subsequently, a signal is sent to the brain (whether it is satietin itself or another signal messenger is still unknown), which consequently sends the message that the process of food ingestion must be ceased. This, in turn, would turn off the feeding system of the brain. Once the satietin signal had completely diminished from the brain, the feeding system would be reinitiated and the feeling of hunger would come back. This is still considered the general scheme of action upon which it works, although the complex system which mediates satiety is still relatively misunderstood and thus satietin's active role in this process is still relatively unknown. What has been confirmed, though, is that satietin's activity in the brain can be localized to the amygdala and the hypothalamus. Further investigation of this activity will give clues to its synthesis, activity, and metabolism.

When injected into rats in either the cerebroventricular space of the brain, intravenously in the blood or subcutaneously in the skin, it has been shown to immediately inhibit feeding of rats and thus can block the hunger drive for up to 46 hours. This has been shown even when the rats themselves were deprived of food for 96 hours. Also, satietin use over an extended period of time resulted in loss of significant body weight in rats. Future therapeutic interventions for the treatment of obesity will look to mitigate the actions of the satiety system such as through drug administration of endogenous satiety signals such as cholecystokynin, bombesin, and satietin.

SEE ALSO: Appetite Control; Appetite Signals; Bombesin; Cholecystokynin.

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School-Based Interventions to Prevent Obesity in Children

THE PREVALENCE OF childhood obesity is increasing throughout the world. In United States, the percentage of children who are considered overweight or obese has doubled, from 15 percent in the 1970s to nearly 30 percent today, while the children who are considered obese has tripled. Obesity poses a serious health threat. There are serious health and economic consequences associated with child obesity. Children who are obese or at risk for obesity have a

greater likelihood of being obese in adulthood and developing heart disease, diabetes, and other serious health conditions. Most obesity interventions have taken place in clinical settings. However, schools also provide an opportunity for preventing and treating obesity, because most children and adolescents are enrolled in schools and spend a significant amount of time there. Schools present a unique opportunity to promote and teach children healthy diet and encourage physical activity.

One of the strategies to plan for prevention of obesity in children at school age is establishing intervention-based school programs which include the following.

Incorporate of nutritional education into the curriculum: All academic institutions and schools should work to develop and implement curricula to teach knowledge and skills needed to adopt healthy nutrition behaviors, such as eating habits, food choices, and lifestyle. Schools should provide opportunities to children to practice those skills, and help them to overcome barriers to adopting positive behavior. Curricula should be culturally appropriate; the program must emphasize reducing the consumption of fat while increasing of fruit and vegetables. It is necessary that school administrators understand that student health, including healthy weight, is a component of successful academic performance.

Improve physical activity standards within schools: Schools can provide multiple opportunities for students to participate and enjoy physical activities. Schools should increase the amount of time that students spend in physical activity. Quality physical education requires at least 150 minutes per week for elementary schools and 225 minutes per week for secondary schools. The physical activity program should keep students active most of the education class time; also, it should be enjoyable for students. Teachers can integrate nutrition and physical activity into existing core subjects such as math and science through movement and the sedentary activities should be discouraged. Schools should implement after-school programs to increase children's physical activity even after school hours; this can be encouraged by opening the school facilities and making them available after school hours and on weekends for recreational and fitness use.

Provide health education for school staff: It is one of the strategies that contribute to student health, by giving the school staff skills and knowledge that they need to become role models for good health. The education program should provide school staff members with the opportunity to participate in nutrition classes and physical activity programs, which can improve their commitment to the health of the students and create positive role modeling. Staff should be aware of the risk factors for obesity in children and how to prevent it. Also, they should be informed about long-term health risks associated with obesity in adults and children among school personnel

Improve school food environment: Meals and snacks provided by schools have a substantial impact on the students' dietary intake, because up to 50 percent of total daily energy intake can be consumed at school. School meals should provide students with essential nutritional components for normal growth and development; it should include a variety of foods so that the child gets the needed nutrients (such as protein, carbohydrate, fat, vitamins, and minerals). School food service must limit access to fast foods, prohibit student access to foods with minimal nutritional value, and eliminate advertising for unhealthy foods on school campuses.

Make environmental changes at school: School administrations and school boards should support the implementation of school health policies that promote environmental changes related to nutrition and physical activity and build an environment that promotes healthy lifestyles and healthy diet, by limiting access to sweetened beverages and other high-calorie vending machines at school stores. They should also modify food prices to promote the purchase of healthy foods and provide guidelines for healthy eating habits to help school food service personnel develop and implement healthy food preparation.

Involve parents in obesity prevention: The role of parents and caregivers in influencing the development of healthy eating behaviors are important, as parents and caregivers can be the primary influence for children. Parents can be a good example for their children by modeling healthful eating behaviors and being physically active. Involving parents makes it easier for them to support their children and reinforce positive behaviors at home. Parents should receive guidance on nutrition and physical activity. They should be taught about nutrition, food labels, food safety, and how to

choose and prepare healthy food for their children; they should be provided with instruction manual and visual materials; and they should avoid using candy as a reward. Parents should be educated about strategies that encourage a reduction in sedentary child behaviors, for example, decrease the amount of time children devote to screen viewing (television, video games, computer) and increase physical activity. Activity Guidelines for Children and Youth recommend that children engage in at least 30 minutes of daily physical activity

Prevent and Treat obesity at schools: it is important to recognize that obesity in many cases is preventable. Schools, healthcare professionals, and parents can work together to reduce the prevalence of overweight and obesity and their consequences. Teaching parents and students about healthy diet and the importance of exercise is essential because obesity is more easily prevented than treated. Preventive strategies should aim at increasing the parents' and students' awareness of risk factors that contribute to overweight, including an sedentary lifestyles, intake of high-caloric foods, parental overweight, low socioeconomic status, high birth weight, early timing or rate of maturation, and psychological factors.

Obesity prevention program should include regular follow-up for all students by routinely measuring their height and weight to assess risk of obesity. For obese children, healthcare providers must provide them with medical and education care.

Work with health care providers: Healthcare providers should design resources to help schools implement a comprehensive and consistent approach to promoting healthy eating among students. Schools should provide health education programs for children and parents that foster healthy activity and eating patterns, and facilitate and lead open discussions with parents about obesity in children and the chronic conditions resulting from being overweight. They also can organize group discussions and encourage peer support toward healthy lifestyle goals, and reduce potential for stigmatization of those who may be overweight.

A healthy school environment can promote healthy habits. The chances for success will be greater if schools, healthcare providers, and parents cooperate and become committed to doing all they can to help the young generation enjoy a healthy life.

SEE ALSO: School Initiatives to Prevent Obesity; School Lunch Programs.

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School Initiatives to Prevent Obesity

CHILDREN SPEND MANY hours in school as they grow, giving schools a unique role to play in promoting healthy living. The vast majority of children, almost 95 percent, attend school and school is the most consistent and intensive influence on children outside of the family. Children spend a third of every weekday in school or roughly 12,000 hours by the time they graduate from high school. One important factor in healthy living is maintaining a healthy weight. Obesity rates are rising in children with approximately 30 percent of school-aged children overweight and 15 percent obese. Overweight children tend to grow into overweight adults. Health issues that used to primarily affect only adults, such as Type 2 diabetes and hypertension, are increasingly affecting overweight children with concomitant costs to the individual and to the healthcare system.

Being overweight as a child is linked to lower academic achievement, greater absences from school, and social problems. Schools offer many opportunities for developing obesity-prevention strategies and these fit within the mandate of schools as health and success in education are highly related. These initiatives fall into four main categories: providing more nutritious food to schoolchildren, offering greater opportunities for physical activity, providing obesity-related health services, and promoting healthy lifestyle choices.

THE ROLE OF FOOD AT SCHOOL

Schoolchildren consume between 20 and 50 percent of their daily food intake while at school. The first way in which schools can help reduce obesity is in the type of foods they serve to children. Children who eat nutritious breakfasts have better cognitive skills and eating a healthy breakfast is also associated with eating fewer calories overall in the day. While many children eat breakfast at home and eat home-prepared lunches either at home or at school, the majority of schools provide sponsored meal programs for both breakfast and lunch. Some schools provide snacks and some schools also provide after-school snacks and dinners in aftercare programs.

School breakfasts and lunches must meet federal nutrition standards, and the sale of certain foods such as soft drinks, water ices, and certain candies during mealtimes is prohibited. However, at other times when food is allowed for sale, there are fewer restrictions and requirements and dietary guidelines do not have to be followed. This means, for example, that while a school cafeteria may not sell soft drinks, sodas can be purchased from vending machines located just outside the cafeteria doors. Some argue that federal standards are inadequate, and stricter state and local regulations should exist.

For budgetary reasons, many schools continue to sell popular but nutritionally poorer foods à la carte in cafeterias, vending machines, snack bars, or for fund-raisers. When children want fries for lunch, if the school cafeteria does not sell them, children may go to a local vendor off school premises. These noncontrolled foods, which are called competitive foods, are not subject to the same regulated dietary guidelines. Availability of these foods increases with age, with almost all high schools offering these kinds of food for sale. The more available these competitive foods are, the less fruit and vegetables are sold and the more fat and calories are consumed.

However, interventions are changing school food environments. Limits can be placed on what is available in vending machines and cafeterias, and on at what times and where these sources of foods can be available. Efforts can be made to reduce the sugar and fat content of foods sold in school cafeterias and in vending machines. For example, making more fruits and vegetables available encourages children to try these choices. When vegetables are sold in salad bars and when food is baked rather than fried, healthier eating is promoted and this encourages children to try items other than sodas, candy bars, and french fries. Vending machines can offer low-fat milk, pure fruit juices, or waters rather than sodas, and fruit-based snacks rather than candy and chips. Portion sizes can be controlled in school cafeterias so that the child learns what amount of food should be considered a normal serving compared to what are often oversized servings in restaurants. Other successful initiatives to promote healthy eating in schools have included hiring specialty chefs, improving the hours and atmosphere of the school cafeteria, and including the children in menu planning and cooking.

PHYSICAL ACTIVITY PROGRAMS

Physically active children have the opportunity to develop social skills and their academic performance also improves. Another initiative that schools can undertake to prevent obesity is to promote physical activity. Schools have the unique ability to promote physical activity through mandatory physical education activities. Physical education includes not only the active participation of children in physical activities such as the typical gym class, it also includes health education about the benefits of a healthy, active lifestyle, including proper nutrition and the benefits of exercise. These benefits include improvements in strength and endurance; the building of healthy bones, muscles, and joints; and weight control. In addition, there can be positive influences that result in reduced anxiety and stress, and increased self-esteem and overall energy level.

Through the education component of physical education, children can acquire the necessary skills and knowledge to lead healthy lives. In the same way children have learned the dangers of smoking, they can learn the dangers of a high-fat, high-sugar diet and sedentary lifestyle. Children need to learn the facts and the skills and they have to have the opportunity to implement these in making healthy lifestyle choices. They also need to learn how to overcome barriers to implementing these strategies.

Inactivity among children is rising and is considered a risk factor for school-aged children to become overweight. There are pressures that can work against efforts to encourage physical activity. As states use standardized tests to hold schools and

students academically accountable, physical education and recess have become a lower priority. But some states are now mandating and promoting more physical activity in schools.

Low levels of physical activity in school children reflect issues both within and without the school. Estimates suggest only half the children walk to school, and nearly half spend four hours or more watching television or playing computer games, on a daily basis. Schools have a role to play in implementing strategies to help children reduce their level of television viewing and at the same time to increase their level of physical activity. Lack of physical activity is a large contributor to the risk of obesity. Some schools do not have scheduled recess breaks even for elementary-aged children. Physical education, once an important part of every child's school day, has been cut back at many schools. Less than half of schoolchildren have access to daily physical education classes. Estimates suggest that 20 percent of children partake in two or fewer bouts of vigorous activity per week while at least 30–60 minutes daily of such activity is recommended.

In schools where physical education classes are offered, it is important to not only ensure that the children are actually physically active and not just listening, but also to ensure that the children are acquiring the sorts of skills that will allow them to remain physically active throughout their lives. This means that in addition to instruction in specific skills and sports, children are now engaging in more activities that emphasize personal fitness and aerobic conditioning including dance. Success should require making an effort not perfecting a skill.

Both competitive and noncompetitive games should be included and all children need to be included. This means the old-fashioned way of choosing players for teams by calling out names is no longer used as the last children picked may give up before the activity begins. Making physical education classes fun and interesting for all students, not just those who are naturally athletic is the key to encouraging children to remain physically active as adults.

Although the recommendation is that high school students spend more time in physical education activities than elementary students, the reality is that fewer high school students participate in these classes as they are not mandatory in these grades.

OBESITY-RELATED HEALTH SERVICES

School health services can also help address obesity by providing screening, health information, and referrals to students, especially low-income students, who tend to be underinsured and may not receive health services elsewhere. These are also the children who are most at high risk for obesity. Because some children may rely almost exclusively on school health services, it is important to ensure that staff can assist the child (and family) in making the right choices.

One initiative recently considered has been to provide information on each student's obesity risk via regular health report cards. While this raises privacy concerns, parental involvement is vital if a child's weight puts the child at risk.

PROMOTING HEALTHY LIFESTYLE CHOICES

An increasing number of schools are also encouraging healthy lifestyle behaviors. This includes not only healthy eating via the meals supplied by the school, but also information about healthy eating incorporated into the school curriculum. These programs can be especially successful when teachers are qualified, parents are involved both individually and through school councils, and health professionals contribute. Schools benefit when they have a coordinated health-oriented plan and a council to develop, implement, and monitor policies affecting nutrition and physical activity.

While the majority of children used to walk to school, in recent years the majority of children have arrived at school in a school bus. When children walk to school and parents or other adults walk with them as safety volunteers, physical activity increases among both children and adults. Similar programs promote riding bikes to school rather than school buses. Some schools offer opportunities for increased physical activity through intramural and competitive sports programs and sports clubs, and more emphasis on good old-fashioned recess. Making school facilities such as gymnasiums and swimming pools available to families after school can promote increased physical activity in both children and adults.

Some schools are able to take advantage of relationships with local farmers. Local high-quality produce gets consumed which benefits the farmers and the children may be able to visit the farm. Other initiatives involve local gardens or school gardens where the school community gets to participate in growing

the produce. There is some evidence that children who garden like vegetables better than children without gardening experience.

Schools are also large-scale employers and initiatives to improve the nutritional quality of food in schools and to promote physical activity benefit the staff as well. Health and activity promotion programs for teachers benefit those they teach. When the adults with whom children spend time model positive behaviors, this also impacts the children.

While parents are a child's primary social environment and while the family sets the stage for healthy eating and active behaviors, schools have a valuable role to play in providing a second environment where both healthy eating and physical activity are modeled. These interventions all hold promise for reducing the risk of obesity in school children. Schools cannot solve the problem on their own, but they are vital parts of the solution.

SEE ALSO: Obesity in Schools; Overweight Children and School Performance; School Based Interventions to Prevent Obesity; School Lunch Programs.

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School Lunch Programs

ESTABLISHED IN 1946, the National School Lunch Program (NSLP) is a federal program that enables schools to provide low-cost or free nutritious lunches to children who attend participating nonprofit private and public residential childcare institutions and schools. Its provisions supply cash subsidies and agricultural commodities to assist in assuring that healthy lunches are made available to our nation's youth, and that farm food prices are stabilized. The program is administered federally by the U.S. Department of Agriculture's (USDA's) Food and Nutrition Service (FNS) and locally through state government agencies who manage agreements with local school district food program directors and authorities.

Participating schools, that is, feeding sites, are responsible for menu formulation, food preparation, food distribution, program activities, and on-site program management. Meals served must meet all applicable recommendations from the Dietary Guidelines for Americans. For example, lunches are designed to meet one-third to one-half of the minimum daily food group requirements. A minimum of one-third the recommended dietary intake of vitamin A, vitamin C, iron, calcium, and calories must be met. The total calories from fat must not exceed 30 percent of total calories provided, and saturated fats must not exceed 10 percent. In exchange for agreeing to follow age-appropriate guidelines, feeding sites are reimbursed either per meal served or for the actual cost of the program, whichever is less.

One program provision mandates that all participating schools must grant students the opportunity to participate in subsidized special lunch pricing. Special pricing provisions grant free lunches to children who come from families with incomes less than or equal to 130 percent of the poverty level, and reduced-priced lunches for children from families with incomes at 130 to 185% of the poverty level.

Due to increased awareness of the link between learning and child hunger and nutrition, the National School Lunch Act was amended in 1962 to increase program efficiency, reduce disparities, and increase program participation. Through continued critical review and other legislative actions and mandates, the NSLP services, quality, funding, educational, and technical support have improved. By 2002, 99 percent of all public and private nonprofit schools participated in the NSLP, and by 2005, more than 29.6 million children were receiving free and reduced-price lunches. Through the success of the NSLP and related school programs, food is made available to students in need while indirectly teaching families the necessities of a good diet.

Significant legislative actions and mandates have served to improve and expand the school lunch program. Amendments to enhance program performance have been adopted.

Other policies that have positively impacted the school lunch program include those that established School Lunch Week, the School Meals Initiative for Healthy Children, Team Nutrition, and federally legislated school wellness policies. National School Lunch Week was established in 1962. A Joint Resolution of Congress guided the president to annually issue a proclamation for Americans to participate in "appropriate ceremonies and activities" during the second week of October to call attention to the program.

In the mid-1990s, school lunch meal quality was greatly impacted by the adoption of the School Meals Initiative for Healthy Children. This legislative mandate was implemented to amend nutrition standards and regulations for the national school feeding programs, including school lunch. This legislature required that school meals meet certain minimum standards for calories and other nutrients, such that they contain less than 30 percent calories from fat and 10 percent calories from saturated fat over the course of a week; provide one-third recommended daily allowance (RDA) for protein, iron, calcium, and vitamins A and C; comply with the Dietary Guidelines for Americans; include decreased levels of sodium and cholesterol and increased amounts of dietary fiber. The legislature further mandated that menus, recipes, food product descriptions, and production records be maintained to show that adequate food items or menu items are offered each given day, and that nutrition standards are met for specific age/grade groupings when averaged over each school week (a school week is defined as a minimum of three consecutive days and a maximum of seven consecutive days), and that state nutrition officials conduct audits at least once every three to five years to ensure compliance.

In addition to the USDA launch of the School Meals Initiative for Healthy Children, in 1994 USDA's

The National School Lunch Program (NSLP) is a federal program that enables schools to provide low-cost or free nutritious lunches to children who attend participating nonprofit private and public residential childcare institutions and schools.

FNS established Team Nutrition. Team Nutrition's aim was, and continues to be, to support the Child Nutrition Programs through training and technical assistance for foodservice personnel, nutrition education for children and their caregivers, and school and community support for healthy eating and physical activity. Team Nutrition's goals are to improve children's lifelong eating and physical activity habits. Team Nutrition resources are available for food service personnel, teachers, and other interested parties via Web sites, materials available for purchase, and so forth. Team Nutrition Training Grants and affiliated local wellness grants have funded work that has demonstrated strong impacts.

The most recent policies that have significantly impacted the NSLP were put into place by Congress during the reauthorization of the Child Nutrition Act in 2004. This legislation mandated that school districts create local school wellness policies to address health, nutrition education, physical activity, and every aspect of the foods and beverages made available during the school day. Local policies were established by the start of the 2006–07 school year; they continue to evolve to improve the health of our nation's youth.

Other programs of interest that are affiliated with and/or similar to the NSLP include the School Breakfast Program (established 1966), the After-School Snack Program (established 1998), the Summer Food Service Program for Children (established 1968), and the Homeless Children Nutrition Program (established 1994). These programs were established with the same student eligibility requirements as the NSLP, and they provide nutritious foods to children under the age of 18 and to the mentally and physically handicapped over the age of 18.

SEE ALSO: Federal Initiatives to Prevent Obesity; Food Stamp Nutrition Education Program; Governmental Policy and Obesity; Healthy People 2010.

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Self-Esteem and Children's Weight

A CLOSE RELATIONSHIP has been documented between weight and self-worth or self-esteem in childhood and adolescence, although the exact relationship is still unclear. Drive for thinness and societal pressure to achieve the ideal but unrealistic body size have reportedly been extended to children as young as 8 years old. Such pressures have caused anxiety regarding body weight in children and adolescents and may influence them to base self-worth on their weight, which may have significant social and emotional consequences. Factors such as weight that influence self-esteem have been found to be critical contributors to a child's all around development.

High self-esteem may be an indicator of outcomes like occupational success, social relationships, well-being, positive perceptions by peers, academic achievement, and improved coping skills. Conversely, low self-esteem is associated with elevated levels of loneliness, sadness, nervousness, and increased likelihood of initiating in high-risk behaviors such as smoking, sexual promiscuity, and alcohol consumption. With the rapid increase in childhood obesity, it is especially important to understand the psychosocial effects of weight on children. Many overweight children and adolescents are believed to be at particularly high risk for developing lower self-esteem and consequently the associated developmental risks.

ARE OBESE CHILDREN AT HIGHER RISK FOR LOW SELF-ESTEEM?

Findings regarding the association between the presence of childhood obesity and decreased levels of self-esteem vary between studies. The relationship between weight and self-esteem is most consistently reported when body esteem or body image are specifically measured as the primary aspect of self-esteem. Many professionals contend that weight additionally affects overall self-esteem as children with lower body image generally report lower global self-esteem. The causality of the relationship between weight and self-esteem also remains undetermined.

Negative attitudes toward overweight children begin young. These social pressures to be thin and high levels of peer teasing and rejection that can

accompany obesity may lower self-evaluation putting overweight children at a higher risk for developing significantly low levels of self-esteem. Other factors associated with obesity, such as decreased levels of physical activity, increased levels of depression, or poorer home environments may also be responsible for lower self-esteem levels in overweight/obese children and adolescents.

DIFFERENCES BY DEMOGRAPHIC VARIABLES

It is becoming more apparent that the relationship between self-esteem and obesity depends in part on children's age. It is not known at what age self-esteem and overweight/obesity become related but the overall self-esteem of overweight children may decrease from childhood to early adolescence. Reviews of studies like that by Richard Strauss and colleagues have found that, while there is a clear and measurable inverse relationship between self-esteem and overweight or obesity in adolescents (13–14), studies of younger children (9–10) have been less consistent. This may support beliefs that early adolescence is a critical period for the formation of self-worth and esteem in obese children.

The association between children's self-esteem and weight is also likely to be influenced by ethnicity. There appears to be differences in self-esteem between all black and white children of any weight, with white children's levels dropping in adolescences, but black's remaining stable throughout adolescence. Biro and colleagues found interactions between race and body mass index (BMI) on global self-esteem in that self-esteem was affected more negatively by increases in BMI in white versus black adolescents. It has also been suggested that black girls who perceived themselves to be overweight were less likely to have lower self-esteem than white girls who perceived themselves as overweight, with differences attributed to greater satisfaction in physical appearance. Additionally, some have suggested that Hispanic girls demonstrated similar weight-based changes in self-esteem as white children. This is consistent with negative weight perceptions, and dieting behaviors similar in both groups.

There also appears to be variance between genders in relationship of weight to self-esteem, which may be explained by gender differences in weight identity. Weight perception and concerns have been shown to vary by gender as girls are more likely to perceive themselves as overweight, to report more body dissatisfaction, and to be concerned about their weight than boys. In the general population, differences between genders in self-esteem emerge in early adolescence, with girls reporting lower general self-esteem than boys; Allen C. Israel and colleagues found the same to be true in overweight teens. Nevertheless, many have suggested that obese adolescent boys still may demonstrate mildly decreased levels of self-esteem compared to their normal-weight peers.

While there does appear to be clear variance across demographics, many have caution against making generalizations that all obese children of certain demographics have low self-esteem as not all obese children are affected, although reasons for their resilience are unclear. Moreover, in addition to demographic variables, other factors such as positive family or social interactions may mitigate the negative psychosocial affects of obesity.

WEIGHT, SELF-ESTEEM, AND TREATMENT

With such important developmental factors at risk, treatment has been at the forefront of obesity research. Findings from multiple studies have demonstrated that weight-loss treatment programs that focus on improving physical activity and dietary behaviors appear to also indirectly improve children's self-esteem. Such findings support the contention that obesity may play a role in the development of psychopathology and that early weight treatment could prevent psychopathology.

While weight appears to play an important causal role in the development of decreased self-esteem in overweight children, some still contend that the relationship between the two may be transactional. Huang and colleagues found that adolescents who lost or maintained their weight over the yearlong study period demonstrated lower baseline body image and self-esteem scores than those who went on to gain weight, suggesting that personal efforts to manage weight may be initially driven by body dissatisfaction. As such, experts are also exploring treatment programs including self-esteem building as a means for weight management.

SELF-ESTEEM AND NORMAL-WEIGHT CHILDREN

While there appears to be a definite association between weight and self-esteem for most overweight children, increasingly, there is a prevalence of body

dissatisfaction and weight-loss behaviors that are inappropriate for normal-weight children. Children are taught from early on that society values a slender body. More and more, children with an appropriate body weight are reporting dissatisfaction with their bodies and weight and engaging in attempts to lose weight, which many view as a factor of poor self-esteem and self-image. Pinheiro and colleagues found that children with appropriate weight but with lower self-esteem had twice the chance of feeling fat, when compared with normal-weight children with higher self-esteem. This relationship is concerning because body dissatisfaction and weight concerns among prepubescent children are associated with eating disorder symptoms in adolescence, particularly among young females.

Interventions aimed at improving self-esteem have been found to better body image and reduce unhealthy weight-loss behaviors in normal-weight children. O'Dea and colleagues found that an educational intervention significantly improved the body image of female participants while helping them to avoid the dieting and weight loss observed in a control group.

SUMMARY

While the prevalence, cause, and exact social and emotional consequences of decreased self-esteem in obese children remain undetermined, there appears to be a consequential relationship between the two. Moreover, this relationship may significantly influence overall development. Indicated in recent studies, more focus should be placed on improving treatment and prevention interventions for weight-related problems as a means of promoting healthy psychosocial development.

SEE ALSO: Body Image; Self-Esteem and Obesity; Self-Esteem in Obese Women.

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Self-Esteem and Obesity

MANY OF THOSE who are overweight and obese suffer with self-esteem issues because of their weight situation. Self-esteem is the experience that one is appropriate to life and to the requirements of life, and the confidence one has in one's ability to think and cope with the basic challenges of life. It is also the value, worth, and respect one pays oneself.

Self-concept, in contrast, is the overall attitude about oneself. Self-concept can be thought of as being on a continuum from positive to negative, or high to low. Do you like yourself? Are you a valuable person? Are you satisfied with the way you live your life? When faced with a challenge, are your thoughts positive or negative? Do you believe that you can meet the challenge or that you are not very good at doing things? If your beliefs and feelings about yourself are positive, you tend to have a high self-concept. Your personality is based, in part, on your self-concept.

Individuals who are overweight and obese in Western society deal with terrible stigmatization in many

aspects of interpersonal and social interaction. This stigmatization can, and does, result in internalization of the negative attitudes, feelings, and beliefs, thus adversely affecting self-esteem and ultimately self-concept. Not all overweight and obese individuals have poor self-esteem, however, and research continues into why some develop poor self-esteem and others do not. Some research suggests that low self-esteem leads to or promotes obesity, rather than obesity leading to and promoting low self-esteem. The current consensus is that each individual's self-esteem issue is somewhat situational.

Obese and overweight individuals often internalize the negative views and attitudes of others through numerous interactions and circumstances. Some of the more common ways that self-esteem has been negatively affected in the overweight and obese are listed below.

PAST EXPERIENCES THAT ARE OBSTACLES TO GOOD SELF-ESTEEM

- Being told that you were not enough or were too much (not enough like the other normal kids, or too fat, too big, too stupid, to undisciplined, too unfit).
- Being told your feelings were not acceptable or valid (feelings of good self-worth were unjustified or invalid because of failure to comport with the cultural body ideal).
- Being ridiculed or humiliated for weight, size, or appearance.
- Having feelings or actions dismissed as unimportant or of no value.
- Attempts at control through shame or guilt.
- Overprotection or obstruction of normal learning and experience, and increase on self-reliance.
- Lack of boundaries and rules, or contradictory and conflicting boundaries and rules.
- Encouragement to doubt self, doubt one's own abilities, or one's capabilities.
- Treating evident and obvious facts as unreal, thus shaking a sense of rationality and reality (mother ignores drunk father falling out of chair at table).
- Physical or sexual violence.
- Being told that you are sinful, bad, or unworthy.

These boil down to inadequacy, shame, guilt, generalized or specific sense of self-distrust, and generalized or specific feeling of unworthiness. The internalization of these circumstances and interactions can severely damage healthy self-esteem, but not all persons who have encountered these circumstances have negatively affected self-esteem.

Research has shown that some individuals are able to protect self-esteem by attributing negative comments and views to causes other than obesity. When able to do so, many individuals will blame other types of discriminatory conduct such as gender, race, or religious prejudice. In such instances, the individual feels less to blame for the prejudice, and places the responsibility for the negative attitudes squarely on the shoulders of the biased person.

In other instances, however, overweight and obese individuals are not able to find external avenues of attribution for the negative behavior or comments, and are forced either directly or indirectly, correctly or incorrectly, to conclude that the negative reaction is based on the individual's weight. In these cases, most individuals then place the responsibility for the negative views on their own shoulders because they do not comport with the prevailing thin body ideal. The negative views are internalized and self-esteem is damaged.

In a study that involved the effects of rejection of obese women by a male in a dating situation, the women in the study reasoned that when rejected by the man who was aware of their weight, that the weight was the major cause of the rejection. The rationalization was fully legitimate to these women. They felt that being overweight or obese made them less attractive and therefore less deserving of male attention. The reasoning extended further to reach the conclusion that their unattractiveness was a legitimate reason for the rejection. Their self-esteem issues both caused the perceptions and suffered from them.

The self-esteem of overweight and obese individuals has been adversely affected by the societal view that weight is a factor totally within the control of the individual, whereas gender or race is not. The stereotype that obese individuals are lazy, self-indulgent, and lacking self-discipline has created an atmosphere in which obese individuals are accountable for their weight and for the negative outcomes and consequences that result from their weight situation. Rejection, then, is seen as justifiable and not prejudicial. Even the law, a reflection of society's beliefs and values, supports this attitude, with only one state offering protection from weight discrimination.

Society's beliefs and values regarding thinness and beauty create externally contingent self-worth mechanisms. People differ in what they believe they must do or be to have value or worth as a person. People may base their self-esteem on a wide variety of things, including their appearance and acceptability to society based on appearance. Research has shown that those who base their self-esteem on external factors such as social acceptability and cultural compliance, are more at risk for negative effects from stigmatization. This same research showed that women who base their self-worth on their appearance have lower self-esteem when they feel they do not look good, and exhibit more symptoms of depression disordered eating.

Conversely, men who based their self-worth on appearance showed significantly fewer psychological issues than women who based their self-esteem and self-worth on the same factor. The cultural ideal for men is based less on physical appearance than on social interaction, success, and other factors. Women in this study rated themselves as being far from the cultural ideal set for a woman, yet men rated themselves as meeting or exceeding the cultural ideal for men. In a subsequent study, these same researchers found that the more women based their self-esteem on appearance, the higher they scored on body shame, body dissatisfaction, or strive for thinness.

An additional factor identified in the research is "all or nothing" thinking, or the "ego gap." Anyone who has worked with the overweight and obese can relate stories of patients stating, "I was born this way, this is not my fault, and others are prejudiced and wrong," or, "I am fat and worthless, therefore I deserve whatever rotten things happen to me." It is an "all or nothing" approach to thinking. Either there is no personal responsibility or there is total personal responsibility. Information that is inconsistent with the self-perception is either denied or it is internalized to a crippling degree.

The limits of this binary thinking present costs and damages for the individual in any circumstance. Feelings of worthlessness and lack of value as a human being create terrible psychological costs for the individual. Relationships, goals, dreams, vocation, and other aspects of the individual's life are also adversely affected. In fact, the ripple effect extends to affect the lives and self-esteem of others as well. The costs to the individual and others become exponential.

People may base their self-esteem on a wide variety of things, including their appearance and acceptability to society based on appearance.

Those individuals who blame others rather than him-/herself also create personal and public costs. Self-esteem may be protected, but the blame and accusation make others feel defensive and reactive. Conflict, separation, and isolation can result, detrimentally affecting both the individual and others in a variety of ways. Either form of "all or nothing" thinking causes disconnection, a blurring of reality, ineffective coping, an inability to deal with the real issues, and an inability to accept the faulted and flawed nature of humanity. In many instances, "all or nothing" thinking can result in the development of pseudo self-esteem as a societal coping mechanism.

Pseudo-self-esteem is the illusion of self-efficacy (belief in one's ability to perform in a specific situation), and self-respect without the reality. It is a protective device to reduce anxiety and provide a false sense of security. It allows one to feel better about oneself while avoiding dealing with the real causes of the low self-esteem.

Instead of seeking good self-esteem through consciousness, responsibility, and integrity, individuals may falsely seek it through popularity, prestige, material acquisitions, or sexual exploits. Instead of valuing personal authenticity, they may value belonging to the

right clubs or keeping up with the Joneses. Instead of meeting their own needs and thinking independently, they may simply practice blind loyalty to a particular group or system. They may try and make themselves feel like good people by rationalizing that they must be good people because they do good things (philanthropy). Instead of being in personal control and having personal confidence, they may seek the power of manipulating or controlling others.

The key to healthy self-esteem is becoming aware of one's personal strengths and accepting oneself as a worthy person despite any real weaknesses that may exist. This also means learning that low self-esteem is partly self-imposed, regardless of original causes. A person maintains his or her own low self-esteem by misperceiving his or her basic worth relative to others. This is because the more he or she criticizes himself or herself, the worse he or she feels. Finding fault with everything around an individual is no more helpful than finding constant fault within, except in providing a bit of immediate relief. The good news is that individuals can change how they *view* themselves—without changing their basic personalities. They can also spend more time doing useful things and less time sitting around thinking about themselves. This builds self-esteem.

Talking to other people can help the obese or overweight individuals see that they are not the only ones with problems. This means focusing on others, not dwelling on oneself, a good diversion. Support groups are an excellent forum for this type of activity. Everyone has something they do not like about themselves or that they feel bad about no matter how hard it may be to see it in them. So, there is nothing unique about obese or overweight individuals in this regard; hence, they have every right to be as happy as anyone else.

Helping others and being a good listener is one way to develop a sense of being good at something and a greater sense of self-worth. Listening and being nice to others is one of the easiest things to change about oneself, something everyone can do, and it is one of the best ways to starting to feel better about oneself. The key point is to do things that get in the way of dwelling on the self. Remaining busy gets one out of "self" mode, especially if what one is doing gives one a sense of achievement.

Each of us is the ultimate creator of our own self-concept. People with good self-concepts are happier and more likable, have better relationships, and are more productive. Real self-esteem, a major component of self-concept is based on effectively dealing with issues faced by the individual, personal achievement, and contributions to relationships, causes, and the world. The solution to low self-esteem issues may be to concentrate on something larger than the self. "Larger" in this context has nothing to do with weight.

SEE ALSO: Anxiety; Appearance; Body Image; Depression; Fat Acceptance; Loneliness; Obesity and the Media; Quality of Life; Self-Esteem in Obese Women; Stereotypes and Obesity; Weight Discrimination.

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Self-Esteem in Obese Women

IN A SOCIETY in which appearance is valued and being thin is glorified, women experience a great deal of pressure regarding their appearance, shape, and weight. Beginning in adolescence, women have been found to be more concerned with their body weight and appearance than men, and researchers have found that women are more concerned with eating, weight, ap-

pearance, and have lower appearance self-esteem than men at all ages. Many posit that this pressure must be felt to a greater extent by women who are overweight, and it has been suggested that the self-esteem of overweight and especially obese women suffer accordingly. The widespread stigmatization experienced by obese individuals in general and obese women in particular further corroborate the assumption that being obese takes a toll on the self-concept of women. However, findings on the levels of self-esteem in obese women are mixed, and research suggests that the relationship between obesity and self-esteem in women is more complicated than many assume.

It appears that from an early age, overweight and obese women are subjected to increased degradation and stigmatization, and some suggest that these early experiences may lay the groundwork for lower levels of self-esteem later in life. A study conducted by Neumark-Sztainer and colleagues found that in a population of overweight adolescent girls, the majority reported offensive comments and differential treatment/rejection as a result of being overweight.

Such comments included weight-related teasing, joking, and name-calling which most girls reported began in elementary school and continued into middle school. These comments were reported to be made by family members, peers, and strangers and occurred most commonly at school and home. Teasing has been found to predict low body satisfaction, increased depressive symptoms, suicidal ideation and attempts, and low self-esteem in both boys and girls. Girls, however, who researchers believe experience greater pressure in regard to appearance, have been found to express the most distress from weight and appearance related teasing.

In addition, a study conducted by Pearce et al. found that compared to normal-weight girls, obese girls report more experiences of relational victimization, which is victimization using interpersonal connections which often results in social marginalization such as exclusion from social events. These findings suggest that obese girls may be subjected both to overt victimization, such as teasing, and to more subtle victimization, such as exclusion, both of which can be damaging to the self-concept and self-esteem of an adolescent.

A research study on adult obese women found that women who reported greater frequency of being teased about their weight and shape in their youth had a more negative evaluation of their appearance and a greater degree of body dissatisfaction as an adult. Interestingly, this study found that low self-esteem was related to poor body image but not to teasing history. Specifically, women with higher levels of self-esteem reported more positive evaluation of their appearance, and women with lower levels of self-esteem had higher levels of body dissatisfaction. This study also reported that women who had been obese since childhood or adolescence reported more childhood teasing about their appearance and weight/size, greater adulthood body dissatisfaction, and lower self-esteem than women who became obese as adults.

While the relationship between self-esteem and obesity in women is complicated, this research suggests that early-onset obesity, and the social costs that accompany it, place obese women at an increased risk of developing negative body image which may have a significant impact on self-esteem later in life. This finding can be connected to research on the harmful effects of childhood stigmatization and appearance and weight-related teasing, and it suggests that at a very early age, women are often evaluated by their weight and appearance.

The overall effect of weight and appearance on women's self-image and self-evaluation has been demonstrated in a study conducted by Sarwer and colleagues on obese and nonobese women. This study found that obese women were more dissatisfied with their body and overall appearance than nonobese women. In addition, compared to nonobese women, more obese women reported thinking about and being upset by their appearance as well as trying to conceal their appearance more than half of the days in the past month. Obese women also reported more intense embarrassment in social situations compared to nonobese women. Interestingly, the obese and normal-weight women did not differ in regard to measured levels of self-esteem. However, among obese women, high levels of body dissatisfaction were associated with more depressive symptoms and lower levels of self-esteem.

A similar but less significant relationship between high levels of body dissatisfaction, more depressive symptoms, and lower levels of self-esteem was also found among nonobese women; this highlights the appearance and weight related pressure felt by women in general, not just women who are overweight. Additionally, body image dissatisfaction did not vary based

on the subject's height-to-weight ratio (body mass index [BMI]) in both the obese and nonobese women, suggesting that body image dissatisfaction may not be directly related to one's weight. Thus, according to these findings, being overweight may not be the single determiner of an individual's level of body dissatisfaction, a measure that some have identified as having associations with levels of self-esteem.

Stigmatization against obese individuals, especially obese women, has been widely documented in many settings. In addition, research has found that obese individuals often internalize this anti-fat bias. This was demonstrated in a study by Latner et al. in which obese and nonobese individuals, when shown pictures of individuals with various physical disabilities and asked to rank them in order of preference, gave the obese person the second lowest rank regardless of their own weight. Because obese women are stigmatized to an even greater extent than obese men, some suggested that women might also internalize this stigmatization to a greater extent and hypothesized about the adverse effects this might have on their self-esteem.

A study by Puhl et al. found that 30 percent of overweight women reported that stereotypes about overweight and obese people were sometimes true and 7 percent reported that they were true. Interestingly, individuals who believed stereotypes to be true, sometimes true, or false had no differences in measures of self-esteem, depression, or diagnosis of binge-eating disorder. These findings emphasize the complexity of the relationship between obesity and self-esteem in women and caution against making assumptions based on findings from other research.

In a study conducted by Crocker et al., normal-weight and overweight college women were given feedback on whether an attractive man, who they knew was aware of their height and weight, was interested in dating them. Crocker and colleagues conducted an initial assessment of self-esteem prior to study participation and found that overweight women had lower levels of self-esteem specifically related to their appearance than women of normal weight. In addition, results showed that overweight women who were told the attractive man was not interested in dating them tended to attribute this response to their weight. However, these women did not report that the man was discriminating against them because of their weight. This suggests that while these overweight women blamed the overweight stigma for this negative judgment, they did not blame external circumstances such as prejudice for this outcome.

This study also found that overweight women who were rejected reported increased negative emotion, hostility, and depression and decreased appearance self-esteem compared to their normal-weight counterparts. In addition, overweight women reported more negative emotions the more they blamed their weight for their rejection. This study found differences among obese and normal-weight women in levels of appearance self-esteem but not overall self-esteem; however, these researchers suggested that similar negative social interactions may recur throughout an obese woman's life and, in this way, take a toll on her self-esteem over time.

A study conducted by Gidi Rubinstein on personality traits in overweight women found interesting results by dividing this group into nondieters and dieters. This study reported no differences in self-esteem or personality traits, defined as "the big five" personality traits (i.e., neuroticism, extroversion, openness, agreeableness, and conscientiousness) between overweight dieting women and women of normal weight. However, overweight nondieting women were found to be more neurotic and less open, agreeable, conscientious, and extroverted as well as to have lower self-esteem than both the normal-weight women and the dieting overweight women.

Thus, according to these results, being involved in a diet group is associated with higher levels of self-esteem. This difference could potentially be explained by the experience of being a part of a group in general or by the motivation necessary on an individual level to join such a group. This study also found that the more conscientious, agreeable, and open a woman is, the more satisfied she is with her weight.

Additionally, more than half of all the women in this study endorsed having a slim ideal of feminine beauty, one-third endorsed having a normal-weight ideal, and only one individual reported that her ideal is an overweight woman despite the fact that two-thirds of the subjects were overweight. This finding reinforces the pervading thin cultural standard of beauty which most likely has a strong impact on the self-concept of overweight women regardless of personality type. While further research needs to be done on this area, these findings provide an intriguing potential explanation for the discrepancy in results of research on self-esteem in overweight women.

Although research on self-esteem in obese women does not report consistent findings, studies have identified certain behaviors and beliefs, specifically binge eating and body image dissatisfaction, which tend to exist in obese women who have low self-esteem. Body image dissatisfaction, or body dislike and disparagement, has been found to be more common in obese as compared to nonobese individuals. Within obese populations, body image dissatisfaction has also been found to be more common among women.

In addition, research has identified a relationship between self-esteem and body image dissatisfaction in overweight populations. A study conducted by Matz and colleagues on overweight and obese women seeking weight-loss treatment found an association between lower self-esteem and body image dissatisfaction. However, this study did not find as association between BMI and body image dissatisfaction. Thus, self-esteem is an important factor to examine because research suggests that it may be able to explain the variety of results found regarding the psychosocial effects obesity had been found to have on the individual and specifically on women.

Current research has not come to a consensus regarding whether self-esteem differs in obese women as compared to normal-weight women and/or obese men. However, some research suggests that self-esteem in obese women may still be an important factor to study because it may be indicative of specific psychosocial effects of obesity.

SEE ALSO: Self-Esteem and Children's Weight; Self-Esteem and Obesity; Obese Women and Social Stigmatization.

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Sensory-Specific Satiety

SENSORY-SPECIFIC SATIETY (SSS) occurs when a particular food is eaten to the point where there is a decrease in satiety and pleasure. Satiety takes place when hunger is satisfied and a pleasant feeling of fullness is present. Satiety is a signal to stop eating as flavor fatigue manifests and the senses are no longer aroused to the stimulus of a particular food.

SSS is subjective and does not appear to be dependent upon the type of food or its macronutrient composition (carbohydrate, protein, fat). Some foods may be more satiating than others, depending on an individual's preferences. SSS is not characteristic to a particular age group or gender. As SSS is subjective, it is unique to the individual's trigger as to when the hedonistic quality of taste diminishes. The sensory sensation of food involves its taste, smell, texture, and appearance. These factors may contribute to a food's overall appeal. The sensory stimulus may increase intake as the desire to experience various sensations increases. The first bite of a food may be the best as the taste buds are excited in anticipation of pleasure. The taste of a food may decline after subsequent bites.

Internal cues such as hunger are appropriate and are a signal to nourish the body. External cues such as surroundings, food availability, time of day, and sight of food may increase and become inappropriate when determining food consumption due to eating for the pleasure instead of the necessity for nourishment. An appetizing smell of food may entice a person as an external cue but that same odor for five minutes will decrease the pleasantness of a food. SSS may occur within two minutes of eating one food, thus creating a change in palatability which may lead to switching to a different food. The two-minute time limitation has little to do with digestion and absorption and is therefore specific to SSS.

A buffet may excite all senses, creating a desire to experience the pleasure of consuming a variety of food. It is theorized that sensory perceptions may promote overeating, thus playing a role in obesity. Research has shown an increase in consumption when a variety of food is presented. It is unclear about the actual amount needed to be ingested for SSS to occur. Other external cues may be present, leading SSS to be prolonged or ignored, but the pleasantness of taste appears to be a regulating component. Foods of similar tastes and textures may promote SSS to occur sooner, thereby decreasing consumption and calorie intake. It is theorized that foods with a weak taste may produce weaker SSS, thereby increasing intake to reach satiety. Studies have theorized that after a sweet meal, the desire for more sweet declines. The same has been demonstrated for a savory meal. This may be the determinant to switch to another food.

SSS may play a role in food choices and eating cessation although there have been no conclusive human studies regarding a possible connection to obesity and SSS. There is no evidence that there is a difference between a nonobese and obese individual in regard to SSS. There may be a hunger component as more food may be required to satisfy an obese person. A diet high in modified foods and variety may have an influence on obesity.

Research is ongoing as to a possible connection between SSS and obesity. There are many factors beyond SSS that may contribute to overweight and obesity.

SEE ALSO: Appetite Signals; Flavor: Taste and Smell; Olfactory System; Variety of Foods and Obesity.

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Serotonergic Medications

SEROTONIN, OR 5-Hyrdoxytryptamine, 5-HT, is a neurotransmitter synthesized by serotonergic neurons in the central nervous system (brain). It is believed to play an important role in a number of biological processes, including body temperature regulation, mood, aggression, sexuality, and appetite. It is sometimes referred to as the "food and mood" chemical. Low levels of serotonin are symptomatic of a number of psychological conditions, including depression and anxiety disorders. Several obesity medications work by increasing levels of serotonin, as this been shown to reduce appetite and food intake. Several classes of serotonergic medications are available for different indications. Selective serotonin reuptake inhibitors are widely used for the treatment of depression. Serotonin 5HT1b/1d agonists are used for the treatment of migraine. Serotonin reuptake inhibitors with additional activities (fenfluramine and sibutramine) are effective for weight loss.

Fenfluramine is a mixture of two chiral isomers, while dexfenfluramine is one of the two isomers. Fenfluramine and dexfenfluramine are the first serotonergic agents used for weight loss. Fenfluramine and its metabolite norfenfluramine have multiple biological activities, including serotonin transporter inhibitor, 5HT releaser, and agonist at several serotonin receptors. Fenfluramine has also been used in combination with phentermine in an off-label fashion. Although fenfluramine is an effective weight loss agent, its long-term use is associated with the development of valvular heart disease. This serious side effect led to the withdrawal of both fenfluramine and dexfenfluramine worldwide in 1997.

Fluoxetine is the first selective serotonin reuptake inhibitor (SSRI) and it is indicated for the treatment of depression. Early clinical studies indicated that short-term use of fluoxetine or other SSRIs is associated with modest weight loss. However, long-term studies showed that the weight loss is transient, and in some cases, long-term weight gain has been observed.

Sibutramine is the only serotonergic agent currently approved for the treatment of obesity. It is a serotonin and norepinephrine transporter inhibitor, along with weak activity at the dopamine transporter. Sibutramine reduces appetite and body weight. Weight loss has been demonstrated in clinical trials lasting two years. Unlike fenfluramine, sibutramine has not been associated with valvular heart disease. While sibutramine at the therapeutic doses is well tolerated, increase in blood pressure has been observed in some patients. Regular monitoring of blood pressure is required when prescribing sibutramine.

SEE ALSO: G-Protein Coupled Receptor; Sibutramine (Meridia).

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Set or Settling Point

SETPOINT IS A term derived from engineering, used to describe a switching point between systems designed to increase or decrease the level of a controlled variable. One example is the setpoint for water level in a reservoir. If the water level rises above the setpoint, an increasing amount of water will be discharged into the outflow. The inflow of water from points upstream might also be reduced. If the water level falls below the optimum level, then the outflow will be reduced in the inflow increased. A setpoint mechanism does not imply full control over a variable, but only a regulated ongoing influence.

Body weight setpoint is thought to operate in a similar manner, with body fat stores as the controlled variable. In the past, the theory of a body weight setpoint was controversial. Some authors misinterpreted the term "setpoint" to mean that body weight was unchangeable, which is clearly not the case. The primary critique of setpoint theory was it required a signal to pass from the adipose tissue to the hypothalamus, and no such signal was known.

This criticism vanished abruptly with the discovery of the hormone leptin. Leptin levels are directly proportion to fat mass, and sufficient levels of leptin trigger decreased input and increased output of stored energy. Falls in leptin levels trigger the opposite effects. Leptin acts as a brake on changes in body fat stores in both the upward and downward direction, precisely the behavior expected of a setpoint process.

Increases in body fat stores trigger several changes that oppose the further accumulation of lipids. (1) Fat storage is an energy requiring process, so some excess calories are consumed transporting and esterifying fatty acids into adipocyte triglyceride droplets. (2) The synthesis of lipids from excess carbohydrates and proteins requires considerable energy, as does the synthesis of the glycerol backbone. (3) Increases in body weight raise the energetic cost of physical activity. (4) Adipose tissue requires a small amount of energy for maintenance, although far less than muscle or other active tissues. (5) Mainly through the influence of leptin, increases in active thyroid hormone and catecholamines raise metabolic rate and increase expression of uncoupling proteins, which increase the generation of heat in competition with energy storage. (6) Most importantly, appetite is suppressed through leptin and other circulating mediators.

Decreases in fat stores trigger the opposite changes. Basal metabolic rate can decline significantly during severe caloric restriction, particularly if there has been loss of lean metabolically active tissue in addition to body fat loss. In countless studies in humans and animals, weight loss has been shown to decelerate over time, with rapid initial losses of body weight slowing to a lower long term rate. Part of the rapid phase is due to fluid loss, especially the loss of water associated with glycogen, but a major portion of the decline in weight loss over time reflects body weight setpoint mechanisms.

SEE ALSO: Assessment of Obesity and Health Risks; Weight Cycling and Yo-Yo Dieting.

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Sexual Abuse and Eating Disorders

WHILE SOME RESEARCH suggests a link between having a history of sexual abuse and developing an eating disorder, the nature and strength of this association is not yet known. For example, some believe sexual

abuse is a risk factor of eating disorders, while others assert that sexual abuse is a risk factor of general psychopathology. The role that sexual abuse plays in the development of eating disorders is complicated and a casual link cannot be established; however, various results of research on this topic are discussed.

The prevalence of reported sexual abuse in individuals with eating disorders varies widely. Sexual abuse is a difficult subject of study because traumatic experiences such as sexual abuse are often unreported. According to a review of the literature on sexual abuse and eating disorders conducted by Connors and Morse, roughly 30 percent of eating disorder patients report having experienced sexual abuse as a child. However, conclusions regarding the relationship between sexual abuse and eating disorders are difficult to determine because of the variability in findings of the prevalence of such a history in noneating disorder populations.

Some research reports similar findings of sexual abuse in the overall population compared to that found by Connors and Morse, while other research reports smaller findings. Preti and colleagues took an interesting approach to researching this topic and examined the sexual abuse history and eating disorder symptoms in a community sample of women aged 18–30. They found that the 20 percent of women who reported having experienced sexual abuse were also more likely to report symptoms of eating disorders compared to those who did not endorse experiencing such abuse. They also found that women who were sexually abused before age 12 were more likely to endorse eating disorder symptoms. While this specific study looked at eating disorder symptoms as opposed to eating disorder diagnosis, these findings are valuable considering the relationship between disordered eating habits and the development of an eating disorder.

Recent research suggests a relationship between purging behaviors, such as vomiting, and a history of sexual abuse. Specifically, Gerko et al. conducted a study on women diagnosed with restrictive subtype anorexia nervosa, bulimia nervosa, and binge-eating disorder and found that overall almost 30 percent of these women reported a history of childhood sexual abuse. History of sexual abuse, however, varied based on eating disorder diagnosis. Specifically, out of the women who did not engage in purging behaviors, that is, those who were diagnosed with restricting type anorexia or binge-eating disorder, about 17 percent reported childhood sexual abuse. However, 34 percent of women who engaged in any purging behavior, which included vomiting and laxative and diuretic use, endorsed suffering sexual abuse as a child.

Treuer et al. reported similar findings in their study of abuse in individuals diagnosed with anorexia or bulimia. They found that 29 percent of individuals diagnosed with anorexia or bulimia had a history of sexual abuse, with the most individuals having a diagnosis of anorexia, binge/purge subtype (38 percent). Interestingly, in contrast with Gerko's findings, Treuer and colleagues reported higher rates of sexual abuse in individuals with anorexia, restrictive subtype compared to individuals diagnosed with bulimia. Carter et al. found that 48 percent of individuals diagnosed with anorexia reported a history of childhood sexual abuse. Of these individuals, 65 percent were diagnosed with binge/purge subtype anorexia, whereas only 37 percent were diagnosed with restricting subtype anorexia.

These studies suggest that childhood sexual abuse may be related to bulimic symptoms (i.e., purging behavior*);* it is possible that sexual abuse may guide eating disorder symptoms, which can originate from a variety of causes, toward bulimic behaviors, as opposed to being the direct cause of such behaviors.

Some research proposes impulsivity as an explanation for the relationship between sexual abuse and eating disorder pathology, specifically bulimic behaviors. Impulsive behaviors, which include bulimic behaviors, alcohol or drug abuse, suicide attempts, self-mutilation, and sexual disinhibition, can be classified as acts that are associated with overwhelming urges to commit the behavior, mounting tension upon attempts to resist engaging in the behavior, and relief after performing/engaging in the behavior. Some believe that these impulsive behaviors help the individual escape awareness, and numerous studies have identified a connection between trauma and impulsive behaviors.

Corstorphine and colleagues examined the trauma history and impulsive behaviors of individuals diagnosed with an eating disorder and found that sexual abuse was reported by about 38 percent of individuals with eating disorders. The highest proportion of these individuals was diagnosed with anorexia binge/purge subtype and the lowest proportion was diagnosed with anorexia restrictive subtype. Reported sexual abuse was also associated with multiimpulsivity which was defined as bulimic behaviors and at least two other impulsive

behaviors as defined above. More research needs to be done to determine whether bulimic behaviors are a part of a class of impulsive responses to traumatic events.

While research on the relationship between sexual abuse and eating disorders tends to focus less on binge eating disorder, possibly because it is not yet classified as an eating disorder in the *Diagnostic and Statistical Manual of Mental Disorders*, studies on the sexual abuse history of obese individuals have revealed interesting findings. A study conducted by Fairburn et al. found that sexual abuse was reported in about 29 percent of women diagnosed with binge-eating disorder, 35 percent of women diagnosed with bulimia, and 26 percent of psychiatric controls (individuals diagnosed with a psychiatric disorder aside from an eating disorder).

These findings all differed significantly from the 11 percent of healthy, nonobese controls who endorsed a history of sexual abuse. However, Grilo and colleagues found that 32 percent of very obese male and female candidates for bariatric surgery reported experiencing sexual abuse but did not uncover any differences between individuals with and without binge-eating disorder. Further research needs to be conducted on the prevalence of sexual abuse in individuals with binge-eating disorder.

SEE ALSO: Anorexia Nervosa; Binge Eating; Bulimia Nervosa.

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Sexual Health

OBESITY CAN IMPACT a person's sexual health as early as puberty. There have been strong correlations between the timing of sexual maturation and a person's body weight, body mass index (BMI), and skin fold thickness. The most common sexual health complaint among obese women is polycystic ovary syndrome (PCOS). The severity of the syndrome and the severity of obesity are often directly correlated. Obesity also impacts pregnancy and infertility. Obesity also has an impact of sexual function and dysfunction with obese men reporting more cases of erectile dysfunction (ED) than nonobese men. A major comorbidity that is listed along with obesity is cancer, especially cancers of the sexual organs.

Girls who experience early sexual maturation are twice as likely to be overweight and obese than girls who experience sexual maturation at the average age. However, the opposite was found in boys. Boys who reach sexual maturation early are less likely to be obese. The evidence that links fatness and maturation in girls does not confirm that obesity causes early sexual maturation. There are several theories that propose

Hormones that affect a woman's ovulation cycle, such as estrogen, tend to be higher in obese females.

a mechanism of interaction. One of the earlier theories looks at the idea of allocation of energy. In women especially, reproduction is very energy expensive. As such, a woman will not reach sexual maturation until her body stores enough energy to maintain a pregnancy. Because overweight and obese females have more stored energy, they reach menarche sooner.

Another theory examines the close relationship between adipose tissue and hormones. Hormones that affect a woman's ovulation cycle, such as estrogen, tend to be higher in obese females. As such, these females experience premature menarche. The relationship may also be bidirectional. Women often experience weight gain once they have reached sexual maturation. As such, an individual may not be obese until after reaching sexual maturation; however, the timing of events may not be well known. There are few theories regarding males and sexual maturation.

Obesity is also strongly correlated to PCOS. The most common complaints associated with PCOS are hyperandrogenism and menstrual irregularities. It is also a leading cause of infertility and will affect 4 to 7 percent of women. The severity of PCOS is proportionate to the severity of obesity. Increased abdominal adiposity is more strongly related to PCOS. The relation between PCOS and obesity is thought to be a result of poor regulation of hormones. An increase in adipose tissue causes changes in insulin, estrogen, iodothyrins, and growth hormone, which all regulate sex hormone-binding protein (SHBG). Significantly lower SHBG plasma levels and worse hyperandrogemia are a main characteristic of obese women with PCOS.

Women with PCOS are characterized by a high prevalence of several metabolic abnormalities that are strongly influenced by the presence of obesity. Weight loss has been reported to be increasingly difficult among women with PCOS. However, weight loss induces significant benefits on hyperandrogenism and the severity of PCOS. Weight loss is a simple, cost-effective, and safe way of treating the symptoms and is highly recommended for women with PCOS.

Obesity impacts pregnancy and infertility. There is a significant amount of evidence concluding that obesity is a major risk factor in adverse pregnancy outcomes, including infertility and miscarriages. Obese women have reported increase difficulty conceiving. This may be a result of the disease burden or hormone irregularities.

In addition, obese women and those with metabolic syndrome are more likely to be diagnosed with gestational diabetes, which can lead to poor pregnancy outcomes. It is important for women with gestational diabetes to seek medical attention and treat the condition. In addition, overweight and obese women are at higher risk for preeclampsia, gestational hypertension, preterm birth, cesarean section, and macrosomia. However, obese women should still follow the recommended weight gain to avoid further complications. A high BMI has also been correlated to low semen quality in men and increased difficulty in inseminating their partner.

Obesity plays a role in many aspects of sexual health. In men, there is a strong correlation between obesity and ED. ED is defined as three months of consistent, recurrent inability of a man to attain and/or maintain penile erection sufficient for sexual activity. It can be either organic or psychogenic. While the causes of this are not known, it is thought that the effects obesity has on ED may be a result of the increase in vascular risk factors. While obesity itself does not cause sexual dysfunction, the impact that it has on the heart and vascular system will result in ED.

Female sexual dysfunction, FSD, is defined as a persistent or recurrent disorder of sexual interest, desire, disorders of subjective and genital arousal, orgasmic disorders, pain, and difficulty with attempted or incomplete sex. There is little evidence that there is an association between obesity and sexual dysfunction in women. In cases where it occurs, it is more likely to be psychogenic than organic. Many obese women report poor body image which can result in FSD.

Cancer is often listed as a comorbidity of obesity. It appears that the majority of cancers associated with obesity are hormone-mediated cancers that affect reproductive organs. This includes breast, prostrate, and endometrial cancers.

SEE ALSO: Hormones; Obesity and Cancer.

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Shape Up America!

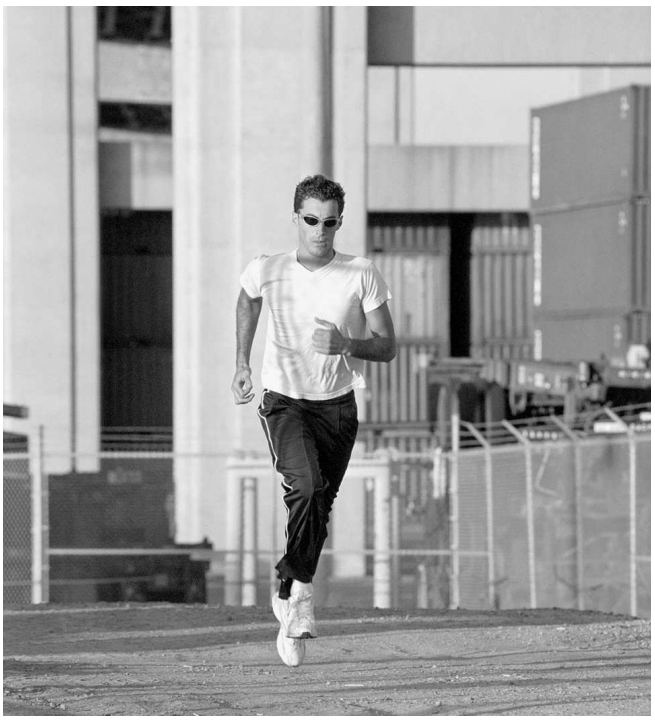
SHAPE UP AMERICA! is a national educational initiative established in 1994, by former Surgeon General C. Everett Koop, MD, to fight obesity. This 501(c) nonprofit corporation views obesity as a major public health problem. Shape Up America! is determined, according to the mission statement, to increase the public's awareness of the importance of achieving and maintaining healthy body weight through increased physical activity and healthy eating.

Shape Up America! has four main educational objectives.

- The first goal involves increasing the American public's understanding of healthy weight loss, maintaining the weight loss, and the importance of eating healthfully and increasing physical activity.
- The second goal involves teaching the public healthy ways to reach an ideal body weight and how to avoid weight gain.
- The third goal involves stimulating continued cooperation among national community organizations to reinforce the Shape Up America! mission.
- The fourth goal involves inspiring healthcare professionals to take the role of first-line combatants fighting obesity through education and treatment.

Shape Up America! has produced two publications that have been well-received by the public. The first was published for physicians in October 1996, entitled *Guidance for the Treatment of Adult Obesity*, a reference book to help physicians identify and treat adult obesity. The second was produced for the public in September 1997, entitled *Healthy Weight, Healthy Living*, which discusses the risks of obesity and encourages partnership with physicians.

Shape Up America! developed an interactive Web site in 1996. The site, www.shapeup.org, is an important vehicle for the promotion of healthy living. The Web site offers access to several "centers" that aid in achieving optimal weight and fitness goals—the BMI center calculates body mass index; the Cyberkitchen assists with balancing food intake with activity level; and recipes and shopping lists are popular features. The Fitness Center assess current level of fitness, and the Support Center offers information and encouragement to overcome



Shape Up America! works to increase public awareness of achieving and maintaining healthy body weight through physical activity.

weight-loss obstacles. The Web site also offers treatment guidelines for professionals, dietary and activity tips for consumers, and press releases for the media. The Web site regularly features a newsletter that discusses recent research and other relevant topics. (Newsletter archives are available.)

The Shape Up America! Coalition is comprised of a group of 46 nonprofit organizations that share a goal of educating the American public. The coalition members strive to help improve the health status of Americans by alerting them to the Shape Up America! principles and activities. In 1996, Take Off Pounds Sensibly (TOPS), a coalition member, sponsored a national walking campaign. In addition, the coalition has produced educational pamphlets on healthy diets and exercise.

Shape Up America! is a unique approach to the public health issue of obesity. The Web site successfully attracts scores of visitors on a monthly basis, and offers a treasure trove of educational information and motivational tips that reflect the credibility of the organization.

SEE ALSO: Exercise; President's Council on Physical Fitness and Sports.

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Sibutramine (Meridia)

WHILE BEHAVIORAL INTERVENTIONS demonstrate successful weight loss in the short term, the effects are difficult to maintain. Pharmacological treatment has yielded positive results in weight loss and weight loss maintenance. Sibutramine, a beta-phenethylamine that inhibits the reuptake of norepinephrine, serotonin, and dopamine to a very limited degree, was approved by the U.S. Food and Drug Administration (FDA) for long-term weight loss for patients 16 years and older.

Substantial evidence based on clinical trials lasting six months up to 24 months has shown that doses of 10 to 15 mg/d (i.e., the recommended dose) produced average weight losses of 8 to 10 percent of initial weight, as compared to reductions of only 1 to 2 percent for placebo-treated patients. Clinical trials have documented these losses in the first six months and showed that they were maintained at 12 months if patients remained on medication.

One of the most notable clinical trials, Sibutramine Trial of Obesity Reduction and Maintenance (STORM), was conducted by W. P. James and colleagues. This multisite randomized trial investigated the efficacy of sibutramine in adults over an 18-month period. Findings from this study revealed that 77 percent of the patients treated with sibutramine lost weight, while 43 percent of the individuals in the sibutramine group maintained at least 80 percent of their weight loss compared to 16 percent of those in the placebo group at the end of 18 months.

BEHAVIORAL AND PHARMACOLOGICAL TREATMENT COMBINED

More recently, research supports a combination of behavioral and pharmacological treatment in achieving weight loss. Thomas Wadden and colleagues examined this among adult women who were randomized to one of three groups (drug alone, drug plus lifestyle modification, or combined drug, lifestyle modification, and portion controlled diet). The researchers found significantly greater reductions in weight in the drug plus lifestyle modification group as well as in the combined group at 12 months.

Robert Berkowitz and colleagues examined the combination of pharmacotherapy and behavioral intervention among adolescents in a single-center, six-month controlled trial combining sibutramine with a comprehensive behavioral program that induced a statistically significant weight loss in obese adolescents compared to a behavioral program alone. Following month six, all subjects were treated with sibutramine plus a behavioral program for an additional six months. At month 12, those adolescents originally treated with placebo in the initial six months lost further weight and those initially treated with sibutramine for six months maintained their weight loss at month 12. Adolescents in both groups lost similar amounts of weight by month 12,

suggesting that sibutramine may be used following six months of behavioral treatment and that sibutramine may also induce a stable weight loss following 12 months of treatment.

Two more studies were recently released supporting the use of sibutramine in the induction of weight loss for adolescents. Amelio Godoy-Matos and colleagues conducted a randomized, double-blind, placebo-controlled trial in which adolescents were randomized to placebo or sibutramine for six months, along with a hypocaloric diet. Adolescents randomized to the sibutramine group lost 22 pounds on average compared to 5 pounds in the placebo group. The change in mean BMI was also significantly greater among the sibutramine group, replicating the earlier results of Berkowitz.

Berkowitz conducted a one-year multicenter, placebo-controlled, randomized study examining the longer-term effects on weight loss by adding sibutramine to behavior therapy in 498 obese adolescents. This study yielded very promising results for the use of sibutramine in adolescents. Specifically, participants in the treatment group using sibutramine lost an average of 15 pounds compared to the participants in the placebo group who gained an average of 4 pounds. In addition, there were statistically greater improvements in body mass index, waist circumference, high-density lipoprotein cholesterol, triglycerides, insulin, and HOMA in the sibutramine group compared to the placebo group. Of note, there were similar results reported in Caucasian, African-American, and Hispanic adolecents. Leticia Garcia-Morales investigated the use of sibutramine in Mexican adolescents who were obese and found that diet, exercise, and sibutramine (10 milligrams) per day for six months demonstrated weight loss averaging 16 pounds.

SAFETY AND SIDE EFFECTS

Several clinical trials have documented the positive effects on metabolic and cardiovascular risk factors (glucose homeostasis, central adiposity, dyslipidemia). While sibutramine is not associated with abuse potential, it does have documented side effects. These effects include asthenia, constipation, dry mouth, and insomnia. Sibutramine has also been associated with increases in blood pressure and pulse rate in both adults and adolescents compared to placebo. Sibutramine may be contraindicated for patients with a history of cardiac arrhythmias, coronary artery disease, congestive heart failure, or stroke. Prescribing physicians should closely monitor blood pressure and pulse rate in patients using sibutramine.

Two unpublished studies conducted by Abbott (formerly Knoll Pharmaceuticals), which were reviewed by the FDA in approving sibutramine, found that sibutramine was not associated with valvular heart disease. In the larger investigation, 210 patients (mean age of 54) who had been assigned to either medication (15 mg/d) or placebo underwent echocardiography after an average of 7.6 months of treatment. The point prevalence of valvular heart disease in the sibutramine group was 2.3 percent (all were mild aortic insufficiency), compared with 2.6 percent in the placebo group (one case of mild aortic insufficiency, one case of severe aortic insufficiency). In the second study, no cases of valvular heart disease were detected, using pre- and postechocardiograms, in 25 patients who were treated by sibutramine for 12 weeks. No cases of primary pulmonary hypertension (PPH), another problem associated with the fenfluramines, have been reported in the more than 6,000 patients treated by sibutramine.

SEE ALSO: Behavioral Treatment for Child Obesity; Family Therapy in Treatment of Overweight Children; Medical Interventions for Children.

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Sisters Together

SISTERS TOGETHER IS a coalition of community organizations and residents who came together to encourage healthy eating and increased physical

activity among young African American women (aged 18–35) with the goal of helping them to achieve and maintain a healthy weight. It began as a pilot program of the Weight-Control Information Network (WIN), was supported by the National Institute of Diabetes and Digestive and Kidney Diseases, and was one of ten model programs highlighted in the first Surgeon General's Report on Physical Activity.

The original Sisters Together program was based in three Boston communities—Dorchester, Mattapan, and Roxbury—and focused on community partnerships, resource development, and cultivation of self-efficacy. Sisters Together was developed in conjunction with the Boston Obesity/Nutrition Research Center, which included a community nutritionist, media specialist, and educator, as well as representatives from the New England Medical Center, the Harvard School of Public Health, and Tufts University School of Nutrition Science and Policy.

Sisters Together focuses on two related aspects of health: eating better and increasing activity levels. It incorporated information from national research and campaigns and adapted them as appropriate for the specific neighborhoods in question to have impact on the lives of young African American women who are at greatest risk of becoming overweight. For instance, to promote healthy eating, Sisters Together encouraged local restaurants to offer healthier versions of traditional foods (such as collard greens cooked with smoked turkey rather than bacon or ham), offered cooking classes, and developed a calendar that included recipes and tips for healthier eating. To promote greater physical activity, Sisters Together created a walking brochure with tips to increase activity and how to find safe times and locations to exercise outdoors, and descriptions of local walking routes. Sisters Together also created a brochure on hair care and exercise because difficulties with hair care is often cited as a disincentive to exercise by African American women.

The pilot program of Sisters Together took place from 1994 to 1998. Much of the information learned during development and application of that program is presented in its program guide, available for download from the Sisters Together Web site. Part of the intent of publishing this information is to encourage the development of analogous programs for similar populations. The program guide also includes reproducible materials including a fact sheet, news release, radio public service announcements, letterhead, flyers, and forms for group sign-ups and exit interviews.

Sisters Together is a coalition that was created to encourage healthy eating and increased physical activity among young black women

SEE ALSO: African Americans; Community Programs to Prevent Obesity; Department of Health and Human Services; Physical Activity and Obesity; Weight-Control Information Network; Women and Dieting.

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Sleep Apnea

SLEEP APNEA IS the temporary cessation of breathing during sleep longer than an interbreath interval. Nine percent of men and 4 percent of women suffer

from sleep apnea and incidence is increasing. Sleep apnea can be obstructive (OSA), where the upper airway is closed; central (CSA), where no breathing effort is made; or a mixture of both. There are shared genetic risk factors between OSA and obesity. Apnea is also a part of obesity hypoventilation syndrome, characterized by obesity, OSA, and hypersomnolence (daytime sleepiness).

Normally, sleep is associated with pharyngeal narrowing, but factors such as obesity or abnormal anatomy can further narrow or completely obstruct the upper airway. Partial narrowing results in hypoventilation or snoring and complete obstruction results in OSA. Obstruction is most often seen near the soft palate, but multiple sites of closure are common. Arousal results in a dilation of the airway, allowing sufficient breathing. Arousals terminate all OSA and most CSA and are also the cause of hypersomnolence.

Apnea presents as loud snoring and severe sleepiness. Restless sleep can result in night sweats, sore throat, dry mouth, and morning confusion or headache. A prototypical OSA patient is an obese, middle-aged man with a large neck, structurally abnormal airway, and high blood pressure. Obesity adds fat to the tongue and enlarges fat pads near the airway, increasing airway obstruction. Obstruction can also be caused by nasal obstruction, a low hanging soft palate, enlarged tonsils or adenoids, a receding chin, or a tumor. High blood pressure, a large neck circumference, and a history of snoring are clinical predictors of sleep apnea. Collar size is a better predictor of OSA than body mass index.

Apnea is in part determined by an apnea index, which measures the average number of apneas per hour of sleep. An index greater than five may manifest as daytime sleepiness, but an index below 15 is usually asymptomatic. An index over 60 means that a person cannot sleep and breathe simultaneously. Diagnosis is confirmed by measurements and observation during sleep.

Nonsurgical management includes weight loss, prevention of sleeping on one's back, avoiding respiratory depressants such as alcohol, and avoiding respiratory irritants such as smoking. Treatment can include use of positive airway pressure devices to open the airway. Apparatuses that protrude the jaw or hold the tongue forward are especially effective in children. Surgical treatment can involve tracheotomy, a removal of the obstruction if it is a tumor or enlarged gland, and surgery to enlarge the airway diameter.

Characterized by unstable breathing control, CSA is often seen as an increased respiratory drive. This can be caused by heart failure, high altitude, pain, or anxiety and results in initial hyperventilation and low blood carbon dioxide levels.

SEE ALSO: Alcohol; Asthma; Genetic Mapping of Obesity Related Genes; Respiratory Problems; Sleep Duration and Obesity.

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Sleep Duration and Obesity

THE MODERN INDUSTRIALIZED society has been experiencing two parallel trends: increasing body weight and decreasing sleep duration. There is evidence to suggest that these seemingly disparate trends are related. Results from both experimental studies and population-based studies point to a strong connection between short sleep duration, weight gain, and obesity. If results from future well-controlled studies continue to support these connections, then public health interventions that address inadequate sleep or poor sleep quality could be initiated to help treat or prevent obesity.

Researchers from the University of Chicago found in experimental studies that when normal healthy young adults were deprived of sleep, their levels of hormones that function to control appetite were affected. In response to sleep deprivation, levels of leptin, a hormone produced by fat cells, decreased and levels of ghrelin, a hormone produced by stomach cells, increased. Low concentrations of leptin in the body function to increase appetite by indicating to the brain that fat stores are inadequate. Ghrelin

Insufficient sleep could contribute toward obesity by making it more difficult to maintain a healthy lifestyle.

stimulates appetite and levels of this hormone are increased prior to meals and decreased after meals. As would be expected from the changes in these hormones, the appetites of the sleep-deprived subjects increased. The subjects were found to particularly crave sweet and salty snacks. It would be expected that subjects who are awake longer would expend more energy and need to increase their caloric consumption to compensate for the increased energy expenditure, but the experimental protocol called for the extra hours of wakefulness to be spent lying in bed or sitting in a comfortable chair, so the increase in energy expenditure in this case was considered to be negligible.

Chronically short sleep durations could therefore lead to weight gain by increasing appetite and caloric consumption greater than what would be necessary to compensate for the increased energy expenditure from staying awake longer. In other experimental studies at the University of Chicago, sleep deprivation has been shown to decrease glucose tolerance and compromise insulin sensitivity which are potent risk factors for weight gain and obesity.

Insufficient sleep could also contribute toward obesity by making it more difficult to maintain a healthy lifestyle. Not getting enough sleep was shown to be associated with irritability, impatience, pessimism, and feeling tired and stressed in results from the National Sleep Foundation's 2002 Sleep in America poll. These feelings and emotional states would presumably function to lessen one's resolve and willpower to maintain an exercise routine or follow a diet plan.

Epidemiological population-based studies have shown significant relationships between short sleep duration and obesity in children, adolescents, and adults. Stronger relationships have been found in young and middle-aged subjects than in elderly subjects. Possible explanations for the discrepancy between results for younger and older subjects include increased mortality associated with obesity and age-related sleep changes. Individuals suffering from obesity would be less likely to survive into their later years and advanced age is associated with increased difficulties initiating and maintaining sleep.

If sleep deprivation leads to metabolic and behavioral changes that contribute toward weight gain and obesity, then interventions designed to increase the amount and improve the quality of sleep could augment the most common clinical interventions for obesity of increasing physical activity and improving nutrition. These interventions could include helping patients to modify maladaptive sleep habits and educating them about healthier sleep hygiene practices.

SEE ALSO: Ghrelin; Leptin; Sleep Apnea.

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Slim-Fast

SLIM-FAST IS A line of meal-replacement products marketed to people who wish to lose weight. The first Slim-Fast product on the market was the Slim-Fast powdered shake formula, introduced in 1977 by S.

Daniel Abraham. The company Slim-Fast Foods was spun off in 1990 from Abraham's company Thompson Medical, and was bought by Unilever in 2000 for about $2.3 billion. At the time of the sale, the worldwide sales of Slim-Fast products were valued at over $610 million per year. Slim-Fast currently sells food replacement products, including shakes, shake powders, and snack bars under the brand names Slim-Fast, Ultra Slim-Fast, and Optima.

Slim-Fast markets a number of products and promotes several diet plans. The company Web site promotes weight loss as part of a healthy lifestyle, and includes a great deal of standard health behavior advice, such as including physical activity as part of one's daily schedule and avoiding consuming empty calories from soft drinks. It also includes tools that could be useful to any individual wishing to lose weight (without regard to purchasing Slim-Fast products), such as a body mass index (BMI) calculator and an interface to create an exercise program and track one's activity level. This standard information is intermingled with promotion for Slim-Fast products and related materials such as success stories of people who have lost weight using them, and access to some of the health-related materials requires registering with the Web site.

The original Slim-Fast product was the Slim-Fast shake, which was intended to be used as a partial meal-replacement system within a diet plan. The Slim-Fast dieter was instructed to consume a Slim-Fast shake each for breakfast and lunch, and have a low-calorie dinner of conventional food. This type of diet has been shown to produce larger weight losses than diets that allow individuals to select conventional foods whose total calories add up to the same amount. They have also been shown to be effective in maintaining weight loss. The primary explanation for this phenomenon is obese people typically underestimate their caloric intake, sometimes by as much as 50 percent, so that if allowed to choose their own foods, they actually consume far more calories than specified by their diet plan.

Use of meal replacements such as Slim-Fast shakes removes this difficulty by presenting the individual with a fixed portion for each meal. In addition, use of meal replacements removes many of the emotional connections people have with food, and the monotony of consuming the same food every day removes the temptation to consume more than prescribed, so the dieter is not distracted from his or her goal of consuming only a fixed amount.

Slim-Fast has diversified its product line over the years, partly in response to the popularity of low-carbohydrate diets, and currently markets five lines of products. In general, all Slim-Fast products are calorie-controlled meal-replacement products that include fiber and are fortified with micronutrients (vitamins and minerals). Slim-Fast Original products include shakes which contain 220 calories and 10 grams of protein and are fortified with a number of vitamins and minerals; and meal bars of a similar nutritional makeup, and breakfast and lunch bars which are intended to be consumed with 8 ounces of skim milk: the bars themselves are 140 or 150 calories, and when consumed with milk the total becomes 230 or 240 calories.

Slim-Fast Optima Products have less sugar than Slim-Fast Original products and were introduced partly in response to the success of low-carbohydrate diet plans. Optima products are available as shakes, shake powders, snack bars, and meal bars, and this is the largest of the Slim-Fast product lines in terms of the variety of products and flavors available. Optima products are similar in nutritional makeup to Original products, but according to company materials, they are designed to control hunger more effectively than the Slim-Fast Original products.

Slim-Fast High Protein shakes and granola bars contain more protein than their Original counterparts (15 grams of protein in both the shakes and the bars), but are otherwise nutritionally similar. Slim-Fast Easy to Digest shakes are lactose-free and gluten-free, but are otherwise similar to Original products. Slim-Fast Lower Carb products include shakes containing 190 calories, 20 grams of fat, and 2 grams of net carbs, and snack bars containing 120 calories, 6 grams of protein, and 1 gram of net carbs.

Some Slim-Fast products are available at ordinary retail outlets such as Walgreens, while others can only be purchased online through the company Web site. Slim-Fast products have been heavily advertised in the mass media, and campaigns using celebrity endorsers such as the baseball manager Tommy Lasorda, former New York City mayor Ed Koch, and the comedian Whoopi Goldberg will be familiar to many Americans. The inclusion of male and of middle-aged (and older) spokespersons distinguishes the Slim-

Fast campaigns from those of some similar products, which focus primarily on young women.

In a *Forbes* magazine study of the cost of 10 popular dieting systems, Slim-Fast was one of the least expensive at just under $78 per week (in 2004), considerably lower than Jenny Craig (about $138 per week) or NutriSystem (about $114 per week) but more expensive than Sugar Busters! (about $70 per week) and Subway (about $69 per week). The costs of the Slim-Fast diet was calculated at 42.8 percent over the median weekly food expenditure in the United States, however.

COMPANY HISTORY

S. Daniel Abraham began his career working for a small drug company in New York. He left that enterprise to found the Thompson Medical Company which purchased the rights to various product lines and also produced modified versions of existing products. Thompson's first diet product was Slim-Mint gum, introduced in 1956, which included benzocaine, which was believed to act as an appetite suppressant. In 1960, Thompson introduced a line of diet pills called Figure-Aid, and in 1976, introduced Dexatrim. This was a diet pill which originally included phenylpropanolamine (PPA) and became the best-selling diet pill on the market; although Dexatrim is still sold as a weight-loss aid, formulations have changed over the years and it no longer includes PPA.

Slim-Fast was introduced by Thompson Medical in 1977 as a powdered formula designed to be mixed with milk to create a shake similar to a milkshake. Slim-Fast was pulled from the market along with many other liquid meal-replacement formulas when a number of dieters died after following a 300-calorie-per-day diet created by a different manufacturer of liquid meal replacement products. Slim-Fast was returned to the market in the 1980s and achieved sales of almost $200 million in 1984.

Abraham pioneered the use of celebrity endorsers with Slim-Fast—one of the first was Los Angeles Dodgers manager Tommy Lasorda, who lost 30 pounds after following the Slim-Fast program for several months. Lasorda was seen as a particularly effective spokesperson because he was a man admitting he needed to lose weight, and because the results of his weight loss were presented on national television through Slim-Fast advertising. In addition, Slim-Fast made a highly publicized contribution of $20,000 to the charity of Lasorda's choice (a group of nuns based in Nashville) in recognition of his adherence to the diet.

Slim-Fast became a separate company in 1991, although Abraham remained majority stockholder. The company changed its advertising tactics after the split and began to promote Slim-Fast and weight loss as part of a healthy lifestyle. This was partly in response to slower sales due to Oprah Winfrey's highly publicized weight gain after going off a liquid diet. The product line was also broadened to include premixed shakes, packaged meals, and snacks. The company introduced an infomercial in 1993 which promoted Slim-Fast products without selling them directly, that is, the consumer was urged to buy the products from a retail store. In 1999, Slim-Fast had a 46-percent share of the diet liquid/powder weight-loss market, and in 2000 was sold to Unilever for $2.3 billion.

SEE ALSO: Dexatrim; Liquid Diets; Low-Calorie Diets.

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Smoking

THERE HAS LONG been a popular tradition that smoking inhibits eating, and as a result, people who smoke are less likely to put on weight. Consequently, the giving up of smoking is often associated with the person gaining weight. Indeed, in 1964, a report given to the U.S. Surgeon General raised the possibility of cigarette smoking having certain laxative effects on the body which served to counter obesity, and according to some people, therefore counterbalanced the health hazard posed by smoking cigarettes. Some

studies have also shown that obese individuals are less likely to smoke than nonobese people.

During the late 19th century, it was observed, anecdotally in Britain and the United States, that heavy smokers were often gaunt, and it was not until the 1964 report by the advisory committee to the U.S. Surgeon General Luther Leonidas Terry that it was publicly acknowledged that although smoking caused death from cancer, it did seem to promote "good intestinal tone and bowel habits," having a medically proven laxative effect which countered obesity to some degree. Furthermore, the smoking of cigarettes was seen to stimulate smokers when they became fatigued, whereas nonsmokers might get more energy from eating food. The report, however, did clearly stipulate that the positive effects of smoking in no way counterbalanced the "significant health hazard" posed by smoking cigarettes.

With campaigns during the 1970s to encourage people to stop smoking, it was soon discovered that it was more difficult to get women in the United States to give up the habit than men. Part of this was believed to have been that women fulfilled so many more different roles than men, and the smoking was used to calm them down, with many believing that by giving up smoking cigarettes, they would put on more weight quickly. Certainly, reports during the 1970s tended to show that in the initial stages after giving up smoking, many people did notice an increased edginess as well as an increase in their body weight. Indeed, this was confirmed by a study by Temple University in Philadelphia, Pennsylvania, which showed that during the late 1970s, nonsmokers weighed on average seven pounds more than smokers. It was argued that this might have been because smoking increased the metabolic rate of people.

Others suggested that the extra energy needed for breathing for smokers, every day, was also contributing to their weight loss. Some people in the cigarette industry leaped at the concept and started to promote their products as being associated with slimness, especially for teenagers, and even more particularly with females who tended to find it more difficult to give up smoking than men. The tobacco manufacturers cultivated the image of the woman who smokes as someone who might be using their product—cigarettes—as a beauty aid, and symbolize their liberation from male oppression.

Smoking stimulates smokers when they became fatigued, whereas nonsmokers might get more energy from eating food.

In recent years, there have been studies that have dealt with the problem in the opposite manner to the research in the 1970s, which had been concerned with why smokers were, on average, less likely to be obese than nonsmokers. Some studies conducted in the 1990s proved that obese individuals are less likely to smoke.

SEE ALSO: Obesity and Cancer; Respiratory Problems.

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SNP Technologies

A SINGLE NUCLEOTIDE polymorphism (SNP or "snip") is a single base pair variation at a particular location in the deoxyribonucleic acid (DNA) sequence that occurs in more than 1 percent of the population. SNPs are used as markers to identify regions in the genome associated with disease states such as obesity, diabetes, heart disease, and Alzheimer's. The discovery of such markers requires that a high number of SNPs be analyzed within large study populations. Many different technologies have been developed for SNP analysis that are rapid, accurate, and cost effective to increase the number of SNP markers suitable for use in determining effective medical care.

The human genome is composed of 3 billion base pairs of DNA, approximately 99.9 percent of which is identical between individuals. The 0.1 percent of genetic variation that occurs between us is responsible for differences in our appearance, personalities, and physiologies. These variations in DNA sequence, or "polymorphisms," occur as deletions, insertions, sequence repeats, or SNPs. Mutations differ from polymorphisms in that they occur in less than 1 percent of the population.

SNPs account for approximately 90 percent of polymorphisms in the human genome, and over 9 million have been identified. Some SNPs are directly responsible for a disease process or for how an individual responds to a drug. When found within the coding region of a gene, a SNP may cause changes in the structure and possibly the function of the protein encoded by that gene. This could result from either a direct amino acid substitution or by affecting the splicing of the messenger ribonucleic acid (mRNA). If a SNP occurs in the regulatory region of a gene, the expression pattern of the gene could be significantly altered, with possible serious consequences to the cell or the organism.

Figure 1. Example of Two Alleles Represented by an SNP

Major allele (85 percent)	GTATCTA**T**GGCCAAT CATAGAT**A**CCGGTTA
Minor allele (15 percent)	GTATCTA**C**GGCCAAT CATAGAT**G**CCGGTTA

Most SNPs, however, are found in the 98 percent of the human genome that does not encode protein. Even when a SNP has no obvious affect on the expression of a gene, it may still be useful as a genetic or physical marker due to its proximity to a specific gene of interest. This is the basis for using SNPs to identify genes that either cause or influence various medical conditions, or to determine if an individual has a particular gene that predisposes him or her to a disease.

To understand how this works, it is first necessary to define a few terms. An *allele* is an alternative DNA sequence that occurs at a specific genetic locus. Figure 1 gives an example of two alleles represented by an SNP. At this location within the genome, a T-A base pair occurs 85 percent of the time, and a C-G base pair 15 percent in a given population. Most SNPs are biallelic, meaning only two sequence variations are found at that location. A few are known to be tri- or tetraallelic.

A particular combination of alleles on a given region of a chromosome is called a *haplotype*, whereas a collection of alleles within the entire genome is called a *genotype*. Figure 2 illustrates two different haplotypes, in which two SNPs are located near one another on a given chromosome.

Note that the combination of SNPs on individual A correlates with a person of normal body weight, while

Figure 2. Two Different Haplotypes

	WEIGHT	HAPLOTYPE
Individual A	Normal	GTATCTATGGCCAAT…CACCTGCAGACTCAG CATAGATACCGGTTA…GTGGACGTCTGAGTC
Individual B	Obese	GTATCTACGGCCAAT…CACCTGAAGACTCAG CATAGATGCCGGTTA…GTGGACTTCTGAGTC

individual B, who has a different set of SNPs (a different haplotype) is overweight. In this example, body weight is the phenotype, the physical appearance of a trait. The example suggests that these two SNPs are in or near a gene that influences body weight.

SNP technologies focus on discovering associations like the one just described. The general approach for identifying genes related to a medical condition such as obesity involves characterizing the SNPs in a population of patients and healthy controls (obtaining their genotypes), then looking for an association between genotype and phenotype, such as body mass index (BMI). Rigorous analysis of the data can reveal connections between specific SNPs and the physical trait being examined. Further analysis of the genome region near the SNP would identify candidate genes to be further studied.

To do this, however, requires the analysis of large numbers of SNPs (>100,000) across the entire genome. Large study populations are also needed to obtain results that are statistically significant. Adding to the complexity is that certain populations may have different frequencies of the same alleles. So an association found in African Americans and Caucasians may not hold true in Asians.

Several efforts are under way to identify optimal sets of SNPs that may be used as markers for disease-association studies. One of these is the International HapMap Project, which seeks to identify a subset of SNP haplotypes covering the entire human genome, and to determine their distribution among different populations. Advances in the development of technologies that allow the simultaneous analysis of hundreds of thousands of SNPs at reduced costs are facilitating this type of research. These methods are collectively referred to as genome-wide SNP (GW-SNP) assays.

Few cases of obesity are monogenetic, meaning they stem from a single dysfunctional gene referred to as a causative gene. Obesity is primarily a polygenetic disease arising from contributions made by many different genes. These genes may be involved in lipid uptake and metabolism, regulation of food uptake, development of adipose tissue, or other aspects related to energy storage and expenditure. Due to the complexity of the disease process, discovering an association between obesity and one of these genes only indicates it as risk factor and not a cause. GW-SNP assays are being employed to scan the entire genome to discover genes that influence obesity, typically using BMI as the primary phenotypic marker. This technology has the capability of identifying not only genes that increase the risk of obesity, but also those that might play a protective role against it. These studies are expected to uncover a number of candidate genes that would further our understanding of the biochemical pathways that control body weight.

SEE ALSO: Body Mass Index; Ethnic Variations in Body Fat Storage; Ethnic Variations in Obesity-Related Health Risks; New Candidate Obesity Genes.

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Social Marketing and Obesity

SOCIAL MARKETING INVOLVES the adaptation of highly successful commercial marketing principles to promote health by effecting behavioral change in members of a target population. Social marketing is not a theory by itself, but rather draws upon theories and models from the behavioral and social sciences for understanding and influencing human behavior. These theories are useful starting points because the underlying causes of health problems are often rooted in individual behaviors.

While the goal of commercial marketing is to generate a profit from the given product or service, social marketing goals are organized around promoting health for the greater good. Social marketing campaigns have been successfully employed to address health concerns such as infant mortality, smoking cessation, human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS), and seat belt use. Social marketing is differentiated from "top-down" health promotion and communication programs by its incorporation

of in-depth research with target audience members (a consumer-driven focus) and an ongoing process of program evaluation (rather than only at the beginning and end of an intervention). In addition, the target audience of social marketing has expanded to include "upstream" policy makers and organizations in order to facilitate an environment that removes barriers to and supports behavioral change. Obesity-related social marketing campaigns typically encourage target audience members to adopt healthier eating practices and increase physical activity levels.

While these two factors are cited as the main contributors to obesity, environmental, genetic and biological mechanisms are still under investigation as related factors. Because obesity is a complex health issue, social marketers will be challenged in new ways as they attempt to address this public health problem.

HISTORY AND KEY COMPONENTS

Social marketing as a discipline began in the 1970s when Philip Kotler and Gerald Zaltman suggested that the same principles used by marketers to sell products could be applied to promoting ideas and behaviors. While marketers in the 1950s were considering application of the techniques to political and social themes, others were concerned that marketing might be used for social control and propaganda. By the 1960s, however, as public health policy began to shift from a treatment-focused model to one of disease prevention, health education campaigns, particularly in developing countries, began to incorporate commercial marketing techniques. By the 1980s, social marketing as an approach in promoting public health became widespread.

Social marketing utilizes an adaptation of the four P's from commercial marketing: product, price, place, and promotion. In social marketing, the product can be a tangible item, but more often, it is the behavior (e.g., smoking cessation), service (e.g., preventive screenings), or idea (e.g., seat belts save lives) that individuals in the target audience are encouraged to adopt or endorse. Price usually equates to financial cost in commercial marketing, but in social marketing, price also pertains to the time, effort, or what must be given up to obtain the product.

Changes in attitudes or behaviors often require a concerted effort and long-term commitment on the part of members of the target audience. The exchange theory in marketing is relevant here in that when consumers enact a behavior (purchasing an item or service, for example, at a particular price), the payback (receipt of that item or service) takes place simultaneously or shortly thereafter. When an individual changes behavior in response to a social marketing campaign, the payback may take place months or years later, if at all.

Furthermore, when perceived costs exceed perceived benefits, individuals are unlikely to make behavior changes. The third component, place, describes how, when, and where the product can be obtained. Finally, promotion pertains to disseminating messages and information through channels such as advertising, the internet, or in community and family settings and peer-to-peer groups.

In addition to the four P's from commercial marketing, social marketing incorporates additional P's: publics, positioning, policy (or politics), partnership, and purse strings. Social marketers need to reach many different audiences, so they must take into account multiple publics. This includes external publics, or members of the target audience, and internal publics, individuals involved with the approval, funding, or implementation of the program. Positioning refers to how members of the target audience perceive the social marketing product in relation to other competing products and messages in the marketplace.

Social marketers must have a good understanding of the competition and be able to clearly demonstrate compelling benefits that will result from a change in behaviors, attitudes, or beliefs. Policy (or politics) is important to social marketers because while a program may be successful in bringing about behavior change in the short term, long-term success may depend upon sociocultural and political environments that support new behaviors. This often requires policy change. In addition, because health issues are complex and difficult for a single agency to address by itself, partnership is another key success component. This may mean partnering with other organizations with similar goals. Finally, purse strings (budgetary restrictions) play a large role in the design, implementation, and maintenance of social marketing campaigns because many organizations rely on limited funding from grants or donations.

RESEARCH AND EVALUATION ARE FUNDAMENTAL

Unlike more traditional health promotion campaigns in which research is conducted at the beginning of

an intervention and again at the end to evaluate outcomes, research and evaluation are the foundations of social marketing and should be engaged throughout the life of the campaign. Social marketing research is consumer driven, and formative research in the beginning stages assists in identifying the target audience, choosing messages that will resonate with the audience, and selecting the most appropriate channels for communicating those messages.

Research can illuminate changes that should be made during the program (process evaluation), and provide evidence of whether the program is achieving its objectives (outcomes evaluation). Ideally, the research should consist of both quantitative and qualitative methods. Quantitative methods, such as analysis of existing data and the conduct of surveys, for example, answer what is happening. Quantitative methods produce data generalizable to a larger population and are appropriate for conducting needs assessments or evaluating outcomes. Qualitative methods include in-depth interviews, ethnographic observation, focus groups, and informal conversations. Qualitative data can reveal why something is happening. Qualitative methods are well suited for uncovering target audience perspectives and understanding meanings and behaviors. Primary research (collecting the data firsthand) and secondary research (utilizing existing sources of data) can be accomplished using quantitative or qualitative methods.

STEPS IN SOCIAL MARKETING

The process of social marketing involves several major steps: planning and strategy, message and materials development, pretesting, program implementation, and evaluation and feedback. The planning and strategy phase consists of gaining an in-depth understanding of the target audience, segmenting that audience, and developing the campaign strategy. Formative research in the planning stage ideally consists of both quantitative and qualitative methods using primary and secondary research sources to understand how target audience members make health-related decisions. Because the target audience is likely to be a heterogeneous mix of individuals who respond in different ways to different messages, social marketers must segment their intended audience by targeting subgroups based on common-ground characteristics. Segments have typically been grouped based on factors such as age, geographic location, race/ethnicity, gender, attitudes, or by behavior (e.g., smokers vs. nonsmokers). However, other factors such as attitudes and beliefs, lifestyle, leisure-time activities, or stages of life may prove to be more relevant, depending upon the goals of the particular campaign.

Developing the campaign strategy involves choosing a theory or model for understanding and influencing behavioral changes. Social marketing is considered a discipline (not a theory), however; social marketers draw upon theories and models of behavior change from disciplines such as psychology, sociology, anthropology, and communication. The use of theories and models is not always explicit in a social marketing campaign nor do social marketers necessarily incorporate all components of a given theory or model. Some of the most common theories and models are summarized here.

The Transtheoretical Model of Health Behavior Change (Stages of Change) suggests that individuals move through a series of stages before adopting a new behavior. At the precontemplation stage, an individual may not perceive that he or she is at risk and therefore the social marketing product is irrelevant. Making the target audience aware of the problem and associated risks can move individuals to the second stage, contemplation. At this point, social marketing messages must maximize perceived benefits and minimize perceived costs of behavior change to move individuals to the third stage, preparation for action. In the fourth stage, action, individuals have begun making behavioral changes. Action is facilitated when the behavior is portrayed as something many other people engage in and agree with. The fifth stage, maintenance, requires motivational and reinforcing messages to keep individuals from relapsing back to prior stages.

The Health Beliefs Model (HBM) was designed to explain why people do not participate in diagnosis or disease prevention programs. Core components of the HBM relevant to social marketing include an individual's perceptions of susceptibility (risk for developing a health problem), severity (seriousness of consequences of the health problem), benefits (to taking action), barriers (negative aspects of taking action), and cues to action (bodily or environmental events triggering action). HBM incorporates the idea of self-efficacy (an individual's perceived control over his or her behavior) as a

Creating a social marketing campaign involves developing messages and identifying how to reach the target audience.

predictor of health behaviors, particularly when lifestyle changes must be maintained over time. In addition, the model gives attention to factors such as socioeconomic status, age, gender, and knowledge as influential on an individual's perceptions and behaviors. The HBM has been useful for addressing "at risk" populations who may not perceive themselves as such.

Social Cognitive Theory (SCT) suggests that human behavior is determined by a dynamic and reciprocal process between personal factors (individualized internal characteristics, e.g., self-efficacy), behavior, and the external environment. In addition, observational or vicarious learning by watching others is a key component of this model in that an individual's behavior can be influenced by observing reinforcements (positive or negative) in response to someone else's behavior. SCT is relevant to social marketing in that it suggests individuals are most likely to model behaviors observed in others if the others are perceived to be similar to themselves, and if the perceived benefits outweigh the perceived costs.

The Theory of Reasoned Action (TRA) posits that the most important predictor of human behavior is intention, and that intention is influenced both by the individual's attitudes toward the behavior and by subjective norms (beliefs about what other people will think about the behavior). Each of the three components of TRA is weighted differently depending upon the individual and the situation. The Theory of Planned Behavior is an extension of TRA and includes self-efficacy as an influencing variable. When social marketing programs utilize these theories, in-depth open-ended interviews are helpful in identifying variables relevant to the population and the behavior of interest.

The Diffusion of Innovations Theory broadens the focus from individuals and their influencers to include how ideas, products, and practices from one group to another. Diffusion refers to how information flows through networks over time to reach the members of a group while innovations encompass the ideas or behaviors that individuals perceive as new. This theory centers on factors that will increase or decrease the probability that new idea, practice, or behavior will be adopted by individuals. In addition, the theory suggests that there are innovators, or early adopters, within a given group who are the first to adopt new ideas, attitudes or behaviors, and who are influential in encouraging others to do the same.

The second major step in creating a social marketing campaign involves developing messages and materials and identifying the most appropriate channels (channel analysis) for reaching the target audience. Effective messages are personal, designed to resonate with the experiences and realities of individuals within the target group. Effective messages also clearly communicate the specific desired behavior to the audience while creating and reinforcing positive attitudes about behavioral change. Channel analysis means finding out how and where members of the target audience obtain information, so that the most relevant channels can be used for communication.

Step three, pretesting, involves testing messages and materials with members of the target audience and other relevant parties to ensure that they evoke the expected response before launching a media campaign. Implementing the campaign is the fourth step and requires close coordination with participating partner agencies and organizations. Although evaluation and feedback are listed as the last step, in reality, these two components should be engaged throughout the campaign (process evaluation) to incorporate feedback. Outcomes evaluation measures campaign results and helps to determine whether or not the objectives were accomplished.

EXAMPLES OF SUCCESSFUL SOCIAL MARKETING CAMPAIGNS

Social marketing campaigns have been successfully implemented with positive results, both interna-

tionally and domestically. Some key examples are summarized here:

Mass Media and Health Practices Program, Honduras. This social marketing campaign to decrease infant mortality was initiated by a partnership between the Academy for Educational Development (AED) and the Honduran Ministry of Health in 1980. At the time, dehydration from diarrhea, the leading cause of infant mortality, accounted for 24 percent of all infant deaths in Honduras. Social marketers wanted mothers to use a new product, oral rehydration therapy (ORT), to increase child survival. Information, with easily remembered slogans and songs, was disseminated in a mass media campaign via print materials and radio. Healthcare workers trained in ORT preparation and administration gave hands-on demonstrations to village assistants, who in turn trained others within the community. Infant mortality due to dehydration from diarrhea decreased from 47.5 to 25 percent in the first year of the campaign.

Stop AIDS, Switzerland. Implemented as a national multimedia campaign in 1987, Stop AIDS is the longest running HIV/AIDS prevention program in the world. The campaign was created in partnership by the Swiss AIDS Foundation and the Swiss Federal Office for Public Health. Specific goals of the campaign included increasing condom use, reducing discrimination against individuals with HIV/AIDS, and increasing understanding among the general population for those members diagnosed with HIV/AIDS. Products included condoms, antidiscrimination, and needle exchange, and campaign messages were disseminated via billboards, posters, advertisements (print, radio, and television), movie theater commercials, and sporting events. Ongoing evaluations of the program measured both condom use and the change in attitudes toward HIV/AIDS. Within 1 year of program launch, more than 90 percent of the population recognized the campaign logo, and within 3 years, condom use among men aged 17–30 increased from 8 to nearly 50 percent.

Florida Truth® Campaign, Florida. The truth campaign, sponsored by the American Legacy Foundation, is a national antitobacco initiative targeted toward youth. The program was started by the State of Florida's Office of Tobacco Control and was funded by monies from the 1998 Master Settlement Agreement (MSA) between 46 states and the tobacco industry. The campaign uses information from tobacco companies to expose both manufacturing and marketing practices of the industry, and provides facts about the health consequences, social costs, and addictiveness of tobacco. Campaign messages are distributed via radio, print advertisements, and television. The target teen audience is segmented by ethnic group, with specific advertising designed to mirror audience segmentation done by tobacco companies. In addition, a grassroots initiative helps teens share information in peer-to-peer settings. In the first two years of the campaign, the number of youth smokers decreased 22 percent.

Click It or Ticket, North Carolina. Launched in 1993, Click It or Ticket was designed to reduce fatal and serious traffic-related injuries by increasing seat belt and child safety seat use. The program was pilot tested before the launch of a major and sustained media campaign in conjunction with increased enforcement of the seat belt law. Click It or Ticket was supported by policy makers, high-ranking state officials, and influential organizations including the Governor, State Insurance Commissioner, Commander of the State Highway Patrol, and the Insurance Institute for Highway Safety. In addition, nearly every one of the state's law enforcement agencies participated in the campaign. Fatal and serious traffic-related injuries decreased by 14 percent, while seat belt use increased to over 80 percent, one of the highest rates in the nation.

SOCIAL MARKETING AND OBESITY

Social marketing campaigns designed to reduce the prevalence of and prevent obesity typically focus in some form on the adoption of healthier eating practices and increased physical activity levels. Both of the social marketing campaigns summarized below focus not only on the "downstream" individuals for behavioral change, but also on influencers in the familial and social spheres and "upstream" policy makers. The campaigns leverage partnerships on a number of levels to maximize resources and extend the reach of key messages. The social marketers who developed these campaigns incorporated behavioral theories and models in their strategies, and use ongoing research and evaluation as critical components to improve the campaigns and measure outcomes.

VERB™ It's What You Do, Centers for Disease Control and Prevention (CDC). VERB is a national, multicultural social marketing campaign designed

to increase and maintain physical activity among the target audience of youth aged 9–13 (tweens). Additional target audiences for the campaign include parents and other influential adults such as teachers and coaches, youth leaders, and health professionals. The campaign markets physical activity as something fun for tweens to do, and provides information via brochures, articles, posters, and stickers. Short-term outcomes focus on increasing tween and parent awareness of the campaign and key messages. Midterm outcomes are related to changes in subjective norms, beliefs, self-efficacy, and perceived behavioral control related to participating in physical activity. The long-term outcomes are focused on getting tweens to engage in and maintain physical activity, which will in turn reduce risk for diseases such as diabetes, hypertension, and sleep apnea.

The campaign engages formative research, process evaluation, and outcome evaluation. Formative research involved reviews of existing research, conduct of primary research with target audiences, consultation from experts, and reviews of other campaigns targeting the same age group. Exploratory research was used to understand the behaviors and mind-sets of tweens and their influencers to identify motivators and barriers to participation in and support of physical activity. The tween audience was then segmented into subgroups based on cultural, ethnic, and economic variables. Messages and communications strategies were tested prior to mass media release to gauge audience reactions and to ensure that the messages and advertisements were understandable, appealing, and motivating.

Process evaluation is ongoing throughout the campaign and includes analysis of campaign advertising. To accomplish this, the campaign utilizes a telephone survey conducted with tweens four times per year to find out what VERB means to them, what they think of the brand, and where they've seen VERB advertisements. VERB-sponsored events are evaluated on site, and follow-up interviews are conducted afterward. Outcomes evaluation assesses changes in awareness, attitudes, knowledge, and behaviors related to physical activity that have been directly influenced by the campaign. This step relies on a longitudinal cohort design telephone survey with children and parents to make measurements over time. After the first year of the campaign, 74 percent of children surveyed were aware of the campaign, and physical activity levels were increased for those children versus others who were not familiar with the campaign.

California Project LEAN (Leaders Encouraging Activity and Nutrition) (CPL). CPL is a collaborative effort between the California Department of Health Services (CDHS) and the Public Health Institute toward the vision that Californians will become physically active, eat healthy foods, and live in communities that support healthy lifestyles. CPL's overall mission, aligned with the Governor's vision for Healthy California, is to increase healthy eating and physical activity, which in turn, will help to reduce the prevalence of obesity and related health conditions such as cardiovascular disease, stroke, and diabetes. The campaigns goals include using policy and environmental changes to create healthier communities, provide education on choosing healthy food choices and physical activity, and to conduct research-based consumer-driven campaigns.

Originally, CPL began as an initiative to encourage low-fat diets for individuals living within the San Francisco Bay area. The CDHS expanded the initiative to work with state and local physical activity and nutrition leaders in conducting programs throughout the state. The CDHS provides funding for 10 regional offices across the state for social marketing programs including *Food on the Run, Successful Students through Healthy Food Policies,* and *Huesos Fuertes, Familia Saludable* (Strong Bones, Healthy Families).

CDHS is also a lead partner on the California Obesity Prevention Initiative (COPI), a campaign that promotes healthy eating and physical activity as mechanisms to reduce the prevalence of obesity and related health conditions. CPL developed *Captive Kids Selling Obesity at Schools: An Action Guide to Stop the Marketing of Unhealthy Foods and Beverages in School*, and the campaign's research on soda and snack foods distributed in schools has contributed to new policies and legislation that will make healthier food choices available in schools.

SEE ALSO: Federal Initiatives to Prevent Obesity; State and Local Initiatives to Prevent Obesity.

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Soda and Soft Drinks

BEVERAGES ARE IMPORTANT components of diet and a route for the intake of caffeine, ethanol, and other bioactive substances. Consumption of fruit drinks and soda represents nearly 81 percent of the increase in caloric sweetener intake in the United States. The largest source of these added sugars is nondiet soft drinks, which account for 47 percent of total added sugars in the diet.

The term *soft drink* encompasses sodas along with other sugar-sweetened beverages such as fruit drinks, lemonade, and iced tea. The term *soda* encompasses sugar-sweetened carbonated beverages such as colas. Consumption of these beverages was shown to increase by 135 percent between 1977 and 2001. In the United States, on average, a 12-ounce serving (12 ounces = 1 can of soda or 1 soda = 1 serving) of soda provides 150 kilocalories and 40–50 grams of sugar in the form of high-fructose corn syrup ([HFCS] approximately 45 percent glucose and 55 percent fructose), which is equivalent to 10 teaspoons of table sugar. If these calories are added to the typical U.S. diet without reducing intake from other sources, one soda per day could lead to a weight gain of 15 pounds or 6.75 kilograms in one year. It has been hypothesized that fructose may lead to greater weight gain and insulin resistance by elevating plasma triacylglycerols and subsequently decreasing the production of insulin and leptin in peripheral tissues—not suppressing ghrelin—thereby decreasing signaling to the central nervous system from insulin and leptin—and possibly ghrelin.

Recent studies have shown that women who consumed one soda per week were 0.47 pounds (0.21 kilograms) heavier than those who reported no soda consumption. Men consuming one soda per week were 0.33 pounds (0.15 kilograms) heavier than those who reported no soda consumption, although this difference was not significant. The weight gain is very significant in women and children. Several studies found significant associations between the intake of sugar-

Cola-type beverages in particular are negatively associated with bone mineral density and positively associated with bone fractures.

sweetened beverages and greater overweight or obesity. Soda consumption of 2 servings or more per day enhances body weight gain in children. It has also been suggested that soft drinks that contain caramel coloring are rich in advanced glycation end products, which may increase insulin resistance and inflammation.

The intake of caffeine from soda (10–16 milligrams/100 grams) may increase blood pressure in adolescents, especially those of African-American background, thereby increasing their risk of hypertension, although this adolescent population's blood pressure may also be affected by dietary and lifestyle practices for which the consumption of caffeinated beverages is a marker. In the Third National Health and Nutrition Examination Survey (1988–94), based on a nationally representative sample of the noninstitutionalized civilian U.S. population, observed that adults with diabetes reported drinking three times as much diet soda as adults without diabetes. Adults with diabetes who had one or more drinks of diet soda per day, HbA1c level was 0.7 units significantly greater compared with those who drank none.

Several studies showed that the consumption of cola-type beverages in particular is negatively associated with bone mineral density and positively associated with bone fractures. Associations between high intakes of fruit juice and obesity were observed with apple juice only, which likely reflects the high fructose (13.9 g/8 oz serving) and sucrose (4.2 g/serving) content of apple juice. In a recent review of 88 studies, it was suggested to reduce soft drinks based on its association with obesity and diabetes in the United States. In a study of 91,249 women followed for eight years, those who consumed one or more soft drinks per day were twice as likely as those who consumed less than one per month to develop diabetes.

SEE ALSO: Beverage Choices in Children; Food "Addictions"; Food Marketing to Children; Obesity in Schools.

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Nutrition 21

South America

PRIOR TO THE European conquest of the continent, South America was divided, anthropologically, into six main groups: the Circum-Caribbean, who also dominated the Caribbean and some of Central America; the Savanna-Orinoco peoples; the tropical forest peoples of modern-day Brazil and surrounding regions; the Andeans, who included the Incas; the Southern peoples who covered the region from the Chaco to Patagonia; and the Atlantic peoples in the southeast of modern-day Brazil. Most of these left little in the way of written records, and the Spanish and Portuguese were not inclined to study the local people except for collecting gold and other precious metals, and their subsequent use as a labor force.

Many of the household objects and votive ornaments of these pre-Columbian peoples were destroyed, but sufficient numbers do survive to indicate that there might have been a problem of overweight with some people. The most well known of these is probably the hollow vessel of a female form in the Capuli style of the Narino (800–1250 C.E.) which was found in a tomb of the Narino tribe which lived on the modern-day Colombian-Ecuadorian border region. However, it is possible that the wide girth of the person is as much to do with the pottery item's main use, as a decorative pottery vessel for holding grain, rather than an accurate representation of the people.

Similarly, a Tairona miniature jar in the figure of a large seated man clearly chewing a coca quid, may similarly have the figure dramatically altered for style. Large squat figures from the Caldas department of Colombia and those of the pre-Inca Mohica warriors may also have their size exaggerated to allow the fig-

ures to stand up, rather than reflect the size of the people. The human-like figures made from gold and other metals, and those that do not have a dual use as a storage vessel, tend to show slim figures. Although the diet of the period included large amounts of potatoes, most of the people did not seem to eat in excess and had plenty of exercise.

The arrival of the Spanish conquistadors dramatically changed the whole of South America, and early line engravings of the Spanish and Portuguese show little evidence of overweight. However, some paintings from the 17th century onward often show large European people. The style of large flowing garments, with puffed sleeves and breeches worn by men, as shown in the paintings of the late-17th century Cuzco school of Archangels in contemporary costume, often makes the people look much larger than they actually were.

Similarly, the heavily pleated dresses often dramatically exaggerated the size of small women. During the Spanish period, the diet of the region changed considerably with new dishes such as yaguarlocro, a potato soup made with chunks of congealed blood sausages, often served with cheese, and still popular in Ecuador, clearly could lead to health problems if not a part of a balanced diet and exercise regime. Undoubtedly, some obese people did exist, but evidence points to them being relatively rare.

During the Portuguese period in Brazil, obesity was generally equated with prosperity, although this fashion changed significantly in the 19th century. Certainly, in studies in Brazil in the 1990s it was shown that socioeconomic class factors correlated against obesity showed that the wealthier people were less likely to be obese, with poor nutrition suggested as a reason for increasing obesity among the poor. In Brazil, as with many other developing countries around the world, there has been an increase in obesity from the second half of the 20th century with increased prosperity, and more meat in the diet, together with much larger consumption of sugar, for which Brazil is the second largest producer of cane sugar in the world.

The rise in obesity levels became particularly pronounced from the mid-1970s with the population of Brazil rapidly equaling that of the United States in the consumption of sugar, accounting for 19 percent of calories consumed. Much of this came from the increased availability of soft drinks for which consumption rates rose 400 percent in the period from 1975–2005. The importance of dietary fat as a major role in obesity in Brazil has, however, come in for criticism from Rosely Sicheri of the Institute of Social Medicine in Rio de Janeiro who viewed the decrease in physical activity as being more important. From 1940, where 80 percent of the population was rural, largely working on farms, to a population in 2000 where 80 percent are urban, has clearly also been a major contributory factor to obesity.

Perhaps the most well-known obese Brazilian was Tim Maia (1942–98) who is regarded as the father of Brazilian soul music. He collapsed while performing at Niterói and died of pulmonary edema a few days later. In recent years, there has been significant focus in the Brazilian medical community into obesity with Dr. Drauzio Varella hosting a television show on obesity, as well as writing in newspapers about weight-loss techniques. In the city of Cascavel in Paraná state, in the south of Brazil, there has been much research at the Genisis and Salete hospitals into obesity management, indicating it is a recognized problem. Policies to combat obesity in Brazil are coordinated by Associação Brasileira para o Estudo da Obesidade with Dr. Henrique de Lacerda Suplicy of São Paulo being the national representative on the International Association for the Study of Obesity (IASO).

For Argentina, a small degree of obesity among country landowners during the colonial period was seen as a mark of prosperity, although this was not as marked as in Brazil. After World War II, there was a large rise in the level of obesity among migrants from southern Europe, especially Italy and Spain, owing to a massive increase in the amount of meat in their diet. This was combined with many of these migrants settling in urban centers with a major decrease in physical activity. Since the 1980s, there have been further significant declines in the average amount of physical activity, along with a massive rise in the consumption of soft drinks, leading to medical problems including an increase in diabetes.

One example of this has been Diego Maradona, who played football for Argentina, becoming recognized as one of the greatest players in the history of the sport, and after he finished playing football, he had drug problems and developed a significant level of overweight, ending up being treated for obesity. In a study from the Nutrition and Diabetes Department

of Hospital Durand, Buenos Aires researchers, show that overweight mothers tend not to be as cognizant of overweight in preschool children which would become a major factor in obesity in children for years ahead. Studies among the Guarani-Mbya from Misiones, in northern Argentina, along the Paraguayan border, has a very significant rise in obesity rates among Amerindians which has resulted from a process of nutritional transition. In recent years, the Sociedad Argentina de Obesidad y Trastornos Alimentarios has been formed with Dr. Rosa Labanca of Buenos Aires being the national representative on the IASO.

The land on the north bank of the River Plate, because of its geographical isolation from the Brazilian city of Rio de Janeiro, and its proximity to the Argentine capital of Buenos Aires, became a popular place for smugglers and cattle farmers during the 18th century, and maintained a Spanish-speaking population, managing to get its independence in 1828, becoming the Eastern Republic of Uruguay.

Although wracked by civil wars during the 19th century, Uruguay was one of the most prosperous countries in Latin America by the early 20th century, largely based on its beef industry. With considerable wealth and a small population, Uruguay has managed to establish one of the best health systems in Latin America, and has been involved in many healthy lifestyle campaigns to combat bad diet and obesity. In spite of these, its general affluence contributes to its having the highest rate of adult obesity in the Americas (about 50 percent according to some studies), being more common among females than males. The Sociedad Uruguaya para el Estudio de la Obesidad has recently been founded, with Dr. Raul Pisabarro being the national representative on the IASO.

The population of Chile is traditionally one of the best-fed in Latin America, with the prevalence of malnutrition among children under the age of 4 being the lowest in the Americas (0.8 percent). However, as a result, Chile has the highest rate of obesity in the Americas for the 20- to 29-year-old population, followed by Costa Rica, Cuba, and Peru. The Sociedad Chilena de Obesidad was founded with Dr. Patricio Mois of Santiago being the national representative on the IASO.

The landlocked Republic of Paraguay was created in 1813 after a declaration of independence by its leader, **José Gaspar Rodríguez de Francia, who ruled the** country until 1840. Francia was notably gaunt, but his successor, **Carlos Antonio López, was well known for** his large size. Journalist Hector Varela who met López wrote, "one rarely sees a more impressive sight than this great tidal wave of human flesh." The British writer Richard Burton, although he did not meet López himself, certainly met people who had, and described the president as "hideous, burly and thick-set … with chops flying over his cravat, his face wears, like the late George IV, a porcine appearance, which, however, as in the case of Gibbon, is not incompatible with high intellect." The U.S. historian Philip Raine wrote that the most impressive feature of López was his obesity, with a double or triple chin, depending on which portrait one used for a likeness. His wife, Dona Juana Paula Carrillo, who took the title "La Presidenta," was also obese. López was a relatively benevolent ruler, but after his death in 1862, his son, Francisco Solano López, overweight but by no means obese, led Paraguay into a destructive war with its neighbors resulting in the destruction of much of the country, and the death of the vast majority of the men.

Compared to many other urban centers in South America, Asuncion has a lower level of obesity than its other counterparts, although cases are rising, often associated with an increase in kidney disease. There was much publicity given to the size of Victor Daniel Pavia, co-owner of the Ycuá Bolanos supermarket which was the location of the August 1, 2004 fire which resulted in the deaths of at least 275 people. The Sociedad Paraguaya para el Estudio de la Obesidad operates in Paraguay with Dr. Maria Cristina Jimenez of Asuncion being the national representative on the IASO.

In Paraguay's western neighbor, Bolivia, obesity is generally associated with poverty and poor diet in the indigenous population, rather than gluttony and overeating, as is the case in many instances in Argentina, Brazil, and Paraguay. Much of this comes from a heavy diet of potatoes with Bolivia still having one of the lowest height for age for children under the age of 4 in the whole of South America (38.3 percent), ahead of Peru (35.3 percent) and Ecuador (34 percent). In 2006, following the swearing-in of Evo Morales as president of Bolivia, there were moves to legalize coca with the suggestion that coca's ability to stunt appetite could result in a natural cure to the obesity problem throughout the world.

Another explanation for the high level of obesity among the poor in Bolivia came to light in a recent

survey by S. Mohanna, R. Baracco, and S. Seclen from the Instituto de Investigaciones de Altura, Universidad Peruana Cayetano Heredia, Lima, which showed that obesity was more common in populations that live at a high altitude, which was evident in a study across various peoples living in Peru.

Certainly, there is some evidence of obesity in colonial times with an early-19th-century watercolor of Doctor Valdez Busioni, displayed in the Museo de Arte de Lima, portrays a very large man covered in an equally large cloak. However, the Peruvian results relying on the high altitudes may also explain the situation in neighboring Ecuador. There have been several studies of the elderly poor in Quito which have shown similar results to those in Bolivia with obesity being common among the poor, owing to poor diet. The Peruvian Association of Atherosclerosis and Obesity coordinates research in the country, with Dr. Mario Zubiate of Lima being the national representative on the IASO.

In recent years, there has been an increase in obesity in women in Colombia, with the result of a rise in the risk of cardiovascular problems. The Colombian neo-figurative artist, Fernando Botero (b. 1932), was interested in the concept of corpulence, and many of his paintings and sculptures show obese animals or humans. Mention should also be made of Fabio Ochoa Restrepo "Don Fabio" (1923–2002) who was patriarch of a Colombian crime family connected with drug dealer Pablo Escobar. Ochoa suffered from severe obesity and was extradited to the United States in September 2001, dying of kidney failure in 2002. Research in Colombia is coordinated by the Asociación Colombiana de Obesidad y Metabolismo with Dr. Rafael Gomez Cuevas of Bogotá being the national representative on the IASO.

The increasing prosperity in Venezuela since the 1960s with the oil boom has led to more obesity with surveys by the Food and Agriculture Organization showing a rise in obesity rates in children under the age of 5 until 1993–1994, and then stabilizing in 1994 at 2.9 to 3 percent. It has increased again since then with one study of Valencia in the north central part of the country by G. Ovedio, M. Moron de Salim, and L. Solano revealing the medical consequences of obesity in the early 21st century. Operating from Caracas, the Asociación Venezolana para el Estudio de la Obesidad coordinates research in Venezuela, with Dr. Carlos Carrera of the Hospital de Clinicas Caracas being the national representative on the IASO.

SEE ALSO: North America; Prevalence of Childhood Obesity Worldwide

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South Beach Diet

THE SOUTH BEACH Diet is a popular diet program developed by the cardiologist Dr. Arthur Agatston in the 1990s and widely publicized after the appearance of his book *The South Beach Diet* in 2003. Agatston has stated that he developed the South Beach Diet to help his overweight cardiac patients lose weight, as he had observed that many of them did not respond well to the low-fat, carbohydrate diet recommended at the time by the American Heart Association. The South Beach Diet program emphasizes moderate consumption of healthy foods prepared by the individual, and except for the first phase in which carbohydrates are restricted, does not depend on any extreme allocation of macronutrients (carbohydrates, protein, or fat).

This differentiates it from both high-carbohydrate and high-protein/high-fat diets, as well as from diets based on the purchase of prepackaged foods or meal replacement items such as meal bars or shakes.

The South Beach Diet is organized into three phases. Phase one, which lasts 14 days, is the most restrictive. All fruits and fruit juices, starchy foods, dairy foods, and alcohol are prohibited during this phase, and the only carbohydrates consumed come from beans and other vegetables. This phase is basically a short-term low-carbohydrate diet in which moderate amounts of healthy fats and lean protein are consumed during Phase 1, and green vegetables such as broccoli and lettuce are also included. The dieter is instructed to eat three meals and two snacks a day during this phase to forestall hunger. The elimination of most carbohydrates from the diet for a short period of time enhances rapid weight loss (although some of that weight is due to decreased water retention), which is psychologically reinforcing to the dieter. However, Agatston also argues that this strategy also reduces cravings for refined carbohydrates and changes the body's metabolism; both of these claims are open to question.

In Phase two of the South Beach Diet, carbohydrates are gradually added into the diet. A distinction between good and bad carbohydrates is observed, so that (for instance) whole fruit is recommended rather fruit juice, and whole grain bread rather than bread made from refined white flour. Weight loss is slower in Phase two than in Phase one, and the individual stays on Phase two until he or she has reached his or her goal weight. Wine is allowed in moderation in Phase two and Phase three.

The South Beach Diet emphasizes moderate consumption of healthy foods prepared by the individual.

Phase three is a maintenance phase intended potentially to last the rest of the person's life. The individual is no longer on a diet per se, but is assumed to have learned healthy eating habits and to be accustomed to monitoring his or her weight and food intake. Snacks are not included in Phase three because meals are larger and it is assumed an individual will not become hungry between meals. A variety of foods are allowed in Phase three, governed by a few basic principles such as avoidance of refined carbohydrates and saturated fat.

The South Beach Diet distinguishes itself from other popular diets such as the Atkins Diet by distinguishing among types of carbohydrates and types of fats. In the South Beach Diet, "good" carbohydrates include fiber and include those found in many vegetables and whole grains, while "bad" carbohydrates have little fiber and typically are found in highly processed foods in which most or all of their fiber has been removed. In general, good carbohydrates have a low glycemic index (GI), while bad carbohydrates have a high GI. Similarly, fats are divided into good and bad: good fats are monosaturated or polyunsaturated and are found in foods such as olive oil and canola oil, while bad fats are saturated fats found in animal products. Consumption of good fats is included in all phases of the diet, while consumption of bad fats is limited throughout.

The South Beach Diet was an overwhelming commercial and popular success, but scientific evidence for its effectiveness remains largely lacking. Despite the fact that the diet was developed by a cardiologist, books and other materials presenting the South Beach Diet plan are written in a popular manner, and support for the diet's effectiveness is largely anecdotal. Some of the science presented in the first presentation of this diet, Agatston's 2003 *The South Beach Diet*, has also been questioned. A review of nutritional claims presented as fact in *The South Beach Diet,* conducted by a team led by S. L. Goff, found that many

of these claims were unsupported and some were contradicted by the current state of scientific knowledge. In fact, Goff's comparison of "facts" presented in *The South Beach Diet* with peer-reviewed medical literature found that only one-third of the claims were supported in the peer-reviewed literature, 17 percent were completely unsupported, 7 percent had not been studied, and evidence for the remaining 43 percent was ambiguous or equivocal.

The South Beach Diet is not easily categorized because it does not rely on extreme diet choices: for instance, although low carbohydrate in comparison to the typical American diet, it is not intended to put the body into a state of ketosis as is, for instance, the low-carbohydrate Atkins Diet. Some critics refer to the South Beach Diet as "Atkins Lite" and view it, particularly the first phase, as a less extreme version of the Atkins Diet. The South Beach Diet also distinguishes itself from the Atkins Diet by a marketing approach which emphasizes that it is based on moderation and healthy food choices, and thus has won greater support among nutritionists. In fact, some attribute the phenomenal success of the South Beach Diet books (the original South Beach Diet book was a New York Times bestseller and it, plus two spinoff books, sold over 14 million copies between 2003 and 2005) to the fact that the first South Beach Diet book appeared the same month that Atkins died. The South Beach Diet is also more acceptable to nutritionists and other medical professionals because the advice after Phase one generally involves eating a balanced diet consisting of moderate amounts of healthy foods, which is standard weight-loss advice.

One controversial aspect of the South Beach Diet is its embrace of the concept of the GI, which is a measure of the effect different carbohydrate-containing foods have on blood glucose in the first two hours after consumption. The theory is that foods with a high GI tend to break down quickly in the digestive system and cause a rapid increase in blood sugar, which causes the body to release large amounts of insulin, resulting in lowered blood sugar, bringing about storage of excess calories as fat and the recurrence of hunger. On the other hand, it is theorized that foods with a low GI break down more slowly, eliminating the spike in blood sugar followed by a spike in insulin and drop in blood sugar, and should therefore satisfy hunger and minimize food cravings longer than high-GI foods. Examples of high-GI foods in the Western diet include (GI in parentheses) rice cakes (110), baked potatoes (158), and glucose (137); examples of lower-GI foods include bran bread (68), oatmeal (70), lentils (36), and whole milk (39). The GI index is somewhat controversial for several reasons, including the fact that individual responses to any food can vary widely, and that the same person's GI response to a particular food depends on the quantity consumed and what other foods are consumed at the same time.

Although licensed South Beach Diet food products may be purchased, including a line of South Beach Diet Foods produced by Kraft Foods including meal bars, cereals, and packaged meals and South Beat Diet Wraps produced by the Santa Fe Tortilla Company, purchase of these foods is not required to follow the diet. In fact, preparing healthy meals using ordinary supermarket food is a point of emphasis in the South Beach Diet materials; large portions of the original South Beach Diet book is taken up with recipes and menus consonant with the phases of the diet, and several books of South Beach Diet recipes have been published. The Web site and newsletter also include recipes, and one of the diet books include advice about selecting foods in restaurants that will fit the South Beach Diet plan.

This emphasis on teaching the dieter to cook healthy meals using ordinary foods is admirable because it both keeps down expenses (relative to purchasing prepackaged meals) and retrains the individual dieter for a lifetime of healthy eating. On the other hand, the South Beach Diet materials have been criticized as elitist because many of the recipes, which call for expensive or obscure and exotic seasonings and main ingredients such as mahi mahi and edamame, are not commonly available in local grocery stores or familiar to many Americans. The variety of foods incorporated into the recipes is not accidental, however, but is consistent with Agatston's philosophy that dieters who consume a variety of foods, and who enjoy each food item and each meal they consume, are the most likely to be successful in controlling their weight in the long term.

A 2005 article in *Forbes* magazine found the South Beach Diet to be a relatively inexpensive choice among 10 popular diet plans. Purchasing foods to follow menus presented for the South Beach Diet was calculated to cost about $78 per week, about 44 percent above the national average food budget for one person. This made it far cheaper than programs such as Jenny Craig ($137

per week) or NutriSystem ($113 per week) which require the dieter to purchase packaged meals from the manufacturer, and slightly more expensive than Slim-Fast ($77) or Sugar Busters! ($69).

Agatston, who developed the South Beach Diet, received his medical degree from New York University and is an associate professor of cardiology at the Miller School of Medicine at the University of Miami. In 2004, Agatston founded the Agatston Research Foundation which initiates research and conducts collaborative studies and educational and prevention activities related to nutrition and health. One of the foundation's major studies is the Healthier Outcomes for Public Schoolchildren (HOPS) study, which consists of nutritional and lifestyle interventions for 15,000 schoolchildren from 17 public schools in the Miami area. The goal of HOPS programs is to test the effectiveness of different interventions and to develop programs that could be used in other public school settings, including the inclusion of whole foods in school lunches, development of curricula to teach children and their parents about nutrition and lifestyle management, and develop programs to create fruit and vegetable gardens in elementary schools.

SEE ALSO: Carbohydrate and Protein Intake; High Protein Diets; Low Calorie Diet; Low Carbohydrate Diet; Portion Control.

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State and Local Initiatives to Prevent Obesity

IN 1999, THE Centers for Disease Control and Prevention (CDC) established NPAO, a cooperative agreement between the CDC's Division of Nutrition, Physical Activity and Obesity (DNPAO) and 28 state health departments. NPAO seeks to prevent and control obesity by supporting states in the development and implementation of nutrition and physical activity interventions, particularly through population-based strategies geared toward balancing caloric intake and expenditure, increasing physical activity, increasing consumption of fruits and vegetables, decreasing television-viewing time, and increasing breastfeeding. States receive funding at two different levels: capacity building and basic implementation.

In the 2007 fiscal year 21 states were funded at the capacity building level and seven states were funded at the more advanced, basic implementation level. The funded states have made progress in establishing the infrastructure needed by gathering data, building partnerships, and creating statewide health plans to get broad based support for their programs.

Additionally, the seven states funded at the basic-implementation level are working to implement a comprehensive nutrition and physical activity state plan to prevent and control obesity and other chronic diseases, provide training and technical assistance to communities, implement and evaluate nutrition and physical activity interventions to prevent obesity and other chronic diseases, and evaluate the progress and impact of both state plans and interventions. An example of an NPAO state intervention is the Colorado Physical Activity and Nutrition Program (COPAN) which funds eight rural communities and has also partnered with Kaiser Permanente to support 11 sites in the metro Denver area. Commerce City has a nutrition, cooking, and literacy program for low-income preschool students and their parents, a community gardening program for youth, a mobile market program that delivers fresh and affordable produce throughout the community, an environmental education camping program for inner-city youth, and an organized walking program for seniors.

In 2006, the National Governors Association (NGA), chaired by Arkansas Governor Mike Hucka-

bee, launched the initiative Healthy America aimed at reducing obesity, improving nutrition, and increasing physical activity.

Numerous grassroots efforts have successfully engaged in obesity prevention efforts and many of the current state efforts and policies are a result of these. Local restaurants in Tiburon, California spawned a national movement against trans fat. Their 18 restaurants voluntarily stopped using trans fats in their cooking oils, making it the first city in the nation to go "trans fat-free." Project Tiburon was an inspiration and model and inspired New York City was the first city in the nation to require by law that all city restaurants to remove trans fat from their restaurants when the Board of Health voted that this be done in all New York City restaurants by July 1, 2008. Since then, other jurisdictions are considering trans fat bans or consumer warning notices and other cities already have voluntary trans fat bans in restaurants.

SEE ALSO: Community Programs to Prevent Obesity; Federal Initiatives to Prevent Obesity; Policy to Prevent Obesity; School Based Interventions to Prevent Obesity.

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Stereotypes and Obesity

THROUGHOUT GENERATIONS, THE stereotypes associated with obesity, or merely being slightly overweight, have always taken mainstream positions. The positions, despite always being present, have not always been consistent. Having excess body fat was once desirable, indicating wealth and plentiful food supplies. Today, however, being overweight is associated with images of laziness and poor self-control. Despite the varying stereotypes, obesity is a medical condition and has some well-established physiological disadvantages that do not change with the changing generations. With a general understanding of some of the issues related to obesity, the modern stereotypes can be tempered around the realities of obesity.

In many of the earliest human societies, having excessive body fat was highly prized. Statuettes were made celebrating the body image of what today would be considered very obese women. This body type was a necessity from 20,000 years ago to the earliest records of human activity. In times of famine and extreme cold, the women with the most body fat would be best able to bear children and live through harsh periods.

After the development of fixed housing and established heating sources, having excessive body fat was still considered a positive trait. Into the early 1200s, monarchs aimed for larger physical statures to indicate wealth and general prosperity. Past that, into the 1500s and 1600s, female plumpness was seen as erotic and many notable paintings were done indicating such. In general, up until more modern times, larger men were seen as strong and rich, and larger women were seen as erotic and caring.

The stereotypes of excessive body fat have shifted dramatically into the modern day. With adequate food supplies, having excess body fat is no longer a necessity for survival. Through modern advertising, the thin ideal has come into effect. This ideal dictates that lean and trim bodies are more desirable. This new ideal image also brought new stereotypes about body types. These stereotypes, however, have developed without the general knowledge of the metabolic disorders that make obesity a far-reaching condition.

Today, a person having excessive body fat can encounter many disadvantages in society. Many general stereotypes are assigned to obese people, with lazy and incompetent being two of the more common. The stereotypes are, in large part, are negative and assigned as a way to explain why a person is fat. For instance, upon first impression, many people assume that an obese individual is lazy, uncontrolled, and unmotivated. Those three characteristics would, in general opinion, explain why and how a person could accumulate a disproportionate amount of body fat.

In a less malicious form, obese individuals may be seen as jolly or funny. Often, an obese person can be assumed to take on a Santa Claus of funny-fat-man role. While these do not seem to be as harmful of stereotypes as lazy and incompetent, the underlying

assumption is that the obese person is less of a person and more of an object of ridicule. While the image of the funny-fat-man has taken many classic roles in American movie history, this image is rarely associated with high levels of respect or responsibility. More commonly, the funny-fat-man is irresponsible, unkempt, and has poor decision-making skills.

Despite the images of obese people being lazy and having uncontrolled eating habits, many obese people put forth significant effort to lose weight, only to face more significant challenges. The causes and mechanisms of obesity are being heavily researched. Many questions exist regarding the ability of an obese person to effectively burn stored energy. What are the effects of having too much body fat on a person's metabolic processes? How does an obese person's daily energy requirements compare to that of a normal-weight person? What options are available if traditional exercise places an unsafe amount of stress and force on an obese person's bones? Such questions have to be addressed and considered in the face of stereotypes.

Often, an obese person will encounter many significant complications while trying to lose weight. Many obese individuals have a diminished ability to effectively burn calories and cannot safely do the amount of exercise needed to produce sufficient calorie loss. Additionally, many established biochemical medical conditions can further hinder an obese person's attempts at weight reduction. Condition resulting in diabetes, high blood pressure, clogged arteries, and poor oxygen capacity are all common to an obese person, and can all markedly reduce the ability of an obese individual to lose weight.

Knowledge of the medical and social challenges facing obese individuals can help alleviate the social stigma placed upon obesity. Many people will not ridicule a quadriplegic for not being able to comfortably navigate narrow grocery store aisles; however, the sight of an obese individual knocking over cans of tomato soup while turning a corner may bring hysterical laughter to onlookers. While both individuals face significant medical and social complications, the basic knowledge exists explaining the need for a wheelchair. Perhaps if a similar knowledge of the metabolic and social issues facing obese individuals was wider spread, more compassion would be felt and the stereotypes would not be as prevalent in society.

The image of overweight people has transformed over time. While being obese was once a necessity for survival, and wildly erotic, the idea of having excessive body fat has been attached to characteristics such as laziness, incompetence, and sloppiness. As research continues to discover more reasons for obesity, the expanding knowledge base has the potential to take the stereotypes out of circulation.

SEE ALSO: Access to Nutritious Foods; American Obesity Association; Appearance; Assessment of Obesity and Health Risks; Back Pain; Central Obesity; Eating Disorders and Obesity.

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Steroids

STEROIDS, OR STEROID hormones, are fat-soluble molecules whose chemical structures are derived from cholesterol. Found in both plants and animals, these small molecules regulate many developmental and physiological processes within the organism. They function by binding to and activating proteins called hormone receptors located in the nucleus of the cell. These activated receptor proteins then bind to DNA, turning on or off specific genes within the cell.

There are five major classes of steroids in the human body: progestagens, androgens, and estrogens, also known as sex hormones, affect sexual development and function; glucocorticoids regulate carbohydrate and lipid metabolism and have an inhibitory effect on inflammation; and mineralocorticoids help regulate the balance of electrolytes in the body and affect blood pressure through regulation of the fluid amounts in the body and vasculature.

Fat storage cells, also known as adipose tissue, play an important role in metabolizing steroid hormones,

A high proportion of fat to muscle in the body can disrupt the normal balance of steroids in the body.

interconverting them between different forms. A high proportion of fat to muscle in the body can therefore disrupt the normal balance of steroids in the body.

Glucocorticoids such as cortisol increase the uptake of lipids into fat cells by increasing the expression of lipoprotein lipase (LPL), an enzyme found on the cell's surface that helps remove fatty acids from lipoproteins circulating in the blood. Excess glucocorticoid levels lead to a redistribution of body fat, mostly in the abdomen but also in the face, producing a "moon face" characteristic of Cushing syndrome. The sex steroid hormones are believed to act in an opposite manner, decreasing lipid uptake into adipose tissue. Testosterone in particular has a strong effect, leading to lower body fat content in men.

The reduction of sex hormone levels that occurs with age is associated with an increase in obesity, bringing with it risks for cardiovascular disease and Type 2 diabetes. Hormone replacement therapies in older women and men can alleviate this problem, but have side effects which limit their use.

Medically, steroids are used to reduce swelling, increase the level of red blood cells, ease breathing, and replace low levels of hormones in the body as well as for treatment for delayed puberty, impotence, or in conditions such as acquired immunodeficiency syndrome (AIDS) or cancer. Side effects of steroid use include an increased appetite, bitter taste in the mouth, and cellular swelling. This may cause a transient rise in a person's weight.

Anabolic steroids, also known as roids, are derived from testosterone and promote the development of muscle mass and enhance bone structure. They have many legitimate medical uses, but have received a bad reputation due to their controversial use in competitive sports. These hormones are often used by athletes to increase muscle mass and strength, but have serious side effects, including virilization (the development of male secondary sexual characteristics in women), severe acne, increased blood pressure, elevated cholesterol levels, and possibly liver damage.

SEE ALSO: Cortisol; Cushing Syndrome; Glucocorticoids; Hormones; Lipoprotein Lipase.

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Stigmas against Overweight Children

ONE OF THE most significant consequences of being an overweight child is the stigmatization and discrimination he or she often faces at school, in playgroups and after-school activities, and, at times, in the home. Although the prevalence of overweight in children has increased greatly over the past three decades, the stigma of being overweight has been an enduring part of childhood and adolescence for much longer. This was first brought to light when Stephen A. Richardson and his colleagues set out to look at the stigmatization of handicaps and found that children stigmatized overweight children; they consistently ranked an overweight child as less desirable to befriend than a child with a broken leg, a child in a

wheelchair, a child with a hand missing, and a child with a disfigured face. This study and finding has been replicated numerous times since the original 1961 study with children of different ages and of varying cultural backgrounds.

Sociologists and psychologists conceptualize stigma as the possession of a trait, either ascribed (from birth, such as race) or achieved (a trait someone acquires later in life). Overweight is a particularly complicated stigma because there is debate as to whether weight is an ascribed trait, such as from one's genes, or if it is an achieved trait that results from overeating and underexercising. Although researchers generally agree that it is both an ascribed and achieved trait, meaning that those with a genetic predisposition are more likely to suffer the consequences of overeating and underexercising, the general public tends to blame people for their weight, hence further stigmatizing them. There are numerous consequences for both adults and children who are stigmatized.

Bruce Link and Jo C. Phelan conceptualize stigma as a combination of being labeled, stereotyped, and separated from the rest of society, in addition to losing status, and being a victim of discrimination. Once a person is labeled as "fat," he or she is also stereotyped as being lazy, sloppy, and ugly. This is particularly harmful for children who are in their formative years. Every experience a child has leaves a lasting effect because children are not as adept at compartmentalizing and rationalizing negative experiences as adults are. Moreover, children's self-concepts start to form at a very young age and they inevitably internalize the fat label and the stereotypes with which it is associated. Therefore, their confidence, sociability, and grades often suffer, leading overweight children onto a path of disadvantage and discrimination.

Recently, schools around the country have received attention for efforts they are making to reduce childhood overweight; however, they may inadvertently add to childhood stigma. Many of these efforts are innocuous efforts to increase physical activity for all children, such as "walking school buses" where children follow the school buses traditional route, picking up other children at various points, yet walk to school instead of bus to school. Other efforts, however, are more controversial and potentially harmful for children. For example, some schools have resorted to placing children's body mass indexes (BMI)—a ratio of weight in kilograms to height in meters squared—on their report cards. Other schools have taken this further by singling out children whose BMI is above the 95th percentile and sending letters to their parents telling them of their child's weight problem and/or inviting overweight and obese children to special fitness programs.

While these programs can have significant benefits for some children, they can also exacerbate the stigma associated with overweight for children, both socially and psychologically. School-aged overweight children are generally aware that they are overweight. Consequently, the self-protective mechanisms they employ to deal with the social isolation and taunting that frequently accompanies it are dismantled when it is reinforced by a letter to the home stating that they are not only not accepted by their peers, but by school officials, and now, potentially their family. Thus, these letters confirm some children's worst fears that they are not accepted by anyone.

Studies have shown that both children and adults are more likely to stigmatize overweight people of all ages if they perceive it to be the overweight person's fault. Instead of blaming the children or their parents for their weight, it would be advisable to look for ways to improve school menus, access to physical activity, proximity of schools to fast food, and how food is advertised to young children. Dr. Kelly Brownell and his colleagues at the Rudd Center for Food Policy and Obesity have pioneered these efforts, and they are slowly being implemented. Community-wide efforts blame everyone and no one. As a result, they reduce the potential for stigmatizing the most vulnerable—the children.

SEE ALSO: Overweight Children and School Performance; School Based Interventions to Prevent Obesity in Children; Self-Esteem and Children's Weight.

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Stress

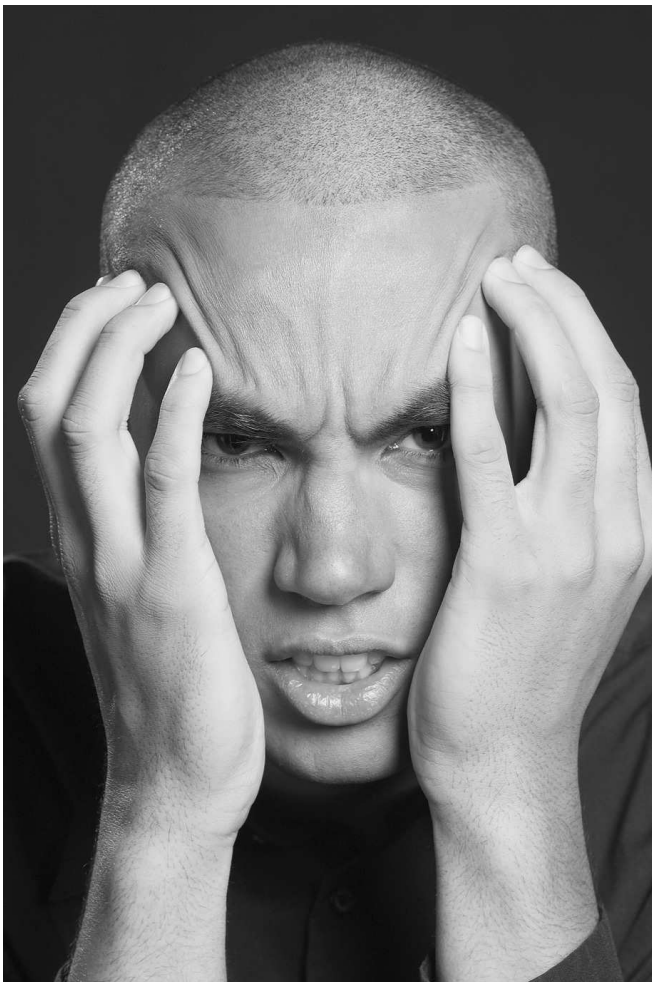
HUMAN STRESS IS the result of too much demand, or strain, on the body. The fight or flight mechanism, where the body chemically responds to a strain by creating adrenalin, glucocorticoids and other hormones that enable extraordinary athletic feats in a time of peril (fight) or rapidly running away from an aggressive predator (flight), is central to the concept of stress.

The downside of stress is that it can be linked to health problems and obesity. Historically, researchers posited that stress was the result of the body's attempt to combat strain and maintain homeostasis. The idea was that when the body ran out of the resources to combat whatever strain it was exposed to, it would start to break down and have mechanical problems such as diabetes. From there, we moved to the belief the problem was really the buildup of the chemicals that the body was creating in an environment that did not allow for fight or flight. The chemical stress response was seen to erode soft tissues and create problems such as ulcers.

Current research is focusing less on the body wearing down or not having enough resources. In fact, we now believe that stress can be alleviated by finding ways to cope with the strain better. Coping can include things like dietary changes to reduce the risk of heart failure (reducing fat and sodium intake), behavioral changes like getting more sleep or taking a walk, mind altering techniques to change the perception of the strain (counseling or participating in yoga), and medications to reduce anxiety.

What is the relationship between stress and obesity? Stress has several dichotomous associations that interact with obesity. The first of these divisions is between good stress called eustress (losing weight from walking more) and stress that we respond to negatively called distress (becoming hungry, tired, and grouchy after a long walk on a hot day). Juxtaposition is also found between stress that results from physical strains (weighing too much) and stress coming from psychological strains (hearing someone refer to you as fat). Stress can additionally be divided by duration: that which is the result of an acute event (trying to exercise more and pulling a ligament in one of your joints because they are not used to moving in that manner under that much weight) and that which is chronic or continuous (not having the energy to cook dinner after going home from work). Last, stress has a cyclical relationship with obesity. Some research supports the idea that stress can precede becoming overweight or obese, and other works demonstrate how being obese causes a lot of stress on the body.

It is important to remember that not everyone responds to strains in the same way. When a person is energized by strain, the result is called eustress. Eustress, or good stress, can result from an overweight person losing weight. The end result may be renewed effort, anticipation of rewards, or the decision to make constructive changes in the environment that surrounds the individual. When looking at obesity, eustress is usually associated with successful and intentional weight loss.



Popular culture abounds with the belief that people will make poor food and exercise choices when under strain.

The same event that is seen as helpful and rewarding by one person may be thought of as disabling or restrictive by another. When someone feels stressed, they tend to respond with coping behaviors. Some coping behaviors attack what caused the problem and others revolve around avoidance. Attacking or correcting the problem allows the individual to minimize the effect of the strain and cope with it. Avoidance may work well in some instances, but frequently the situation remains in the individual's mind as a stressor. For instance, drinking alcohol or eating ice cream after being fired does not help the situation.

These behaviors allow the person to feel better for a limited amount of time. They may even become addictive behaviors, but the stressor is still there. Coping behaviors are learned behaviors, such as conditioned food preferences. Many of these behavoirs were established during childhood. We often think of food as symbolic of a familial love and comfort, so reaching for comfort food in times of strain is a common response. Distress, or bad stress, is also represented by the relationship between obesity and depression.

Being overweight puts a physical strain on the body. Excess weight is considered to be a risk factor for mortality and a variety of physical ailments, like Type 2 diabetes, certain cancers, high blood pressure, heart problems, and a variety of muscle and joint problems. Epidemiology has not yet shown if the physical distress on the body from being overweight causes the diseases that obesity has been labeled a risk factor for, or if the etiology of these diseases also leads to obesity. The risk associated with these diseases often increases for individuals who yo-yo diet or lose and regain weight over and over again.

When we think of psychological strain, we tend to think about people ruminating over events that have happened in the past, thinking about them over and over again, and creating anxiety. Psychological strain can originate from prejudice and inequality. Caloric restriction and repeated diet failures also create psychological strains. People can learn a feeling of inadequacy without ever realizing why they feel the way they do. Psychological strains alter both health and future health behaviors.

Acute strain comes from a significant life-changing event. One acute strain is parental divorce. Some life-course researchers look to see if there are lasting relationships between experiencing parental divorce as a child and adult behaviors like forming bonds with other adults or emotional eating. The adult behaviors that are associated with traumatic childhood events are often also associated with health behaviors such as using food for comfort and compulsive overeating.

The hormones that are involved in human stress are epinephrine, norepinephrine, and cortisol. A challenge to a human can result in the release of norepinephrine, the primary hormone released in the "fight" response. If the stress continues, the "flight" response may be activated, and this coincides with release of epinephrine. One of the primary hormones linking stress and obesity is cortisol

Stress may lead to weight change. There are many correlations between strain and the physical ailments associated with obesity. Popular culture abounds with the belief that people will make poor food and exercise choices when under strain. It is believed that some people will lose their appetite when unable to cope with stress and lose weight. Other people talk about eating more comfort foods, such as potato chips, ice cream, and chocolate, when exposed to chronic stressors such as paying bills. The literature shows that increased exercise helps decrease the physical strain that stress puts on the body, but very few people think about increasing their exercise when exposed to chronic strains, and increased exercise also adds physical strain to a body that is carrying extra weight.

Weight gain may also lead to distress. There is the obvious strain of carrying extra pounds. Another strain is created by body image. Living in an environment that disapproves of the way overweight people look drives people to want to be thin. The reality is that very few overweight people have been able to achieve and maintain a thinner body. Yet, overweight individuals are blamed for their own failure.

SEE ALSO: Addictive Behaviors; Anxiety; Caloric Restriction; Compulsive Overeating; Conditioned Food Preferences; Cortisol; Depression; Dieting: Good or Bad?; Economics of Obesity; Ethnic Disparities among Obesity in Women; Exercise; External Controls; Food "Addictions"; Food Intake Patterns; Food Reward; Glucocorticoids; Hypertension; Impulsivity; Loneliness; Metabolic Rate; Mood

and Food; Obese Women and Social Stigmatization; Obesity and Socioeconomic Status; Personal Relationships and Obesity; Self-Esteem and Obesity; Stereotypes and Obesity; Stigmas against Overweight Children; Support Groups for Obese Women; Toxic Environment; Waist-to-Hip Ratio; Weight Cycling and Yo-Yo Dieting; Weight Discrimination; Well-Being; Women and Dieting; Worksite Interventions to Prevent Obesity.

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Stress Urinary Incontinence

STRESS URINARY INCONTINENCE (SUI) is defined as the complaint of involuntary leakage of urine that occurs with a sudden increase in intraabdominal pressure, such as that seen with physical activity, effort, exertion, coughing, or sneezing. This occurs without a simultaneous rise in detrusor (bladder-generated) pressure.

The detrusor urinae muscle expels urine from the bladder. Urge urinary incontinence (UUI) is defined as incontinence due to involuntary detrusor contractions whereby the complaint of involuntary leakage is accompanied by or immediately preceded by urgency. This condition is classified as detrusor overactivity incontinence or bladder overactivity incontinence. SUI seems to be more common than UUI, although some women may have mixed urinary incontinence (MUI).

SUI is a common condition in women of all ages and occurs at least weekly in one-third of adult women. However, many women are reluctant to consult their doctors about their condition even though quality of life is affected. Therefore, it is thought that SUI is greatly underdiagnosed. The three most common risk factors for SUI include aging, obesity, and heavy smoking. In regard to obesity, both UUI and SUI increase proportionately with a rising body mass index (BMI). Additional risk factors included parity, chronic cough, depression, poor health, lower urinary tract symptoms, previous hysterectomy, and stroke. However, the data available regarding the role of pregnancy and route of delivery on pelvic floor disorders such as SUI are not well understood. Dietary factors may include the consumption of carbonated drinks and tea. In addition, SUI may be caused by anatomical problems related to factors such as age, obesity, parity, and menopause.

The cause of incontinence differs by age group with older women more likely to experience UUI and younger women more likely to experience SUI. Ultrastructural changes in the bladder and distinct changes in receptor response provide a partial explanation for the rising prevalence of UUI with increasing age. SUI with advancing age may reflect a general loss of muscle tone, long-term effects of denervation injuries experienced during childbirth, and/or changes in hormonal stimulation, as well as unknown factors. The relationship between smoking and urinary incontinence may be due to a direct effect or indirectly through smoking-related illnesses that cause increased coughing, such as chronic obstructive pulmonary disease.

SUI involves four primary structural defects: (1) increased tonic stress on the pelvic fascia due to pelvic floor muscle (PFM) tears; (2) fascial tearing due to PFM denervation; (3) fascial weakness resulting from tears; and (4) inefficient PFM contraction due to altered motor control. These four components collectively allow urine leakage under conditions of increased intraabdominal pressure.

The basic evaluation of women with SUI includes a history, physical examination, cough stress test, voiding diary, postvoid residual urine volume, and urinalysis. Treatment may include weight loss, pelvic floor exercises, vaginal cones, bladder training, urethral inserts, and general lifestyle modification advice. It can also be treated surgically with procedures such as Burch colposuspension, vaginal slings

or tension-free tapes and injection of bulking agents alongside the urethra.

SEE ALSO: Stress; Urinary Incontinence in Severe Obesity in Women.

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Stroke

OBESITY IS A condition in which excess fat has accumulated in the body, usually considered to be 20 percent above the recommended weight for height and age. Obesity is seen as a worldwide chronic disease with high prevalence that has been associated with increased morbidity from many conditions including stroke.

Obesity is clinically defined as a body mass index (BMI) of between 30.0–39.9; a BMI of 25–30 usually indicates that a person is overweight. A person who is overweight or obese has an increased risk factor for developing hypertension, hyperlipidemia, heart disease, and diabetes mellitus, all of which increase the risk of stroke. Obesity, however, is an important modifiable risk factor for stroke. Risk-factor modification is the most important aspect of prevention of stroke in obesity. This includes lifestyle modifications and different therapeutic modalities to control it. Cardiovascular morbidity and mortality related to obesity are due to increased levels of leptin (hormone which plays a role in regulating energy intake and expenditure), dysregulation of adipocyte proteins, increased insulin resistance, and elevated levels (>3mg/L) of basal C-reactive protein.

Stroke is the third leading cause of death in developed countries and a leading cause of severe long-term disability. The prevalence of stroke worldwide is between five and 10 per 1,000 population. Stroke is an important cause of morbidity and mortality, and is an economic burden.

Stroke occurs as a result of a sudden occlusion of an artery (or less commonly vein) supplying the brain. The blood supply of the brain is provided by two pairs of arteries, (the right and left internal carotid arteries and the right and left vertebral arteries) and their branches and also the anastomotic circle of Willis which enables a continuous interrupted blood flow to the brain in the event of interruption of blood flow. There are two forms of stroke: ischemic or hemorrhagic. The former is the most common type; it occurs when an artery is obstructed by a clot reducing the amount of blood and oxygen reaching the brain. Hemorrhagic strokes are caused when an artery ruptures.

In general, surgery, medications, hospital care, and rehabilitation (e.g., physical therapy, speech therapy, and occupational therapy) are all accepted stroke treatments. Ischemic strokes may be treated in the immediate period with a clot-dissolving drug tissue plasminogen activator (tPA). Antiplatelet drugs such as aspirin are used to reduce the risk for recurrent stroke; aspirin may also be used to improve the outcome of stroke when administered within 48 hours. Some patients may also take an anticoagulant such warfarin. Hemorrhagic stroke usually requires surgery. Types of surgery employed in stroke treatment include carotid endarterectomy (removes plaques) and cerebral angioplasty.

There are times where one may not suffer a full stroke but instead a transient ischemic attack (TIA) which is an obstruction in the supply of blood to the brain for a short period ranging from a few minutes to around 24 hours. Patients often recover from TIA; however, they are advised to be cautious because they now have an increased risk of suffering a full stroke.

The symptoms of a stroke include weakness such as hemiplegia (paralysis in vertical half of body), hemiparesis (partial paralysis in vertical half of body), vision problems, confusion, dizziness, and incoordination.

There are many risk factors for developing stroke; these include diabetes mellitus, and modifiable factors such as hypertension, cigarette smoking, and obesity. It is reported that for each unit increase in BMI (equivalent to a weight gain of ~7.4 lb for a 6-foot man and 6.2 lb for a 5-foot 6-inch man), the chances of having a stroke increase by six percent.

There are many measures advised to prevent the occurrence of stroke. As BMI is a modifiable risk factor, the prevention of stroke may be another benefit associated with preventing obesity in adults. A healthy diet is paramount; minimal salt (sodium chloride) intake is important as an increased salt intake increases blood pressure. It is also recommended that the intake of fats should be of the polyunsaturated type, such as omega-3 fatty acids, and low in trans-fat and glycemic load. Intake of omega-3, however, should be monitored as an abnormal increase in intake can increase the possibility of hemorrhagic stroke.

The intake of fiber found in foods such as wholemeal bread and brown rice and a diet high in folate and potassium is encouraged. A diet with high intake of fruits and vegetables (recommended five portions of fruit and vegetables a day) and decreased intake of fats (which reduce levels in serum cholesterol), decreases fasting lipids by 5 to 10 percent and hence reduce the risk of stroke. Statin drugs are used in lowering the levels of fasting cholesterol as they can lower fasting lipids by 50 to 60 percent.

Studies suggest a Cretan Mediterranean diet which is high in beneficial oils, whole grains, fruits, and vegetables and low in cholesterol and animal fat, has been shown to reduce stroke and myocardial infarction by 60 percent and hence it is suggested that this type of diet may be even more effective than statins as it has additional antioxidants from the many fruits and vegetables. A diet that replaces animal protein with vegetable protein is recommended, as eating less meat has been suggested to reduce the risk from coronary heart disease. Other ways to prevent stroke include exercise, refraining from smoking and drinking alcohol.

A stroke, which can occur suddenly, is the third leading cause of death in developed countries and a leading cause of disability.

Undoubtedly, obesity is major risk factor for the development of stroke. Controlling weight is an issue that is paramount in reducing the detrimental effects not only on the individual but also on society. Issues such as the fact that patients are more likely to be kept in the hospital after a stroke than their leaner counterparts demonstrates that obesity associated with stroke also places an increased burden on the economy.

SEE ALSO: Cardiovascular Disease in African Americans; Cardiovascular Disease in Asian Americans; Cardiovascular Disease in Hispanic Americans.

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Sugar and Fat Substitutes

SUGAR AND FAT substitutes are used in foods to mimic the taste and organoleptic properties of sugar and fat, yet provide fewer calories and/or lipid. The development of sugar and fat substitutes has enabled the creation of a wide array of dietetic foods, but the efficacy of these food products in weight-loss programs remains unproven. Research indicates that sugar and fat substitutes, when used to reduce overall calorie intake, produce weight loss in feeding studies. However, in public use, there is limited evidence whether these products actually aid consumers' weight-control efforts. Sugar and fat substitutes are widely accepted by the public and are used by millions of people every day in popular foods, but some consumers question the safety of these artificial ingredients and choose to avoid them.

As with other novel food ingredients, sugar and fat substitutes must be preceded by extensive safety testing prior to approval for use in the food supply, and the U.S. Food and Drug Administration (FDA) maintains that they are safe. There is currently no consensus on whether sugar and fat substitutes should be recommended by nutrition and healthcare professionals as dietary modifications to prevent or treat obesity, and whether to consume sugar and fat-modified food products remains an individual's decision.

Sugar substitutes are accepted by the public but some consumers question the safety of artificial ingredients and choose to avoid them.

Sugar and fat substitutes exist in the modern food supply in a variety of forms. The number of available FDA-approved fat and sugar substitutes continues to grow in number, each possessing unique attributes for food and beverage application. These ingredients have enabled the development of highly palatable food, beverage, and confectionary products with reduced or eliminated fat, sugar, and total calories.

While research shows that use of sugar- and fat-modified foods results in decreased energy intake during short-term blinded studies, the widespread use of these compounds in food has been insufficient to slow the climbing obesity rate on an epidemiological scale. One proposed theory maintains that with time, dissociation of perceived sweet taste with energy intake may interfere with appetite regulation. Psychological and behavioral issues also influence the success of sugar and fat substitutes in producing weight loss. Clearly, sugar and fat substitutes can only elicit weight reduction if their consumption leads to a cumulative energy deficit. Use of diet foods as an excuse to eat unlimited quantities is a common problem for unsuccessful dieters. However, the ability to consume highly palatable foods in a less calorically dense form can favorably affect dieter adherence to reduced-calorie regimens, improve quality of life, and avoid weight regain.

SUGAR SUBSTITUTES

Sugar substitutes, also known artificial sweeteners, impart a sweet taste to foods but either are not metabolized by the body (nonnutritive sweeteners) or are used in such small amounts as to provide negligible calories. The FDA currently has approved five artificial sweeteners as food additives: saccharin, aspartame, acesulfame-K, sucralose, and neotame. Because these products are intensely sweet, only small amounts are needed to replace the sucrose in foods. Thus, even caloric sugar substitutes contribute insignificant energy to the food or beverage. Commonly, recipes using ar-

tificial sweeteners must also include starches or other ingredients to account for bulking, moisture-retaining, or browning properties of sugar, which the substitutes lack. In addition to reducing the calorie content of foods, sugar substitutes offer additional benefits that appeal to some consumers. Diabetics may benefit from improved glycemic control when replacing digestible sugars with noncaloric sweeteners, and unlike sugar, artificial sweeteners do not promote tooth decay.

Saccharin, available under the trade name Sweet 'N Low®, is 300 times sweeter by weight than table sugar. Discovered accidentally by scientists at Johns Hopkins University in 1879, it was the first artificial sugar substitute. Saccharine passes through the digestive tract without being absorbed, thus contributing no food energy. Health concerns over saccharin mounted in 1977 after a large animal study linked the sweetener to bladder cancer in rats. A proposed ban on saccharin was met by strong public opposition, particularly among diabetics. While the sweetener remained on the market, legislation required a mandatory warning be included upon packaging of products containing saccharin. Extensive studies since then have not established causality between saccharin consumption and cancer in humans, and review of prior research has cast doubt over the conclusions drawn from the early experiments. In 2000, Congress repealed the law requiring health warning labels on saccharin products.

Use of saccharin in the food industry has been replaced in many instances with newer artificial sweeteners that more closely resemble the taste of sugar, that is, without saccharin's bitter aftertaste. Yet saccharin still maintains a market share due to its advantageous long shelf life. In many products such as fountain soft drinks, a combination of saccharin and aspartame is used to yield the most shelf-stable and palatable product.

Aspartame, commonly seen as Equal®, Canderel®, and NutraSweet® brands, is about 200 times sweeter than sugar. Aspartame is formed by joining two amino acids, aspartic acid and phenylalanine, with an additional methyl group. Upon digestion, these compounds are broken down and absorbed, similar to amino acids provided by proteins. Like other peptides, aspartame contributes 4 kilocalories per gram, but because the amount used in food products is minute, the energy provided by the sweetener is negligible. Aspartame is widely used in diet soft drinks, powdered drinks, sugarless gum, chewable vitamins, and yogurts. It is undesirable in baked products, however, because its sweetness is lost when exposed to high heat.

Like saccharin, aspartame has been a topic of health controversy. Some people report side effects with aspartame use, and urban myths are widely circulated via the Internet alleging a link between aspartame and brain tumors. More than 200 studies over a 30-year period have supported the safety of aspartame, and the consensus among regulatory agencies is that aspartame is safe. However, people with the inherited disease phenylketonuria (PKU) must avoid aspartame entirely. This rare disease disrupts the body's ability to metabolize the amino acid phenylalanine, and PKU patients must adhere to a strict low-phenylalanine diet. Because aspartame contains phenylalanine, foods containing aspartame must say "Phenylketonurics: Contains Phenylalanine" in the United States.

Acesulfame potassium, also known as acesulfame K, is a noncaloric sweetener which is not metabolized by the body. Acesulfame potassium is sold under trade names Sunett® and Sweet One®. It was discovered in 1967 and granted general purpose approval in the United States in 2003. Acesulfame potassium is 130 to 200 times sweeter than sugar, and is used in a wide variety of products such as chewing gum, candies, baked goods, ice cream, and carbonated and alcoholic beverages. Because acesulfame potassium is heat stable, it is suitable for baked products and pasteurized beverages. When combined with other caloric or noncaloric sweeteners, acesulfame potassium has a noted synergistic effect on the sweetness. Likewise, it is known to heighten other flavors in food, an additional desirable trait.

Sucralose is an artificial sweetener which is 600 times sweeter than table sugar. Discovered in 1976, sucralose was approved for general use in the United States in 1999. Marketed as Splenda®, sucralose rapidly gained popularity. Within seven years of its introduction, sucralose accounted for nearly 60 percent of the artificial sweetener market previously dominated by aspartame. Heat-stable and suitable for baking and cooking applications, Splenda is available in yellow packets as a tabletop sweetener, as well as in granular forms for home cooking use. For ease of measuring, maltodextrin and dextrose are blended with sucralose to yield a product which can be measured cup for cup like sugar. One cup of Splenda granular provides 96 calories, compared to 774 calories in a cup of sugar.

McNeil Nutritionals, the maker of Splenda, faced a lawsuit from Merisant Co., the maker of Equal, over its advertising claims that Splenda is "Made from sugar, so it tastes like sugar." Merisant disputed that this statement misled consumers to believe that Splenda was a natural product and healthier than other artificial sweeteners. Splenda contests that the manufacturing process begins with real sugar and that its product is simply backed by better marketing. The suit was settled in May 2007 for an undisclosed amount.

Neotame is the most recently approved artificial sweetener in the United States, approved in 2002. Neotame is moderately heat stable and intensely sweet: 8,000 to 13,000 times sweeter than sugar by weight. Aspartame and neotame are structurally similar; however, neotame is not cleaved by proteases into its respective amino acids. For this reason, the phenylalanine in neotame is unabsorbed and it is safe for use by people with PKU. Neotame is not yet widely used in food products, but it is uniquely desirable to food manufacturers for its cost effectiveness, being more economical than other artificial and natural sweeteners.

FAT SUBSTITUTES

Fat substitutes, or fat replacers, are used to mimic one or more properties of fat in food. Some evidence indicates that consuming fat-modified foods may help achieve a lower overall fat intake, but whether these products significantly aid weight loss is still unclear. Fat substitutes can be classified into three structural categories: carbohydrate-, protein-, or lipid-based ingredients.

Carbohydrate- and protein-based fat substitutes generally produce acceptable fat-like textures in food products, but without contributing the flavors associated with fat. These types of fat replacers are optimally used in foods with high water content which are not subjected to cooking or great fluctuations in temperature. Carbohydrate- and protein-based fat replacers are viewed as safe and well understood, posing no physiological concerns. They are widely used in many products today, including reduced-fat salad dressings and frozen desserts.

Carbohydrate-based ingredients which can replace fat in foods include modified glucose polymers, modified starches, pectins, gums, and cellulose derivatives. Protein-based ingredients include native proteins from dairy or plant sources as well as microparticulated proteins. Simplesse®, a microparticulated protein fat replacer introduced by the Nutrasweet Co., consists of whey and egg proteins which are processed into uniform spheres, approximately 1 micron in diameter. When incorporated into foods, these tiny balls create a smooth, creamy sensation on the tongue. Carbohydrate- and protein-based fat substitutes are generally fully metabolized, yet can reduce the energy content of fat-modified foods owing to their low caloric density.

Lipid-based fat replacers have different properties than those made from proteins or carbohydrates. Fat replacers which are derived from lipid can often provide a flavor profile and sensory properties indistinguishable from the native fat, yet are formulated to completely or partially bypass absorption. Therefore, these fat replacers may produce foods with the same caloric density as unmodified foods, yet the actual energy derived from the product may be significantly less.

The most well-known lipid-derived fat substitute is olestra, sold under the trade name Olean® by Procter & Gamble. Structurally, olestra is a sucrose polyester. In other words, it is a sugar molecule to which six to eight fatty acid chains have been attached. The resulting molecule is much larger than a standard triglyceride found in nature and cannot be hydrolyzed by digestive enzymes. Olestra tastes and cooks just like regular fats, but because it is indigestible, olestra contributes zero calories and zero fat to the food. Potato chips, tortilla chips, and other fried snacks can be cooked in olestra instead of traditional oils, and have been available on store shelves since 1996.

Concerns that have arisen relating to olestra consumption stem from the side effects that may occur when large amounts are consumed. Because the olestra molecule is undigested, it passes through the digestive tract and is excreted intact. Consuming large amounts of olestra can cause diarrhea and reduces the absorption of fat-soluble vitamins which bind to the lipid chains. To address these concerns, fat-soluble vitamins are added to foods containing olestra to compensate for inhibited absorption, and products containing olestra are required by the FDA to carry the following label: "This Product Contains Olestra. Olestra may cause abdominal cramping and loose stools. Olestra inhibits the absorption of some vitamins and other nutrients. Vitamins A, D, E, and K have been added." Consumption of olestra-containing snack foods in single servings is unlikely to cause adverse effects, but this unap-

pealing label likely raised consumer concerns enough to slow sales of the product.

Most nutrition experts agree that sugar and fat substitutes, when used in moderation, can have a place in a healthy diet. Weight-loss diets that incorporate sugar and fat substitutes may have better patient compliance due to the increased flexibility and palatability of allowed foods while maintaining energy restriction. Available sugar and fat substitutes have undergone safety and toxicology testing prior to approval by the FDA, and no evidence indicates that these ingredients pose threats to consumer health when consumed according to guidelines.

SEE ALSO: Fat Taste; Flavor: Taste and Smell; Sweet Taste.

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Suicidality

INDIVIDUALS WITH EATING disorders and weight concerns are at increased risk for suicide and suicidal behavior. Suicide, or the act of intentionally killing oneself, and related thoughts and behaviors are of serious concern, with suicide deaths claiming the lives of over 32,000 individuals annually in the United States alone. Eating disorders and obesity have been linked to depressive symptomology, which is the strongest predictor of suicide risk. Additionally, suicidal ideation, or thoughts and/or plans of killing oneself, suicide attempt, and completed suicide have been found to be associated with weight, body image perceptions, and eating habits. Therefore, risk of suicidal thoughts and behavior is an important factor for consideration in populations of the obese, severely underweight, or those suffering from an eating disorder.

Weight extremes in either direction have been found to be associated with increased risk of suicidal ideation and behavior. Both individuals who are underweight and those who are obese exhibit increased depressive symptoms and suicidality, compared to those of average weight. This may be a function of actual weight status, or may be a reflection of weight dissatisfaction. Weight perceptions and dissatisfaction, independent of body mass index (BMI), have been linked with consideration of suicide and suicidal actions. Research indicates that weight dissatisfaction may be more influential on suicidality than actual weight. Those who believe themselves to be extremely underweight or extremely overweight in relation to the norm are at elevated risk for suicidal ideation and attempt. Even those who perceive themselves as deviating slightly from what they believe to be their proper weight demonstrate higher rates of suicidal ideation than those satisfied with the status of their weight.

Others' perceptions of and reactions to an individual's weight can also play a role in development of suicidal thoughts and behaviors. Individuals who have been teased about their weight by family members and/or peers report experiencing more thoughts of suicide and making more suicide attempts. The link between weight-based teasing and suicidal behavior appears to exist independent of an individual's actual weight status. Risk of suicide in obese individuals in some cases may not decrease with weight loss, especially in the presence of a preexisting mood disorder, which suggests that psychological processes play a significant role in the link between weight and suicidality.

Deviant eating behavior is also linked to suicidal behavior. Higher rates of suicide have been consistently demonstrated in individuals with anorexia nervosa and, to a lesser degree, in bulimia nervosa as compared to the general population. Disordered weight control methods (e.g., purging, fasting, diet pill use) in general have been linked to increases in suicidality. No research currently exists regarding the rates of suicidality in individuals with binge-eating disorder. However, self-reported overeating and binge eating, behaviors that are

associated with higher BMI, are noted correlates of thinking about and attempting suicide.

Differences exist in the link between weight and suicidality for males and females. In general, higher BMI is associated with increased suicidality for adult and adolescent females. However, this relationship may not exist or the converse may be true for males. Underweight status has been more consistently linked with suicide ideation, attempt, and completed suicide in males. In some cases, a higher BMI has been associated with greater reported quality of life and decreased risk of suicide in male cohorts. There are several proposed explanations for this discrepancy.

In Western countries, a slender body size is typically idealized and expected for women. In contrast, an average or overweight body size is seen as more acceptable for males, and musculature is more often idealized. In fact, males who are underweight are often taunted for their body size as much as, if not more than, overweight males. It could be expected that higher suicidality would be reported for those who deviate from culture's idealized body standard for their gender. In the case of females, this would refer to overweight women, whereas for males it would refer to underweight men.

Some suggest that there may be a common biological mechanism, such as circulating levels of insulin or cholesterol, linking a higher BMI with a decreased risk of suicide, which is being expressed in males. If such a mechanism exists, the particular demands of cultural body ideals may be strong enough to override its expression in females. However, researchers urge caution in interpreting the results of these studies. It is not advisable to counsel men of average weight to gain in order to protect against suicide risk due to the numerous negative health consequences associated with obesity.

Ethnic differences have been suggested as well; however, evidence is mixed and results thus far are unclear. In one study of high school students, Caucasian students were more likely to attempt suicide if they perceived themselves to be very underweight or very overweight, whereas African-American and Hispanic students were more likely to attempt suicide only if they perceived themselves as very underweight. This may be a reflection of differing cultural body ideals and increased acceptance of overweight in groups of ethnic minorities. However, other studies have failed to find such an effect of ethnicity; therefore, further examination is required before further speculation can be made.

Research indicates that risk of suicide increases with the number of risk factors present at a given time. Therefore, clinicians who observe one or more risk factors for suicidal behavior are urged to evaluate other factors that may put an individual at danger. Noted predictors of suicidal behavior include life stresses (such as problems with family life, social status, employment, and finances), troubled childhood (including history of physical and sexual abuse), family history of suicide, mental illness, addiction, and history of self-harm and/or prior suicide attempt. Some individual characteristics, such as impulsivity, low self-esteem, rigid thinking, and hopelessness, are also associated with suicide risk. Because weight, body size perception, and disordered eating have been linked to suicidal behavior, clinicians are urged to thoroughly assess suicidality when dealing with those who are obese, severely underweight, and those who exhibit eating disturbances.

SEE ALSO: Depression; Loneliness; Mortality and Obesity; Quality of Life; Self-Esteem and Obesity.

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Supersizing

THE TERM SUPERSIZE entered the American lexicon by way of fast-food restaurants. In an effort to increase sales and profits, many fast-food restaurant chains introduced larger food and beverage portion sizes and packaged them together with other items at prices cheaper than if they were purchased separately. The use of the term undoubtedly increased after the release of the documentary film *Super Size Me* by director and protagonist Morgan Spurlock. In the film,

Spurlock subsists exclusively on food from McDonald's for 30 days. During this time, he consumes an average of 5,000 calories per day and limits his physical activity to walking no more than 5,000 steps per day. As a consequence, he gains 25 pounds, his triglycerides go up, and his liver function deteriorates. The regimen that he exposed himself to was certainly extreme, but it pointed out the potentially harmful effects of being physically inactive and consuming a high-calorie diet made up exclusively of fast food.

Economic considerations have motivated the practice of supersizing for both restaurants and consumers. The practice is particularly profitable for fast-food restaurants because the cost of the food ingredients represent only a small proportion of the total cost of selling the products. They can sell their larger-portion-size products at higher prices without substantially raising their costs. In the process, consumers get more food for less money. Consumers also do not have to purchase more than one order to get more food. Many people want more of a particular food item, say fries, but feel self-conscious purchasing more than one order, believing that they may be perceived as being gluttonous. Supersizing allows people to get more fries and seemingly not be seen as overdoing it, making it more socially acceptable to eat more food. Supersizing would almost seem like a win–win proposition for the fast-food industry and consumers, except for the fact that more food, and in particular more fast food, is very likely to have contributed to increases in rates of obesity.

Many of the foods sold at fast-food restaurants are highly processed, low in fiber, and high in sugar, salt, fat, saturated fat, and calories. These foods are also highly palatable and their consumption is very reinforcing. Americans are eating a larger proportion of their meals at restaurants and at fast-food restaurants in particular. Americans, finding themselves too busy to prepare meals at home, have begun to rely more and more on the increasing number of fast-food locations with increasingly extended hours of operation to do their cooking for them.

In at attempt to shed light on the question of why French adults, who have a penchant for foods high in fat, are significantly thinner than American adults, one study compared the portion sizes and time spent consuming meals in restaurants in the United States and in France. The portion sizes in the United States were significantly larger and the time spent eating meals was considerably shorter. Individuals presented with larger portion sizes tend to consume more calories than they would otherwise and food served fast and consumed fast can contribute toward overeating because there is a lag time between consumption and satiety.

Many fast-food chains recently stopped offering the gargantuan-size options after receiving negative public attention for their apparent contribution to the problem of obesity. Many chains have also made attempts to offer more nutritious alternatives such as salads and fruits.

SEE ALSO: Fast Food; Food Intake Patterns; Portion Control.

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Supplements and Obesity

DIETARY SUPPLEMENTS ARE regulated in the United States as foods, not as drugs. Dietary supplements can be consumed by the obese to supplement a restricted diet, or, more likely, in an attempt to aid in weight loss. Although billions of dollars are spent each year in the United States by consumers buying supplements to aid weight loss, there is little to no scientific evidence at this time that dietary supplements aid in significant weight loss.

DEFINITION OF A SUPPLEMENT AND REGULATION

In the United States, a dietary supplement is defined under the Dietary Supplement Health and Education Act (DSHEA) of 1994 as a product that is intended to supplement the diet and contains one or more of the following dietary ingredients:

Individual vitamin and mineral supplements taken in large amounts (that is, well above the recommended daily allowance) can be toxic.

- a vitamin,
- a mineral,
- an herb or other botanical (excluding tobacco),
- an amino acid,
- a dietary substance for use to supplement the diet by increasing the total dietary intake, or,
- a concentrate, metabolite, constituent, extract, or combination of any of the above.

Furthermore, it must be:

- intended for ingestion in pill, capsule, tablet, powder, or liquid form; and,
- not represented for use as a conventional food or as the sole item of a meal or diet,
- labeled as a "dietary supplement."

The U.S. Food and Drug Administration (FDA) regulates dietary supplements as foods, and not as drugs. A supplement manufacturer does not need to prove efficacy or safety to sell their supplements. The FDA may step in to regulate supplements only after evidence shows the supplement harmful.

The claims that a dietary supplement makes are subject to regulation by the FDA. If a dietary supplement claims to cure, mitigate, or treat a disease, it would be considered to be an unauthorized new drug and in violation of applicable regulations and statutes.

Adoption of good manufacturing practices, mandatory in manufacturing of processed food items, is not mandatory in manufacturing supplements. Therefore, product quality (contamination, accuracy of labeling, etc.) of supplements is variable and uncertain and is not subject to regulation. Some supplement manufacturers have voluntarily adopted good manufacturing practices and indicate this with seals on their supplements. An example of contamination occurred recently when an herbal weight-loss supplement was found to be contaminated with amphetamines.

USE OF WEIGHT-LOSS SUPPLEMENTS

It is estimated that approximately 7 to 15 percent of U.S. adults use a weight-loss supplement, and almost 75 percent of users consume a supplement containing stimulants such as caffeine and/or bitter orange. The highest use of weight-loss supplements was among obese young women. Recent estimates of sales of weight-loss supplements were approximately $2 billion per year. Interestingly, sales of weight-loss supplements appear to have slipped after the FDA (in 2004) banned supplements containing ephedrine (ephedra).

People with obesity consider supplements for several reasons. Supplements can provide nutrients for diets that are restricted, such as a calorie restriction. Special dietary restrictions can limit essential nutrients, requiring supplementation. For instance, consumers on diets of less than 1,200 calories typically have a hard time obtaining all the recommend dietary allowances (RDA) or adequate intake (AI) levels of certain vitamins and minerals. Sometimes, a multivitamin-mineral supplement may be suggested for consumers on these restricted diets. Single-nutrient supplements may also be indicated. For instance, if the consumer was restricting diary products and did not have an adequate intake of calcium, then a calcium supplement could be used to make up the deficiency. It should be noted that individual vitamin and mineral supplements taken in large amounts (i.e., well above the RDA) can be toxic.

People with obesity most likely consider supplements to aid in weight loss. Certainly, a simple, nondemanding way to lose weight that is available without a prescrip-

tion would be welcome. The social stigma of obesity in America is still strong. Frequently, consumers may be frustrated by lack of progress with dieting and/or exercise. Additionally, sometimes weight-loss supplements are touted as "natural," and many people assume (falsely) that natural means safe. People may also assume that "natural" is better than a prescription drug. Supplements may also be cheaper than prescription drugs. Finally, advertising in various mediums plays a role in marketing weight-loss supplements. It is not unreasonable that an obese person would want a magic bullet that results in weight loss and is as simple as popping a pill.

SAFETY OF WEIGHT-LOSS SUPPLEMENTS

There are safety problems associated with the use of some weight-loss supplements. For instance, in response to a number of deaths and adverse medical effects, the FDA in 2004 banned the use of the supplement ephedra (also known as Ma huang or ephedrine) from use in weight-loss supplements. There can also be adverse health effects with the use of herbal supplements for weight loss. Herbal and other botanical ingredients of dietary supplements include processed or unprocessed plant parts (bark, leaves, flowers, fruits, and stems), as well as extracts and essential oils. They are available in a variety of forms, including water infusions (teas), powders, tablets, and capsules, and may be marketed as single substances or in combination with other supplements.

Because herbal ingredients are obtained from "natural" sources, that is, can be obtained in the wild, it is assumed by many consumers that natural means safe. However, it is well known that many natural plants (such as Deadly Nightshade) have toxic effects when ingested by humans. Finally, some weight-loss supplements have interactions with legal drugs consumers obtain by prescription. For instance, guar gum has been suggested to increase the effects of insulin and decrease the absorption of oral contraceptives.

In 1998, the U.S. National Institutes of Health issued clinical guidelines for the identification, evaluation, and treatment of overweight and obese adults. In these guidelines, the use of herbal and other supplements was specifically not recommended as part of a weight-loss program due to concerns about efficacy and safety.

Finally, a number of studies have also noted that a majority of weight-loss supplement users did not consult appropriate healthcare practitioners (such as family physicians) prior to weight-loss supplement use. With the possibility of dangerous drug-supplement interactions, not informing a healthcare professional about weight-loss supplement use can be a safety issue.

SPECIFIC SUPPLEMENTS

Below, several popular supplements with which there are associated scientific data are reviewed. The supplements' purported mechanisms are also discussed.

Bitter orange (citrus aurantium) is a plant that is commonly used in "ephedra-free" products. It is also known as Seville orange or green orange. It contains synephrine, a chemical very similar in structure to ephedrine (ephedra). Like ephedra, synephrine is a stimulant that is purported to increase energy expenditure. There is little evidence that supplements containing bitter orange contribute to significant weight loss. Some evidence does suggest, however, increases in blood pressure in subjects taking this supplement.

Chitosan is a polymer derived from chitin, which is found in the exoskeleton of shellfish. It is claimed to decrease fat absorption. However, there is little evidence that supplements containing chitosan contribute to significant weight loss. Some evidence suggests gastrointestinal complaints are frequent in users of chitosan.

Chromium picolinate is a compound consisting of trivalent chromium and picolinic acid. Chromium has been suggested to increase metabolism by potentiating insulin. There is little evidence that supplements containing chromium contribute to significant weight loss.

Conjugated linoleic acid refers to family of trans-fatty acids. This supplement has been purported to increase the breakdown of fat. In obese mice, there is some evidence that conjugated linoleic acid reduces fat in these animals. However, no human data support the use of supplements containing conjugated linoleic acid as contributing to significant weight loss.

Guar gum is a soluble fiber, which, in theory, can absorb water in the gut, increasing the feeling of fullness. There is little evidence that supplements containing guar gum contribute to significant weight loss. Some evidence does suggest, however, that gastrointestinal distress occurs in subjects taking guar gum. As noted above, this supplement may also interfere with the function of certain prescription drugs.

Pyruvate is a metabolite generated during metabolism. Pyruvate has been purported to work by increasing fat breakdown through a reduction in breakdown

of carbohydrate. There is little evidence that supplements containing pyruvate contribute to significant weight loss.

Yerba maté is an evergreen tree native to South America. It is commonly used in preparations containing high amounts of caffeine. Like ephedra, yerba maté is a stimulant that is purported to increase energy expenditure. There is little evidence that supplements containing yerba maté contribute to significant weight loss.

Yohimbe is an evergreen tree native to Central America. It is commonly used in preparations containing high amounts of caffeine. Like ephedra, yohimbe is a stimulant that is purported to increase energy expenditure. There is little evidence that supplements containing yohimbe contribute to significant weight loss.

Recently, the FDA approved the use of the drug Xenical® (orlistat) for over-the-counter (nonprescription) use in lower dosages. Although technically not a supplement, this drug became available without a prescription in July 2007 and is called Alli®. Orlistat works by inhibiting fat absorption from the gastrointestinal tract. There are side effects and possible drug interactions for some people, so literature associated with the purchase of Alli® should be consulted.

New supplements are appearing all the time. It is a challenge to keep up with the newest supplements on the market. Readers are referred to the Web sites in the Bibliography to keep abreast of scientific information on new supplements.

SEE ALSO: Amphetamines; Caffeine; Calcium and Dairy Products; Drug Targets that Decrease Food Intake/Appetite; Drugs that Block Fat Cell Formation; Ephedra; Food and Drug Administration; Leptin Supplements; New Drug Targets; Nondiet Approaches; Office of Dietary Supplements; Tryptophan.

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Support Groups

WHEN REFERRING TO the support groups in the context of the obese patient population, support groups should be viewed as support, *not* therapy. It is helpful to examine the simple definitions of the two words *support* and *therapy*. *Support* is defined as promoting the interests or cause of; to advocate; to assist or help; to maintain. Conversely, *therapy* is defined as an agency (as treatment) designed to deal with a bodily disorder or to bring about social adjustment as with psychotherapy. Although obese patients may indeed require therapy, the support group setting is an inappropriate forum for such a personal and individualized undertaking.

Support groups should be a safe place for patients to learn the skills and information they lack and yet need to be successful in their weight-loss endeavors. Groups should strive to educate, motivate, invigorate, celebrate, advocate, inspire, and promote critical thinking. Patients should not be afraid to learn and should be encouraged to do so by taking overcoming the many types of resistance that new group members often display. Support groups should be viewed as adult, nontraditional learning forums, and should also be treated as such.

Thus, those leading support groups should be intimately familiar with appropriate adult-learning concepts and practices to facilitate such a learning and progressive environment. All too often, those selected to lead weight-management support groups are woefully underqualified for the task. They are often medical or allied health professionals, or well-meaning fellow patients who lack the understanding, experience, knowledge, and skills necessary to balance the many aspects of support group organization, leadership, and management.

Effective weight-management support group leadership involves the application of advanced theory and skills in communication, group dynamics, psychosocial factors, leadership and facilitation, management and administration, education, motivation, obesity medicine, sociology, critical thinking, interpersonal relations, change management, law, advocacy, and community involvement. The balance of the applicable portions of these disciplines must be accomplished without treading into the unauthorized practice of any regulated occupation or profession

such as medicine, law, insurance brokerage, counseling, pharmacy, or dietetics, among others.

Running a weight-management support group involves both leadership and facilitation skills. Leadership is the ability to influence the actions of others, helping others to work toward a goal, and motivating others to do things they would not ordinarily do. Facilitation is the ability to take an active part in the group process without having decision-making authority, create a comfortable atmosphere for participation, and when possible or practical, to provide additional relevant information and resources for the group.

From these two simple definitions, one can see that good support group leaders embody the traits of both a leader and a facilitator. Some say that support groups should only have facilitators and not leaders, while others say that support groups should have leaders and not facilitators. A good support group leader, however, must be both, and must know when it is appropriate to take on more of one role than the other. Small groups are more susceptible of facilitation, and large groups require more leadership and less facilitation.

Each group, no matter the size, will take on its own group dynamics and roles. Support group leaders should be familiar with the general workings of group dynamics, and understand in which instances they should foster or redirect the dynamic. In any case, medical practices and programs should also understand the important nature and benefit derived from the group setting and dynamic, and give support groups the importance they deserve as part of a multidisciplinary treatment program for obesity.

At present, bariatric practices and programs do not tend to give support group programs the importance and attention they deserve, as evidenced by the severe lack of published data on the subject. The Surgical Review Corporation (SRC) requires any program applying for Center of Excellence (COE) certification to have in place a support group program, but supplies no standards, requirements, or criteria for the formulation or administration of such groups. Many practices have adequate programs, but unfortunately, research has shown that an alarming number of practices, COE certified or not, lack the appropriate educational, motivational, sociological, communication, group dynamics, administrative, and advocacy principles necessary to form exceptional and highly effective support group programs. This lack of basic principles leads to programs that misunderstand the very nature and purpose of support groups, the structure, leadership, and administration of them, and leads to patient and practice needs not being met. Bariatric patients are an extremely complex patient population. They require more than a one-dimensional support endeavor. Bariatric patients require assistance not only in the emotional domain, but also in the intellectual, psychosocial, communication, interpersonal relation, sociological, cultural, educational, vocational, nutritional, spiritual, physical, financial, skill building, and a host of other domains. The bariatric patient and his or her attendant personal support circle members require assistance transitioning from one lifestyle to another. The purpose of support groups is to assist individuals in making a successful transition such that the individuals benefit in all aspects of their lives.

To help ensure that benefit, support group leaders should be utilizing a total person approach to help patients by dealing with the whole person, not just a

Support groups should be a safe place for patients to learn how to be successful in their weight-loss endeavors.

particular set of characteristics or qualities possessed by that individual (such as having a weight problem, or utilizing a particular weight-loss treatment option), or a particular issue or portion of a person's very complex life. People play many roles throughout their lives, and indeed, throughout each day. Support group members are more than just weight-loss surgery patients, or lap band patients, or medically managed weight loss patients. They are parents, daughters, engineers, teachers, choir members, students, and more. When they attend support group, they will not completely discard all these other roles, or the issues that accompany them.

Similarly, patients bring all of their personal aspects with them as well—emotional, intellectual, spiritual, physical, medical, legal, social, cultural, financial, educational, career, and personal lives. Should attention be given only to their psychological or emotional aspects on an ongoing basis? And do all patients need ongoing attention for their emotional and/or psychological issues, or are they often resolved through attention paid to other aspects of their personal selves, or through private counseling? All aspects of a patient's life need ongoing attention at support groups, and any patient requiring psychological attention for unresolved issues should seek treatment outside the support group setting.

SEE ALSO: Anxiety; Appearance; Body Dysmorphic Disorder; Body Image; Depression; Fat Acceptance; Loneliness; Mood and Food; Obese Women and Social Stigmatization; Quality of Life; Self-Esteem and Obesity; Self-Esteem in Obese Women; Stereotypes and Obesity; Stigmas against Overweight Children; Weight Discrimination.

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Support Groups for Obese Women

WEIGHT-RELATED SUPPORT GROUPS for women provide the mutual support necessary for members to achieve compliance with weight-loss protocols and to understand the underlying issues that accompany and exacerbate obesity. The validation and support shared among members assists in the facilitation of personal growth and change in a way that individual therapy cannot. Although experts and professionals can provide support and positive direction, the mutual exchange of information between group members is a powerful experience that can promote positive and lasting change.

The strength of support groups lies in the members and their willingness to share their own experiences, challenges, and solutions in the context of the group. Because studies have proven what many already know about the experiences and quality-of-life differences between male and female obese individuals, people often have strong feelings about the kind of group that they want to join. Some feel that only someone who has been there will understand and make a good group member or leader. For these reasons, some believe that "women only" support groups are best.

Successful support groups specifically for women recognize that women are more likely to define themselves through relationships with others most like themselves. This type of peer support group consists of those who have similar experiences and is built on the relationship theory approach based on the assumption that women value relationships differently than men. Women-only support groups can provide a way for women to explore their experi-

ences and to form healthy relationships with others in what they consider a safe environment. Laughing about and exploring the unique experiences one has had as the result of a common situation or circumstance is best done with others who have walked a mile in the same shoes.

Many women prefer this type of single-gender support environment where they can regain their confidence, deal with problems specific to themselves, and generally feel that they can really open up. For many, it is the first time they are free from feeling like some terrible misfit, or that they are being judged. It is easier for them to establish trust and ensure confidentiality with other women, although neither issue should be taken lightly. Members of female obesity support groups feel free and open to discuss the most intimate and controversial aspects of their lives.

As a result, there exists a rare form of unconditional acceptance between the members. That is not to say there is no conflict or differences of opinion; however, the differences are respected and generally accepted. Women connect with other women in a very unique way—the more homogeneous the group, the greater the members' ability to bond with one another.

GIVING ENCOURAGEMENT

A main difference in communications styles between men and women in support group settings is that men are more than twice as likely to give information and women are more than twice as likely to give encouragement and support. Where women provide emotional support and empathy, men offer instrumental support or try to minimize the importance of problems. Men tend to devote a greater proportion of their time to talk about unrelated issues, and a larger proportion of any actual problem talk involves denying or convoluting the problem. For many women, this is not a productive means of support and is why many prefer women-only support groups where they are more likely to receive the emotional support they are seeking.

Obesity or weight support groups for women are considered peer support groups. A support group of this nature provides members with a sense of connection with regard to an experience that can feel isolating. The group provides support in hard times from other women with whom the members feel a stronger connection. It provides gender-specific information and coping skills, and helps members to feel less helpless about themselves and others because they are able to help others like themselves through their participation and experience sharing. It is a forum for sharing tips that only other women in the same situation can provide, and allows for powerful emotions to be shared.

Whether the support group is for women only, support group leaders should be utilizing the total person approach to help patients by dealing with the whole person, not just a particular set of characteristics or qualities possessed by that individual (e.g., having a weight problem or utilizing a particular weight-loss treatment option), or a particular issue or portion of a person's very complex life. People play many roles throughout their lives and, indeed, throughout each day. Support group members are more than just individuals suffering from the disease of obesity. They are parents, daughters, engineers, teachers, choir members, students, and more. When they attend support group, they will not completely discard all these other roles, or the issues that accompany them.

Overall, support groups for women should not be administered any differently than support groups in general. Other than a homogeneous makeup based on gender, all theories and applications on support groups in general apply and should be reviewed and integrated into all weight-related support groups.

SEE ALSO: Anxiety; Appearance; Body Dysmorphic Disorder; Body Image; Depression; Fat Acceptance; Loneliness; Mood and Food; Obese Women and Social Stigmatization; Quality of Life; Self-Esteem and Obesity; Self-Esteem in Obese Women; Stereotypes and Obesity; Weight Discrimination.

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Legal Consulting & Training

Sweet Taste

SWEET TASTE IS a sensation produced on the tongue by molecules that bind to sweet taste receptors, whose structure and genetic origin have recently been uncovered. The sensation of sweetness begins with the transduction by receptors throughout the tongue, or in whatever receptor organ these receptors are present for lower organisms, of the taste signal from a solution containing sweet-tasting molecules. The receptors are heterodimers of two G protein coupled receptors, T1R2 and T1R3, which are expressed by genes at the Sac locus on mouse chromosome 4 and the corresponding Tas1r3 locus in humans.

Taste transduction is the process by which a neural signal is generated following the binding of the tasted molecule to the receptor proteins. It involves a series of chemical reactions that change the resting membrane potential on neurons that produce action potentials carried along the seventh and ninth cranial nerves. These nerves synapse in the brain stem at the parabrachial nucleus where inputs from other visceral organs are combined and then relayed to the thalamus, hypothalamus, and cortex of the brain. Processing at each of these levels results in the ability to perceive taste quality, intensity, and affect (the positive or negative acceptance or rejection magnitude).

It has long been proposed that sweet taste leads to overconsumption of energy and, consequently, obesity.

Sweet solutions that occur in foods and beverages have both positive and negative acceptance/rejection reactions depending upon the individual, state of health or disease, previous consumption, and learned associations with the consequences of the consuming the taste. Generally, humans prefer concentrations of sucrose at approximately 10 percent (weight/volume). Lower and higher concentrations are rejected in comparison to the preferred level, which is also the level at which ratings of liking are highest. In rats, used frequently as animal models for obesity, however, increases in sweetness (independent of postingestional signals) lead to increased responding to obtain it.

The affective reactions to sweet taste are mediated by several neurotransmitters released into various regions of the brain, including the striatum and nucleus accombens. Both dopamine and opioid neurotransmitters appear to be involved in the mediation of the positive rewarding effects of sweet taste. Because sweet tastes are frequently present as sugars in energy-containing foods and beverages, it has long been proposed that sweet taste leads to overconsumption of energy and, consequently, obesity.

However, careful examination of the evidence is not definitive. Although sugar accounts for 22 percent of the American diet, obese individuals have been reported to prefer lower levels of sweetness than nonobese individuals by some investigators, but higher levels by others. Scaling of the level of liking may have led to these differences and more definitive work is needed. On the other hand, recent study demonstrated that in a group that included obese Pima Indians subjects who had a higher preference for sweet tasting food also had higher rates of weight gain after a 2-year follow-up.

Despite the lack of strong evidence, artificial sweeteners will probably continue to be used by many people who wish to limit energy consumption. Provided the energy spared by substituting nonenergy-containing sweeteners for sugars in foods and beverages is not made

up from other foods, this could be an effective strategy. However, there is little doubt that sweet-tasting foods of equivalent nutritional content will be chosen more often and eaten in larger quantities than nonsweet tasting foods. Therefore, an even better strategy would be for people to learn to consume less sweet foods. Sweet tastes also can induce metabolic changes by means of cephalic phase insulin release, which could potentially contribute to overconsumption or excessive fat deposition.

SEE ALSO: Genetic Taste Factors; Palatability.

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Sympathetic Nervous System

THE SYMPATHETIC NERVOUS system (SNS) plays a significant role in the regulation of metabolic and cardiovascular homeostasis. Aberrations in SNS behavior have been implicated in the pathophysiology of obesity and associated disorders including diabetes and cardiovascular disease. Low SNS activity has been suggested as a risk factor for future weight gain. In addition, obesity potentiates SNS activation in patients with hypertension and congestive heart failure (CHF). Therefore, an understanding of SNS behavior in human obesity is important from a clinical perspective.

SYMPATHETIC NEURAL ACTIVATION

The SNS is regulated in a regional-specific manner, and elevated SNS activity to one organ may not be similar to SNS activity targeting other organs. For example, cardiac SNS activity is lower in obese individuals compared to normal-weight individuals. However, SNS activity to the kidney as well as skeletal muscle sympathetic nervous activity (MSNA) is higher in obese individuals. Furthermore, the relationship between SNS to these regions and adiposity is linear and appears to be evident throughout the nonobese range. Given that the SNS is regulated in a region specific manner, it is not surprising that the available evidence suggests that whole-body SNS activity is not consistently altered in obesity.

MSNA is highly variable among individuals with similar age, sex, and body mass index (BMI). One possibility for this variation is that SNS activity is dependent, at least in part, on the distribution of body fat. Indeed, MSNA is higher in men with increased abdominal visceral fat compared to age- and total body fat–matched individuals. In fact, the relationship between abdominal visceral fat and MSNA is evident in normal-weight humans. Unfortunately, there is currently no information on the relationship between visceral obesity and SNS activity in other organs. This is an area that needs to be further explored.

CAUSES OF INCREASED SNS

There are a number of potential mechanisms that may contribute to increased SNS activity in obese individuals. For example, both insulin and leptin have been related to skeletal muscle SNS activity. However, in both cases, the available data do not consistently provide support for a role of either insulin or leptin in having direct effects on SNS activity in humans.

The renin angiotensin-aldosterone system is activated in obesity and angiotensin II has been shown to increase SNS activity in animals. Multiple components of the renin angiotensin system are expressed in adipose tissue, and angiotensinogen, the precursor to angiotensin II, is expressed more in visceral than subcutaneous adipose tissue. Therefore, it is possible that angiotensin II could be increasing SNS activity in obesity, particularly visceral obesity

Finally, obesity itself is thought of as a neuroendocrine disorder characterized by hypothalamic pituitary axis (HPA) dysregulation and SNS activation. However, it is not clear whether dysregulation of the HPA axis is critical to the activation of the SNS in the context of obesity.

CONSEQUENCES OF ACTIVATION

The consequences of SNS activation in obesity are unclear. One hypothesis is that elevated SNS activity

in obese individuals is a compensatory mechanism to prevent further weight gain. Unfortunately, there is little information to support this idea in humans. Interestingly, skeletal muscle SNS activity is lower than predicted in individuals with subcutaneous obesity. However, it is not known whether these individuals demonstrate reduced energy expenditure and are predisposed to future weight gain.

In conclusion, the renal and skeletal muscle circulations, but not the heart, appear to be targets for SNS activation in obesity. In addition, abdominal visceral fat may be an important depot linking obesity to skeletal muscle SNS activity. However, the impact of this depot on the kidney or other regions is unknown. The consequences of SNS activity are also unclear, but may include protection against further weight gain. Future research in this area is needed to begin addressing these issues.

SEE ALSO: Adrenergic Receptors; Autonomic Nervous System.

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Taste Aversion Learning

TASTE AVERSION LEARNING, or conditioned taste aversion, refers to a distaste or repugnance for a particular type of food due to a previous incident or stimulus with unfavorable consequences. Taste aversion learning, which tends to fall in the realm of psychology, can be attributed to certain molecular signaling and physiological pathways. The concept of taste aversion is based on a cause-and-effect relationship; a specific food can trigger memories of illness. In the case of obesity, taste aversion learning is a potentially useful tool for weight control and healthy eating choices.

Taste aversion learning is a type of associative learning known as classical conditioning. Classical conditioning requires the presence of an unconditioned stimulus that automatically evokes an unconditioned response. The point of classical conditioning is to manipulate the relationship of the unconditioned response and stimulus and promote the onset of a certain conditioned response. In behavioral therapy the conditioned response will be more beneficial to the subject, in whatever area the therapy is seeking to address, and repetition will eventually lead to the removal of the more detrimental unconditioned response from the subject's lifestyle.

Aversion therapy is a type of psychological therapy. The treatment occurs when a patient is exposed to a stimulus that is immediately followed by some kind of discomfort. The objective of aversion therapy is to condition the patient to associate the stimulus with something unpleasant that leads the patient to cease the targeted behavior. An aversive stimulus is an unconditioned stimulus, and this stimulus produces an unconditioned response. With aversion therapy, the patient should learn to experience a new, conditioned response with each stimulus, and successful treatment will cause the compulsion to succumb to the former, unconditioned response to subside and eventually disappear. Aversion therapy has been used in the treatment of substance abuse, but the application of its principles to combat overeating and poor eating choices needs to be further explored.

Taste aversion learning occurs when one learns to associate the taste of one food with unpleasant symptoms such as nausea or vomiting. Taste aversion is a trait that can be adapted and is actually a survival mechanism that ensures that one stays away from potentially harmful foods or beverages that are poisonous or spoiled. The association that develops between ingestion of the harmful substance and knowledge of the symptoms that occur after its ingestion ensures the prevention of future consumption.

Taste aversion learning can also occur when an association is taught that links the ingestion of a specific food with a forced task. Animal studies have compared the strengths of conditioned taste aversion with forced activities such as running and swimming

for varying lengths of time. Injections with substances such as lithium chloride have also exhibited strong taste aversion learning.

Certain molecular events allow for the acquisition of taste aversion, and many of these processes are due to an association with fos-like immunoreactivity and taste novelty. Hormonal relationships have also been considered. Some studies have targeted leptin as an influence in the development of taste aversion learning. While results for leptin studies have suggested that it is not related to the attainment of taste aversions, the nonspecific melanocortin agonist MTII was found to produce conditioned taste aversions.

SEE ALSO: Appetite Control; Leptin; Metformin; Taste Reactivity.

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Taste Reactivity

THE DISCRIMINATION ABILITY of taste seems to pale in comparison to those of the other senses. Taste can only distinguish between a few qualities: bitterness, saltiness, sweetness, sourness, and possibly a few others, including umami or savoriness. Despite its seeming simplicity, the sense of taste interacts strongly with the central nervous system (CNS) to regulate appetite for certain foods, and there is a relationship between the need for a specific nutrient and an increased desire for foods with a specific taste. This relationship can be quantified by the taste reactivity test.

The taste reactivity test is performed by injecting liquids with a specific taste through a surgically implanted intraoral catheter in animals (usually rats) and grading the animal's response as either ingestive or aversive. The value of the taste reactivity test in understanding how taste and nutrient need interact is best illustrated by specific experiments.

A simple experiment to demonstrate this interaction compares sodium-replete and sodium-deficient rats when a taste sensitivity test is performed using a solution of sodium chloride and water. Both groups will demonstrate aversive responses to intraoral quinine injection, a strongly bitter stimulus. Sodium-deficient rats, however, will demonstrate a strong ingestive response to salt water administration, whereas sodium-replete rats demonstrate the opposite reaction.

Taste sensitivity experiments have demonstrated a sophisticated interaction between the nutritive needs of animals and taste preferences. Fat-deprived rats will demonstrate stronger ingestive responses to normally avoided, nonnutritive oils, such as mineral oil, than to solutions containing artificial sweeteners. Evidence in both rats and humans suggests that taste receptors may be able to distinguish fatty acids as having a distinct taste and that this is sensitized in fat-deprived individuals. There is also evidence to suggest interindividual variability in the sensitivity for detection of fatty acids by these receptors. Responses to nonessential nutritive substances, such as ethanol, can also be influenced by taste preferences. For example, in rats, there is a correlation between a preference for sweet solutions and greater ethanol intake that has also been observed in human alcoholics.

The mechanisms that link taste and food-seeking behavior likely involve multiple pathways in the CNS. Modulation of neurotransmitters can lead to changes in taste sensitivity test preferences. For instance, rats given naltrexone, a drug that blocks opioid receptors, demonstrate decreased responsiveness to sweet solutions during taste sensitivity testing. This property may be exploited to reduce positive stimulatory feedback in individuals with addictive patterns of food ingestion or excessive cravings for specific nonnutritive tastes (e.g., ethanol). Improved understanding of the interplay between specific tastes and patterns of food ingestion will hopefully allow better prescription of dietary modifications for individuals with certain taste preferences.

SEE ALSO: Neurotransmitters; Nutrient Reward; Taste Aversion Learning.

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Taxation on Unhealthy Foods

IN 1994, KELLY Brownell wrote an op-ed piece in the *New York Times* advocating a tax on foods with low nutritional value such as soda and snack foods. This proposition was dubbed the "Twinkie tax," and has since been called the "snack tax," "fat tax," and "junk food tax." This tax would be similar to those imposed on alcohol and tobacco. Brownell's argument was that foods with a high-sugar and fat-content are affordable, easy to get, and taste good, which encourages increased consumption of these foods, which in turn is feeding the obesity epidemic. Brownell is a prominent Yale University psychologist and obesity researcher, and he has been interested in fixing the "toxic environment" that he believes is the cause of the current obesity epidemic. According to Brownell, a small tax on soda alone could raise millions of dollars a year, which could be used to subsidize healthier food options or used to fund health programs and health education.

According to Brownell, if a tax on unhealthy foods is implemented, there are many positive results that may occur. First, a large tax on these foods may decrease the consumption of these foods in the same fashion as tobacco and alcohol. The tax money will then go to subsidize healthier food options, thereby increasing the consumption of healthy foods. If a small tax is implemented, the money will go to health promotion programs such as those to encourage healthier eating, healthier food options in schools, and physical education programs promoting exercise and a healthy lifestyle. Promoting healthy eating and a healthy lifestyle will help counteract the effect of billions of dollars of advertising spent on promoting unhealthy items. The result of all of this, then, would be to decrease the incidence of obesity and nutrition-related diseases such as diabetes and heart disease.

Individuals of lower socioeconomic status spend a larger percentage of their income on food and bear most of the tax burden.

There are several states that enacted variations of the "Twinkie tax" before the Brownell piece. The majority, such as Indiana, Kentucky, Maine, New York, and Minnesota, imposed sales tax on items such as soft drinks, candy, and gum. California and Illinois taxed only soft drinks. Other states such as Rhode Island, Tennessee, and Virginia levied taxes on the manufacturers or wholesalers of soft drinks. Despite the fact that these taxes raised millions of dollars a year for their respective states, more than half of the taxes were repealed during the 1990s, with the majority of the taxes repealed after Brownell's argument for such taxes.

There are numerous points of debate for a tax on unhealthy foods. First, there is the issue of defining what an unhealthy food is. The states that implemented these taxes mainly targeted food groups that were already defined, such as soft drinks and candy. Even these items may have room for interpretation. The terms *snack food* and *junk food*, however, are very vague, creating the suspicion that it is an umbrella term under which the majority of food may fall. The suggestions for what foods should be taxed include those high in saturated fat, trans-fat, and sugar, and

those with high calories and low nutrient density, meaning the individual is not receiving the proper amount of nutrients needed compared to the amount of calories taken in. The complexity of defining what an unhealthy food is makes implementing a tax based on the above criteria a complicated process.

The second issue with the tax on unhealthy foods is what will be done with the money generated. While Brownell suggested the money be used to promote health education, none of the states with an unhealthy food tax used the money in that way. The majority of the money raised from the taxes were placed in general funds to be used where it was needed. West Virginia allocates the money to support medical, dental, and nursing schools, and Arkansas uses the money to fund Medicaid. By not allocating these funds to a specific program to encourage health and health education, the tax is not doing what it is "supposed to do" in battling obesity.

The third issue is the effect such a tax will have, especially on individuals of lower socioeconomic status. A large tax on unhealthy foods would certainly affect these individuals the most because they are more likely to buy the less expensive, unhealthy foods. Also, individuals of lower socioeconomic status spend a larger percentage of their income on food, causing them to bear most of the burden of such a tax.

A fourth issue is the concern of too much government intervention in the lives of individuals. Critics claim that allowing the government to impose taxes on things that are unhealthy is just another method of control and a way to squeeze more money out of taxpayers' pockets. Although the tax on unhealthy foods has been likened to that of alcohol and tobacco, unhealthy food is still food, while alcohol and tobacco have no nutritional value. Critics claim that it is the right of individuals to choose which foods they consume.

A final issue is whether a tax on unhealthy foods will actually decrease their consumption. This tax has been compared to the taxes on alcohol and tobacco, both of which showed a reduced consumption rate after the tax was imposed. There is no guarantee, however, that the consumption of unhealthy foods will decrease.

Despite all of the arguments against an unhealthy food tax, such a tax may have a significant effect on the rate of obesity. Even if consumption of unhealthy foods does not decrease a significant amount, the consumption of healthy foods may increase due to subsidy or increased consumer knowledge. A small tax may fund a health education program that advertises the benefits of healthy foods to counteract the billions of dollars spent annually on advertising for unhealthy foods. The money could also fund education programs in schools, providing healthier school lunches and increased physical education to encourage physical activity as part of a healthy lifestyle.

The jury is still out as to whether a tax on unhealthy foods would have an effect on obesity. In California in 2002, a soda tax bill was proposed that allocated the money raised to school districts with healthy eating and physical education programs, obesity and oral health prevention programs, and a preventative program for childhood obesity. The bill was not successful. It is unclear whether a tax on unhealthy foods will have an effect on obesity.

SEE ALSO: Federal Initiatives to Prevent Obesity; Government Subsidizing of Energy Dense Foods; School Initiatives to Prevent Obesity; State and Local Initiatives to Prevent Obesity.

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Television

TELEVISION WATCHING IS a popular leisure-time activity in all but the most remote areas of the world. In fact, watching television is often the most time-consuming leisure activity of people of all ages and nationalities. Those living in developed countries watch an average of approximately 3 hours of television each day. In addition to its entertainment value, television is a primary source of health information. Considerable evidence exists that television is a potent influence on knowledge, attitudes, behaviors, and values and its impact may be equal to that of school, religion, parents, or books. Concerns about television watch-

ing and the effect of programming are many and have been voiced by numerous public health authorities. These concerns focus on the content and influence of television programming on many health behaviors (e.g., violence, alcohol and tobacco use, sexual behaviors), including dietary intake and the sedentary nature of television watching. Television is thought to influence dietary intake, and subsequently body weight, by frequently promoting the consumption of calorie-dense foods and providing food and nutrition information that is misleading. Television further affects body weight via the sedentary nature of television watching itself. Available academic research suggests that watching television is associated with increased snacking and poorer quality meals; television advertising increases the number of food purchase requests children make; television advertising influences food preferences, choices, and intake; television advertising and programming affects children's knowledge of nutrition and health.

The vast majority of research evidence concerning the effects of television was conducted in the United States using television programs produced and broadcast there. However, the widespread export of U.S.–produced television programs shares their content with and extends their potential effects to viewers in other countries. Although findings related to advertisements may have less generalizability internationally because advertisements produced for a particular national market are seldom broadcast outside that market, advertising goals and strategies are similar regardless of market and the developmental stages during childhood and adolescence, the audience thought to be most vulnerable to its effects, are generally regarded to be universal.

Research related to television and body weight is divided into content analyses of television advertising and program content, effects of television advertising and programs, and time invested in television watching. Most research has focused on children's television because this population group is cognitively immature and, thus, has difficulty in both interpreting information provided via television and discerning reality from fantasy.

TELEVISION ADVERTISEMENT CONTENT

Most content analyses of television advertising focus children's television programming hours with lesser attention given to other broadcast time periods. Advertisements during children's television programming hours tend to be primarily for food during all months except December when toy advertisements predominate. The types of foods advertised rarely include fruits, vegetables, protein-rich foods, or dairy products. Foods in the breads and cereals group, particularly high-sugar breakfast cereals, are well represented. Additionally, foods high in fats and nutritive sweeteners are advertised frequently, with food sold by fast-food restaurant chains, candy, and sweetened soft drinks being most prevalent. The foods advertised during children's television programs tend to be high in fat, sodium, and sugar, and generally judged to be of low nutritional value. Explicit messages related to the healthfulness or nutritional qualities of the foods are seldom used in advertising; however, nearly half of food advertisements imply a food has healthful or nutritional qualities, likely erroneously so given the nutrient content of most advertised foods.

The limited evidence available indicates that food advertisements also are commonly broadcast during television broadcast periods with an audience largely comprised of women aged 18–35 years (e.g., afternoon serials such as soap operas). The most commonly advertised foods are desserts and sweets, convenience foods, and breads and cereals. The vast majority of advertised foods are high in saturated fat, cholesterol, and sodium. Food advertisements infrequently include health claims.

During prime-time television programming time slots, food advertisements tend to be the largest single category of advertisements. Advertised foods are mostly high in fat, sodium, and sugar. Consumer-related promotional claims, especially those focusing on flavor, quality, and economy, are used to promote most foods. The majority of health and nutrition information embedded in food advertisements is judged misleading or inaccurate often because it is incomplete or exaggerated. The types of foods advertised during prime-time programs may vary with the target audience. For instance, programs with a high African-American viewership have fewer alcohol advertisements and more soft drink and chocolate candy advertisements than programs with a more general audience. Health claims are made more frequently in food advertisements on Spanish-language programs broadcast in the United States than on English-language programs. Regardless

of the differences in the frequency with which food types are advertised to different target audiences, the foods advertised during prime-time television time slots are high in fat and/or sugar.

TELEVISION PROGRAM CONTENT

Television programs frequently include scenes that reference food verbally, visually (as a prop), and/or being actively consumed. During children's programming hours, the most commonly shown foods (fruits, protein-rich foods, and sweets) tend to be of greater nutritional quality than those in advertisements. Foods are primarily portrayed in a positive context; that is, one that constructively describes the food's qualities or encourages the viewer to try the food

Among prime-time programs, those of the situation comedy genre include the most scenes with nutrition or food references and those of the real-life reenactment genre include the least. Low nutrient density foods and beverages, especially alcoholic beverages, are commonly referenced in prime-time television programs. Foods are mostly consumed as snacks rather than meals, with the most common snacks being sweets or salty snack foods. Snacks are less nutrient dense than meals. Adults tend to eat less nutritious foods and snacks than children. Black actors are more likely to eat nutritious foods than white characters. Although the quantity of food consumed by actors tends to be very small, heavy males are shown eating greater amounts than average weight or thin actors. Overweight actors are less likely to be shown with high-calorie foods than average-weight characters. Scenes containing food or nutrition references were most commonly set at kitchen tables, outdoors, and in dining rooms, living rooms, and restaurants. These settings seldom relate to the main point of the scene. Approximately one in five scenes with food or nutrition references promote healthful behaviors, such as eating fruit for a snack or drinking juice to restore blood glucose levels and a similar amount promoted negative, nonrecommended health behaviors. Few actors in prime-time television programs are overweight or obese and substantial numbers are far more likely to be underweight than their real-life

Approximately one in five scenes with food or nutrition references promote healthful behaviors, such as eating fruit for a snack or drinking juice to restore blood glucose levels and a similar amount promoted negative, nonrecommended health behaviors.

counterparts. Black actors tend to have larger body types than white actors.

TELEVISION ADVERTISEMENT EFFECTS

Television advertising has the potential to shape nutrition-related knowledge, attitudes, and food preferences and choices. Increased exposure to advertisements broadcast during children's television programming hours is positively correlated with lower nutrition knowledge and lower understanding of nutrition terminology commonly used in television advertisements. Repeated exposure to highly persuasive advertising messages also may confuse children about the nutritional value of foods. Children who frequently view television have distorted concepts of what constitutes a healthful diet.

Brief exposures to advertisements for low nutrient density foods can influence young children's food preferences, choices, and intake. The more often a food is advertised, the more likely it is to be requested by children and purchased by adults. The first purchase requests occur at about age 2. Children frequently request food items advertised on television and ask to visit restaurants advertised. The more time children spend watching television, the more likely they are to request advertised foods. The most frequently requested foods are breakfast cereals, snacks, and beverages. Requested foods tend to be high in sugar, fat, and/or salt foods. In comparison to younger children, older children make fewer food purchase requests. Foods are requested by children and purchased by parents in the same frequency they are advertised during children's television programs. Advertised foods requested by children and purchased by parents are among the most commonly reported as eaten in national dietary intake surveys.

TELEVISION PROGRAM EFFECTS

The effect of television programs on nutrition knowledge, beliefs, and behaviors remains largely unexplored; however, social learning theory and cultivation theory indicate that television must be recognized as a major source of health and nutrition information and a potentially powerful influence on nutrition and health practices. (Social learning theory is based on the notion that behavior, particularly that of children, is acquired through the process of observing the behavior of others and subsequently modeling one's behavior on the observations. Cultivation theory posits that television shapes or cultivates viewers' perceptions of what they consider to be normal, common, or acceptable behavior.)

Considerable research evidence suggests that those who watch relatively more television tend to emulate observed television behaviors because they perceive that the real world is much like that portrayed on television. In addition, as exposure to television increases, emulation of the behaviors portrayed often becomes increasingly desirable. Television programs also may affect children's nutrition knowledge, attitudes, and food and alcohol intake intentions. The contrast between idealized female body size portrayed by most television actors can negatively affect mental and physical health of female viewers and trigger extreme dieting and disordered eating. Television programs can increase adult's nutrition knowledge levels as well as motivate them to practice healthy dietary behaviors, increase concern about food and fitness, and raise self-confidence in their nutrition knowledge base.

TIME INVESTMENT IN TELEVISION WATCHING

Time spent watching television (using media of all types) may increase obesity risk. One reason this may occur focuses on energy expenditure. The time spent watching television displaces time that could be used for physical activity, the relaxing quality of television watching may lower resting metabolic rate to a level below that of sleeping, and the extent of fidgeting during television watching may be lower than that which occurs during other sedentary activities.

A second reason is the relationship between television viewing habits and nutrition knowledge. As children's total television viewing time increases, so does the rate of inaccurate knowledge about nutrition, unhealthy conceptions about food, and misperceptions that an unhealthy food choice is healthier than it actually is.

A third reason focuses on dietary intake. Watching television is associated with increased snacking, poorer quality meals, and higher dietary fat intake. As children's total television viewing time increases, so does the rate of poor eating habits. In addition, time spent watching television is positively correlated with children's preferences for and consumption of unhealthy foods. Routine television watching during mealtime is associated with poorer eating choices, lower intakes of fruits and vegetables, increased

snacking, and higher consumption levels of pizza, snack foods, and soft drinks. Among adult women, watching more than 2 hours of television daily is associated with greater tendency toward disordered eating and weight dissatisfaction as well as numerous other negative psychological states.

SEE ALSO: Advertising; Beverage Choices in Children; Fast Food; Overweight Children in the Media.

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Three-D Image Reconstruction

THE CONCEPT OF central obesity, or visceral adiposity, was first suggested as an adverse health factor for coronary artery disease as early as 1950, but has only been validated as an important marker for the development of the array of obesity-related medical conditions collectively known as the *metabolic syndrome* in the last decade. With the advent of computerized tomography (CT) and magnetic resonance imaging (MRI), the significance of visceral "intraabdominal" obesity (rather than subcutaneous fat storage) has been documented in an increasing number of body composition studies.

This apple-shape pattern of intraabdominal obesity, although correlated with the amount of total body fat, body mass index (BMI), and subcutaneous fat in an individual, is unique in that it alone is the source of body fat that has been shown to influence the comorbidities associated with obesity and the metabolic syndrome. In fact, previous studies have shown that even among men of healthy BMI, the amount of visceral fat is correlated with increased prevalence of insulin resistance, hypertriglyceridemia, hypertension, and coronary artery disease—all hallmark markers of the metabolic syndrome. While it is generally thought that women tend to carry the majority of their excess adipose tissue in their thighs and hips, rather than centrally, women do indeed show similar patterns of developing symptoms of the syndrome in direct correlation to the amount of intraabdominal fat that they possess.

Thus, the emergence of research techniques that can accurately access total body composition, measure body fat distribution, and quantify the amount of visceral fat can be profoundly appreciated. The technological advancements of the past decade—particularly in the improvements of CT and MRI scanners—has allowed for greater accessibility of CT and MRI outside the realm of clinical medicine, and a grand impact on body composition research.

Both CT and MRI technology have allowed for the production of three-dimensional (three-D) images. Until their development, body composition analysis by way of radiogrametry and conventional X-rays have been limited to fuzzy two-dimensional images. The three-D image reconstruction offered by CT and MRI scanning allows for three-D tissue volume measurements and the production of sharp contrast images with clearer tissue boundaries. To date, none of the other competitive technologies being explored, such as ultrasound, has been able to assess tissue composition three dimensionally as accurately as in CT and MRI.

CT and MRI technology is founded in a two-stage image reconstruction strategy. The first task at hand is to accurately gauge the amount of specific tissue that is expressed in an individual image (in cm^2). Second, this flat image's area is superimposed on various slices of itself to calculate a total volume (in cm^3). This is one of the great advantages in using three-D image reconstruction, as it allows for the

cross-sectional imaging that can compute areas and volumes of tissue structures inside of body cavities. As in brain and heart volume calculations quantified by MRI and CT for clinical purposes, visceral adipose tissue has also been accurately quantified using this technique. Another important advantage to this technology is that it can easily calculate whole-body estimates of tissue-level composition, whereas competing technologies are currently limited to making only regional measurements. It is worth noting that today, three-D image reconstruction technologies are becoming greatly accessible globally; virtually all major medical research centers in the United States have both a CT and MRI scanner capable of full-body composition analysis, and many developing nations have increasing access to one, if not both, of these tools at major hospitals.

For the purposes of human body composition analysis, MRI is currently the more favored method of three-D analysis as it does not expose a patient to the risk of ionizing radiation. MRI scanning, thus, can take multiple images without any known risk to the patient and may be useful in longitudinal studies and clinical trials where multiple measurements are necessary. The one potential disadvantage to certain types of MRI scanners is that the enclosing magnet may prove uncomfortable for obese or claustrophobic patients. MRI is also restricted in patients with implanted metal objects, as the process generates magnetic fields.

Presently, the primary use of MRI scanners in human body composition studies is to quantitatively measure the quantity and distribution of fat and skeletal muscle in tissues. To date, MRI has been used to assess the body composition of many subjects, from fetuses to the elderly, both healthy and diseased populations, and in both males and females. MRI technology is particularly useful in weight-loss studies, as a whole-body MRI image can be obtained in little over a half hour with little discomfort to the subject, and a series of images can accurately display the regional changes in muscle and fat composition that occur with weight reduction by means of diet and/or exercise. For example, a gain of skeletal muscle tissue in one part of the body may go unrecognized by other measurements of body composition analysis and BMI, but MRI can clearly illustrate that this muscle gained is masking the loss of adipose tissue weight in another region of the body.

It is clear now that the advancements in three-D imaging technologies over the past decade have vastly influenced not only the quality of body composition research, but also its impact on the prevention and treatment of obesity and its comorbid conditions. If the rapid increase in both accessibility and utilization of these machines over the past several years is any indication, we can hope that three-D reconstruction imaging will continue to pave new ground in body composition research and prove an essential tool in the fight against obesity.

SEE ALSO: Central Obesity; Computerized Tomography; Magnetic Resonance Imaging Scans, Visceral Adipose Tissue.

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Thrifty Gene Hypothesis

THE THRIFTY GENE hypothesis is the idea that humans can efficiently store nutrients that were limited in supply 10,000–15,000 years ago (known as the Paleolithic era) and that this increased efficiency in storage is related to many of the metabolic problems observed today. This hypothesis was first proposed by James Neel in 1962. Unfortunately, it takes a significant amount of time for genes to evolve into more desirable traits (approximately 5,000–10,000 years). Therefore, our current genetic information is helpful for an environment which no longer exists.

One component of the thrifty gene hypothesis is the ability of humans to store fat very efficiently. The

idea is that during the Paleolithic era, humans would often go long periods of time without sufficient food intake (i.e., only when large animals were caught), and subsequently, these humans became very efficient at storing the calories; the stored calories would be used during times of famine. In modern times, unfortunately, there is a plethora of food available to the residents of many developed countries. Therefore, when humans eat today, they store their extra calories very efficiently. This efficient storage system, combined with a decrease in physical activity, may contribute to the current obesity epidemic and all of the diseases that are associated with obesity (i.e., insulin resistance, Type 2 diabetes mellitus, heart disease, and high blood pressure).

In regard to the development of Type 2 diabetes mellitus, it is believed that early humans rarely consumed simple carbohydrates (sugars) and that the actions of insulin were different 10,000–15,000 years ago. Currently, humans require a large amount of insulin to handle the requirements of the modern-day diet. This increased demand of insulin in people who are also overweight increases the likelihood of developing insulin resistance and eventually Type 2 diabetes mellitus.

In addition to this hypothesis about body weight, the thrifty gene hypothesis includes speculation about high blood pressure. Early humans consumed a diet low in sodium chloride (table salt) and thus established an efficient ability to store sodium. Today, the food supply contains much greater sodium content and thus when people consume sodium, their bodies do their best to hold on to it. This is the believed mechanism for why people get salt-induced high blood pressure.

It has been viewed that storing sodium efficiently is a genetic defect. In fact, according to the thrifty gene hypothesis, the opposite would be true. People who do not get the rise in their blood pressure in response to sodium intake might have mutated their thrifty gene to adapt to the modernized world. Alternatively, because our ancestors consumed a diet high in potassium (found in many fruits and vegetables), humans have not developed a good method to conserve excess potassium. Therefore, it has been suspected that low potassium levels from current diets may also be a factor in the development of health problems in developed countries.

Another component in regard to the link of obesity and the thrifty gene hypothesis is the decrease in physical activity. During the Paleolithic era, humans were extremely physically active. They exerted a great amount of energy in acquiring food. During the last 100 years, physical activity has significantly diminished. Not only do humans have easy access to large quantities of food, but there has also been a significant decrease for other physical tasks (i.e., travel is no longer limited to the legs). The increased food intake in combination with decreased physical activity is unfavorable for the thrifty gene environment.

Often cited with the thrifty gene hypothesis is the Barker hypothesis (also called the thrifty phenotype hypothesis). Barker had hypothesized that the environment a baby is in during gestation can be related to health problems later on in life. He reported that if a pregnant woman had low food intake, the baby would develop more fat cells as a system to ensure enough energy stores once they were born. Subsequently, this will lead to an increase in the percent of body fat that the baby, then child, and finally adult will have. Excess body fat is related to many metabolic diseases and this would increase the risk significantly and Barker hypothesized that these babies would have an increased prevalence of certain diseases such as Type 2 diabetes mellitus, insulin resistance, high blood pressure, and heart disease.

Many large epidemiological studies have confirmed this hypothesis such as the Dutch famine study. During the winter of 1944–45, there was a famine in the Netherlands. Women who were in the first half of their pregnancies during the famine were more likely to have children who were obese by age 19 compared to women in the second half of their pregnancies. Other studies have linked low birth weight with impaired insulin functioning, insulin resistance, and eventually Type 2 diabetes mellitus. This signified the importance of nutrition at very early stages as they related to development.

Another interesting component of the thrifty gene hypothesis is relating the food consumption of people living in underdeveloped or non-Westernized countries. Many of residents in these areas still consume a diet similar to the one of our ancestors which includes high amounts of fruits, vegetables, and fiber and low amount of protein, fat, and salt. People who follow this diet do not suffer from the conditions that affect people who live in the Western world. Also, some researchers have compared animals (particularly monkeys, chimpanzees, and other animals with similar DNA to humans) that live in captivity (i.e.,

zoos) compared to animals that are living in the free world. The zoo animals tend to follow a more Westernized lifestyle, filled with lots of high-fat and protein foods and decreased physical activity. These animals are more likely to become obese, diabetic, and have high blood pressure. Alternatively, the animals that live in the free world follow a traditional lifestyle of hunting and gathering their food supply. These animals tend not to develop the problems that are afflicting the zoo animals.

SEE ALSO: Metabolic Disorders and Childhood Obesity; Type 2 Diabetes.

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Thyroid Gland

THE THYROID GLAND is an organ in the neck that regulates many features of metabolism. There are several steps in regulation of the thyroid gland, and disruption of these controls can lead to thyroid disease. The hypothalamus secretes thyroid-releasing hormone (TRH) which travels to the pituitary gland, stimulating secretion of thyroid-stimulating hormone (TSH), which travels through the blood to the thyroid gland. TSH then stimulates an increased release of thyroid hormones T3 and T4 into the blood stream. There is a feedback loop that allows high levels of thyroid hormone to then decrease production of TRH and TSH. Production of thyroid hormone is dependent on iodine, and iodine deficiency can cause thyroid dysfunction. T3 and T4 have a variety of effects on multiple organ systems of the body. Detection of thyroid disease involves testing blood levels of TSH, T3, and T4 as well as physical exam and history.

Hypothyroidism can be caused by a defect in release of TSH, by decrease production of thyroid hormones (T3 & T4) secondary to iodine deficiency, or can be secondary to autoimmune dysfunction and destruction of the thyroid gland, as in Hashimoto's thyroiditis. People with hypothyroidism have a slowing of metabolic processes, with fatigue, slow movement and speech, moderate weight gain, and intolerance of cold. There is decreased blood flow to the skin, leading to development of dry and rough skin, coarse hair or hair loss, brittle nails, swelling (edema) from water retention, and discoloration of the skin. There may be a decrease in cardiac output because there is a slowing of heart rate and decreased muscle contractility.

In combination with increased fatigue of respiratory muscles, there is a decrease in exercise capacity and shortness of breath. Enlargement of the tongue (macroglossia) can lead to sleep apnea, a condition characterized by multiple episodes of breathing cessation during sleep. Motility of the gut decreases, leading to constipation. Red blood cell mass decreases and may lead to iron deficiency anemia in younger women. Disturbances in metabolism contributes to irregular menstruation and decreased fertility. Hypothyroidism can be treated with replacement of thyroid hormone in the form of pills taken daily, with monitoring of symptoms and TSH levels.

Hyperthyroidism, as suggested by the prefix "hyper," is characterized by an increase in metabolism. The cause of this condition can be due to an increased secretion of TSH, an increased production or release of thyroid hormone (T3 & T4), or secondary to an autoimmune disease such as Grave's disease. People with hyperthyroidism have anxiety, emotional instability, muscle weakness, tremor, heart palpitations, increased sweating, heat intolerance and weight loss, even though both appetite and caloric intake are typically increased. Stimulation of gut motility may lead to frequent stooling or loose stools, and there may be an increase in urinary frequency. Woman may experience irregular menstruation. Skin tends to be

warm and moist, with decreased hair. Patients with Grave's disease may experience exopthalmos, or forward projection of the eyes, with difficulty in eyelid closure and eye movement. Treatment of hyperthyroidism can involve medication to decrease thyroid hormone production, or removal of the thyroid gland with subsequent replacement of thyroid hormone.

The prevalence of true hypothyroidism is actually quite low, and is typically not the root cause of obesity. According to the recent National Health and Nutrition Examination Survey (NHANES III), approximately 4.6 percent of the population has hypothyroidism, but 4.3 percent of these cases are considered subclinical. Thus, contrary to popular belief, most cases of excess body weight are not the result of hypothyroidism, just as most lean individuals are not hyperthyroid.

SEE ALSO: Hypothalamus; Hypothyroidism; Thyroid Medications.

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Thyroid Medications

THE THYROID GLAND produces thyroid hormone, an important regulator of the body's metabolism and a wide array of other functions. There are two basic forms of thyroid disease: overproduction or release of thyroid hormone (hyperthyroidism) and underproduction or release of it (hypothyroidism). Despite the widespread belief that most cases of obesity are caused by having thyroid problems, particularly hypothyroidism, true cases of thyroid problems are rare, and do not explain the majority of obese individuals. However, in rare cases, hypothyroidism can result in weight gain, and potentially obesity. The underlying causes and consequences of the two are quite different, as are the medications used to treat them. To understand how the medications work and how the appropriate dosages are determined, it is important to review thyroid physiology. The thyroid gland produces two types of thyroid hormone. The most common is T4, an inactive or storage form. The T4 can be converted to T3—the active form—in the bloodstream based on the body's demand for it. The thyroid also produces small amounts of T3 directly.

Production of thyroid hormones (T3 & T4) is regulated using complex feedback loops. The pituitary gland, found in the brain, produces thyroid-stimulating hormone (TSH) in response to signals from the brain and the levels of thyroid hormone in the blood. TSH in turn stimulates the thyroid gland to make more hormone. High levels of TSH correspond to low levels of hormone (hypothyroidism), while very low levels of TSH indicate excess levels of it (hyperthyroidism).

Hypothyroidism is treated by taking supplemental thyroid hormone, generally in pill form. Synthetic versions of both thyroid hormones (T3 & T4) are available, either as single agents (levothyroxine and liothyronine, respectively) or in combination. A preparation derived from animal tissue is also available that contains both T4 and T3.

Levothyroxine is the most commonly used medication for treating hypothyroidism and is advocated by many physicians because it relies upon the body's normal regulatory system for converting the synthetic T4 to active T3. In most cases, thyroid replacement medication dosage is adjusted based upon measurement of TSH. In people at high risk for complications related to sudden increases in their metabolism, such as the elderly or those with heart conditions, the medicine is started at very low doses and gradually increased over a period of weeks to months.

Taking thyroid hormone pills with food or other medications can block its absorption through the gut. Therefore, it is important to take the medicine on an empty stomach, either 1 hour before or 2 hours after a meal. On the other hand, it is also possible to cause hyperthyroidism by taking too much medicine, so careful supervision by a healthcare provider with regular measurements of TSH are important.

Hyperthyroidism can be cause by overproduction of thyroid hormone or by injury of the gland with transient release of the hormone into the bloodstream. The first type is most common, and often requires surgical removal or radioactive destruction of the gland for definitive treatment. In this type, medications can also be used for long-term control for or in emergencies to bring hormone levels down quickly. The most commonly used

medications—propylthiouricil and methimazole—block production of T4 and T3 by the thyroid gland. Dosages are adjusted based on measurements of TSH over time or, in emergency cases, the level of T4 as this changes more quickly than does TSH. The medicines do not cure the hyperthyroidism for most people, but rather just control it. Therefore, relapse of the symptoms of hyperthyroidism are common when medication is stopped. Both propylthiouricil and methimazole can affect the bone marrow, liver, gastrointestinal tract, and skin in addition to the thyroid gland, so monitoring for side effects is very important. Potassium iodide is a medication that can be used for short periods to stop production of T4 and T3 by the thyroid gland and to prevent release of the hormone. It is also given to people who have been exposed accidentally to high levels of radiation, as with nuclear accidents, to suppress the thyroid gland and prevent the development of thyroid cancer from uptake of radioactive substances into it.

In the cases when hyperthyroidism is caused by injury to the gland with release of hormone, propylthiouricil, methimazole, and potassium iodide have little or no role. Instead, medicines that block the effects of T3 on organs like the heart, such as nonselective beta-blockers, help control symptoms until the excess thyroid hormone finally leaves the bloodstream. Other medications, such as steroids, can also be used to block the conversion of T4 to T3.

SEE ALSO: Hypothalamus; Hypothyroidism; Thyroid Gland.

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TNF (Tumor Necrosis Factor)

TUMOR NECROSIS FACTOR (cachexin, cachectin, TNF) is a multifunction cytokine. When produced in normal amounts, it is a beneficial part of the immune inflammatory system. However, when it is produced in abnormally large quantities, numerous pathologic conditions result. This is especially true in obesity.

TNF is but one cytokine of the tumor necosis factor family, all of which have 157 amino acids and a tetramer structure. In normal circumstances, TNF plays a vital role in programmed cell death, cellular proliferation, differentiation, inflammation, tumrogenesis, fever, acute phase reactions, appetite regulation, neutrophil migration, tumor cell death, coagulation, and endothelial cell function. It is usually thought of as a product of macrophages, but it is also produced by lymphoid cells, adipocytes, fibroblasts, neuronal tissue, and mast cells.

In obesity, high levels of TNF are produced by adipocytes throughout the body, but especially in the waist-hip region. Adipocyte TNF messenger RNA expression rises with elevated body mass index (BMI) and falls with weight loss. This affects numerous organ systems and is in part responsible for the morbidity that affects the obese.

In the coagulation system serum levels of TNF seem to induce production of plasminogen activator inhibitor (which impairs the breakdown of blood clots) and increase plasma levels of von Willebrand factor (which increases the likelyhood of blood clots, and is a marker of inflammation). This is believed to contribute to the higher occurrence of thrombotic events such as deep venous thrombosis, pulmonary embolism, myocardial infarction, and stroke.

The higher incidence of insulin resistance and diabetes in obesity is believed to result from adipocytes and macrophages, which are found in white adipose tissue that secrete TNF and other cytokines (adiponectin, resistin, interleukin-6 [IL-6]) which exert lipotoxic effects on pancreatic beta cells. TNF impairs insulin signaling by downregulating the activity of the insulin receptor. It also increases serum glucose due to a reduced number of muscle cell glucose receptors. These impairments that are partly caused by obesity may promote the cycle of insulin resistance and encourage the development of diabetes.

TNF along with IL-6 plays a role in dyslipidemia (high levels of blood lipids) in obesity. These cytokines inhibit lipoprotein lipase which increases very-low-density lipids and increases the secretion of triglycerides. These cytokines also increase lipolysis which causes elevation in free fatty acids. The cytokines also activate serum amyloid A in the liver which causes a

drop in high-density lipoproteins. TNF alone stimulates hormone-sensitive lipase, and downregulates fatty acid binding protein and fatty acid synthetase.

It seems that there are numerous other effects of TNF in obesity being discovered almost daily. Weight loss, however achieved, results in falling levels of TNF. This causes improvement in the organ system affected.

SEE ALSO: Cytokine; Fertility; Interleukins.

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Toxic Environment

PSYCHOLOGIST KELLY BROWNELL coined the term *toxic environment* in 1994 to describe the obesigenic effects of our physical surroundings. Brownell argued that despite promising advances in our understanding of biological risk factors for overweight, the precipitous increase in obesity at the population level has occurred so quickly that it cannot be attributed solely to genetic factors. Instead, he proposed that the most straightforward explanation for the upsurge of obesity in the last decades of the 20th century is that the modern environment systematically promotes the consumption of unhealthy foods and discourages physical activity. The toxic environment provides a parsimonious explanation for the well-established finding that individually targeted behavioral weight-loss programs and community-based nutrition education campaigns produce modest reductions in population body weight. Proponents of the toxic environment theory predict that curbing the rising tide of obesity will require public policy changes that encourage healthful diet and exercise choices.

THE TOXIC FOOD ENVIRONMENT

Unhealthy foods such as ice cream, potato chips, and candy not only prey upon biological preferences for sugar and fat, but are also conveniently accessible, heartily portioned, cleverly marketed, and relatively inexpensive in the contemporary environment. These powerful influences converge to render unhealthy food choices the default option for modern consumers.

With regard to convenience, fast-food restaurants boast the 24-hour availability of high-calorie menu items, such as cheeseburgers, french fries, and sugared soft drinks. In addition, prepackaged snack foods are readily available at sites previously unrelated to food consumption, including gas stations, hospitals, and schools. Experimental paradigms highlight the surprising influence of tiny changes in the convenience and accessibility of unhealthy foods; in one study, secretaries ate nearly three times as many candies per day when candy bowls were placed on their own desks versus a desk two meters away. The pervasive influence of convenience is further illustrated by findings from a recent epidemiological survey indicating that approximately one-third of American children aged 4–19 consume fast food on a typical day.

Another facet of the toxic food environment is increasingly large portion sizes, which have expanded in tandem with national waistlines. Marketplace research indicates that typical portions of convenience foods such as bagels, hamburgers, muffins, and soft drinks surpass U.S. Food and Drug Administration serving size recommendations by two- to eight times. For example, the original Coca-Cola bottle held just 8 ounces of soft drink, while contemporary vending machines stock 20-ounce bottles. The first Hershey chocolate bar weighed just 0.6 ounces, while current versions weigh from 1.6 to 8 ounces. A growing number of experimental studies have linked increasing portion sizes to excess energy intake. In one investigation, participants who served themselves from four-liter bowls ate approximately 50 percent more nuts, chips, and pretzels than participants who served themselves from two-liter bowls.

Moreover, unhealthy foods are very heavily marketed, especially to young people. In 1998 alone, soft drink companies collectively spent $115.5 million advertising

their products, and fast-food chain McDonald's spent more than $1 billion. In contrast, the National Cancer Institute recently spent just $1 million to promote its "5 a day" fruit and vegetable campaign. On average, American children view 10,000 food advertisements per year, most of which promote the purchase of energy-dense foods such as candy, soft drinks, sugared breakfast cereals, and fast foods. In addition to traditional advertisements, insidious guerilla marketing techniques—including product placements in television shows and video games and the placement of snack food logos on school-based athletic scoreboards—blur the line between advertising and entertainment.

In addition to being more convenient, accessible, and heavily marketed than healthy foods, data also indicate that unhealthy foods are more affordable. A recent analysis of grocery store prices identified an inverse relationship between energy density and cost. Specifically, foods high in calories but low in nutrients (such as margarine, mayonnaise, and sugar) were sold at significantly lower prices than foods low in calories but high in nutrients (such as green peppers, lettuce, and fresh tomatoes). Epidemiological data also suggest that there may be fewer grocery stores and more fast-food restaurants in poorer versus wealthier communities, making healthy food choices even more difficult for low-income families.

THE TOXIC PHYSICAL ACTIVITY ENVIRONMENT

Despite governmental recommendations that Americans engage in at least 30 minutes of moderate-intensity physical activity each day, epidemiological research indicates that the majority of American adults are not regularly active. Available data suggest that environmental factors such as energy-saving devices, zoning regulations, and urban design converge to discourage physical activity in the modern environment.

Modern energy-saving devices ranging from dishwashers to elevators have rendered physical activity an increasingly less integral part of daily life. In the early part of the 20th century, agricultural and industrial occupations required intensive manual labor. Modern employment opportunities have become increasingly sedentary in response to dizzying technological advances. Similarly, outdoor leisure activities such as sports have been displaced by less active pastimes such as watching television, playing video games, and surfing the internet. A recent prospective study revealed that young children who watched at least three hours of television per day weighed significantly more as adolescents than those who watched less than 1.75 hours per day as children.

Furthermore, zoning regulations that prohibit the co-aggregation of residential neighborhoods and local businesses promote increasing reliance on automobiles for transportation. The suburban sprawl emanating from these regulations necessitate high-speed, high-traffic streets on which pedestrian and bicycle travel are both impractical and unsafe. Against this backdrop, researchers have begun to identify characteristics of the built environment that foster higher levels of physical activity among community residents.

For example, a recent observational study found that residents of neighborhoods featuring high population density, proximity of homes and businesses, and high street connectivity engaged in 70 more minutes of physical activity per week and exhibited significantly lower rates of obesity than residents of neighborhoods that did not possess these features. Similarly, another study found that adults residing in neighborhoods with a high density of recreational resources (e.g., parks, trails, swimming pools) were

A recent study revealed that children who watched at least three hours of television were more likely to be overweight.

more likely to engage in physical activity during a typical week than adults residing in neighborhoods with a low density of recreational facilities.

The major lesson of the toxic environment is that individually targeted behavioral weight-loss interventions and community-based nutrition education campaigns will continue to produce a very modest impact on population body weight if the seductive environmental forces that promote obesity remain unchecked. Therefore, proponents of the toxic environment theory argue that policy changes geared toward neutralizing the toxic environment will be essential to the success of future obesity prevention efforts.

Specifically, public health advocates have proposed that removing soft drinks from schools, restricting food advertising targeted toward children, and levying taxes on unhealthy foods (or creating subsidies for healthy foods) could have a salutary effect on the national diet. Similarly, proponents of lifestyle activity have suggested that zoning reforms encouraging the proximity of business and residential buildings, as well as the creation and maintenance of neighborhood parks, trails, and greenways, could promote systematic increases in physical activity at the population level.

SEE ALSO: Access to Nutritious Foods; Advertising; Built Environments; Economics of Food; Fast Food; Increasing Portion Sizes: Taxation on Unhealthy Foods.

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Transgenics and Knockouts for Obesity-Related Genes

A TRANSGENIC MODEL is created through the manipulation of an animal's genome causing a change in the expression of an endogenous gene. The gene(s) can be overexpressed, inactivated, or otherwise influenced. Trangenics can also involve the additional expression of a human gene or a gene foreign to the model's genome. Alternatively, a genetic knockout is a complete loss of a specific gene, typically through homologous recombination techniques. Creation of genetically modified models with obese phenotypes is useful for studying the factors affecting the onset of obesity as well as understanding associated comorbidities.

Mice are the most commonly used animal in transgenic and knockout studies due to their similarity in physiology and gene function to humans. In addition, mouse genetics has been well studied, and the techniques for manipulation have made the process of creating a transgenic model relatively simple. In mice, there have been 248 genes that were identified as being involved in weight maintenance or adiposity through transgenic model studies or through naturally occurring mutations. Some of the more frequently studied transgenic and knockout strains will be discussed in this entry.

The melanocortin system is involved in feeding regulation and many of the associated proteins and receptors have been studied using transgenic and knockout models. Melanocortin (MC) and α-MSH, a product of proopiomelanocortin (POMC), inhibits feeding. A knockout of the POMC gene becomes obese within the first two months of life. These mice were unable to regulate their energy intake or their metabolism with changes in diet. Treatment of these mice with α-MSH reversed the associated obese phenotype.

Knockouts in the melanocortin receptor MC3-R and MC4-R gene have been established, both of which cause an obese phenotype. MC4-R mutations occur occasionally in the human obese population at about 4 percent, so this model is important in studying this population subset. Inactivation of MC3-R causes obesity primarily through metabolic changes, and reduced physical activity. A mouse model with inactivation of both MC3-R and MC4-R is heavier than the single knockouts, implicating that the genes are functionally different.

The protein agouti-related protein (AGRP) is an inhibitor both MC3-R and MC4-R. The transgenic overexpression of AGRP in mice exhibits obesity and altered insulin levels, and for this reason, this strain is typically used to study obesity accompanied by insulin resistance. Overexpression of the syndecan-1 gene, which codes for a protein involved in regulating

the binding of AGRP to the MC receptors, resembles the AGRP overexpression strain, also showing an increase in body weight due to excess caloric intake.

In addition to these transgenic strains, overexpression of melanin concentrating hormone (MCH) gene, and a knockout of cocaine- and amphetamine-regulated transcript (CART) have also been studied and show an increase in susceptibility to weight gain and an increase in caloric increase, respectively. These proteins are also associated with the melanocortin system.

In addition to the melanocortin-associated genes, there are several other obesity-related neurotransmitters, proteins, and receptor genes which have been altered via transgenic technology. The neurotransmitter NPY regulates several physiological pathways, including feeding behavior. Two of its receptors, neuropeptide-Y1 (NPY-Y1) receptor and neuropeptide-Y2 (NPY-Y2) receptor, have been disrupted in mice. NPY-Y1 receptor knockout presents with obesity, most frequently in females due to metabolic dysregulation. Alternatively, NPY-Y2 knockouts show leptin resistance, with moderate obesity but exhibit normal caloric intake and body weight regulation

Gene disruption models of serotonin, a transmitter involved in mammalian behavior, are used as a model for behavior changes in food consumption. Two serotonin receptors knockouts 5-HT_{1B} and 5-HT_{2C} were established, and displayed notably different phenotypes. Knockouts of receptor 5-HT_{2C} are heavier than their controls, and their obesity is due to increased appetite and food consumption (hyperphasia), while strains lacking expression of receptor 5-HT_{1B} did not show an obese phenotype. A knockout of orexin-A and B, neuropeptides also influencing feeding behavior and metabolic rate, also present with late-onset obesity.

Mice deficient in brown adipose tissue (BAT) are studied as a model for decreased energy expenditure. BAT regulates energy through uncoupling proteins which dissipates fatty acid derived energy as heat. These mice are obese due to a decrease in thermogenesis and a shift in energy balance. β_3-adrenergic receptor (β_3-AR) is also involved in the activation of uncoupling proteins. β_3-AR receptors are present in adipose tissue and when they are disrupted become mice have a decrease in energy expenditure and become moderately obese, especially in females.

In a transgenic model of GLUT4, which is a glucose transporter seen in fat and muscle tissue, GLUT4 was overexpressed specifically in adipose tissue but not in muscle. This manipulation caused an increase in the number of fat cells, but not the size of these cells, making this model useful in understanding fat cell replication.

In addition to these models, a transgenic rodent strain containing a useful "on/off" switch for an obesity phenotype also exists. This strain contains an ovine growth hormone transgene that can be inactivated to change the levels of circulating growth hormone (GH). GH is able to regulate the breakdown of fat, and when it is inactivated, mice become obese. Similarly, a severe late-onset obesity rat (SLOB) containing transgenic expression of growth hormone has been created. These rats are still not completely understood, but are different from all other rodent obesity models that have been studied up to this point.

SEE ALSO: Agouti and Agouti Related Protein; Animal Models of Obesity; Monogenic Effects that Result in Obesity; New Candidate Obesity Genes.

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Translational Research

TRANSLATIONAL RESEARCH CAN be defined as the process by which discoveries made at the molecular or cellular level in basic scientific research are applied, or translated, into diagnostic tests or therapies for the purpose of identifying, preventing, or treating disease. Successful translational research depends on the cooperation of multidisciplinary teams consisting of basic and clinical researchers, healthcare providers, community members, and other agency

partners. Translational research is a key component of national obesity prevention and treatment initiatives, such as those supported by the National Institutes of Health (NIH).

There are two distinct segments, or phases, of translational research. The first phase involves the movement of ideas, insights, and discoveries from the "bench" of basic science to clinical research with humans. This clinical research, carried out under controlled conditions through a series of clinical trials, is designed to test the safety, effectiveness, and proper dosing of medical treatments. The second phase of translational research involves applying the clinical trials results to real-world situations with diverse populations in uncontrolled settings, either within the clinic (the "bedside") for diagnosis, prevention, or treatment of disease, or as an intervention within the larger community.

In theory, translational research is intended to function as a two-way communication process. Ideally, as scientific discoveries move from laboratory to clinical trials and then to the clinic and community settings, feedback from clinical trials, clinics, and community settings should flow back to scientists conducting basic research. The two-way communication process has proven challenging, however, because it requires, on the one hand, that clinicians understand the research conducted at the bench by scientists, and that the scientists understand the complexities of treating disease in real-world settings.

To facilitate a two-way communication process in translational research on obesity, the Strategic Plan for NIH Obesity Research serves as a guide for coordinating obesity-related research activities within the NIH and with partner agencies, clinical researchers, and communities. The Strategic Plan includes translational research as a key component of cross-cutting research topics to address obesity as a public health problem. As part of the Strategic Plan, the NIH supports Obesity/Nutrition Research Centers (ONRCs) and Clinical Nutrition Research Units (CNRUs) located throughout the country. The Centers conduct basic, clinical, multidisciplinary, and translational research on obesity, eating disorders, and weight management. In addition, the NIH collaborates with partners such as the Agency for Healthcare Research and Quality (AHRQ), the Centers for Disease Control and Prevention (CDC), and the American Diabetes Association to disseminate clinical trials results to the community and facilitate the two-way communication process.

Finally, the NIH *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults* provides an example of translational research in action. The guidelines are designed to help practitioners identify and treat patients with obesity by encouraging decreased calorie consumption and increased physical activity. The Guidelines' recommendations are based on a combination of clinical trials data and observations of actual medical practice activities.

SEE ALSO: National Cancer Institute; National Institutes of Health; NIDDK.

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Treatment Centers for Eating Disorders

SERVICES FOR TREATING eating disorders range on a continuum of intensity from inpatient hospitalization to residential care to partial hospitalization programs to varying levels of outpatient treatment. Decisions about the best treatment setting in which to manage a person with an eating disorder depend on the nature of the disorder, the level of risk, physical and psychological complications, and patient preference. There is evidence that those with eating disorders have better outcomes when treated in specialized eating disorder facilities where staff has experience and expertise in treating eating disorders. Major deterrents in selecting the most appropriate treatment include geographic

proximity to specialist centers that limit access, lack of data for comparing cost and effectiveness of different settings, inadequate insurance coverage, and managed care decisions that limit access to effective care.

DIAGNOSTIC GROUPS

Anorexia nervosa occurs in about 1 in 250 females and 1 in 2,000 males and is estimated to be the third most common chronic medical illness in girls 15–19 years of age. Bulimia nervosa is about five times more common than anorexia nervosa. The mortality rates for anorexia nervosa are more than 12-fold the number of deaths expected from all causes among women 15–24 years of age and two to three times as high as any other psychiatric disorder. Mortality rates for bulimia nervosa are lower but still not insignificant. Medical complications are typical during the acute phase of an eating disorder and persist among those not successfully treated, leading to a wide range of physical and emotional disorders into early adulthood.

An even larger diagnostic category consists of patients with "atypical eating disorders," or "eating disorders not otherwise specified." These are conditions of clinical severity that do not conform to the diagnostic criteria for anorexia nervosa or bulimia nervosa, but nevertheless closely resemble these disorders. An example would be someone who possesses all of the features of bulimia nervosa but purges only after consuming small amounts of food (does not have objectively large binge-eating episodes). Another example is an individual at low weight with the features of anorexia nervosa who is still menstruating. Yet another example would be a person who maintains a low normal weight by engaging in extreme restrictive dieting, compulsive exercise and vomits only one time a week on average.

Many people with atypical eating disorders have suffered with anorexia nervosa or bulimia nervosa in the past. Binge eating disorder (BED) has been more recently identified and refers to individuals who engage in uncontrollable episodes of binge eating but do not use compensatory behaviors. Many BED patients are obese and may or may not be accepted into treatment facilities that routinely accept those with anorexia and bulimia nervosa.

Current systems of diagnosing eating disorders into mutually exclusive categories based largely on body weight, bingeing, and purging fail to emphasize the overlap between these conditions in terms of core psychological features, behavioral symptoms, medical symptoms, motivation for change, and social supports. Moreover, there is extraordinary variability within each of the diagnostic subgroups on these key variables. It is well known that individuals with eating disorders can move between the diagnostic categories at different points in time based only on variations in body weight and symptoms such as binge eating and vomiting. Therefore, it is important to avoid generalizations based only upon diagnosis.

SELECTING A TREATMENT SITE

Eating disorders treatment facilities can be distinguished in terms of treatment philosophy, types of clients served, levels of care provided, program structure, staff training, cost, and documented effectiveness. In determining the optimal initial treatment or whether a change to a different type or level of treatment, it is important to consider the patient's age, the nature of the eating disorder, level of risk, physical and psychological complications, motivation, social circumstances, and patient preference.

Pretreatment evaluation of the patient is essential for determining the appropriate treatment setting. Patient weight, rate of weight loss, cardiac function, and metabolic status are the most important physical parameters for making this choice. Eating disorders should be recognized and early treatment implemented as soon as possible after the onset of symptoms. This is especially true in children, adolescents, and young adults to avoid the disorder becoming chronic.

FACTORS TO CONSIDER IN THE CHOICE OF A TREATMENT SITE

A comprehensive clinical assessment is the first step in deciding what is the most appropriate form of treatment. A proper clinical assessment is a careful assessment of the patient's history and current circumstances and usually, if done thoroughly, requires several hours. It includes a thorough review of the patient's height and weight history; current and past symptoms, restrictive eating behaviors, binge eating, exercise patterns, purging and other compensatory behaviors; attitudes regarding weight, shape, and eating as well as the presence of other psychological disorders.

A full physical examination of the patient is strongly recommended and may be performed by a physician familiar with common findings in patients

with eating disorders physical health and associated risks. For children and adolescents, family involvement in the assessment is considered essential. It is desirable for older patients. An assessment should include a family history of eating disorders or other psychiatric and medical disorders, family stressors, and family attitudes toward eating, exercise, and appearance. Family members usually approach treatment with considerable guilt about their role in the development of the eating disorder, and theories that imply blame of family members can alienate family members and interfere with the patient's care and recovery. Therefore, it is important to take the focus off of these theories when interacting with patients' families. Standardized and empirically validated measures of eating disorder symptoms and associated psychological features should be part of the initial assessment and discharge.

SPECIALIZED EATING DISORDER PROGRAMS

Specialized eating disorder programs are not available in all geographic areas, and their financial requirements may limit access. However, there is evidence to suggest that patients with eating disorders have better outcomes when treated in settings specializing in the treatment of these disorders than when treated in general settings where staff lack expertise and experience in treating eating disorders.

MULTIDISCIPLINARY APPROACH

Optimal treatment requires a highly skilled interdisciplinary team, whose members are specifically trained in the management of the full spectrum of disorders. Ideally, staff should include experienced psychiatrists, psychologists, social workers, registered nurses, dietitians, mental health workers, and school personnel to coordinate ongoing education with schools as well as provide tutoring. Medical consults should be readily available from cardiology, endocrinology, gynecology, and internal medicine.

ONGOING ASSESSMENT OF EATING DISORDER SYMPTOMS AND MEDICAL CONDITION

There is considerable variability across treatment centers in the frequency with which body eating disorder symptoms (weight, body, medical status) are part of the ongoing assessment. The treatment team must be continually vigilant regarding shifts in weight, blood pressure, pulse, other cardiovascular indices, and behaviors that are likely to precipitate physiological decline and collapse. Patients should be continually monitored for safety with particular attention given to suicidal thoughts, plans, and intentions, as well as to impulsive behavior including self-harm behaviors.

DO PROGRAMS MEET NEEDS ACROSS THE CONTINUUM OF CARE?

Not all specialized eating disorders programs provide all available levels of care—inpatient hospitalization, partial hospitalization, intensive outpatient, and outpatient treatment. However, providing a range of services can facilitate continuity of care. Programs must give consideration to the age continuum (from adolescents to adults) and disorder severity (from mild to severe and chronic). It is important for the setting to be age appropriate to the educational and social needs of children and adolescents.

Stepping down from one level of care to a less intensive level may be destabilizing for a patient and can lead to resistance to change. Patients may erroneously interpret moving to a less restrictive level of care as meaning they should be expected to be fully improved. Programs that encourage continuation of treatment with familiar and trusted staff across different levels of care are optimal, because they may contribute to the success of aftercare planning. If this is not possible, there must be careful coordination between clinicians planning a move from one treatment setting to another to ensure continuity and attention to important aspects of treatment.

INDIVIDUALIZED SERVICES

Eating disorder programs vary considerably in the degree to which services are tailored to meet the individual needs of patients. Specific treatment protocols are followed but are adapted to meet the unique needs of individual patients. An individualized treatment plan should be developed for each patient based on a comprehensive assessment, and it should be grounded in evidence-based treatment principles. This plan should also be continually revised, with active input from patients and families based on changes that occur during treatment. Programs of fixed duration or those identified too closely to one philosophical orientation (e.g., faith-based) or exclusive treatment

model (e.g., addiction model) are obviously limited in their ability to adapt to individual patient needs.

EVIDENCE-BASED TREATMENT RECOMMENDATIONS

One of the most important advancements in the treatment of eating disorders has been the dissemination of evidence-based practice guidelines from the American Psychiatric Association (APA) in 2006, the National Institute for Clinical Excellence (NICE) in 2004, and the Evidence Based Practice Center in 2006. These clinical practice guidelines provide clinicians with specific treatment recommendations based on the best available empirical evidence. Where evidence is lacking, they provide recommendations based on the consensus view of experts in the field. Treatment centers should endeavor to follow these guidelines where possible. They should also be dedicated to improving clinical practice by evaluating therapeutic outcomes using well-established measures of change. Data should be collected prospectively using standardized measures that can be used as a benchmark for progress and to determine the outcomes and effectiveness of current practices. Patient evaluations of treatment are also part of routine assessment, and are used to assess the effective components of treatment, as well as to evaluate therapists for the purpose of remedying deficits and adding new skills.

INVOLVEMENT OF THE FAMILY AND CARERS

There are ethical, financial, and practical grounds for including parents in treatment of younger patients. There are also benefits in involving the family in the treatment of adult patients to the degree that the patient is at risk and the degree to which parental involvement is likely to reduce that risk. Many adult patients live with parents or other caregivers and, depending on the extent to which they require nutritional and behavioral management, family members need to be involved if management is to be effective. In some cases, the involvement is limited to information about the disorder and management principles.

On one hand, involvement in therapy can lead to recommendations for parents to be less involved in practical day-to-day management of food and symptom control. On the other hand, caregivers may assume a significant role in shopping for food, meal planning, and managing meal times. In most cases, families benefit from education and support to deal with the stress they experience. Although clear parameters need to be established related to confidentiality, it is also important to consider the health and safety concerns that may take priority.

OUTPATIENT TREATMENT

In general, those with bulimia nervosa and binge-eating disorder can be effectively treated with outpatient therapy. Some patients with anorexia nervosa can begin effectively treated in an outpatient setting; however, if progress is not made, they should be referred to specialized partial hospitalization or inpatient programs. Similar recommendations are appropriate for bulimia nervosa patients who fail outpatient treatment or who develop serious medical complications. Failure to refer patients who remain highly symptomatic (low weight, high frequency of bingeing and purging) is not recommended by current practice guidelines.

INPATIENT HOSPITALIZATION

Inpatient hospitalization should be considered for individuals who are markedly underweight and for children and adolescents whose weight has deviated below their expected growth curves. Generally, adult patients with anorexia nervosa have difficulty gaining weight without the structure provided by inpatient treatment. Hospitalization may also be appropriate for some eating disorder patients who are not at a low body weight. Factors suggesting hospitalization may be appropriate include rapid or persistent decline in food intake, medical instability manifested in abnormalities in vital signs or abnormal laboratory tests, continuing weight loss while in a lower level of care, uncontrolled bingeing and purging, the presence of family or social stressors leading to psychological instability, or other psychiatric problems that merit hospitalization. Ideally, hospitalization should occur before the onset of medical instability.

AN ECONOMICAL ALTERNATIVE TO INPATIENT AND RESIDENTIAL TREATMENT

Anorexia nervosa can be particularly costly to treat because the duration of inpatient treatment required for complete weight restoration can be between two to three months and relapses are common during the first year after discharge. The costs of inpatient treatment have increased dramatically in the past two decades,

placing an onerous burden on insurers and those individuals who must pay for treatment privately. One approach to cost containment has been shorter lengths of inpatient treatment emphasizing medical stabilization of acute medical symptoms. However, shorter lengths of stay result in lower discharge weights and low discharge weights are one of the most reliable predictors of relapse and poor long-term outcome.

Partial hospitalization programs (PHPs) that specialize in eating disorders are more economical than inpatient treatment and have produced encouraging outcomes with adults. Patients participating in a PHP generally participate in treatment at least five days a week for seven hours a day. Higher quality programs are able to achieve about the same rate of weight gain as inpatient hospitalization. The cost savings can be considerable with some PHPs being able to provide as much as three months of treatment for the same cost as 10–14 days of inpatient hospitalization. An added advantage of the PHP level of care is that the time outside of the program on evenings and weekends permits patients to practice eating, socialization, and relapse prevention, while participating in treatment that is relatively intense and structured.

GEOGRAPHIC PROXIMITY TO TREATMENT CENTERS

There has been a proliferation in specialized treatment centers in recent years and some have invested heavily in aggressively marketing their programs. Some of these programs provide quality care; however, it is important for the consumer not to confuse marketing skills with treatment effectiveness. In general, seeking treatment at specialized centers, with closer geographic proximity to home, makes ongoing family involvement during treatment more practical. Moreover, the recovery process can be fraught with advancements followed by backslides and brief periods of readmission can be a valuable option. Treatment centers should work closely with patients to develop a realistic plan after discharge and this usually will involve making referrals to therapists in the patients' communities. Nevertheless, if complications or a relapse should occur, being relatively near the center providing care can be reassuring and lead to better aftercare.

EXPECTED OUTCOMES

There have been major advancements in the treatment of both anorexia nervosa and bulimia nervosa in recent years. Although there are large variations across outcome studies, approximately 70 percent of adolescents with anorexia nervosa recover from their eating disorder. The course can be difficult for these patients, and a significant proportion remain impaired in terms of psychological adjustment and social functioning. Long-term follow-up studies of adults with anorexia nervosa indicate a less favorable outcome with approximately 50 percent recovery rates; however, again, there are significant variations in outcome across follow-up studies. Despite these promising results, there is tremendous variability in treatment outcome from different centers, suggesting that the components for effective treatment exist but are not well established or consistently applied.

There is more evidence of effective treatment for bulimia nervosa, with as many as 70 percent recovering within three months of an empirically validated outpatient cognitive-behavior therapy. However, recent studies have emphasized the fluctuating nature of symptoms over the follow-up period with many patients who have pervasive psychological and interpersonal problems. This cannot be attributed simply to selective bias associated with treatment seeking, because prospective studies of representative community-based cases indicate that poor psychosocial functioning and psychological maladjustment affect a significant subgroup of patients with anorexia and bulimia nervosa. Even among those who recover from anorexia nervosa, there is evidence of elevated rates of persistent perfectionism, obsessionality, and poor social functioning, as well as an increased risk for a range of health problems during early adulthood.

SEE ALSO: Anorexia Nervosa; Binge Eating; Bulimia Nervosa.

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Tryptophan

TRYPTOPHAN IS AN essential amino acid. It has been implicated in playing a role in obesity because of its function as a precursor to various neurotransmitters that regulate homeostasis in the body. Once tryptophan is ingested, it can be converted to serotonin by serotonergic neurons in the brain. Serotonin neurons can affect the brain function of food intake—the serotonin neurons participate in the neuronal pathway that reduces the intake of food. Therefore, any abnormal metabolism of tryptophan could potentially be one of many causes of obesity and normalizing the level of tryptophan and correcting the abnormal metabolism pathway of tryptophan and serotonin can be considered a treatment for the obese.

Tryptophan is required in human diet and it is often found in dairy, meat, seeds, and nuts. It is mainly involved in protein synthesis; however, it can serve as a precursor to neurotransmitters or niacin. Tryptophan has been recognized to play a possible role in obesity due to its ability to convert to serotonin—serotonin has been known to participate in controlling sleep, appetite, mood, sexual behavior, and pain sensation. Serotonin neurons in the brain control appetite and a negative correlation is observed between the level of stimulation of those neurons and the amount of food intake.

When tryptophan is ingested as part of diet, it can be metabolized in various ways. First, tryptophan is normally modified to one of the two metabolites: 5-hydroxytryptophan or N-formylkynurenine. In the presence of the enzyme tryptophan 5-monooxygenase, tryptophan is converted to 5-hydroxytryptophan, which will be further metabolized to serotonin with the help of vitamin B6. Under certain conditions, such as stress, elevated cortisol level, vitamin B6 deficiency, or a high dosage of tryptophan (above 2,000 mg), the pathway to the production of N-formylkynurenine is preferred, reducing the amount of available serum tryptophan and 5-hydroxytryptophan.

Because serotonin is impermeable to the blood–brain barrier and needs to be synthesized by neuronal cells in the central nervous system after the uptake of tryptophan, the decreased availability of tryptophan in the central nervous system may compromise the adequate production of serotonin. Furthermore, lowered proportion of tryptophan to five other amino acids—tyrosine, phenylalanine, valine, leucine, and isoleucine, all of which share the same transporter molecule with tryptophan—can negatively affect the production of serotonin.

The serum concentration of tryptophan can fluctuate based on a variety of factors including stress and dieting. The drop in the serum concentration of tryptophan will lead to the reduced uptake of the amino acid by the central nervous system, which will in turn result in a decreased level of serotonin in the central nervous system due to its heavy dependence on the concentration of tryptophan to produce serotonin in the central nervous system. Because 5-hydroxytryptophan can readily cross the blood–brain barrier and serves as a precursor to serotonin, 5-hydroxytryptophan is being studied as an agent that reduces food intake by increasing the availability of serotonin.

SEE ALSO: Neurotransmitters; Obesity and the Brain; Serotonin.

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Tubby Candidate Gene

IN THE 1970S, the tubby gene (normal: tub; mutated: *tub*) caused severe obesity in mice. Several other animal species, including humans, were subsequently found to have the tubby gene. This gene is one of several genes which, when mutated, lead to severe obesity. It provides an avenue for research into the genetic causes of obesity, and therefore potential therapies and cures.

Mice are excellent animal models for numerous diseases, disorders, and genetic characterizations. One reason for this utility is that mice can be raised in inbred colonies such that every mouse is of identical genetic background. Researchers can then manipulate one gene at a time and exercise selective breeding

to create colonies with some mice that differ at only one particular gene. Sometimes, spontaneous mutations arise in a colony. Scientists can then use genetic analysis techniques to determine which gene spontaneously mutated, and the result of the mutation. At the Jackson Laboratory, a leading mouse supplier, two such spontaneous mutations arose, and the mice harboring the mutations developed severe obesity. One gene was named *fat* and the other *tubby*.

Fat and tubby mutants result in slow-onset severe obesity that does not ultimately progress to diabetes. Nevertheless, fat and tubby mice show hyperinsulinemia, hyperactivity of beta cells (in the islets of Langerhans of the pancreas), and degranulation of the beta cells.

In a study published in 1990 in the *Journal of Heredity*, scientists at the Jackson Laboratory determined that the inheritance of the *tub* mutation was autosomal recessive, that is, the gene is not on a sex chromosome but rather on an autosome, and a mouse must have both copies of its genes mutated to develop the severe obesity. A mouse with one *tub* gene and one normal gene would pass the *tub* gene to one-half of its offspring; these offspring, like the original mouse, would be carriers, showing no symptoms of obesity.

Tubby mice have a slow progression to obesity. The obesity becomes apparent within a few months of age. A mouse a few months of age is considered an adolescent. In addition, males become obese more rapidly than females. Similarly, males have a quicker progression of plasma insulin concentration and declining morphology of the pancreas.

Tubby mice are not sterile; however, they are relatively infertile in that they can produce offspring only if mated before the onset of severe obesity. Tubby mice have even fat distribution over the whole body. There are no regions with more pronounced fat deposits.

The tubby gene is located on the seventh murine autosomal chromosome. Researchers point out that although the gene for insulin II is also present on chromosome 7, the *tub* mutation does not have an effect on insulin structure or ratio of insulin I to insulin II levels.

In 1996, an article was published in the journal *Cell* outlining the molecular basis of the *tub* mutation. The scientists sequenced the normal gene and the mutant gene. They found that the normal gene is predominantly expressed in the brain, specifically in the hypothalamus. The hypothalamus is a region of the brain that, among other tasks, monitors body weight. It does so by regulating energy expenditure, and also has some control over appetite. Additionally, the scientists discovered that the normal mouse tub gene is 89 percent identical to a human gene, now also named tub. When the gene is read into a protein, the amino acid sequences of the two proteins (mouse and human) are 94 percent identical, with near-exact identity in the latter portion of the protein. These scientists also showed that the protein is hydrophilic (water soluble), and most likely not a cellular transmembrane protein.

By 2002, four tubby gene family members had been found in vertebrates. The *tub* mutation was also known to cause neurosensory degeneration in the photoreceptors in the retina and also the cochlea. Programmed cell death, or apoptosis, of neuronal cells is seen in all four *tub* mutations; the normal gene could then be involved in controlling the cell's normal life cycle, or more likely, the mutant cells might be committing suicide because they are irreparably ill.

A review published in 2002 by Akihiro Ikeda et al. outlines three possible cellular roles for tub—two roles are involved in reception of cell signaling and one in intracellular transport of signaling compounds. Therefore, if the tub gene is mutated and the cellular signaling cannot occur as normal, the most affected cells would be those with the most tub activity normally, such as the hypothalamus. Given that the hypothalamus is a regulator of body weight, it is understandable how the *tub* mutation leads to obesity, via disrupted signaling between neurons in the brain region that controls body weight.

Several mouse lines, each with a single gene mutation resulting in obesity, have been reported; therefore, there is credence to the theory that some people with obesity may have a genetic component to their obesity. Because these genes can be unrelated, it is not highly plausible that there will be one master gene which causes obesity.

SEE ALSO: G-Protein Coupled Receptors; Genetics; Genomics; Hunger; Hypothalamus; Insulin; Neuropeptides; Neurotransmitters; Ob/Ob Mouse.

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Twin Studies and Genetics of Obesity

MUCH RESEARCH EFFORT and expense is expended trying to find the causes of obesity, both genetic and environmental. Researchers have used one of nature's own experiments, the phenomenon of twin births, to derive valuable evidence that has informed our understanding of the causes of obesity.

The study of twins has played a vital part in quantitating the heritable influences on obesity and explains how this naturally occurring human model has helped researchers detect and quantify the effect of environmental influences independent of genetic factors. Twin studies play a part in understanding causation of obesity and in improving efficiency in the hunt for gene loci (sites).

When many believed that obesity was a matter of willpower, influenced only by environmental effects, the adoption studies of Dr. A. J. Stunkard were a historic turning point in scientific thinking. His landmark study showed strong genetic influence on the weight of Danish adults adopted between 1924 and 1947. He demonstrated that the offsprings' adult weight related strongly to the weight of the biological parents, rather than the adoptive parents who had provided their environment. For further evidence, he performed twin studies that confirmed the relatively stronger importance of inheritance than environment on adiposity.

One study compared the heritability of adiposity in adult twin men who had been reared together, compared to twin men reared apart. Stunkard's studies showed that there was no difference in the estimated heritability of fatness (which was high, at about 70 percent) between adult twins, whether reared together or apart. This also confirmed that genetic influences on fatness were greater than the environmental ones. The method for calculating heritability is described below.

Identical or monozygotic (MZ) twins occur about 4 in every 1,000 confinements and nonidentical or dizygotic (DZ) twins about 8 per 1,000 in Caucasians, with increasing age a factor in producing DZ twins. MZ twins share all their genes because they come from the same egg, and DZ twins, like ordinary siblings, come from two separately fertilized eggs and share half their genes. Unlike siblings, DZ twins are, of course, identical in age. Twins share very closely their pre- and postnatal gestational influences and rearing.

It is accepted that well-conducted twin studies have been able to estimate the relative importance of genes and environment by comparing the concordance (similarity) of the adiposity trait in MZ versus DZ twins. Heritability, a statistic which indicates the size of the heritable effect, refers to the amount of observed variation in the trait (e.g., fatness) that can be attributed to genetic variation. Heritability approximates to the doubling of the difference between MZ and DZ correlations for the trait, that is, $2x(r^2\ MZ - r^2\ DZ)$ where r is the correlation between twin pairs for that trait. The remainder of the variation is made up of environmental influences, which are either shared or unique to the individual. In most twin studies, this estimate for heritable fatness is in the range of 40 to 70 percent, indicating that 40 to 70 percent of the population variance in fatness across the study group is attributable to genetic factors. Some of the difference in heritability estimates is due to the estimate of fatness used, although most results are within this range whether measuring body mass index or more accurate, direct measures of total body fat, waist, or the more accurate and direct measures of central body fat.

Elegant intervention studies have also been performed in MZ twins. These showed that identical overfeeding (under close supervision) led to variable amounts of weight gain between twin pairs but very similar weight gain within MZ pairs. These important studies suggest familial factors determine the amount of weight gain in overfeeding. A similar familial effect of excess exercise on body compositional changes and weight loss has subsequently been shown in MZ twin pairs, suggesting that heritable factors regulate weight loss as well.

Another benefit from use of the twin model is to examine whether discordance (difference) for environmental factors commonly held to contribute to obesity explains the within-twin pair differences in body fat in genetically identical individuals. As the

Twin studies play a part in understanding causation of obesity and in improving efficiency in the hunt for gene loci (sites).

MZ twins are genetically identical, the relationship (correlation) between the discordance in environmental influence and the difference in adiposity (after correction for any known influences), can be quantitated, for example, between regular physical activity and adiposity. Further calculations are also possible to assess the gene–environment interactions determining the final adiposity of the twin subjects.

Current genetic theory suggests that genetic influence on common complex traits such as body fatness is likely to be due to multiple genes of varying effect size. There have been benefits from statistically modeling the clinical data available from such studies in statistical packages specially designed for this work. This type of multivariate analysis has allowed the correlations between clinical traits to be analyzed and broken down into "specific" and "in common" (or shared) genetic and environmental influences, guiding the search for genes as well as the overall understanding of the pathophysiology of disturbances of weight regulation. Accurate measurement of the traits associated with obesity should improve success. There have now been large population and volunteer-based twin registers set up in several countries as the need for large numbers of subjects has been recognized to detect those genes having small individual effects.

SEE ALSO: Genetic Mapping of Obesity-Related Genes; Genetics; Human QTLs; Mendelian Disorders Related to Obesity.

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Type 2 Diabetes

TYPE 2 DIABETES mellitus (T2DM) is a progressive metabolic disorder characterized by insulin resistance, insulin deficiency, and hyperglycemia. Nearly 21 million Americans have diabetes, and at least 54 million people over age 20 reported to have prediabetes. About 151,000 people below the age of 20 have been diagnosed with diabetes. The World Health Organization (WHO) estimates that more than 180 million people worldwide have diabetes and this number will double by 2030. In 2005, it was reported that an estimated 1.1 million people died from diabetes and 80 percent of diabetes deaths occur in low- and middle-income countries. Most of the deaths occur in people under the age of 70; 55 percent of diabetes deaths are in women.

The metabolic changes in T2DM may lead to organ damage, impairment of organ functions, and mortality due to cardiovascular disease. T2DM is often associated with obesity, hypertension, hypercholesterolemia, hypertriglyceridemia, proinflammatory cytokines, and coagulation factors, and cluster of all these risk factors may lead to cardiometabolic

syndrome. The common symptoms of T2DM are increased thirst, increased hunger, fatigue, increased urination especially at night, weight loss, blurred vision, and sores that do not heal.

DIAGNOSIS OF T2DM

The diagnosis criteria of T2DM is based on fasting blood glucose, postprandial blood glucose, and glycosylated hemoglobin (HbA1c). The normal fasting plasma glucose levels is considered normal if it is less than 100 milligrams per deciliter (mg/dl). If the fasting plasma glucose levels of more than 126 mg/dl on two or more tests on different days indicate diabetes of an individual. A random blood glucose test can also be used to diagnose diabetes. A blood glucose level of 200 mg/dl or higher indicates diabetes. When fasting blood glucose stays above 100mg/dl but in the range of 100–126mg/dl, this is known as impaired fasting glucose (IFG). A person is said to have a normal response when the 2-hour glucose level is less than 140 mg/dl, and all values between 0 and 2 hours are less than 200 mg/dl. A person is said to have impaired glucose tolerance when the fasting plasma glucose is less than 126 mg/dl and the 2-hour glucose level is between 140 and 199 mg/dl.

A person has diabetes when two diagnostic tests done on different days show that the blood glucose level is high. A woman has gestational diabetes when she has any two of the following: a 100g OGTT, a fasting plasma glucose of more than 95 mg/dl, a 1-hour glucose level of more than 180 mg/dl, a 2-hour glucose level of more than 155 mg/dl, or a 3-hour glucose level of more than 140 mg/dl. Glycosylated hemoglobin test ($HbA1_c$) measures blood sugar control over an extended period in people with diabetes.

In general, the higher the $HbA1_c$ value, the higher the risk that develop complications such as eye disease, kidney disease, nerve damage, heart disease, and stroke. The American Diabetes Association (ADA) currently recommends an $A1_c$ goal of less than 7.0 percent. The $HbA1_c$ is linearly related to the average blood sugar over the past 1 to 3 months.

ACUTE COMPLICATIONS

Elevated blood sugar levels due to lack of insulin or a relative deficiency of insulin or abnormally low levels of insulin causes acute complications in diabetic individuals. This leads to increased urine glucose, which in turn leads to excessive loss of fluid and electrolytes in urine. Lack of insulin also causes the inability to store fat and protein along with breakdown of existing fat and protein stores. This dysregulation results in the process of ketosis and the release of ketones into the blood. Ketones turn the blood acidic, a condition called diabetic ketoacidosis. Ketoacidosis can rapidly go into shock, coma, and death. A hyperosmolar coma usually occurs in elderly patients with T2DM.

In cohort studies, direct relationships were observed between higher systolic blood pressure levels and death, coronary artery disease, nephropathy, and proliferative retinopathy. Diabetes and hypertension are interrelated and they strongly predispose to end-stage renal disease, coronary artery disease, and peripheral vascular and cerebrovascular disease.

CHRONIC COMPLICATIONS

The chronic diabetes complications are related to blood vessel diseases and are generally classified into small vessel disease, such as those involving the eyes, kidneys, and nerves (microvascular disease), and large vessel disease involving the heart and blood vessels (macrovascular disease). Diabetes accelerates hardening of the arteries (atherosclerosis) of the larger blood vessels, leading to coronary heart disease, strokes, and pain in the lower extremities because of lack of blood supply (claudication), which in turn lead to peripheral arterial disease. Diseased small blood vessels in the back of the eye cause the leakage of protein and blood in the retina. Disease in these blood vessels also causes the formation of small aneurysms (microaneurysms), and new but brittle blood vessels (neovascularization). Diseased small blood vessels in the kidneys cause the leakage of protein in the urine. Later on, the kidneys lose their ability to cleanse and filter blood.

Diabetic nerve damage includes numbness, burning, and aching of the feet and lower extremities. Minor foot injuries can lead to serious infection, ulcers, and even gangrene necessitating surgical amputation of toes, feet, and other infected parts. In men, diabetic nerve damage can affect the nerves that are important for penile erection, causing erectile dysfunction and impotence.

DYSLIPIDEMIA

The most common lipid pattern in T2DM consists of hypertriglyceridemia (hyper-TG), low high-density

lipoprotein cholesterol (HDL-C), and normal plasma concentrations of low-density lipoprotein cholesterol (LDL-C). However, in the presence of even mild hyper-TG, LDL-C particles are typically small and dense and may be more susceptible to oxidation. Chronic hyperglycemia promotes the glycation of LDL-C and both these processes are believed to increase the atherogenicity of LDL-C.

OBESITY

Excessive upper body fat, or abdominal obesity, is a strong independent predictor of metabolic comorbidities. Waist circumference values 102 centimeters or more (40 inches) in men and 88 centimeters or more (35 inches) in women are associated with substantially increased abdominal fat accumulation and health risks. People at high risk for T2DM can prevent or delay the onset of the disease by losing 5 to 7 percent of their body weight.

ENDOTHELIAL DYSFUNCTION

DM is associated with increased prevalence of endothelial cell dysfunction and vascular diseases. Mechanisms leading to alterations in endothelial cell function are poorly understood. High glucose concentration activates endothelial cells leading to monocytes adhesion providing further evidence that hyperglycemia might be implicated in vessel wall lesions contributing to diabetic vascular disease. Diabetes-associated pathophysiological conditions in the endothelium are modifications of lipoproteins, formation of advanced glycation end-products and circulating lipoprotein immune complexes, alteration of the nitric oxide pathway, and elevated levels of homocysteine. The main goals in restoration of endothelial function are optimal glycemic control, lipid lowering, cessation of smoking, normalization of elevated blood pressure, supplementation of antioxidants for scaveging free radicals, and normalization of homocysteine and insulin levels. There is abundant evidence that some pharmacological agents exert direct beneficial effects on endothelium, suggesting that at least part of their therapeutic action is associated with improvement in endothelial dysfunction.

DEPRESSION

Depression is twice as common in people with diabetes as in the general population. Depression may develop because of stress but may also result from the metabolic effects of diabetes on the brain. Depression and T2DM increases the risk of death for heart patients.

DIABETES MANAGEMENT

T2DM is first treated with weight reduction, a diabetic diet, and exercise. When these measures fail to control the elevated blood sugars, oral medications are used. If oral medications are still insufficient, insulin medications are considered. In prevention trials, it was clearly observed that aggressive and intensive control of elevated levels of blood sugar in patients with T1DM and T2DM decreases the complications of nephropathy, neuropathy, retinopathy, and may reduce the occurrence and severity of large blood vessel diseases. Aggressive control with intensive therapy means achieving fasting glucose levels between 70–120 mg/dl; glucose levels of less than 160 mg/dl after meals; and a near-normal hemoglobin A1C levels. Studies have shown that there is a 10 percent decrease in relative risk for every 1 percent reduction in A1C.

SEE ALSO: American Diabetes Association; Insulin; Metabolic Disorders and Childhood Obesity.

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Uncoupling Proteins

UNCOUPLING PROTEINS (UCP) are a group of proteins that produce energy. They were first discovered in brown adipose tissue (fat cells) and were originally called thermogenin. Brown adipose tissue differs from white adipose tissue, which is the predominant type of fat in adult humans. Brown adipose tissue is much more metabolically active than white adipose tissue. It can utilize a greater amount of fatty acids compared to the white adipose tissue. The reason for this is that brown adipose tissue has a greater amount of mitochondria (to use more fatty acids) and a greater amount of blood vessel around it (to deliver fatty acids). White adipose tissue is primarily used for the storage of fatty acids.

Normally, the mitochondria are responsible for creating energy in the form ATP. It creates the majority of energy through a process known as an electron transport chain. One of the final steps in the electron transport chain involves moving hydrogen ions (H^+) from the mitochondrial membrane back inside of the mitochondria. This movement of hydrogen ions is responsible for the creation of ATP. In brown adipose tissue, in addition to the electron transport chain, a UCP also sits within the mitochondrial membrane. It also moves hydrogen ions back inside of the mitochondria. This, in turn, will lead to the generation of heat. Therefore, one of the primary functions of UCP is to generate heat for the body. Catecholamines, such as epinephrine and norepinephrine, are major stimulators for UCP. They will increase the activities of the proteins.

Because UCP was originally discovered in brown adipose tissue, several other types of UCP have been discovered in the body. UCP1 is found in brown adipose tissue. UCP2 is found in many tissues throughout the body, UCP3 is found in skeletal muscle, and UCP4 is found in the brain. The function of UCP2 and UCP3 is not completely known at this time. The activity of UCP2 and UCP3 increases when a person has been fasting, probably as a way to generate energy for them. Also, animals that were fed a high-fat diet had an increase in their UCP2 activity. This is probably a way for the body to increase the amount of energy used when it takes in extra calories. The function of UCP4 remains unknown at this time.

Some people hypothesized that low levels of UCP1 may be related to obesity. However, adult humans have little to no brown adipose tissue. Rather, adult humans have mainly white adipose tissue which is less metabolically active. Human infants do contain brown adipose tissue and thus UCP1. This is a way for infants to generate extra energy while they are developing. Soon after development is completed, the brown adipose tissue no longer functions. Many researchers have had an interest in finding a way to stimulate UCP1 activity in adults. They believe that this may be useful for treating obesity. UCP2 has also been suspected to be related to

the development of obesity. The gene that makes UCP2 is located on chromosome 11 (in humans). Defects in the expression of some genes on chromosome 11 have been linked to elevated insulin levels and obesity. However, no direct link between UCP2 and obesity has been established at this time.

While adult humans do not have brown adipose tissue (and subsequently UCP1), many other mammals do. For example, a hibernating animal, like a bear, has a significant amount of brown adipose tissue and UCP1. While in hibernation, the UCP1 activity is decreased. However, as the hibernation season comes to an end, UCP1 activity is increased as a way to generate heat and raise the body temperature of the bear. Animals that are obese have an alteration of their brown adipose tissue. The UCP1 content is very low and the animal has an impaired ability to generate heat. Also, the brown adipose tissue has atrophied, another sign of decreased ability to generate energy.

SEE ALSO: Adipocytes; Metabolic Disorders and Childhood Obesity.

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Urinary Incontinence in Severe Obesity in Women

URINARY INCONTINENCE (UI) is a common and complex problem that affects women of all ages. Incontinence has been associated with devastating effects on an individual's social, professional, and family life. Approximately one-half of all women will develop UI during their lifetime. The significant decrements in function and quality of life have a societal cost to the United States of $26.3 billion annually. Several potentially modifiable lifestyle factors are associated with UI. Three of the eight most common risk factors are obesity, constitution and physical work, age, length of menstrual cycle, number of pregnancies, education, and level of health awareness. Treatment may include weight loss, pelvic floor exercises, vaginal cones, bladder training, urethral inserts, and general lifestyle modification advice; or it can be treated surgically with procedures such as Burch colposuspension, vaginal slings, or tension-free tapes and injection of bulking agents alongside the urethra. Weight reduction is an effective treatment for overweight and obese women with UI. Weight loss of 5 to 10 percent has an efficacy similar to that of other nonsurgical treatments and should be considered the first-line therapy for incontinence. The highest odds for urinary incontinence have been reported for body mass index (BMI), heavy smoking, and tea drinking.

UI can be defined and classified as stress, urge, and mixed incontinence in women. Women with stress, urge, and mixed urinary incontinence seem to have a primary neuromuscular disorder in the urethra. The opening mechanism is overactive and occurs with a fall in urethral pressure instead of a stimulated increased pressure during the filling phase of the bladder. Also, during bladder emptying, there is a more efficient opening of the bladder outlet than in normal women. Stress urinary incontinence (SUI) may also occur secondary to pelvic floor muscle (PFM) tears, PFM denervation, fascial weakness, and inefficient PFM contraction due to altered motor control.

SUI is defined by the International Continence Society as "the complaint of involuntary leakage on effort or exertion, or on sneezing or coughing." SUI is the most common form of UI, reported by approximately 50 percent of the incontinent women. The three most common risk factors for SUI include aging, obesity, and heavy smoking. Additional risk factors include parity, chronic cough, depression, poor health, lower urinary tract symptoms, previous hysterectomy, and stroke. Pregnancy and childbirth may also be a risk factor for SUI. The basic evaluation of women with SUI includes a history, physical examination, cough stress test, voiding diary, postvoid residual urine volume, and urinalysis.

Alternatively, patients may suffer from urge urinary incontinence (UUI), characterized by the "complaint of involuntary leakage accompanied or immediately preceded by urgency." This is reported by 10 to 20 per-

cent of incontinent women. In regard to obesity, both UUI and SUI increase proportionately with a rising BMI. The cause of incontinence differs by age group with older women more likely to experience urge incontinence and younger women more likely to experience SUI. Overactive bladder (frequency-urgency syndrome) is the most common bladder problem in late life, affecting up to 41 percent of over-75-year-olds, and the elderly experience more severe disease.

Patients having symptoms of SUI and UUI may have mixed urine incontinence (MUI). This complaint is defined by the International Continence Society as the "involuntary leakage associated with urgency and also with exertion, effort, sneezing or coughing." MUI has been reported by 30 to 40 percent of incontinent women. The management of MUI involves a combination of treatments for both stress and urgency incontinence. Initial first-line therapy includes patient education and lifestyle interventions such as weight loss. Additional interventions include pelvic floor muscle training and bladder training. Oral pharmacotherapy often acts synergistically with the previous treatments. Invasive procedures include minor and major surgical procedures. However, at this time, surgery treats the stress or urgency component of MUI but not both. In addition, surgical procedures carry the risk of infection, hemorrhage, and failure.

Obesity has often been shown to be a risk factor for UI. There are several mechanical and physiologic reasons why an increased BMI may cause or be associated with urinary incontinence. The increased intraabdominal pressure due to obesity adversely stresses the pelvic floor and may contribute to the development of urinary incontinence. In addition, obesity may affect the neuromuscular function of the genitourinary tract, thereby also contributing to incontinence. Evidence suggests that the prevalence of both UUI and SUI increases proportionately to a rising BMI. The increase in the bladder pressure created by a high BMI may reduce the continence gradient between the urethra and the bladder. In this situation, the magnitude of increased intraabdominal pressure necessary to force urine through the urethra is reduced because the static pressure within the bladder is higher.

Unfortunately, the majority of the patients suffering from UI delay seeking medical help or may never consult a physician regarding their problem. This occurs even with the considerable negative impact on their quality of life. The delay or avoidance of treatment may be related to feelings of shame and embarrassment. In addition, some individuals believe that UI is a normal and inevitable consequence of the aging process. Research has shown an association between UI and depression. Both disorders are associated with social stigma, underreporting by patients, and lack of recognition by physicians. Therefore, it is important that those suffering from UI are aware of treatment options.

SEE ALSO: Obese Women and Social Stigmatization; Stress Urinary Incontinence.

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Uterine Cancers

UTERINE CANCER IS considered the fourth most common cancer in women in the United States. It is estimated that as many as 7,400 women will die from this type of cancer in 2007. Uterine malignancies most commonly affect women after the reproductive years, between the ages 50–70. Obese women are at higher risk for developing uterine cancer.

The uterus is a hollow reproductive organ that houses the fetus and supports the development of the baby during pregnancy. The uterus can be viewed as a pear divided into two parts, or two ends. The lower end extends into the vagina (the female genital canal) and is referred to as the cervix. The higher, wider part is called the body (or corpus, meaning *body* in Latin). The uterine body is lined by two layers of different cells. The inner lining is called the

endometrium. The outer lining is called the myometrium. These two layers of the uterus can give rise to different types of cancer.

Endometrial cancer arising from the inner endometrial lining of the uterus is most often (95 percent) adenocarcinoma. The endometrial lining of the uterine corpus contains tiny cells that resemble glands. The cancer of these cells is called adenocarcinoma. Another type of cancer that can occur in the endometrial lining is called adenosquamous carcinoma. This malignant tumor arises from another type of cell in the endometrial layer—the squamous cell. This type of cell is found on various surfaces of the body, such as skin.

Uterine cancers that may arise in myometrium are called sarcomas (5 percent incidence rate). The myometrium has a similar composition to muscle. These types of cancer tend to be more aggressive, more invasive, and more resistant to treatment.

Another type of uterine cancer that may occur includes features of cancers arising from the endometrial as well as myometrial lining. This type of cancer is called uterine carcinosarcoma.

There are numerous risk factors for developing uterine cancer. One of the major risks is prolonged exposure to estrogen (hormone produced by reproductive organs that stimulates sexual maturation and growth and regulates menstrual cycle). This exposure may be due to certain diseases, tumors, or medicines. Another important risk factor is obesity. It has been found that an individual 20–50 pounds overweight has tripled her risk for developing uterine cancer. Moreover, if an individual is more than 50 pounds overweight, the risk goes up to 10 times. It has been observed that fat cells in the human body produce estrogen, a hormone linked to increased incidence of uterine cancer. Some other risks include nulliparity (not having given birth to children), diabetes, hypertension, gallbladder disease, and late onset of menopause.

Any unusual discharge, bleeding, or spotting from the vagina, especially in women after menopause, may be a presenting symptom of uterine cancer. Additional symptoms may include abdominal pain, mass, and weight loss.

The diagnosis of uterine malignancy is accomplished through several steps. First, a gynecologist (a doctor specializing in treating the female reproductive system) must examine any woman suspected of having uterine cancer. The doctor may take an endometrial biopsy (a sample of the endometrium for close examination under the microscope) and perform an ultrasound. There are some other procedures that may be suitable, depending on the patient. Hysteroscopy is a method that allows the doctor to look closely inside the uterus and inspect it with a small telescope. If there is any evidence of malignant area, a biopsy can be taken. Dilation and curettage (D&C) procedure provides more information if biopsy results are inconclusive. During this procedure, the cervix (lower part of the uterus) is dilated (enlarged) and a special instrument is inserted into the uterus to scrape off a sample of the tissue inside.

When the biopsy sample is examined under the microscope, the presentation of the cells is significant to determine the severity of the disease. Cancerous cells are grouped into different stages, depending on how they look under the microscope. Higher grade cancers are usually more malignant, difficult to treat, have poor prognosis, and may have spread to other organs in the body.

The basic treatment options for uterine cancers include radiation therapy, hormonal therapy, chemotherapy, and surgery.

SEE ALSO: Endometrial and Uterine Cancers; Estrogen Levels; Hormones; Menopause; Obesity and Cancer.

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Variety of Foods and Obesity

THE COMPLEX RELATIONSHIP between food intake and weight has never been more relevant as it is in the current cultural context of abundance exceeding necessity in developed nations. An unlimited amount of food choices inundates the public, yet these options have sent Americans into a worsening obesity epidemic despite knowledge about the complications of obesity. Unless a major reversal of dietary trends is established, the overindulged will face the long-term consequences of weight gain and obesity.

The total number of packaged food products available to American consumers is over 300,000. Each grocery store holds approximately 40,000 different food items for purchase. Over 10,000 new products are introduced to consumers yearly. The top new food groups manufactured are candy and snacks, condiments, beverages, bakery products, and dairy products.

The prevalence of obesity has doubled among adults over age 20 in the past two decades and this trend continues to rise. Using the body mass index (BMI) at or above 25 kilograms per meters squared, it is estimated that 65 to 80 percent of adults in the United States are overweight or obese. Children also have exhibited increasing trends toward obesity, tripling prevalence rates in the same period. An estimated 15 percent of children are overweight or obese. Researchers are investigating suggested relationships between the massive amount of food products available and the obesity epidemic that continues to afflict these same consumers.

How humans evolved into this culture of abundance from one thought to be of scarcity has antediluvian origins. Anthropologists have investigated evidence of traditional diets at the origin of the human species. They have found that these ancestors also had a variety of food choices available, but these were mainly choices from plant sources. It is also suggested that this variety of plant sources provided the majority of human sustenance, with hunting and eating animal protein at a smaller percentage of sustenance than was previously thought.

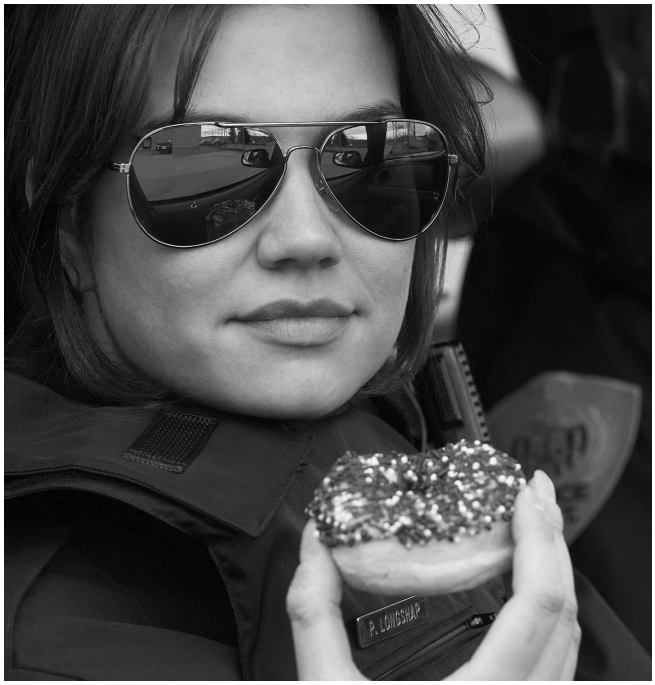
The human genome has changed little for the greater part of human evolution, so the recent obesity trend would not seem to stem from purely genetic components. Rather, obesity seems to be a multifactorial genetic disorder that is greatly influenced in a complex manner by the environment. The greater amount of food available at affordable prices has been a liability for genotypes that afforded survivability in times of food scarcity. In addition, so called fast food is convenient and inexpensive, often a lure to parents and those with limited time for food preparation and with limited income.

Despite abundant food choices currently available, the main ingredients in many American food products are from four basic crops: corn, soybeans, wheat, and

rice. These four staples are expected to continue to be in demand and are exhibiting record sales/growth levels. The average American in 2000 consumed about 300 calories a day more than the average American in 1985. Grains account for 46 percent of this increase, and those are mainly refined.

Decreased fruit and vegetable consumption contributes to higher obesity rates in low-income households as shown by expenditures on these items. In 2000, the average low-income household spent $3.59 per capita per week on fruits/vegetables, compared to $5.02 per capita per week that higher-income households spent. This significant difference might be reflected in other food choices for low-income households that are more calorie dense and do not contain the nutrients that fruits and vegetables provide.

Similarly, energy-dense sugary soft drinks continue to replace beverages such as milk and fruit juice in children. Studies suggest that 4.2 ounces of soft drink replaces one ounce of milk, which provides a net gain of 31 calories and a loss of 34 milligrams of calcium. The inverse relationship continually increases as children get older. In 1985, the average consumption of milk per capita per year was 26.7 gallons. In 2005, that number has dropped to 21 gallons per capita per year. In 1985, the average consumption of regular soft drinks was 29.8 gallons per capita per year. In 2005, that number has risen to 35.5 gallons per capita per year.



Foods that are commonplace at fast-food establishments are often highly energy dense, sugar laden, and loaded with saturated fat.

Fast-food restaurants provide a large variety of food that is available at low cost and is convenient. The large portion sizes of these highly palatable, cheap meals are contributing to ever-increasing waistlines. The foods that are commonplace at fast-food establishments are often highly energy dense, sugar laden, and loaded with saturated fat. A population-based prospective cardiovascular study showed that subjects who consumed fast food frequently (more than twice per week) gained an extra 9.9 pounds and had a doubled increase in insulin resistance over 15 years compared to those who ate fast food infrequently (less than once a week). Thus, frequent fast food consumption is suggested to increase the risk of Type 2 diabetes as well as body weight, with accompanying health risks thereof.

This fast food analysis is not surprising given the fact that dietary fat has been linked to obesity and cardiovascular disease since the 1950s. Dietary fat is the densest macronutrient that commonly contributes to weight gain. However, fat content per capita in the United States has been fairly constant over time even as the total calories per capita have risen. This trend suggests that consumption of highly processed, fat-reduced foods has contributed to filling the calorie gap, while not providing enough satiety.

A common misconception is that by eliminating a macronutrient, weight loss will follow. This has been attempted in many fad diets by reducing amounts of carbohydrates (as in the Atkins Diet) or fats (low-fat diet) consumed. These restrictions have not been proven to have any lasting effects for weight loss or general health. Until further evidence can be brought forth regarding the effects of macronutrient redistribution and energy expenditure, a diet with a variety of macronutrients is still recommended. The general overriding principle for weight loss remains that energy output must exceed energy input, regardless of the type of energy. For most people, that simply means burning more calories than one is consuming.

This is not to say that the quality of energy is not important. While the particular methods of consumption differ, the consensus for an optimal diet is essentially the same. Guidelines released in 2002 from the National Academies of Science's Institute of Medicine (IOM) emphasized that 45 to 65 percent of calories should

come from carbohydrates, 20 to 30 percent of calories from fat, and 10 to 35 percent of calories should come from protein sources. The context of these macronutrients is what complicates things further.

Dietary goals should be met with plenty of fruits and vegetables, whole grains, protein from lean sources, and fats derived from monosaturated and polysaturated oils. Restraint should be applied when possible to refined starches and sugar, animal fats, and trans-fat from processed foods. This is to say that while a variety of nutrients should be incorporated into a dietary plan, food variety can manifest in other ways as well.

Food variety is hypothesized to contribute to obesity as suggested by these differing dietary means. Overall, dietary variety, the first type, is when a person's overall diet is composed of many different foods and is often referred to a cafeteria diet. The other common types of food variety exist in a single meal. Variety in a meal can be presented at the same time and be composed of different foods. Conversely, variety can be introduced successively with each course of a meal containing several foods. It has been shown that eating a meal that contains several courses of different foods presented in succession causes dietary intake to increase compared to a single-course meal with the same foods.

Sensory-specific satiety is another hypothesis that relates the consumption of different types of foods with the gustatory senses. Over the duration of consuming a certain type of food, its perceived satisfaction decreases. A greater intake of calories exists if there is an opportunity to eat a variety of foods. This suggests that satiation is not based on a set amount of calories, but rather as sensory characteristics of food consumed. It also indicates that sensory qualities may exert a larger effect on hunger and satiety than postabsorptive effects of consuming these same foods.

Sensory qualities of foods can differ in taste, texture, color, shape, and specific flavor. Other studies have suggested that macronutrients and energy density do play a factor in sensory-specific satiety. Further research is needed to determine these roles and also individual genetic variety that may influence weight control and hunger mechanisms.

Despite knowing the adverse effects of weight gain, ingestion habits can continue to predominate because of the powerful learning mechanisms that started the habit. The increased convenience of eating energy-dense comforting foods has been a motivating factor in learning these behaviors. Individuals who are genetically influenced by these seductive high-caloric foods would be more susceptible to weight gain and ultimately obesity.

Habituation is a model that has been proposed to explain the sensory response that occurs when a stimulus is repeatedly presented and the sensory response is decreased. After eating the same type of meal consecutively over time, the presentation of a novel food will cause increased caloric consumption of this new food. Different food factors can contribute to a faster habituation rate than others such as sensory qualities of food, density differences, and individual variability.

The complexity of food consumption is modulated by multiple central and peripheral signals that are activated in reward situations and decision making. These pathways vary individually and differ in responses to food. Neurobiological adaptations also contribute to environmental cues of dietary variety by influencing these reward centers and decision-making abilities toward behavior favorable toward greater food consumption.

Obesity treatment may be possible by modifying and decreasing dietary variety. Restriction of seductive foods will make this more possible. Meals that are composed of similar sensory qualities presented as one course may help reduce dietary intake. Repeating the same meals over time would also help to control hyperphagia. Similarly, limiting the overall access to variety of foods consumed can also contribute to weight loss.

Obesity is a chronic condition, and dietary and lifestyle changes that are made must be sustainable over time. It is difficult for large-scale prevention and treatment programs to be effective when environmental cues constantly facilitate compulsive eating. In addition, food is necessary for survival and thus it is difficult to break behavior that sustains life.

The food industry could also contribute to the prevention and control of obesity by manufacturing healthier food that is more palatable and more reasonably priced. Instead of stocking grocery shelves with massive amounts of cheap, high-fat, and high-calorie foods, healthier attractive food options could replace that role. However, regulations to enforce these policies might be difficult to achieve, and each individual must confront the factors in his or her life that contribute to his or her eating habits.

SEE ALSO: Disinhibited Eating; Eating Out in the United States; Economics of Food; Epistatic Effects of Genes on Obesity; Flavor Nutrient Learning; Flavor: Taste and Smell; Genetics Taste Factors; Nutrient Reward; Toxic Environment.

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Vegetarianism

THERE ARE MANY variations of the vegetarian diet practiced throughout the world. The vegan, or strict or pure vegetarian, does not consume any animal products. Considered a philosophical approach to life, the vegan abstains from using any animal products including leather, silk, honey, etc. Products that are tested on animals are also excluded. Raw veganism includes a diet of fresh fruit, nuts, seeds, and vegetables. Fruitarianism, another form of veganism, consists of only fruit, nuts, seeds, and other plant matter that can be gathered without harming the plant. The lacto-vegetarian includes milk and milk products. This diet is often what is meant by the term *vegetarian*. The ovo-vegetarian consumes eggs. The lacto-ovo vegetarian consumes both milk and eggs. In addition, there are various forms of the semivegetarian diet. The lacto-ovo-pesco-vegetarian consumes fish in addition to eggs and milk products (sometimes shortened to pescotarian). The flexitarian consumes poultry, fish, milk, and eggs.

Hominoid primates are largely vegetarian and current hunter-gatherer groups rely on foods that can be obtained most conveniently. It has been suggested that the human gastrointestinal tract features the anatomical modifications consistent with an herbivorous diet. However, the size of the gastrointestinal tract is dependent on both body size and the quality of the diet. It is argued that humans and other primates developed a relatively large brain while adopting a high-quality diet, which permitted a reduction in the relative size of the gastrointestinal tract. The total mass of the GI tract is only about 60 percent of that expected for a similar-sized primate.

Although the brain, heart, kidneys, liver, and gastrointestinal tract make up just under 7 percent of total body mass, they account for just under 70 percent of the total basal metabolic rate of the body. The mass of the splanchnic organs (the liver and the gastrointestinal tract) is approximately 900 grams less than expected and the mass of the encephalized human brain is 850 grams larger than would be expected. This may suggest that changes to such a high-quality diet involved an increased proportion of animal-based products. One example mainly derived from animal products is B-12. Although plant foods can contain vitamin B-12 on their surface from soil residues, this is not a reliable source of B-12 for vegetarians. Much of the vitamin B-12 present in spirulina, sea vegetables, tempeh, and miso has been shown to be inactive B-12 analog rather than the active vitamin. Supplementation or use of fortified foods including fortified soy milk and whole grain cereals may be advised.

There are many advantages to a vegetarian diet. Well-balanced vegetarian diets are appropriate for all stages of the life cycle, including children, adolescents, pregnant and lactating women, the elderly, and competitive athletes. Diets largely based on plant foods, such as well-balanced vegetarian diets, could best prevent nutrient deficiencies as well as diet-related chronic diseases. Vegetarians living in affluent countries enjoy remarkably good health, exemplified

by low rates of obesity, coronary diseases, diabetes, many cancers, and increased longevity. In most cases, vegetarian diets are beneficial in the prevention and treatment of certain diseases, such as cardiovascular disease, hypertension, diabetes, cancer, osteoporosis, renal disease, dementia, diverticular disease, gallstones, and rheumatoid arthritis. Shown to be efficacious for weight and cholesterol control, vegetarian diets may represent an advantage for adult sedentary populations and the prevention of chronic diseases.

Fruits and vegetables are rich sources of not only vitamins, such as carotenoids, ascorbic acid, tocopherols, and folic acid, but also fiber, indoles, thiocyanates, cumarins, phenols, flavonoids, terpenes, protease inhibitors, plant sterols, and a host of other yet-unknown and unnamed phytochemicals and nonnutrient compounds that may protect humans from many cancers and other diseases.

The increased risk of cancer and cardiovascular disease experienced by populations following diets largely based on animal foods may be due to an excess of energy, total and saturated fat, and other nutrients, but also a deficiency or very marginal intake of phytochemicals and other compounds abundant in plant foods but not yet labeled as nutrients. The vegetarian diet not only contributes to weight loss and the prevention of many chronic diseases, but also results in the same plasma cholesterol lowering as achieved by statin drugs. Results have also shown a 40-percent reduction in low-density lipoprotein cholesterol after one year, and the reversal of heart disease.

There are concerns regarding deficiencies in the vegetarian diet. However, deficiencies are usually due to poor meal planning. Restrictive or unbalanced vegetarian diets may lead to nutritional deficiencies, particularly in situations of high metabolic demand. For example, there has been some concern that vegetarian female athletes are at increased risk for oligoamenorrhea, but evidence suggests that low energy intake, not dietary quality, is the major cause.

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Plant proteins have a reduced content of essential amino acids compared to animal proteins. For example, grains are low in the amino acid; lysine and legumes are low in methionine. However, combining foods such as beans and rice provides a complete protein. A significant reduction of limiting amino acids (methionine, lysine, tryptophan) means lower protein synthesis. However, lower intake of these amino acids provides a preventive effect against cardiovascular disease via cholesterol regulation.

Iron and zinc are currently the trace minerals of greatest concern when considering the nutritional value of vegetarian diets. With elimination of meat and increased intake of phytate-containing legumes and whole grains, the absorption of both iron and zinc is lower with vegetarian than nonvegetarian diets. Although vegetarians have lower iron stores, adverse health effects from lower iron and zinc absorption have not been demonstrated with varied vegetarian diets in developed countries. Moderately lower iron stores have even been hypothesized to reduce the risk of chronic diseases. Improved assessment methods are required to determine whether vegetarians are at risk of zinc deficiency.

Vegans have lower bone mineral density than their nonvegans, but evidence regarding calcium, vitamin D, and fracture incidence is inconclusive. In addition, vegans have lower calcium needs than nonvegetarians because diets that are low in total protein and more alkaline have been shown to have a calcium-sparing effect.

Vegetarians may also have subtle nutritional deficiencies which have been related to the occurrence of an unrecognized malabsorption syndrome. The excess phytate content in cereals, nuts, legumes, and oilseeds seems to play a central role in the pathogenesis as an inverse relationship has been shown to link the phytate content of the diet with the intestinal absorption of trace minerals and proteins. The regular intake of probiotic preparation is an inexpensive and safe tool to convert a diet with a low potential for bioavailability of trace minerals and proteins, such as the vegetarian diet, into a diet with a high bioavailability potential.

The potential adverse effect of a vegetarian diet on iron status is based on the bioavailability of iron from plant foods rather than the amount of total iron present in the diet. Vegetarian and nonvegetarian athletes alike must consume sufficient iron to prevent deficiency, which will adversely affect performance. Other nutrients of concern for vegetarian athletes include zinc, vitamin B-12 (cyanocobalamin), vitamin D (cholecalciferol), and calcium. The main sources of these nutrients are animal products; however, they can be found in many food sources suitable for vegetarians, including fortified soy milk and whole-grain cereals.

Vegetarians have higher antioxidant status for vitamin C (ascorbic acid), vitamin E (tocopherol), and beta-carotene than omnivores, which might help reduce exercise-induced oxidative stress.

Well-planned, appropriately supplemented vegetarian diets appear to effectively support athletic performance including elite athletes. Plant and animal protein sources appear to provide equivalent support to athletic training and performance. It has not been shown that exclusion of meat from the diet would impair repetitive short-term performance, whereas reduction of protein intake and a concomitant increase of carbohydrate intake during a period of three to five days improve anaerobic (two to seven minutes) performance.

Vegetarians (particularly women) are at increased risk for nonanemic iron deficiency, which may limit endurance performance. In addition, vegetarians have lower mean muscle creatine concentrations than omnivores, and this may affect supramaximal exercise performance. However, because their initial muscle creatine concentrations are lower, vegetarians are likely to experience greater performance increments after creatine loading.

The increasing global health problems of overweight and obesity are associated with coronary heart disease, hypertension, diabetes, osteoarthritis, and certain cancers. Vegetarian diets are associated with reduced body weight, lower incidence of certain chronic disease, and lower medical costs compared with nonvegetarian diets.

Worldwide, an estimated 2 billion people live primarily on a meat-based diet, while an estimated 4 billion live primarily on a plant-based diet. The U.S. food production system uses about 50 percent of the total U.S. land area, 80 percent of the fresh water and 17 percent of the fossil fuel energy. The meat-based food system requires more energy, land, and water resources than the lacto-ovo-vegetarian diet and more sustainable than the average American meat-based diet. Therefore, the vegetarian diet may be one of the most suitable diets for the environment and humans alike.

SEE ALSO: Diet Myths; Dieting: Good or Bad?; Fruits and Vegetables; High Carbohydrate Diets; High Protein Diets; Low Calorie Diets; Low Fat Diets; Macrodiets; Very Low Calorie Diets; Weight Cycling and Yo-Yo Dieting; Western Diet; Women and Dieting.

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Vertical Banded Gastroplasty

THERE ARE SEVERAL surgical treatments for obesity called bariatic surgery. Vertical banded gastroplasty seems to be the least intrusive of these surgeries with a shorter recovery time and an overall commendable weight-loss success rate. This treatment modifies the size of the stomach to promote weight loss. The surgeon fashions a small pouch at the upper portion of the stomach with a narrow outlet that measures only ½ inch. The surgeon then reinforces the pouch with a mesh band to prevent stretching. The newly created pouch holds four to eight ounces of food. The weight loss results from the limited food consumption. There are two approaches that a surgeon can take to perform vertical banded gastroplasty: the open method, or the laparoscopic method. A laparoscope is a thin arm-like tool with a miniature camera attached, which is inserted into the abdomen. The camera projects images onto a screen for the surgeon.

IDEAL CANDIDATES FOR VERTICAL BANDED GASTROPLASTY

Body Mass Index, or BMI, is a unit that physicians use to figure out the client's level of obesity that takes into account both the height and weight of the person being measured. The surgical procedures for obesity are recommended for people who are morbidly or severely obese with a BMI greater than 40, or 100 pounds or more overweight. A physician may also recommend vertical banded gastroplasty to a person who has a BMI of 35–39 who also has a serious medical condition such as diabetes mellitus, cardiac disease, or hypertension. Other considerations that would make someone with a BMI of 35–39 a likely candidate for the bariatric surgery would include impaired physical performance that affects ability to get around safely and ability to perform necessary tasks at home and at work.

BENEFITS

Research has linked severe obesity with heart disease, metabolic changes (e.g., Type 2 diabetes), gastroesophogeal disease, depression, premature death, infertility, asthma, dyslipidemia, menstrual irregularities, and cancer. The good news is that as the client loses even as little as 10 percent of his or her excess body weight, there is a diminished need for medications, such as

antihypertensive and antidiabetic medications. In some instances, a need for medication is completely eliminated with the reversal of the medical conditions.

Sleep apnea and shortness of breath associated with walking is often remarkably improved. After the surgery, the committed person will be encouraged to participate in a personalized exercise program and enjoy an increased activity level.

RISK FACTORS

Vertical banded gastroplasty, whether the open technique or the laparoscopic technique, is major surgery and comes with associated risks. Clients have a 5 to 8 percent risk in developing complications from the surgery. The risk of death is low at 0.5 percent. Reported complications include pulmonary embolism, bleeding or injury to the spleen, ulcers, anemia/malnutrition, adhesions, scarring causing gastrointestinal obstruction, chronic vomiting, chronic diarrhea, wound infection (related to breakdown of staples allowing leakage from stomach/intestines into abdomen), vomiting related to overeating, slipping or wearing away of the band, enlargement of the pouch, reflux esophagitis, vitamin deficiencies (lifelong supplementation is required), bleeding, abdominal hernia (10 to 20 percent occurrence, which needs surgical repair), gallstones, lung problems, and complications of general anesthesia. Even though risks may be serious, it should be noted that VBG has less risks than Gastric Bypass procedures, particularly when done laproscopically.

BARIATRIC SURGERY SUPPORT

Hospitals that promote bariatric surgeries establish programs that are both educational and supportive. The trained hospital staff are available to increase the client's awareness on topics such as (1) requirements prior to the surgery, (2) insurance coverage, (3) benefits of the surgery, (4) lifestyle changes after the surgery, and (5) available resources, educational materials, and seminars.

Before the surgery, the client will undergo a complete physical examination with a medical history questionnaire, a psychiatric evaluation and counseling, consultation with a dietitian, and a discussion of prior weight-loss attempts. The surgeon will review the client's list of current medications, an important step as it may be necessary to reduce or temporarily discontinue certain medications such as aspirin or coumadin, which may cause excessive bleeding.

After the surgery, the client will have access to a registered dietitian, weight-loss support groups, nursing staff to monitor progress, and educational seminars.

THE PROCEDURE

(1) An intravenous (IV) line is placed in the client's arm for the administration of fluids and medications.
(2) The client will receive general anesthesia, administered by an anesthesiologist. The anesthesia will allow the client to sleep during the entire procedure and prevent the sensation of pain. The surgery generally lasts two hours.
(3) A trache-tube, or breathing tube, is placed in the throat, inserted through the mouth. This aids in breathing during the surgery.
(4) A urinary catheter is inserted into the bladder to drain urine.

When implementing the open method, the surgeon opens the abdomen with an 8–10 inch incision. When the laparoscopic method is used, the surgeon makes a series of small incisions in the abdomen. The abdomen is then inflated with gas to allow better visibility for the surgeon. The surgeon inserts the laparoscope and other tools through the incisions. In both the open method and the laparoscopic method, staples are used to divide the stomach into two sections. The upper part is the new pouch that empties through a narrow port into the lower, larger portion of the stomach. Then, the surgeon, using the band of polypropalene mesh, wraps the opening to prevent stretching. The band is adjustable, even after the surgery. As a reminder, the pouch can hold only ½–1 cup of food; a normal stomach can hold 5–6 cups of food. The narrow port/opening slows down the movement of food causing a feeling of fullness early during the meal and for longer periods of time. Normal digestion is not impeded by this surgery.

The surgery is completed when the incisions are sutured or stapled. The trache-tube is then removed and the client is taken to the recovery room for monitoring of vital signs (blood pressure, temperature, respirations, pulse, pulse ox). Medications are given for pain. Hospital stay is two to five days. Laparoscopic clients are often discharged earlier than open-method clients.

POST-OP CARE IN THE HOSPITAL

After the surgery, the client can expect a regimen of care which includes pain medications, elastic surgi-

cal stockings to enhance circulation, deep breathing exercises, and early ambulation.

The diet in the hospital will be simple and progressive to accommodate the "new" tender stomach.

- Day of surgery: No food or drink.
- Day 1 after the surgery: Upper gastrointestinal X-ray (to search for leaks from pouch). If no leaks are found, one ounce of liquid every 20 minutes.
- Day 2 after surgery: one to two tablespoons of pureed food or one to two ounces of liquid every 20 minutes.

If leaks are found, the client is nourished with IV therapy until leaks are repaired.

POST-OP CARE AT HOME

The client should notify the doctor if he or she experiences signs and symptoms of infection such as fever, chills, redness, pain, or drainage at the incision site. Also, notify the physician if there is blood in the stool or urine. The client should prearrange to be at home from work for approximately two to six weeks. Laparoscopic clients return to work earlier than open-method clients. Postsurgical clients are cautioned to avoid driving and heavy lifting for at least two weeks. However, walking regularly is encouraged. Once the client returns home, the support team plays a vital role in his or her daily life. The team members will monitor his or her progress, educate, and offer emotional support.

For morbid obesity, surgery remains the method with the highest success rate. Success is defined as the ability to lose and maintain the loss of 50 percent of excessive weight. Reportedly, the majority of people who have the bariatric surgery are able to maintain the weight loss. Vertical banded gastroplasty, performed on a client who is committed to the healthy lifestyle changes, is a combination for success.

SEE ALSO: Health Coverage for Gastric Surgeries; Lap Band; Roux-en-Y Gastric Bypass.

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Very Low-Calorie Diets

A VERY LOW-CALORIE diet (VLCD) is one that, as its name suggests, is extremely low in calories, lower than is generally required by adult humans. While all diets typically restrict calories, a VLCD is one that restricts patients to 800 or less kilocalories per day. Following a VLCD scrupulously can result in rapid weight loss up to two kilograms or more (three to five pounds) in adults on a sustained basis (VLCDs should not be used for children). A medical practitioner will recommend a VLCD to a patient who is morbidly, perhaps dangerously, obese whose health would improve from rapid weight loss. VLCDs are particularly useful in the case of patients who have Type 2 diabetes and who are obese. The diet can rapidly reduce problems with glycemic control and this leads to swift weight loss. However, in the case of these patients and others, there are heightened risks arising from the regimen.

Humans derive their energy from food and the amount of energy, hence food, required per day varies considerably based on the size and age of the person concerned, his or her general health, and the type of diet and exercise he or she customarily pursues. Because of enhanced agricultural productivity, economic development, and changing lifestyles, many people take more food than they need. In developed countries, particularly in urban populations, obesity has emerged as an increasingly important phenomenon. Eating less and exercising more works as a corrective lifestyle for some but not all people. In extreme cases, VLCDs are indicated. They customarily consist of around 800 calories of food energy per day, which is almost certain to reduce weight for any adult. Nutritionists have designed a series of different powders which are designed not just for the energy freight they contain, but also provide a balanced diet with respect to such necessary components as protein, vitamins, fatty acids, and various trace elements. The powders

Eating less and exercising more does not work as a corrective lifestyle for everyone. In extreme cases, very low-calorie diets are used.

are intended to be rehydrated by mixing with water or another suitable liquid and form the totality of the daily intake of food. Initially, weight loss results primarily from the elimination of excess liquids, but more sustainable weight loss soon follows. Alternatives include bars and these, like powders, are usually made available on a commercial basis. VLCD products should be distinguished from meal-replacement products which may appear to be similar but which are not intended to represent the entirety of a person's diet. The dangers of a VLCD are such that they should only be followed under medical supervision. Doctors will wish to calculate body mass index (BMI) before deciding whether a VLCD is suitable. Because the diet reduces both body fat and lean body tissue, it is rarely, if at all, appropriate for adolescents or elderly people.

Health issues that might arise as a result of a VLCD include not just the loss of needed lean body tissue, but also the effect on the brain, which will adjust its level of action to a drastically reduced intake of food energy. In addition, the loss of trace elements, which is very difficult to provide in a VLCD on an individualized basis, can result in such problems as loss of menstruation, osteoporosis, depression resulting from lack of serotonin, problems with the gall bladder and the liver, and a range of other side effects. Less serious but still potentially debilitating side effects include nausea, diarrhea, and feelings of torpor. Studies suggest that sustaining a VLCD for as long as six months can result in a loss of as much as 25 percent of body weight. Unfortunately, it appears that in common with most severe weight reduction programs, VLCDs are subject to the law of diminishing returns, that is, the longer they are pursued, the less effective they become, and if pursued beyond their effective range, weight regain may even occur. Adult obesity generally results from a complex combination of factors which require, in the long term, behavior modification and lifestyle restructuring (i.e., in terms of physical rehabilitation and exercise) in order for sustainable and positive health outcomes to be achieved.

SEE ALSO: Caloric Restriction; Children and Diets; Diet Myths; Dieting: Good or Bad?; High Carbohydrate Diets; High Protein Diets; Liquid Diets; Low Calorie Diets; Low Fat Diets; Women and Dieting.

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Viral Causes

IN 1967, DR. William Stewart, the Surgeon General of the United States, declared that it was time to close the book on infectious diseases. The advent of effective antibiotic therapy had turned previously fatal infections into entirely treatable conditions. History has, however, proven Dr. Stewart's opinion premature. From 1967 to the present day, infectious diseases have remained a significant global cause of morbidity and mortality, and new infectious syndromes—including

Lyme disease, acquired immunodeficiency syndrome (AIDS), drug-resistant tuberculosis, and Legionnaire's disease—have emerged.

More important, infectious agents have been implicated in the pathogenesis of a number of unexpected medical conditions. When Dr. Barry Marshall of Australia suggested in the 1980s that peptic ulcers—long thought to have been caused by stress, poor eating habits, and alcohol use—were due to an infection with the bacterium *Helicobacter pylori*, he was ridiculed. In 2003, he received the Nobel Prize for his unconventional observations, and with antibiotic therapy against *H. pylori*, peptic ulcer disease has become a curable condition. Just as revolutionary is the fact that it is now possible to prevent cervical and liver cancer through vaccination with the human papillomavirus (HPV) and hepatitis B vaccines, respectively. Recent research has looked into the role bacteria and viruses may play in the pathogenesis of a wide array of noninfectious medical conditions, including atherosclerosis, multiple sclerosis, and lupus.

PROPERTIES OF VIRUSES AS INFECTIOUS AGENTS

Along with these other medical illnesses, scientists have investigated infectious agents—specifically viruses—as being a possible factor in the development of obesity in some individuals. Viruses have been focused on because of their unique characteristics. A virus is a simple noncellular parasite composed of two parts: an internal viral genome (either composed of deoxyribonucleic acid [DNA] or ribonucleic acid [RNA]) and an external protein coat called a capsid. The complete viral particle is referred to as a virion. Viruses cannot replicate their genome on their own; rather, they require cellular machinery to do so. The protein coat protects the viral genome in the external environment and also enables the virus to bind to surface proteins on cells targeted for infection. Once the virus has entered a cell, enzymes contained in the viral core hijack the cell's internal genetic transcription processes in order to replicate the viral genome at the expense of the cellular genome.

Other viruses, however, employ a different mechanism of cellular parasitism. They lie dormant in the cell by integrating their own viral genes within the larger cellular genome. The viral genes are not expressed until such time that external conditions are more favorable for virion survival outside of the cell. When that occurs, the viral genes are spliced from the cellular genes to begin the viral replication process.

Another intriguing feature of viruses is their cellular specificity. The viral capsid proteins usually have an affinity for specific cell surface proteins only expressed on certain cells. For instance, the human immunodeficiency virus (HIV) virion can preferentially enter those cells that express the CD4 protein on their cell surface, which includes most of the population of T cells known as helper T cells. The selective infection and death of helper T cells that express CD4 leads to the clinical manifestations of HIV infection. Another example of this viral specificity is the herpes virus, which infects and lies dormant in neurons in the spinal cord after initial infection.

Because viruses hide intracellularly, they manage to avoid many of the human body's immune defenses. The specific response of the immune system against virally infected cells is a tightly regulated interplay between multiple cell types. Virally infected cells will process viral proteins and display them on the cell membrane, where circulating immune surveillance cells can recognize and respond to their presence. Two cell types—the cytotoxic T cell subset and natural killer (NK) cell subset—are responsible for destroying cells that display viral proteins on their surface, and another cell type—the helper T cell—stimulates and supports this process. Antibodies, produced by another class of immune cell known as the B cell, bind to virions in the extracellular environment, thus preventing them from being able to bind to surface proteins on their target cells. Defects in the T cell population lead to an increased susceptibility to viral infections and increased severity of viral infections once acquired.

SPECIFIC ADIPOGENIC VIRUSES

In 1982, the first adipogenic (i.e., obesity-causing) virus was identified when it was shown that infection with the canine distemper virus (CDV) leads to increased body mass in mice. Soon after, other viruses were identified that led to obesity in infected animals, including RAV-7 (Rous-associated virus type 7) and BDV (Borna disease virus). More recently, a family of viruses—adenoviridae, also known as the adenovirus family—has been correlated with obesity not just in animal models, but also in humans.

Adenoviridae are viruses that contain a linear, double-stranded DNA genome and are common infectious

pathogens in both humans and animals. They have a protein coat but lack a lipid membrane (derived in viruses from the host cell membrane). Most often, infection with an adenovirus subtype leads to pharyngitis, conjunctivitis, gastroenteritis, or a nonspecific upper respiratory infection in humans. Most people have antibodies against adenovirus subtypes, indicating prior exposure and infection, and a large number of people have adenoviral DNA incorporated into their immune cells' genomes. There are roughly 50 subtypes of human adenovirus and a similarly large number of subtypes specific to certain animal species.

In animal models, experimental infection with SMAM-1 (avian adenovirus) and adenovirus-36 (a human adenovirus) has led to increased visceral fat deposition. Furthermore, in some of the studies that had found increased adiposity in SMAM-infected animals, transmission of SMAM-1 to co-caged animals led to obesity in the noninoculated animals, as would be expected if a contagious agent were the cause of the observed obesity. Although the animal data were well documented and reproducible, it was not until 20 years after the initial animal data were available that human data linking viruses to obesity began to emerge.

Work done by Dr. Richard Atkinson and his associates was the first evidence in humans to support a connection between viral infection and obesity. Their approach was essentially observational. They screened a large population of subjects—both obese and nonobese—for antibodies against adenovirus-36 (Ad-36), which had already been shown to cause obesity in chickens, mice, and marmosets. Approximately three times as many obese subjects had antibodies against Ad-36 than did nonobese subjects, and this difference was large enough that it could not have been due to chance alone.

Furthermore, the mean body mass index (BMI) of individuals with antibodies against Ad-36 was greater than those who were seronegative (i.e., no antibodies against Ad-36). To control for the genetic aspects of obesity, twin pairs were also compared in which one twin was seropositive for Ad-36 and the other seronegative, and the relationship between seropositivity and higher BMI was still observed. It is worth cautioning, however, that the presence of antibodies to a specific virus does not necessarily indicate active, ongoing infection, but can also signify prior exposure or a chronic, indolent infection. This epidemiologic study also cannot prove causality because there may be other, unknown characteristics about obese individuals that would predispose them to being infected by Ad-36, such as reduced immunity to adenoviruses. Nonetheless, the combination of causal experimental data in animals and epidemiologic data in humans suggests that viral infection should be considered seriously as a potential contributor to obesity in some individuals.

MECHANISMS OF VIRALLY INDUCED OBESITY

There are multiple mechanisms by which viruses might cause obesity. The current hypotheses—none of which are mutually exclusive—concern viral infection of cortical neurons, liver cells, thyroid cells, and adipocytes (i.e., fat cells) as leading to the development of increased adiposity (i.e., visceral and subcutaneous fat storage).

Regarding viral effects on fat storage cells, Ad-36 has been shown to have an array of effects. Infection with Ad-36 leads to the upregulation of multiple genes responsible for the differentiation of human and animal adipocytes, including PPAR-γ (peroxisome proliferator-activated receptor), GPDH (glycerol-3-phosphate dehydrogenase), and CEBP-β (cholesterol ester binding protein). When these genes are activated, dormant preadipocytes are induced to become active, mature adipocytes capable of fat synthesis and storage. The same upregulation of these genes was not observed when preadipocytes were infected with a strain of adenovirus not associated with obesity. Ad-36 also induces cell division in preadipocytes, thus generating a larger potential pool of future fat cells.

Metabolically, Ad-36 has been shown to increase glucose uptake by infected adipocytes, which in turn can lead to increased triglyceride synthesis and fat storage. Finally, Ad-36 has been shown to decrease leptin expression and secretion by infected adipocytes. Leptin is a hormone produced by mature adipocytes that interacts with centers in the brain to regulate appetite and energy metabolism. When leptin levels fall, it can lead to increased appetite and decreased energy expenditure in both humans and animals.

Direct infection of brain tissue by adipogenic viruses has also been postulated to cause obesity. The first virus shown to cause obesity—CDV—is neurotropic, which means that it has a predilection for infecting nervous tissue. One of the brain structures targeted by CDV is the hypothalamus, which is the brain center responsible for the control of a number of bodily

functions, including hunger and satiety. Some of the initial animal inoculation experiments did show an increase in food intake in those animals infected with CDV as opposed to control animals. CDV infection of the hypothalamus also leads to reduced expression of certain appetite-suppressing hormones, including melanin concentrating hormone (MCH).

Infection of liver cells has also been shown with some of the adipogenic viruses. RAV-7 infects the liver cells of chickens, and leads to a pattern of injury often observed in obese individuals with a syndrome known as nonalcoholic fatty liver disease (NAFLD). SMAM-1, the avian adenovirus, has also been observed to cause a similar pattern of liver cell injury. The microscopic pattern of damage in NAFLD resembles the liver damage seen in response to chronic alcohol ingestion, and is thought to be caused in part by obesity. This pattern of injury involves fatty infiltration of the liver with microscopic damage to the liver cell mitochondria, the intracellular organelle responsible for energy production within the cell. Although the liver cell changes induced by viral infection may be a consequence of the virus's effects on other cell types, dysregulation of liver function can lead to changes in glucose and insulin sensitivity than can predispose to the development of obesity.

RAV-7 also infects thyroid cells in chickens, leading to infiltration of the thyroid gland by inflammatory cells and destruction of hormone-producing cells. Thyroid hormone, also known as thyroxine, is essential for controlling the metabolic rate in both humans and animals. Decreased levels of thyroid hormone lead to decreased caloric expenditure at rest, and this tends to promote weight gain.

INTERACTION BETWEEN VIRAL INFECTIONS AND OBESITY: IMMUNOLOGIC RESPONSES

It is unclear what factors might predispose certain animals or humans to develop obesity in response to infection with an adipogenic viral strain. Host factors certainly must play a role in this interaction, and it may be those host factors that predispose to viral infection that actually lead to the observed obesity. In other words, infection with viruses such as Ad-36 may not lead to obesity, but rather, maybe only people prone to becoming obese are susceptible to being infected by Ad-36. For instance, a predisposition to insulin insensitivity—which is correlated with obesity—may also lead to increased susceptibility to adenoviral infection. Using other viruses as a model, the relationship between viral infection and obesity is bidirectional, thus making the determination of causality in cases of adipogenic viral infection difficult.

An example of the complex interaction between obesity and viral infections can be seen in the relationship between obesity and hepatitis C. Hepatitis C is a chronic infection of liver cells caused by the hepatitis C virus. This virus belongs in the flavivirus family, which are small, single-stranded RNA viruses. This infection is spread by shared blood contact, usually either by blood transfusion or by sharing needles used for intravenous injection. Over time, infection with hepatitis C can lead to cirrhosis (i.e., scarring of the liver) and end-stage liver disease. Obesity and insulin resistance can independently lead to liver cell damage as previously mentioned, and in obese individuals with hepatitis C, there is an additive effect and more rapid progression to end-stage liver failure. Conversely, individuals infected with both hepatitis C virus as well as HIV are predisposed to developing insulin resistance, which in turn can lead to obesity, thus making hepatitis C a possible candidate human adipogenic virus.

The complicated effects of obesity on preexisting viral infections may be due to changes in the immune system observed in obese individuals. The body responds to chronic obesity as if it were an inflammatory process, similar to diseases such as rheumatoid arthritis or lupus. Obese individuals have been observed to have increased circulating levels of inflammatory markers, such as tumor necrosis factor alpha (TNF-alpha) and interleukin-6 (IL-6). In animal studies, obese individuals have increased susceptibility to viral infections as a consequence of both chronic inflammation and downregulation of natural antiviral proteins, such as interferon-α and interferon-β. Obesity also predisposes individuals to developing other medical conditions that interfere with immunity, namely insulin resistance and diabetes.

CONCLUSIONS

Ultimately, the implications of the research linking viral infection and obesity are unclear. There is no specific antiviral therapy against the human adenovirus nor is there an effective vaccine, and the links between adenoviral infection and human obesity are still being explored.

More important than identifying the specific adipogenic viruses in humans, however, is using our current knowledge about adipogenic viruses to better understand the immunology of obesity. Through the animal and human studies of these viruses, the general mechanisms that promote the development and maintenance of obesity can be better understood. Furthermore, the observation that obese individuals may be predisposed to developing certain viral infections more than nonobese people can aid in a better understanding of the complex interaction between metabolic disorders and the immune system. The chapter on infectious diseases and their effect on the human organism has not yet been closed, but as can be seen, is still in the process of being written.

SEE ALSO: Inflammation; Intestinal Microflora Concentrations; Obesity and Viruses.

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Visceral Adipose Tissue

THE WORD *VISCERA* originates from Latin and is defined as "organs in the cavities of the body." Visceral adipose tissue is the adipose tissue distributed around the internal organs of the trunk, mainly in abdomen and pelvis. Visceral adipose tissue and intraabdominal adipose tissue are terms that were often used interchangeably in publications. On average, visceral adipose tissue consists about 12 percent of total adipose tissue in men and about 6 percent in women. Elevated visceral adipose tissue is a major component of central obesity. The relatively small visceral adipose tissue compartment is now widely recognized as conveying the highest health risks in humans.

Anatomically, visceral adipose tissue includes adipose tissue distributed in body cavities (i.e., intrathoracic and intraabdominopelvic). The intrathoracic adipose tissue includes intrapericardial and extrapericardial adipose tissue. The intraabdominopelvic component includes intraperitoneal and extraperitoneal adipose tissue. The major components of intraperitoneal adipose tissue are omental and mesenteric adipose tissue. Extraperitoneal adipose tissue can be further divided into pre- and retroperitoneal adipose tissue. The latter serves mainly as mechanical cushions of organs and these depots are named accordingly, perirenal, pararenal, periaortic, peripancreatic, parametrial, retropubic, paravesical, retrouterine, pararectal, and retrorectal adipose tissue.

In humans, visceral adipose tissue can be accurately quantified by in vivo high-resolution imaging methods, such as computerized axial tomography (CAT) and magnetic resonance imaging (MRI). The advantage of CAT and MRI methods is their capability in quantifying visceral adipose tissue and subcutaneous adipose tissue separately. It usually requires approximately 40 contiguous cross-sectional images or approximately seven every five centimeter slices to cover the whole abdominopelvic region.

Visceral adipose tissue is either reported in volume (L) or converted to mass (g) by multiplying the volume by the adipose tissue density, which is assumed constant at 0.92 g/cm^3. Because of the cost of image analysis and concerns over exposure to radiation, single-slice imaging studies have often been used as a compromise. Single-slice studies provide only an area (cm^2) when reporting visceral adipose tissue, in contrast to the volumes or masses reported in multiple-slice studies. Other measures of abdominal obesity or central obesity, such as trunk fat measured by dual energy X-ray absorptiometry (DEXA) or waist circumference measured by tape, cannot differentiate abdominal subcutaneous and visceral adipose tissue. Nonetheless, trunk fat and waist circumference have high correlations with visceral adipose tissue amount. Due to the high cost and high technical requirement of imaging methods, waist circumference has been widely used as a surrogate measure of visceral adipose tissue and abdominal obesity in large-scale studies.

Although women store more fat than men, men on average have more visceral adipose tissue than women. There are also ethnical differences in the amount of visceral adipose tissue. Asians have a higher proportion of visceral adipose tissue as total adipose tissue than African Americans, Caucasians, and Hispanics. African Americans have the least visceral adipose tissue among ethnic groups. Visceral adipose tissue increases with age and women tend to accumulate visceral adipose tissue post menopause.

Visceral adipose tissue is associated with obesity-related health risk factors including insulin resistance, dyslipidemia, and inflammation. Increased visceral adipose tissue has also been found to be a risk factor for metabolic syndrome, Type 2 diabetes, cardiovascular disease, and all-cause mortality. Waist circumference is used as the abdominal obesity component of metabolic syndrome, which includes a cluster of insulin-resistance-related risk factors. During interventions including exercise and low-calorie diet in initially abdominally obese subjects, a preferential mobilization of visceral adipose tissue leads to substantial reduction of risk of coronary heart disease and of Type 2 diabetes. Nonetheless, there is still controversy surrounding the question of whether visceral adipose tissue is the cause of obesity-related health risks and diseases and whether visceral adipose tissue is a superior measure of obesity than total fat or anthropometric measures.

It is hypothesized that the metabolic importance of visceral adipose tissue, especially of intraperitoneal adipose tissue, may be due to the delivery of free fatty acids into the portal system exerting potent and direct effects on the liver. This is consistent with the knowledge that omental and mesenteric adipocytes are more lipolytically active and are more resistant to the antilipolytic effects of insulin than subcutaneous adipocytes. Although visceral adipose tissue is not the major contribution of systemic free fatty acids level and its incurred peripheral insulin resistance, there is an increase in visceral adipose tissue free fatty acids delivery to liver in viscerally obese subjects. Visceral adipose tissue is thus likely related to hepatic insulin resistance and hepatic very-low-density lipoprotein triglyceride production.

SEE ALSO: Central Obesity; Waist Circumference.

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Volumetrics

VOLUMETRICS IS A weight-loss diet designed by Dr. Barbara Rolls, who teaches nutrition at Pennsylvania State University. This dietary approach allows patients to eat satisfying amounts of food while controlling calories and meeting nutrition requirements.

The types of food emphasized in this program are high in water content, and fiber, such as fruits and vegetables, which allows patients to eat satisfying volumes of food without eating lots of calories. This follows the principal of energy density. Briefly, the amount of calories per gram of food is the energy density of that food. The higher the energy density of food, the more calories per gram of food. So patients design meals with lower energy density foods; thus, they eat more yet consume fewer calories. Fiber has a very low energy density and can also be added to food to lower the energy density. Fat is the most energy-dense food and increases calories dramatically and should be avoided as much as possible. Several large population-based studies have shown that higher energy dense foods tend to be positively correlated to increases in patients' body weights.

Another principal of volumetrics relies on the fact that most people eat the same amount of food by weight day in day out. Additionally, if you eat less food by weight one day, you tend to make that up the next day by eating more food by weight. Therefore, if you lower the energy density of food and eat the same weight of food, you will lose weight. Amazingly, because people eat according to weight, lower calorie foods can bring patients as much satiety as foods with higher energy density.

To teach these principals to patients, Dr. Rolls breaks down food into four categories: Category 1,

very low energy-dense foods—energy density less than 0.6; Category 2, low energy-dense foods—energy density between 0.6–1.5; Category 3, medium energy-dense foods—energy density between 1.5–4.0; Category 4, high energy-dense foods—energy density between 4–9.0. Patients need to consume as much as possible of categories 1 and 2 and limit 3 and 4.

The volumetrics approach to a meal not only works by satisfying the patient's need to have a certain weight of food, but it also satisfies the mind by thinking you are eating a normal-sized meal. The bigger the meal, the longer it lasts, thus sending more sensory signals to your brain. Additionally, the larger volume activates more stretch receptors in the stomach, thus increasing satiety.

The principals of volumetrics are classically shown in the casserole experiment. Patients were invited to eat one of three choices: the first was a vegetable-and-rice casserole alone, the second was the same casserole served alongside a 10-ounce glass of water, and the third was a soup made by cooking the water and casserole together. Then researchers measured the quantity of lunch eaten a few minutes later. Compared with intake of the casserole alone, water consumed as a beverage with the casserole provided no additional reduction in lunch intake, whereas incorporating water into the casserole (to make soup) resulted in a 100-calorie reduction in later intake.

These types of small changes day in and day out offer people a viable alternative to quick weight-loss diets that leave patients hungry with slowed metabolism. Long-term adoption of these principals will lead to weight maintenance throughout a person's life.

SEE ALSO: Diet Myths; Dietary Restraints; Dieting: Good or Bad?; Fiber and Obesity; High-Carbohydrate Diets; Low-Fat Diets; Water and Obesity.

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Waist Circumference

MANY STUDIES NOW show a link between central adiposity and an increased risk of Type 2 diabetes, cardiovascular disease, and mortality. Specifically, waist circumference, an anthropometric measure at waistline, provides a simple and practical anthropometric measure for assessing central adiposity. Recent studies showed that waist circumference was associated with obesity-related risk factors independent of body mass index (BMI; in kg/m^2). Waist circumference has been increasingly used as a convenient measure of abdominal adipose tissue in epidemiological studies as well as in weight-loss intervention trials.

Waist circumference measured in a physician's office or in research studies is different from a person's self-preferred clothing waistline. There is no universally accepted method of measuring waist circumference and the most commonly used four body sites for waist circumference measurements are as follows: (1) Waist circumference is measured immediately below the lowest ribs, which is the easiest site to locate, even in obese persons; (2) Waist circumference is measured at the narrowest waist. This location is probably the most frequently recommended and is suggested in the Anthropometric Standardization Report. However, for some subjects, there is no single narrowest point because of either a large amount of abdominal fat or extreme thinness; (3) Waist circumference is measured immediately above the iliac crest. This site is recommended in the National Institutes of Health Guidelines and as applied in the third National Health and Nutrition Examination Survey; (4) Waist circumference is measured at the midpoint between the lowest rib and iliac crest. This site is suggested in the World Health Organization Guidelines. Requiring identifying two anatomical landmarks, this method is more time consuming than the other three methods. From a technical standpoint, it is very difficult to stabilize the tape on a sharply curved skin surface of females. The reproducibility of waist circumference measurements at any site depends on the observer's skill.

All four sites had high reproducibility and the variability is less than 1 percent when waist circumference is measured by well-trained staff. Because waist circumference values vary between the four commonly used measurement sites especially in females, the need for a universally accepted waist circumference measurement site should therefore be addressed.

No matter which measurement site is chosen for waist circumference measurement, it is important to make sure that the tape is snug but does not compress the skin, and is parallel to the floor. Waist circumference should be read at the end of exhalation of a normal breathing. While heavy-duty tape is inelastic, firm, and easy to place around the trunk in the same plane even with very obese subjects, soft tapes attached with a tension meter has the advantage of

Waist circumference is replacing waist-to-hip ratio as a simple and convenient measure of central obesity.

standardizing the tape tension but is difficult to place in the same plane. A potential source of measurement error for all waist circumference sites is incorrectly positioning the measure tape on the subject's body. This may account for the larger measurement errors reported.

Waist circumference correlates highly with both total adiposity and central adiposity, namely the amount of visceral adipose tissue. The magnitudes of the associations are stronger in females than in males, and are much stronger for abdominal fat than for total body fat. Waist circumference is more closely associated with visceral adipose tissue and central adiposity than is either waist-to-hip ratio or BMI. Therefore, waist circumference is considered most practical anthropometric measurement for assessing a patient's visceral fat mass and total abdominal fat. Waist circumference also correlates highly with waist-to-hip ratio or BMI.

Waist circumference is a risk factor for insulin resistance, cardiovascular diseases, and mortality independent of BMI. In adults, it has also been reported that the relationship between waist circumference and health risks is independent of height. In children, waist-to-height ratio is reported to be more appropriate than waist circumference to be used as cardiovascular risk factors. Waist circumference is also one of the diagnosis criteria of metabolic syndrome, which is a cluster of risk factors including abdominal adiposity, insulin resistance, dyslipidemia, and high blood pressure.

According to the World Health Organization guidelines, overweight and obese condition is defined by BMI ranges. However, many studies found that waist circumference is a better indicator of cardiovascular diseases and diabetes risks than is BMI. This is because BMI is an index of total body fat rather than body fat distribution, whereas waist circumference represents abdominal adipose tissue which is highly related with cardiovascular disease risk factors and diabetes. Waist circumference measurement could therefore further refine the screening of the obese individual with increased health risk. Although BMI is still used as the primary clinical diagnosis criteria of obesity internationally, there is an increasing emphasis on including waist circumference as a clinical measure to identify obesity-related health risks.

Waist-to-hip ratio was traditionally used to indicate central obesity, but accumulating evidence has shown that this ratio is not as useful as waist circumference in assessing cardiovascular risk factors in both adults and adolescents. Waist circumference is replacing waist-to-hip ratio as a simple and convenient measure of central obesity.

Currently, there is no global standard of waist circumference for obesity as a predictor for cardiovascular disease (CVD). Waist circumference cutoffs proposed by the National Institutes of Health and the World Health Organization were derived by identifying waist circumference values corresponding to BMI cutoffs for overweight (BMI = 25 kg/m^2) or obesity (BMI = 30 kg/m^2). As more and more studies support that waist circumference has an independent or a stronger association with risk factors than BMI has, waist circumference cutoff points that are equivalent to BMI cutoff points for identifying CVD risks have also been proposed. However, because there are sex and ethnic differences in waist

circumference, and populations may differ in the level of risk associated with a particular waist circumference, the development of sex- and ethnicity-specific cutoffs for identifying obesity-related health risks is thus warranted.

For instance, women have a greater relative risk of cardiovascular disease at lower waist circumferences than men. Also, Asians appear to have higher morbidity at lower cutoff points for waist circumference than do other ethnicities. Furthermore, there is ethnic diversity within Asians and thus the cutoff points for Asians may depend on ethnic groups. Children's waist circumference reference values have been developed for age groups.

SEE ALSO: Central Obesity; Visceral Adipose Tissue.

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Waist-to-Hip Ratio

WAIST-TO-HIP RATIO (WHR) is used as measurement for body composition to determine how much fat a person has around his or her abdomen (stomach area). This measurement is obtained by determining the circumference of both the waist and the hips in centimeters or inches and then dividing the value for the waist by the value for the hips. People who have a high WHR tend to have more of their body weight deposited around their abdomen. This is sometimes referred to as an anoid or apple-shaped body. People who have a low WHR tend to have more of their body weight deposited in the lower portion of their body. This is sometimes referred to as a gynoid or pear-shaped body. When males gain weight, it tends to be around their abdomen and thus are more likely to have a high WHR when they become overweight or obese. Some women will also gain weight around their abdomen, but women may also gain weight in the lower portion of the body (i.e., around their thighs and buttocks) and these women would have a low WHR. Other tests may also be used to determine how much fat a person deposits around his or her abdomen and these include skin fold measurements, bioelectrical impedance analysis (BIA), magnetic resonance imaging (MRI) scans, computed tomography (CT) scans, and dual energy X-ray absorptiometry (DEXA) scans.

Numerous studies have explored the use of the WHR for both clinical and research purposes. There appears to be an increased risk of developing certain diseases such as heart disease and Type 2 diabetes mellitus with an increased WHR. Likewise, having a low WHR is protective against the development of these diseases. WHR is good predictor for the development of these diseases. Body mass index (BMI) is another body composition measure. BMI is also a ratio; BMI is a person's weight per the amount of height (the height is squared). WHR is a better predictor than BMI for heart disease and Type 2 diabetes mellitus, but BMI is a very good predictor of many other diseases including kidney disease. A high WHR can also be associated with elevated levels of many of the proinflammatory proteins in the body including C-reactive protein, tumor necrosis factor-alpha, and the interleukins. Elevated levels of these inflammatory proteins are usually associated with increased risk of developing heart disease. Several cancers including breast, colon, and prostate are more likely to develop in people who have a high WHR.

There are several advantages and disadvantages for using WHR as a tool to measure how much fat is around the abdomen. One advantage of using WHR is that it is a simple measurement to acquire. Once a researcher has been properly trained, he or she can easily measure the WHR of many subjects. Additionally, this measurement is very useful for physicians. Physicians do not need to buy expensive equipment or to send their patients for expensive tests to acquire these measurements. Physicians can assess the risk that patients have for developing diseases such as heart disease and Type 2 diabetes mellitus with just a simple tape measure. The WHR measurements can be performed during an annual physical exam as a marker of general

health status and be used to evaluate the change in a patient's body composition over time.

One component of the WHR measurements, waist circumference, has become a useful diagnostic tool by itself. An elevated waist circumference (men 40 inches or more; women 36 inches or more) is considered an eligible criterion for a patient to be diagnosed with the metabolic syndrome. The other criteria to be diagnosed with metabolic syndrome include elevated triglycerides (≥150 mg/dl), low high-density lipoprotein (HDL) cholesterol (the good cholesterol; men <40 mg/dl; women <50 mg/dl), elevated blood pressure (≥130/≥85 mmHg), and elevated fasting glucose (sugar; ≥110 mg/dl; a new cutoff of ≥100 mg/dl is sometimes used). Patients who have three of the five criteria can be diagnosed with metabolic syndrome. Metabolic syndrome is a term used to describe the clustering of risk factors associated with the development of heart disease and/or Type 2 diabetes mellitus. It is sometimes referred to as syndrome X and was first described by Dr. Gerald Reaven in 1988. Treatments for metabolic syndrome include weight loss which would decrease the prevalence of all the symptoms as well as targeting a specific symptom such as taking medication for high blood pressure or the high blood glucose.

WHR measurements are ideal for large population-based studies. These large studies are sometimes referred to as epidemiological studies. Epidemiological studies may require the researchers to go into a community to collect measurements and these researchers need to be able to acquire a vast amount of information without any difficulty. WHR measurements are easy to collect; WHR measurements are both inexpensive (the costs are the tape measures and labor) and easily acquired (many people can be trained to collect this measurement). It is also very easy to perform this measurement multiple times during the course of a study (i.e., a weight loss study, before during or after weight loss).

Another use for WHR measurements is in studies that assess attractiveness and health of people. Some, but not all, studies have associated low WHR as an important feature necessary for a women's ability to become pregnant. Some, but not all, studies have shown that a low WHR in women is associated with increased attractiveness.

There are several disadvantages to the use of WHR. First, there may be a large variation in the measurements obtained by different researchers or physicians. This may be the result of improper training. Also, there have been debates about where to define the waist and the hip on a measurement. The measurements are taken based upon the location of the rib and hip bones. It is often difficult to find the hip and rib bones in patients who are overweight or obese, thus resulting in greater error in the measurements of people as body weight increases. A high WHR signifies that a person has an increased amount of weight, particularly fat around the abdomen. However, there are different types of fat that could be around the abdomen, subcutaneous or visceral, with the latter have a greater association of developing heart disease or Type 2 diabetes mellitus compared to the former. Additionally, it is difficult to relate which part of the ratio (either the waist or the hip measurement) is responsible for the outcome (i.e., is the high waist circumference more detrimental compared to the low hip circumference?).

To summarize, WHR is a useful tool to measure body composition. This measure is practical for both researchers and physicians to determine the amount of fat a patient may have around their abdomen. A person with a high WHR would have a greater risk in the development of many diseases including, but not limited to, heart disease and Type 2 diabetes mellitus. Alternatively, people with a low WHR are protected from the development of these diseases.

SEE ALSO: Body Mass Index; High Density Lipoproteins.

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Water and Obesity

WATER IS AN essential part of the human diet. However, the amount of recommended water intake per day varies greatly from study to study and remains controversial. The availability and variety of sweetened and caloric drinks have been increasing at a similar pace and pattern with the prevalence of obesity worldwide and it is believed that there is a positive correlation between increased energy intake from caloric beverages and obesity. That is the reason many diet guidelines and weight control programs recommend increasing water intake as a preventative measure to reduce the intake of calories from caloric beverages. Still, the direct impact of increasing water intake or the level of consumption on obesity has not been clearly demonstrated.

It is possible that people who tend to be more health conscious—hence they watch their weight fluctuation more carefully—choose to drink more water in place of caloric beverages, falsely concluding that increasing water intake will lead to a reduced prevalence of obesity. Regardless, water is an excellent hydration option without additional calories and it may need to be investigated more closely to determine the link between the level of water consumption, in combination with other factors, and the likelihood of being obese.

In an attempt to curb the increasing rate of obesity prevalence, different ways of reducing calorie intake have been explored by researchers and obesity prevention program developers. Because the proportion of energy intake from beverages has been increasing rapidly—it is approximated that over 20 percent of total calories come from beverages for an average American—reducing the number of calories in liquid form can be a possible preventive measure against obesity or part of a weight-loss regimen. However, another factor needs to be considered when discussing energy intake and obesity: satiety. Satiety is the sensation of being full in the stomach after food intake and it can be a contributing factor to obesity because different types and volume of food can affect the level of satiety. For instance, one will experience a greater level of satiety when one eats large quantities of food or food that is high in energy density. That is the

Even though further investigations are needed, educating the public about the benefits of healthy lifestyle, rather than increasing water consumption alone, may be more beneficial in preventing or treating obesity.

reason some researchers believe that drinking more water, which contains no calories and can provide satiety from the volume, can be helpful in reducing the prevalence of obesity.

Still, the impact of satiety from beverage intake, whether caloric or not, needs to be examined. While some studies show that the amount of food consumed following consumption of beverages decreased, others demonstrate that the amount of beverages consumed prior to meals had no substantial relationship to the calorie intake. Hence, it is possible that drinking sweetened or caloric beverages before or during meals have no impact on the amount or calories of food intake. It is also possible that consuming caloric drinks prior to eating may add to the total calories consumed without substantially reducing the energy intake from the food. Replacing caloric beverages with water may be recommended to those who are at risk of developing or suffering from obesity. However, there is currently no agreement on the compensational mechanism of increasing water intake on the amount of food intake and the number of total calories consumed.

The pattern of water consumption is related to lifestyle as well as health consciousness. In the analysis of the relationship between water consumption and food intake, Barry Popkin demonstrated that water consumers tend to drink fewer portions of soft or fruit drinks and consumed an average of 194 fewer calories. People who consumed water also tended to eat more fruits, vegetables, and low-fat dairy products that are low in energy density. Even though further investigations are needed, educating the public about the benefits of healthy lifestyle, rather than increasing water consumption alone, may be more beneficial in preventing or treating obesity.

Another way to fight obesity is by increasing energy expenditure. Maximizing thermogenesis is viewed as an excellent way of increasing energy expenditure in the obese. Thermogenesis is controlled via the sympathetic activity and an agent that increases the sympathetic activity can be used to increase the thermogenesis in the body, which will increase the energy expenditure and lead to weight reduction. Because water intake increases the level of activity in the sympathetic nervous system, some believe water can stimulate thermogenesis—in addition, water has no side effects that other thermogenic substances may have. On the other hand, the effectiveness of using water as an agent to induce thermogenesis needs to be investigated further because research shows conflicting outcomes. Some researchers have previously reported increases of resting energy expenditure after water consumption, but more recent data presented by Clive Brown show that water at room temperature did not stimulate any additional thermogenesis while cold water at 3 degrees increased thermogenesis marginally but insignificantly. Although water does not appear to be an effective agent to fight obesity, water is still an excellent option for hydration without any calories.

SEE ALSO: Calcium and Dairy Products; Liquid Diets.

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Weight-Control Information Network

THE WEIGHT-CONTROL INFORMATION Network (WIN) is an information service of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), which is one of the National Institutes of Health, which is part of the U.S. Public Health Service within the Department of Health and Human Services. The NIDDK provides scientifically based, current information about obesity, weight control, physical activity, and related issues to the general public, health professionals, the media, and Congress. Besides the WIN Web site, WIN maintains offices in Bethesda, Maryland, and a toll-free telephone line.

The WIN Web site includes a summary of basic information about overweight and obesity in the United States, including definitions of overweight and obesity, a body mass index (BMI) table, prevalence and economic costs of overweight and obesity, and the prevalence and costs of physical inactivity. This information draws primarily on federal surveys, which are cited. A link to information about the National Nutritional Monitoring Program of the National Center for Health Statistics is also provided. More technical information for researchers, including links to current programs, clinical trials, and funding initiatives is also provided through the WIN Web site.

WIN produces a number of publications about obesity for the general public and for healthcare professionals; some materials are available in Spanish as well as English. *WIN Notes* is an electronic newsletter for health professionals which reports on activities of the NIDDK Clinical Obesity Research Panel, lists resources available from various organizations, and reports the latest information on obesity and related topics for NIDDK and other organizations. The newsletter is delivered by email and subscription is free; instructions on how to subscribe and newsletter archives dating back to 1996 are available from the WIN Web site.

WIN pamphlets are available for free download in .pdf format from the WIN Web site, and up to 25 hard copies of WIN publications may be ordered for free using an order form available on the WIN Web site. A number of videotaped lectures on topics related to obesity and nutrition, which were originally delivered between 1992 and 1996, may be ordered from WIN.

Typical WIN pamphlets produced for the general public include *Active at Any Size* (a guide to exercise for the overweight individual), *Choosing a Safe and Successful Weight-Loss Program*, *Dieting and Gallstones* (explains the risks of both overweight and rapid weight loss related to developing gallstones), *Improving Your Health: Tips for African-American Men and Women*, *Just Enough for You: About Food Portions* (which explains serving sizes in terms of common objects, and explains strategies for portion control), and *Take Charge of Your Health! A Guide for Teenagers*. Popular materials available in Spanish include *Camina ... Un Pas en la Dirección Correct* (Walking ... A Step in the Right Direction) and the series *Cómo Alimentarse y Mantenerse Activo Durante Toda la Vida* (Healthy Eating and Physical Activity across Your Life Span). Publications produced for healthcare professionals include *Gastrointestinal Surgery for Severe Obesity*, *Longitudinal Assessment of Bariatric Surgery*, *Prescription Medications for the Treatment of Obesity*, and *Talking with Patients about Weight Loss: Tips for Primary Care Professionals*.

The WIN website includes a summary of basic information about overweight and obesity in the United States.

Sisters Together: Move More, Eat Better is a national initiative of WIN aimed to encourage African-American adult women to become more physically active and eat healthier foods. Sisters Together maintains a Web site with downloadable publications and works with the news media, schools, and consumer and professional organizations to promote physical activity and healthy weight maintenance among African-American women.

SEE ALSO: African Americans; Department of Health and Human Services; Exercise; National Institutes of Health; Nutrition Education; Sisters Together.

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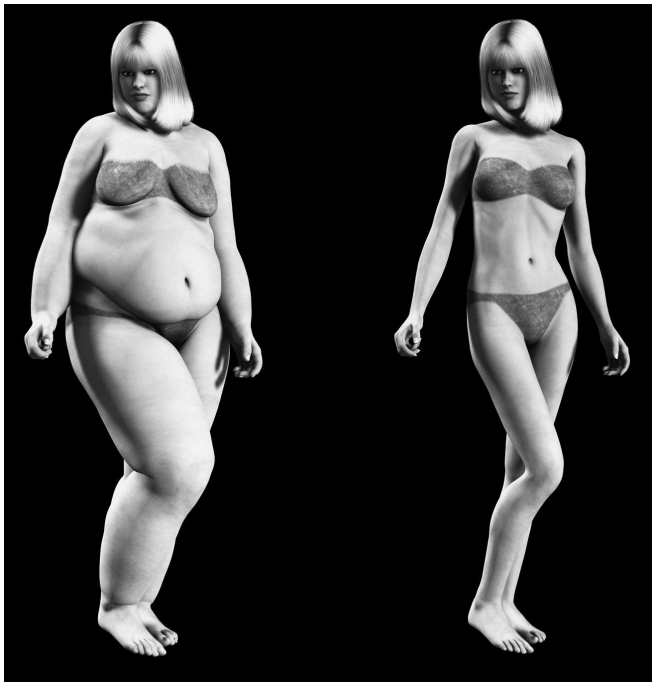
Weight Cycling and Yo-Yo Dieting

YO-YO DIETING IS the common term in lay language for repeated successful weight-loss episodes interspersed with periods of weight gain. A yo-yo dieter is typically described as someone whose weight is constantly on its way up or down, but seldom stable.

Weight cycling is the scientific term for repetitive loss and regain of weight. Weight cycling is defined as going through one or more weight cycles. The phases of a weight cycle are illustrated here, with time on the horizontal axis and weight on the vertical axis. First, there is an initial baseline, which may be a stable weight or a gradual gain associated with aging, typically less than 1 kg a year in mature adults. The weight-loss phase is characterized by an initial rapid loss which decelerates over time. This deceleration reflects two factors: the initial loss of body fluid and the fall in the rate of fat loss as metabolism decreases during adaptation to caloric restriction. After the weight loss, there is a maintenance phase which may be brief or may be permanent. The maintenance phase is marked by reduced calorie requirements, due to reduced body mass and to long-term adaptations to caloric restriction.



A yo-yo dieter is typically described as someone whose weight is constantly on its way up or down, but seldom stable.

The regain phase almost always requires more time than the weight-loss phase. Because caloric requirements are reduced, gradual regain can result from a return to baseline caloric intake. This has been convincingly demonstrated in experimental animals under controlled conditions. Often for humans, the regain phase is marked by increased caloric intake relative to the initial baseline. Binge eating can contribute to rapid regain of lost weight, although it is not required for regain. Most obese humans and animals will return to the baseline weight (dotted line). However, some fraction of subjects will demonstrate overshoot, resulting in a higher weight than the baseline. Animal studies indicate that a genetic predisposition to obesity may be required for the phenomenon of overshoot following weight regain.

In experimental studies, weight cycling is often studied over multiple cycles of weight loss and regain. Generally, the same pattern of physiological changes are observed during the first and subsequent cycles. Thus, there are no unique changes observed during weight cycling that are not observed during a single weight cycle. Periods of weight loss are accompanied by beneficial changes in cardiometabolic markers, as is well known, but periods of weight regain are associated with adverse changes. These adverse effects go beyond a reversal of the beneficial effects of weight loss. For example, blood pressure falls during weight loss, but during weight regain blood pressure may spike beyond the starting level. Importantly, the harmful effects of weight regain are not permanent and tend to fade over time. Unfortunately, many experimental and human studies have failed to take into account recovery time since weight regain episodes.

In clinical studies, weight cycling has been quantified by diverse methods. When many weight measurements are available, the variance of weight over time has been quantified. Most studies have used a retrospective analysis of weight history, and tallied the number of weight-loss attempts or summed the total amount of weight loss by an obese person over their lifetime. Unfortunately, the assumption is usually made that the effects of weight cycling are permanent rather than transient, which likely contributes to conflicting results. Weight cycling, through the harmful effects of rapid weight gain during the regain phase, may be important as a trigger for acute

health events more than a factor responsible for chronic disease.

SEE ALSO: Dietary Restraint; Dieting: Good or Bad?

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Weight Discrimination

FAT PREJUDICE AND discrimination is endemic to our society. We have all been indoctrinated to it, even those of us who are obese or morbidly obese. Each of us carries our own beliefs about obese people, their abilities, and their value, most of which are based on misinformation and lack of education. As a society, we are prejudice against obese people, and "obesism" is rampant.

Prejudice is the prejudging of a person or situation based on attitudes, not perceptions. Discrimination is behavior for or against a person or situation. Discrimination under the law is the behavior or actions in specific situations of an individual, business entity, governmental entity, or their agents for or against a person based on the protected characteristics of race, religion, disability, gender, age, nationality, ethnic origin, pregnancy, and in some states, marital status and sexual orientation. Weight is generally not a protected characteristic.

Weight may now draw more open and widespread discrimination than race, gender, or age, and the prejudice turns up in almost all spheres of life. Obese people are far less organized as a group to fight discrimination than minorities, women, or the elderly, and thus prejudice and discrimination rage through our society. The only state to have legislated against weight-based discrimination is Michigan, but recently the District of Columbia has followed suit. Weight discrimination can be considered under the Americans with Disabilities Act (ADA), but not always. The ADA defines *disability* as "a physical or mental impairment that substantially limits one or more of the major life activities of [an] individual." Disability also includes a record of having such an impairment, or being regarded as having such an impairment. The statute, then, neither expressly includes nor excludes obesity. The Department of Labor's implementing regulations suggest that obesity was not a consideration for a disabling impairment except in unusual circumstances. Courts tended to discount ADA claims based on obesity, and many still do, particularly when all that is presented is a plaintiff who is not morbidly obese, or who does not suffer from a physiological disorder causing the plaintiff to be obese, or one who is simply heavier than average.

Recently, however, courts have been taking a different view of ADA obesity-based claims. In a 1993 Rhode Island case, the federal court held that although simple overweight or obesity probably would not qualify, morbid obesity caused by a physiological disorder would be a disability entitling the plaintiff to ADA protection. The court concluded that the disorder was permanent, and that the plaintiff's weight gain was not meaningfully voluntary. A 1997 decision of the New York federal district court agreed that morbid obesity could be a qualifying disability under the ADA, although it denied the plaintiff's claim because she could not demonstrate that her obesity substantially limited her ability to work as required under the Act.

In 1996, the New Hampshire federal district court held that a teacher had adequately stated an ADA claim by alleging she had been fired because of her weight, and student perceptions that she was less intelligent based on her size. The Pennsylvania federal district court awarded damages to a fired employee in 1997 when he showed that his former manager had made derogatory comments about his weight. And a 1996 Texas decision found that a bus company had improperly decided against hiring an obese woman as a driver because the company could not demonstrate that her obesity would prevent her from performing the necessary functions of the job.

Job discrimination is one of the prevalent forms of discrimination against overweight and obese individuals. The bias and discrimination ranges from stereotypes of lowered intelligence to a perceived inability to do even the simplest of physical tasks due to limited mobility. Obese individuals, especially the morbidly obese, are frequently rejected as candidates

for more highly and publicly visible positions because of their weight. Less qualified candidates are often selected simply because they are more within socially accepted weight parameters.

Employment against the obese is common in the workplace, but litigation of it is not. Those who have suffered the discrimination are reluctant to press their cases because of the pervasive nature of the stigmatization and shame associated with obesity. It is often viewed by courts and juries as a personal failure rather than a disease state or disability. In a 2005 study from Rutgers University, researchers found that compared to normal-weight persons, obese persons with a BMI of 35 or higher were more likely to report institutional and day-to-day interpersonal discrimination. Among this same population, professional workers were more likely than non-professionals to report employment discrimination and interpersonal mistreatment. In any instance, weight discrimination in employment is severely underreported.

Obese people suffer some form of bias or discrimination in every forum and aspect of their lives. The participants of a 2004 qualitative study from Indiana University School of Nursing gave us some modicum of insight into the daily lives of obese individuals. The participants revealed frequent experiences of stigmatization and discrimination on the basis of their obesity, reported being reminded through their everyday encounters with family members, peers, healthcare providers, and strangers that their very being deviates from social norms, and that they are inferior to those who are not obese. Obese subjects experience a pattern of denigration and condemnation that is so pervasive as to constitute what has been labeled civilized oppression.

As practitioners and general members of society, it is incumbent upon us to internalize the fact that obese and morbidly obese individuals are no longer really in control of their disease state. Medical and allied health personnel must put forth an effort to be understanding, sensitive, and helpful. Numerous studies have shown that members of the health and social science professions are severely misinformed about the nature of obesity, and that their biases and perceptions are originated in general society and popular culture. Patients are acutely aware of these biases and are less willing to seek treatment or interaction with the medical profession because they are not willing to voluntarily subject themselves to any more abuse or mistreatment.

SEE ALSO: Appearance; Council on Size and Weight Discrimination; Governmental Policy and Obesity; Obese Women and Social Stigmatization; Obesity Action Coalition; Obesity and Socioeconomic Status; Patient Sensitivity; Quality of Life; Self-Esteem and Obesity; Stigmas against Overweight Children.

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Weight Watchers

POPULAR DIETS HAVE become increasingly prevalent. Each year, millions of Americans enroll in commercial and self-help weight-loss programs. For example, in the United States, approximately 45 percent of women and 30 percent of men are attempting to lose weight at any given time. Unlike diets that may not be based on scientific recommendations, Weight

Watchers International is based on long-standing medical advice which recommends restriction of portion sizes and calories. Started in the early 1960s by Jean Nidetch, Weight Watchers International modestly started with a group of a few friends talking about weight loss to an international company with millions of members around the world.

Weight Watchers International applies a comprehensive approach including a food plan, an activity plan, and a behavior modification plan focused primarily on cognitive restructuring. It is based on the belief that sustained weight loss comes from taking a holistic approach which addresses food, exercise, behavior, and a supportive atmosphere. Individual members determine a goal weight which must be at least five pounds less than their joining weight. In addition, for health benefits, members are encouraged to select a goal weight within a healthy body mass index (BMI) range of 20–25.

Weight Watchers International awards lifetime membership eligibility to members who reaches goal weight and maintain the six-week maintenance program. Successful completion of the program is defined as achieving and maintaining the goal weight within two pounds. The goal weight may be prescribed by a qualified health professional who has stated that the recommended weight is considered healthy based on an individual assessment of the member. With lifetime status, the member receives free services. Because most members have a substantial amount to lose, the considerable majority choose goal weights equivalent to a BMI of 25. The initial goal is to lose 10 percent of body weight because a weight loss of 10 percent confers significant health benefits.

Although rooted in the fellowship of community, Weight Watchers adopts a more behavioristic model which supplies more practical strategies for managing overeating, and a framework for developing positive, adaptive, and self-nurturing modalities.

The program utilizes weekly group meetings with confidential weigh-ins to help members track progress. Lasting less than an hour, the meetings are once a week. Weight Watchers does not utilize a contract and members pay as they attend. The weekly group meetings are led by a group leader who is a lifetime member, and currently within 10 lb of his or her weight goal range. Considered the primary role model, the leaders conduct group meetings, weigh members, and show them how to follow the Weight Watchers weight-loss plan. The leader also provides motivation and social support. The meetings also provide the exchange of tips, recipes, and others' practical experiences.

Using a nutritionally balanced food plan, the Weight Watchers diet is designed to produce a moderate weight loss of up to 2 pounds per week. The activity plan recommends 30 minutes of physical activity on most, if not all, days of the week. Practitioners of Weight Watchers try to keep total daily "points" in a range determined by their current weight. The point value for each food serving is calculated in accordance to an algorithm which takes into account kilocalories, fat, and fiber. Point calculation may be done by slide rule or calculator. In some instances, the point value may be obtained by the consumer from lists or packaging, giving even more ease to the consumer. This provides points for each of a variety of food servings whereby the points are approximately equal to the kilocalories in the food serving divided by 50. In addition, the point value increases by about 1 point for each 12 grams of fat content and is reduced by about 1 point for each 5 grams of dietary fiber content of the serving. Each "point" is approximately 50 calories.

A range of total points are allotted per day based on current body weight. During a day, food servings are selected to stay within the range. The steps are repeated every day to achieve the desired weight control and points may be adjusted secondary to changes in body weight. The minimum and maximum of daily points are between 18 and 35 points. Members are instructed to consume at least the minimum each day to consume adequate minimum calories. In addition, the unused points may be carried over to another day within the week up to a predetermined maximum. This permits the member to have an occasional meal or treat which would otherwise violate the daily maximum, while providing long-term control over food intake. In addition, points may be added to the maximum daily point total based on daily exercise. Points from physical activity are also calculated with a slide rule in accordance to the appropriate point algorithm. Individuals are encouraged to construct an activity plan that provides both weight- and health-related benefits that exercise offers.

An advantage of the system is that the points are small, whole numbers and are calculated for many common foods and serving sizes. The member learns

Using a nutritionally balanced food plan, the Weight Watchers diet is designed to produce a weight loss of up to 2 pounds per week.

the benefit of dietary fiber and the value of avoiding dietary fat. The Flex Plan allows members to personalize daily points by gender, age, weight, height, and daily activity level.

For members who do not wish to count points, the Core Plan instructs members to focus on healthy foods without counting. While avoiding empty kilocalories, the member may eat healthy foods from all the food groups, including fruits and vegetables, grains and starches, lean meats and poultry, and eggs and dairy products while having the occasional snack in controlled amounts. Members may also sign up for an Internet weight-loss companion called Weight Watchers eTools whereby an individual may chart progress online utilizing interactive tools help manage daily food choices and activities.

Weight Watchers Online is also specially customized to meet the unique needs of both men and women. The online tools include over 1,000 recipes and meal ideas, a restaurant guide to assist in healthy choices, daily tips, and expert ideas.

Research has shown that programs including group support, such as Weight Watchers, have been associated with psychological benefits independent of the amount of weight lost. Individuals who successfully lose and maintain weight have been shown to experience improved mood, self-confidence, and quality of life. Furthermore, dieters who regain lost weight do not appear to experience adverse psychological consequences. Additionally, decreasing levels of psychological and behavioral symptoms have been associated with increasing duration of weight-loss maintenance. It can be concluded that quality of life and other psychological measures improve in individuals on comprehensive weight-management programs.

Weight Watchers International offers a weight-loss program which guides food choices that not only reduce calories, but also meets current scientific recommendations for nutritional completeness and reduced disease risk. It has been shown to produce a rate of weight loss of up to two pounds per week after the first three weeks, where losses may be greater due to water loss. Participants are also encouraged to exercise. The program is modeled to be sustainable by utilizing the flex or core diet plans. In addition, because healthy weight loss is regarded as weight loss that lasts, Weight Watchers provides the tools for maintenance with lifetime membership.

SEE ALSO: Atkins Diet; Jenny Craig; South Beach Diet; The Zone.

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Well-Being

IN 1947, THE World Health Organization (WHO) recognized that health should not just be defined as the absence of disease. Instead, it chose a more inclusive and affirmative definition—"Health is a positive state of physical, mental, and social well-being." This recognition has resulted in an increase of clinical, scientific, and public interest in the assessment and treatment of mental illnesses and the assessment and promotion of well-being. This entry defines well-being and discusses the relationship between well-being and obesity and possible mechanisms through which well-being can be enhanced.

DEFINING WELL-BEING

Well-being is a state of optimal experience and functioning. Traditionally, well-being has been viewed from two different perspectives: hedonic well-being and eudaimonic well-being. The hedonic perspective views well-being in terms of pleasure attainment and pain avoidance. Hedonic well-being is most commonly assessed by measurements of subjective well-being (SWB): one's cognitive and affective evaluation of one's life. There are four components of SWB: life satisfaction (global judgments of one's life), satisfaction with important domains (e.g., work satisfaction), positive affect (experiencing many pleasant emotions and moods), and low levels of negative affect (experiencing few unpleasant emotions and moods).

The eudaimonic perspective goes beyond the assessment of simple pleasure and pain and views well-being as the extent to which a person is fully functioning, living a life of meaning and self-realization. From the eudaimonic perspective, feeling a complete range of emotional experiences is seen as a positive characteristic of a fully functioning individual. Further, well-being is defined by the degree to which a person embodies such traits as autonomy, personal growth, self-acceptance, life purpose, mastery, and positive relatedness. Whereas hedonic well-being does not necessarily influence eudaimonic well-being in that many pleasurable activities do not produce wellness, the reverse is usually true; engaging in eudaimonic activities increases SWB in terms of positive affect and general life satisfaction.

WELL-BEING AND OBESITY

The majority of studies suggest that there is strong link between being overweight and experiencing negative psychological consequences. However, this link is often complex, expressing multiple covariations rather than a single pattern of association. In the case of depression, for example, the relationship between obesity and well-being is dependent upon gender, race, and sex. Further, the connection between obesity and well-being is not a direct one, but is linked through several different pathways including physical health ailments, social stigma and diminished self-concept, and behavior patterns such as physical inactivity.

Well-being has become a major topic in studies of chronic diseases as physical illness is often associated with displeasure and pain and can affect well-being because functional limitations often detract from opportunities to increase positive affect and life satisfaction. Of course, there are several biological reasons for the connection between physical health and well-being, including changes in physiological, biological, and hormonal functioning. However, the moderate correlation between physical health and well-being is complex. Interestingly, the majority of the research suggests that it is perceived physical health rather than actual physical health that predicts SWB (i.e., some people with objectively poor health have high SWB, whereas some people with poor well-being have no physical problems).

Many authors have suggested that the less acceptable it is to be overweight, the greater the psychological impact. The societal pressure for thinness along with increased attention to the severity of obesity can have dire consequences on self-concept. This stigmatization of obesity has shown to impact psychological well-being both directly through reduced self-esteem and indirectly through its affects on interpersonal relationships. Again, perceived weight is more important than actual weight in predicting well-being; for example, an average-weight individual who perceives himself or herself as overweight will have similarly adverse effects on well-being as an individual who is actually overweight.

Finally, physical activity has proven to have significant potential to influence subjective well-being along several dimensions including decreasing negative affect and increasing positive affect, improving self-concept (self-esteem, body image, self-efficacy, and cognitive age), health-related quality of life (HRQL) (pain, fatigue, and energy), and life satisfaction. While there are several physiological reasons for the beneficial psychological effects of exercise such as increases in levels of endorphins and improved regulation of norepinephrine, several studies suggest that the benefits arise as a result of improved self-perception.

ENHANCING WELL-BEING

Research suggests that SWB is determined to a substantial degree by genetic factors and is relatively stable across the life span. This may be due to differences in attributional styles under which characteristically happy people and unhappy people view the same life events differently. Another reason that SWB is resistant to change is that people tend to exist on hedonic treadmills; once they reach their accomplishments, their expectations also rise. However, while the literature indicates that well-being in general is resistant to change, improving well-being can and does happen. Whereas the attainment of things such as physical attractiveness, income, number of friends, religious faith, intelligence, and education have been shown not to substantially affect well-being, focusing on eudaimonic activities such as engaging in meaningful activities and setting self-concordant goals has proven to have a significant impact.

Weight reduction in the obese is typically followed by improvements in psychosocial functioning and well-being.

Weight reduction in the obese is typically followed by improvements in psychosocial functioning and well-being (improvements in body image, self-esteem, mood). However, studies indicate that weight fluctuation is associated with negative psychological attributes in both normal-weight and obese individuals. Further, eating disorders are also associated with diminished well-being, which again suggests that the link between weight and obesity depends more on changing internal self-perception rather than actual weight.

Finally, it is likely that the connection between obesity and well-being is bidirectional. Studies have pointed to the powerfully preventative effects of social support and well-being in preventing obesity and other major health outcomes. Thus, focusing on improving well-being is not only likely to have powerful effects on improving self-perception, but it might also inadvertently in the service of attaining and maintaining a healthy weight.

SEE ALSO: Anxiety; Body Image; Depression; Eating Disorders and Obesity; Mood and Food; Quality of Life; Self-Esteem and Obesity; Stress; Weight Cycling and Yo-Yo Dieting.

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Western Diet

THERE IS NO strict definition of *Western diet*, but it is a common concept in discussions of food, nutrition, and obesity. For the purposes of this article, the Western diet will be described in two ways. First, the diet that is common throughout much of the modern industrialized world will be discussed in contrast to that which predated the introduction of agriculture and animal husbandry to human societies, or the diet consumed today by isolated hunter-gatherer societies. Second, the modern Western diet as typical in countries such as the United States and northern Europe will be compared to diets based on food consumption patterns common in the Mediterranean and in rural Asia.

MODERN WESTERN VERSUS PREAGRICULTURAL DIET

Many scholars believe that major changes in diet and lifestyle occurred in human society approximately 10,000 years ago, which created a discordance with our genetic makeup, because human evolution occurs on a much slower scale. This discordance is blamed by some for the development and/or widespread nature of the so-called "diseases of civilization" including many common chronic diseases such as diabetes, hypertension, and osteoporosis. Of course, these claims must be evaluated with respect to other possible explanations, including the fact that improved medicine has reduced the death rates from communicable disease and thus allowed individuals to survive long enough to develop chronic diseases. Important developments in the human diet due to the introduction of agriculture and animal husband include changes in glycemic load, fatty acid composition, macronutrient composition, micronutrient density, acid-base balance, sodium-potassium ratio, and fiber content.

Prior to the introduction of agriculture and animal husbandry, human diets are believed to have consisted primarily of wild plant and animal foods, which were subjected to minimal processing, and any foods common in the modern diet would not have been available. This includes dairy products other than the milk of one's own species during the suckling period (about 11 percent of a typical modern diet), cereal grains (about 24 percent of a typical modern diet, with refined grains making up about 20 percent), refined sugars (about 18 percent of the typical modern diet), refined vegetable oils (about 18 percent of the typical modern diet), and alcohol (about 1.5 percent of the typical modern diet). In addition, added salt would have been unknown to most people prior to agriculture. Of course, the modern diet has undergone many changes since the introduction of agriculture.

Although meat was consumed before the introduction of animal husbandry, it would have been derived from wild animals, which are in general much lower in body fat than domesticated animals. In addition, most edible fatty acids in wild animals are polyunsaturated or monounsaturated, and the amount and composition of body fat varies over the year. The introduction of animal husbandry made it possible to maintain higher levels of fat in animals intended to be slaughtered for food, and increased the amount of saturated fat in their meat. Development of food-processing methods, such as the salting and drying of meat, and making cheese and butter from milk, allowed the storage of animal products, including their fats, and their consumption throughout the year. Technological developments in the 19th and 20th centuries led to further refinements in animal husbandry, including the practice of feeding cattle primarily on corn (rather than grass), resulting in more rapid maturation as well as a higher proportion of saturated fat.

The glycemic index was developed in 1981 as a method to describe the effect of various foods or food combinations on blood glucose. In 1997, the concept of glycemic load was introduced; it is calculated by multiplying the glycemic index of a food by the amount of carbohydrate in a serving. Refined grains and sugars typical in the modern diet carry a higher glycemic load than the unrefined plant foods which are consumed in modern hunter-gatherer societies and presumably by most humans before the introduction of agriculture. Long-term consumption of high glycemic load foods has been shown to promote insulin resistance, which has been related to many chronic conditions including obesity, Type 2 diabetes, hypertension, and heart disease. Consumption of dairy products has also been positively related to insulin resistance.

The fat content of the modern Western diet is high in saturated and trans-fatty acids and low in monounsaturated and polyunsaturated fats; the main dietary sources are fatty meats, baked goods, cheese, milk, margarine, and butter. This balance, which is

opposite of that hypothesized in the preagricultural diet, is related in particular to cardiovascular disease, which has been shown to be more sensitive to the ratio of different types of fat than to the absolute amount consumed. The widespread consumption of processed vegetable fats in the modern diet has also changed the ratio of fats concerned, with an increase in n-6 polyunsaturated fats and decrease in n-3 polyunsaturated fats. The development of a process to hydrogenate vegetable fats in 1897, which allows the creation of solid vegetable shortening and margarine, introduced a new type of fat, trans-fatty acid, into the modern diet. Consumption of trans-fatty acids is associated with increased blood cholesterol and increased risk of heart disease.

In the typical modern American diet, about 52 percent of the calories are derived from carbohydrates, 33 percent from fat, and the remainder from protein. A typical hunter-gatherer population is estimated to consume more protein (19 to 35 percent) and less carbohydrate (22 to 40 percent). Clinical and experimental evidence supports the theory that increasing protein in the diet can improve the blood lipid profile, lower blood pressure, improve insulin sensitivity, and reduce risk for heart disease and stroke.

Micronutrient (vitamin and mineral) density is lower in most refined foods than in wild plant foods and the muscle meat of wild animals, and micronutrients are practically nonexistent in some dietary products common in the modern diet, including refined sugar and some vegetable oils. This produces the paradox that although many Americans consume too many calories, many also suffer from nutritional deficiencies. For instance, at least half of all Americans do not consumed recommended amounts of vitamin A, vitamin B-6, magnesium, calcium, and zinc, and one-third do not consume sufficient folate. Inadequate consumption of micronutrients is implicated in a number of both vitamin-deficiency disease and chronic diseases.

Most foods release either acid or base into the circulation after digestion and absorption. Preagricultural diets are nearly always net base, while modern diets are net acid due to the addition of salt, increased consumption of meat, fish, dairy products, and cereal grains, and decreased consumption of fruits, vegetables, and nuts. Net-base diets are associated with a reduction or slowing in many diseases, including osteoporosis, hypertension, exercise-induced asthma, and chronic renal insufficiency.

The typical modern diet is substantially higher in sodium than in potassium, the reverse of preagricultural diets; the increase in sodium comes primarily from the addition of manufactured salt, while the decrease in potassium comes primarily from decreased consumption of fruits and vegetables. A high-sodium, low-potassium diet is implicated in many chronic illnesses, including hypertension, stroke, osteoporosis, asthma, and kidney stones.

Fiber content in the typical modern diet (15 grams a day is typical for the United States) is much lower than that consumed in contemporary hunger-gatherer societies and lower than recommended levels (25–30 grams a day). The decrease in fiber stems largely from the displacement of plant foods by meat, milk, oils, sugars, and refined grains. Increased fiber consumption is associated with better weight control and decreased blood cholesterol; diets lacking in fiber are implicated in many conditions, including constipation, appendicitis, hemorrhoids, varicose veins, and diverticulitis.

THE MEDITERRANEAN DIET

The Mediterranean diet is a version of the modern diet which is characteristic of many regions bordering the Mediterranean Sea, including Spain, France, Italy, Greece, Portugal, and the countries of North Africa. Of course, people living in those countries today eat a modern diet compared to that typical of preagricultural societies. However, the Mediterranean diet differs in many ways from the modern Western diet typical of, for instance, the United States or northern Europe. The Mediterranean diet is distinguished by the consumption of more whole and unprocessed foods than is common in the United States, more consumption of large amounts of fish, olive oil, fruits and vegetables, and cereals, and less consumption of meat, eggs, and dairy products. This results in a diet that is rich in antioxidants, carotenoids, monounsaturated fats, phytochemicals, fiber, and omega-3 fatty acids, all of which are thought to protect against many chronic diseases. The Mediterranean diet is also lower in saturated fat, and contains little or no trans-fat.

The potential benefits of the Mediterranean diet were first promoted by the American physician Ancel Keys in the 1940s, but did not gain widespread recognition in the United States until the 1990s. At

that time, nutritionists recognized that people living in Mediterranean countries have lower rates of heart disease than people in the United States, although they consume similar amounts of fat, a fact often described as "the French paradox" or "the Mediterranean paradox." Additionally, although the Mediterranean is higher in fat than many diets recommended in the United States for weight loss and control, adherence to a Mediterranean diet has been found to be neutral or negatively related to overweight and obesity.

THE ASIAN DIET

The Asian diet does not refer to the specific diet of any particular country or region, but is based on food consumption patterns typical in the traditional diet of much of rural Asia. This diet is based on the consumption of plant products supplemented with small amounts of dairy, meat, and fish, and is associated with lower rates of some types of cancer, heart disease, obesity, and other chronic diseases. Researchers from Cornell University and Harvard University developed an Asian diet pyramid to explain the principles of the diet to Americans and as a rival to the food guide pyramid developed by the U.S. Department of Agriculture in 1992, which lumped together foods of plant and animal origin.

The base layer of the Asian diet pyramid is rice and grain products, including wheat, millet, and corn, preferably whole grain and minimally processed. The second layer is occupied by other plant products: fruits, vegetables, legumes, nuts, and seeds. Physical activity is also recommend on a daily basis, as is consumption of small amounts of vegetable oil and moderate amounts of plant-based beverages (tea, sake, beer, and wine). Daily consumption of small amounts of fish, shellfish, and/or low-fat dairy products are optional. Eggs, poultry, and swe ets are to be consumed no more often than weekly, and red meat no more than monthly. The Asian diet is lower in fat than the Mediterranean diet, and despite the optional consumption of dairy products, is not associated with development of osteoporosis.

SEE ALSO: Carbohydrate and Protein Intake; Fat Intake; Fiber and Obesity; Portion Control; Thrifty Gene Hypothesis.

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Whole-Body Potassium Counting

QUANTITATIVE IN VIVO measurements of radioactivity in the human body started in the 1940s with the emergence of scintillation detectors. The Whole-Body Counter (WBC) is a device used to identify and measure the radioactive material in the body of human beings and animals resulting from the accumulation of naturally occurring radioactivity and of radiotracers and radioactivity introduced by humans, and from the accidental absorption of radio contaminants.

The WBC uses heavy shielding to exclude natural background radiation from reaching the ultrasensitive radiation detectors and electronic counting equipment. In the most sensitive WBCs, the detectors surround the body and the entire system is kept in room with low background radiation. A second system, the one most frequently encountered, is the chair configuration in which a seated subject is counted with fewer detectors than the former, while a third popular system, containing minimum shielding material, is one with a shadow shield in which a body is scanned by shielded detectors.

Natural potassium (K) is encountered in three isotopic states: 93.1 percent ^{39}K, 0.0118 percent ^{40}K, and 6.9 percent ^{41}K, of which ^{40}K is radioactive with a half-life ($t_{½}$) of 1.3×10^9 years. Thus, it is in a secular equilibrium

with the stable isotopes and 1 g of K contains about 1.8 x 10^{18} atoms of ^{40}K. From the basic laws of physics, ^{40}K with a decay constant λ ($\lambda = \ln 2 / t_{1/2}$) will emit about 200 gamma rays at 1.46 MeV per minute per gram of natural K. Because the human body contains 140 g K on average, about 28,000 gamma rays will be emitted per minute. Considering self-attenuation (multiple scatterings) of the gamma rays in the human body, the variable size and weight of humans, and the total detection efficiency of the counting system, this emission rate is adequate for observing and monitoring K in infants.

The uniqueness of in vivo whole body counting is the high specificity of the counted gamma rays to the originative radioisotope. Therefore, there is no ambiguity with elemental identification, and because these systems are totally insensitive to the underlying chemistry, there is a linear relationship between the response and the abundance of the radioisotope of interest. Quantitative analyses can be carried out with WBC systems by first administering to a subject a suitable analog radiotracer (by injection or drinking), counting it, and establishing a conversion factor from counts to grams of the element.

Another, simpler, method is using a series of different sized anthropomorphic-shaped phantoms filled with solutions containing known amounts of the element of interest, as for example, KCl dissolved in water for K phantoms. Attention must be paid to account properly for bony structures in humans, and to the invariance of phantom positioning in the counting space of the counter. In all cases, the validity of the conversion factor must be demonstrated experimentally or by proper modeling. There is an extensive literature on the use of total body potassium (TBK) measured by the WBC in studies of body composition. Ellis (2005) comprehensively discussed them, along with many of the issues mentioned above.

A recent demonstration suggested that an assessment of partial body potassium (PBK), which entails much simpler systems, is a viable method for following local changes in K content in specific organs. A system was build for monitoring K in the arm of the general population including children, while a mobile system was constructed for monitoring K in the legs of patients with spinal cord injuries.

SEE ALSO: Frontiers in Maintenance and Prevention; Functional Magnetic Resonance Imaging.

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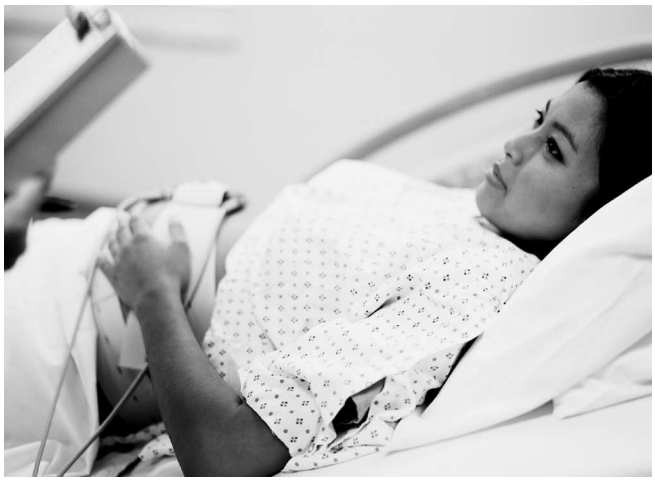
Women and Diabetes

TYPE 2 DIABETES has an equal prevalence in men and women. In many studies, the relationship between BMI and diabetes incidence is stronger in women than in men. The relative risk conferred by diabetic status differs between genders. Diabetes is a powerful risk factor for cardiovascular disease, and the overwhelming majority of excess deaths in diabetes are from cardiovascular causes. In women, is an even stronger cardiovascular risk marker than in men. In fact, diabetes erases the survival advantage of the female gender, so that men and women with diabetes have similar risk of cardiovascular disease and death. The higher relative risk of diabetes in women is not explained by any known risk factor and the cause is currently unknown.

After menopause, the risk of diabetes rises along with body weight. Contrary to expectations, however, menopause or hormonal status have no impact on the incidence of diabetes. Instead, age is the primary factor in diabetes after menopause, with lesser contributions from body weight and waist circumference. There does not appear to be a direct role of sex hormones in the onset of diabetes in women.

Gestational diabetes is a powerful risk factor for the later development of Type 2 diabetes. Women who have had gestational diabetes are strong candidates for diabetes prevention, by increased physical activity and a diet rich in plant based foods. A comparable early warning marker is absent for men. The use of antidiabetic drugs postpartum, particularly the insulin sensitizing agent metformin, may have a role in preventing diabetes in this high risk population.

Polycystic ovary disease is a reproductive syndrome marked by excessive androgens in women, leading to reduced fertility and insulin resistance. The syndrome greatly increases the risk of diabetes. While polycystic



Gestational diabetes is a warning for the later development of Type 2 diabetes. A comparable early warning marker is absent for men.

ovary disease is associated with obesity, at least part of the weight gain is the result of syndrome of insulin resistance, which leads to compensatory increases in insulin secretion thereby promoting further weight gain. Metformin, by reducing insulin resistance, can be effective in countering the syndrome and in favoring weight loss. While polycystic ovary disease increases multiple risk factors for coronary heart disease, the actual increase in cardiovascular events is small or even absent.The reason for this paradox is not clear, but the contribution of polycystic ovary disease to diabetes risk is a major concern.

SEE ALSO: Coronary Heart Disease in Women; Insulin; Stroke; Type 2 Diabietes.

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Women and Dieting

THE IMPETUS FOR dieting among women appears to have several key sources. First is the concern regarding the increasing prevalence of overweight and obesity and associated health risks observed in many countries. This source has led to public health campaigns to remedial or prevention efforts by recommending limiting caloric intake. The second, more pernicious, motive is the mounting pressure on women to be thin to meet cultural ideals for physical attractiveness.

The mass media's emphasis on ultra-thinness as a standard for beauty in Western culture has been linked to the high prevalence of body dissatisfaction and restrictive dieting among adolescent girls and women. It is well documented that individuals who are dissatisfied with their body weight are at a significantly higher risk of developing eating disorders. Along with the pervasiveness and risks of dieting, there is also evidence that weight-loss efforts may backfire by leading to mental and physical and health problems as well as higher weights over time. Ironically, rather than being a solution to obesity, dieting may actually be one of the causes. Because restrictive dieting is the standard treatment recommendation for overweight and obese individuals, consideration of the potential risks of dieting is of utmost importance in a comprehensive volume on the topic of obesity.

DEFINITIONS

The term *dieting* can have different meanings. It can refer to attempts to limit certain types of foods for medical or health reasons (e.g., dairy products in lactose intolerance) with no intent of weight loss, or it can denote restricting amount eaten for the purpose of weight loss. Dieting can refer to behaviors designed to lose weight, or cognitive restraint, where the individual has a clear intent to lose weight, which may not be evident in specific behaviors. Finally, dieting in an attempt to lose weight can be defined as healthy, such as moderate limitation of food intake, or unhealthy, such as fasting, vomiting and so forth.

The term *dieting* will be used in this chapter to denote the intent to restrict food intake to reduce body weight without designating whether the behavior is healthy or whether it actually results in weight loss unless these factors are specifically relevant to the topic of discussion.

DIETING AND CULTURE

One of the strongest predictors of dieting in preadolescents, adolescents, and adult women is body dissatisfaction. Body dissatisfaction and dieting behaviors have been fostered by a clash between unrealistic cultural imperatives to be thin and biological realities that preclude most women from ever achieving the shape standards portrayed in popular women's magazines. In the past 50 years, the perceived cultural ideals of feminine beauty have become even thinner with the burgeoning dieting and weight-loss industry successfully marketing the vision that ultra-thin shape ideals are attainable. This is evidenced by the industry's annual revenues in North America, which are estimated between $35 and $50 billion.

There is compelling evidence that women in Western culture increasingly have been socialized to view their body weight or shape as a marker for attractiveness, self-esteem, social desirability, and competence. The impact of Westernization and globalization has propagated the gaunt standard of beauty to non-Western countries and has, coincidentally, led to the proliferation of dieting and dieting disorders. Studies have shown that as young women from other more weight-tolerant cultures (e.g., Egyptian, Japanese, and Chinese) are assimilated into thinness-conscious Western culture, weight concerns and dieting behaviors in the previously weight-tolerant cultures proliferate.

PERVASIVENESS OF DIETING

The pervasiveness of body dissatisfaction and dieting among adolescent girls and women in Western culture is remarkable because it reflects normative discontentment with an aspect of physical appearance that has also been shown as integral to feelings of self-worth. Research has shown that body dissatisfaction and weight-control behaviors are more prevalent in girls than in boys, and that these attitudes and behaviors peak during adolescence when girls experience conspicuous changes in body fat and shape. Studies indicate that 70 to 85 percent of female adolescents and college students want to lose weight.

Almost 50 percent of 14-year-old girls compared to about 20 percent of boys are actively trying to lose weight. These seeds of discontentment begin even earlier with studies documenting body dissatisfaction in children as young as 6 years old, an age much before they reach pubertal milestones. Then, rather than subsiding in adulthood, concerns about body weight appear to intensify; however, there is some evidence that actual attempts to lose weight may diminish over time along with a decline in disordered eating patterns.

BODY MASS INDEX (BMI) AND DIETING

BMI is one of the most robust predictors of body dissatisfaction and dieting among women. High levels of weight concern are reported in almost 60 percent of women with a BMI of 25 or higher compared to about 25 percent of women with a BMI between 19 and 24.9. Also of interest, more than 10 percent of women with a BMI less than 19 report very high levels of weight concern. Studies indicate that between 40 and 60 percent of high school and college women tried to lose weight in the previous year. Dieting is not uncommon among women who are in the normal weight range and is reported among the 10 to 15 percent of college students who are objectively underweight (BMI less than 19). Interestingly, more than 10 percent of women who report being content with their weight still indicate they are trying to lose weight. Several studies have found that perceived weight status is a better predictor of extreme dieting methods than actual body weight.

It is important to emphasize that measures of body dissatisfaction and dieting have different clinical significance for individuals at different weights. For example, patients with anorexia nervosa and nonclinical college women report similar levels of body dissatisfaction on standardized measures; however, the patients are emaciated compared to the average college student. Although a significant proportion of both groups describe being relatively satisfied with their weight, anorexia nervosa patients are satisfied with a clearly aberrant body weight. Nevertheless, the potential clinical significance of high levels of body dissatisfaction and obsessive dieting among women at the heavier end of the weight spectrum should not be dismissed because these behaviors may be associated with clinical levels of emotional distress.

DIETING AND EATING DISORDER RISK

It could be argued that the high percentage of women who are dieting may reflect that this behavior is relatively benign. However, the evidence does not support this view. Prospective studies indicate that body

dissatisfaction and restrictive dieting are powerful predictors of the development of full-blown eating disorders as well as partial syndromes characterized by dangerous weight-controlling behaviors such as self-induced vomiting, fasting, and laxative abuse. The magnitude of this risk is considerable. Fifteen-year-old girls classified as dieters have eight times higher risk of developing an eating disorder compared to nondieters, and the risk is about 18 times higher for those classified as severe dieters. The starting point for risk can be traced to maternal eating behavior. Prospective studies of mothers and their newborns indicate that maternal dieting and eating disorder symptoms are strong predictors of the development of early childhood eating disturbances.

However, we should be aware of the risk of oversimplifying the relationship between body dissatisfaction, dieting, and eating disorders. Other variables such as media exposure to thin-ideal body stereotypes, internalization of the thin ideal, social comparison, self-esteem, family support, and perfectionism have been shown to improve the prediction of both body dissatisfaction and dieting. For example, perfectionistic women who perceive themselves as overweight engage in extreme dieting and bulimic symptoms more often if they have low self-esteem. They seem to doubt their ability to achieve their high standards. In contrast, perfectionistic women with high self-esteem seem to be protected from severe dieting and bulimic symptoms. Studies of Eating Attitudes Test (EAT-26) results from normal twin pairs have also provided evidence for a substantial genetic contribution to body dissatisfaction and eating concerns.

Finally, understanding the role of dieting as a risk factor for eating disorders must reconcile the fact that trying to lose weight is endemic to young women in Western culture; however, it leads to the expression of eating disorders in only a small fraction of the population. This observation could also be used to minimize the risks associated with dieting, but rather is should be equated to the association between cigarette smoking and cancer. The risks of dieting are real and should be treated as such.

GENDER CONSIDERATIONS

Epidemiological studies of dieting have consistently found that body dissatisfaction is more common among women than men. Moreover, the form, substance, and behavioral correlates of body dissatisfaction appear to be different in men and women. Men are concerned with muscularity as well as body size and many men who are dissatisfied with their weight want to gain weight rather than lose. Men who do perceive themselves as overweight tend to use exercise and healthier dieting patterns than women. Nevertheless, there is evidence that body dissatisfaction and unhealthy dieting patterns are becoming more common among adolescent boys and adult men.

More than 10 percent of women who report being content with their weight still indicate they are trying to lose weight.

RACIAL AND ETHNIC DIFFERENCES

Research has indicated that there are racial and ethnic differences in body dissatisfaction and dieting in Western culture. Historically, weight concerns, dieting, and eating disorders have been attributed to primarily white, middle-class women; however, it is now well recognized that the pattern of racial and ethnic

associations is more complex. Although generalizations must be made with caution, weight concerns appear to be less common among African Americans than among Caucasian and Hispanic women. Asian women show the fewest concerns and Native Americans show the most. The same pattern of findings applies for dieting. Although African-American women report less body dissatisfaction and dieting than other groups, they report binge eating more often. While racial and ethnic differences in body dissatisfaction and dieting do exist, it is important to be mindful of generalizations that could minimize the significance of attitudes and behavior reported by minorities.

Emerging research has shown that body image may be improving, possibly decreasing the risk of unhealthy dieting behaviors. In an article published by Cash and colleagues, it is suggested that body image in college-aged women is currently evolving positively. The study spanned 19 years and evaluated body image using several different measures. Using the data collected, it was determined that although there was a significant decline in body satisfaction from the early 1980s until the mid-1990s, this decline began to reverse in the mid-1990s, causing an overall increase in body satisfaction at the last point of evaluation.

PREVENTION OF EATING DISORDER RISK

There are a number of promising programs found to decrease risk factors for eating disorders such as body dissatisfaction and dieting. In general, there are more positive findings for programs that are interactive versus didactic, multisession versus single session, and with participants older than 15 years of age with high levels of weight and shape concerns. Barr Taylor and colleagues report impressive results from an 8-week, internet-based cognitive-behavior intervention, and the improvements remained significant at one-year follow-up.

INEFFECTIVENESS OF DIETING

The steadily increasing prevalence of obesity in developed countries continues to be a major health concern. The promotion of various forms of dieting as a solution to obesity has remained popular in the absence of evidence for long-term effectiveness except for a small minority of cases. Participation in weight-loss programs usually results in small to moderate amounts of weight loss followed by regain. Moreover, continued participation in weight-loss programs is associated a pattern of weight loss and regain ("weight cycling"). The health effects of weight cycling remain controversial; however, animal studies have indicated that weight cycling may increase metabolic efficiency, insulin resistance, blood pressure, and the consumption of dietary fat. Epidemiological studies of humans have linked weight cycling to increased mortality risk. Even in the absence of serious health consequences, it would be imprudent to underestimate the effects of guilt, shame, and self-disparagement following failed attempts to maintain weight loss as well as the significance of the economic burden associated with participation in weight-loss programs.

The high prevalence of dieting, weight concerns, and obesity among adolescents as well as their potentially serious physical and psychological consequences has raised questions regarding the possible effects of dieting on unintentional weight gain. Again, this is a controversial area with inconsistent findings across studies. However, more recent longitudinal research has found that dieting not only is associated with the development of severe weight control behaviors but also with weight gain. A recent 5-year follow-up study by Neumark-Sztainer and colleagues found that dieting and particularly unhealthy weight-control behaviors predicted subsequent weight gain, overweight status, binge eating, extreme weight control behaviors, and the onset of eating disorders.

Moreover, even behaviors commonly recommended as part of healthful weight management (e.g., increase fruit and vegetable consumption and physical activity) were not predictive of greater weight control five years later. More than half of female adolescents and one-quarter of male adolescents were dieting at the onset of the study. The pervasiveness of serious health consequences and the paradoxical association with weight gain over time rather than weight loss mean that dieting cannot be considered either innocuous or as a conservative approach to health. Taken together, these findings suggest the need for a major shift in thinking about the advisability of recommending dieting for weight control.

ALTERNATIVES TO DIETING

If chronic restrictive dieting has untoward effects, what are the alternatives for those with obesity or

eating disorders? Details of the available alternatives go beyond the scope of this chapter; however, alternatives to the restrictive dieting approach to obesity have moved from the unorthodox (described by David Garner and Susan Wooley) to the mainstream. The new paradigm for the management of obesity more recently described in a special issue of the *Journal of Social Issues* in 1999 and introduced by Jeanine Cogan and Paul Earnsberger emphasizes trying to improve the health and well-being of individuals who are obese without requiring restrictive dieting or weight loss. The main principles involve increasing activity and making healthful food choices within the context of "fat acceptance."

Individuals with eating disorders typically engage in an extreme form of dietary restraint characterized by rigid dietary rules that constitute extreme interpretations of sensible dietary guidelines. For instance, red meat may be excluded altogether, not just minimized; dietary fat and sugar may be totally eliminated rather than reduced; only food considered not fattening is permitted; food intake is limited to 800 calories a day; food will not be consumed after 7:00 p.m. Deviating from these highly restrictive dieting is usually met with intense anxiety. Psychoeducation may be used to correct these nutritional myths; however, a form of structured meal planning is usually required to assist in graded exposure to fears associated with eating certain types and amounts of food. Meal planning involves a detailed written plan specifying meal times as well as the exact types and amounts of food to be consumed. It is an important first step in assisting those with eating disorders to overcome highly restrictive eating patterns.

SEE ALSO: Body Mass Index; Depression; Dieting: Good or Bad?; Fat Acceptance.

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World Patterns

CONCERNS OF WEIGHT and obesity are generally thought to be confined to higher-income nations such as the United States and parts of the European Union. This idea is due to the persistent idea that Western lifestyle choices accompany many of the predisposing behaviors relative to obesity such as overeating and aversion toward regular physical activity. However, a survey of low- and medium-income nations reveals quite a different pattern, one of growing obesity, especially within urban settings.

Indeed, the World Health Organization (WHO) has coined the term *globesity* to indicate the pandemic nature of obesity and concomitant public health outcomes. Without consistent international analysis and oversight, public health consequences of obesity are sure to amplify the global burden of disease as well as exacerbate the already-elevated weight-related prevalence of mortality. Such events may lead to higher economic and social costs and could subsequently reduce a nation's ability to properly sustain itself.

INTERNATIONAL PREVALENCE

In 2005, it was estimated that 1.6 billion adults 15 years of age and over were overweight with an additional 400 million obese. These figures are projected to increase to 2.3 billion and 700 million, respectively, by the year 2015. In addition, approximately 20 million children younger than 5 years of age were overweight.

According to current estimates, the United States represents a large percentage of the world's obese population, accounting for 23 percent. Approximately 35 percent of the overall global population has a body mass index (BMI) greater than 25 with the United

States reporting an estimate of 74 percent. Other nations with relatively high percentages overweight and/or obese persons include Mexico (68 percent), United Kingdom (64 percent), and Germany (60 percent).

Other nations, even those considered low and middle income, have estimated rates of obesity similar to estimates reported among high-income nations (members of the international community are classified according to the United Nations as high-, middle-, or low-income nations based upon their gross national income per capita [GNI PC]. High-income nations reported a GNI PC in 2002 of greater than $9,076, followed by middle-income [$736–$9,076] and low-income nations [$735 or less]). For example, about 25 percent of those living in the Middle East are either overweight or obese. Similarly, South Africa has reported high obesity measures. Overweight estimates range from 11 percent in Indonesia to 40 percent in Slovenia, while obesity measures range form 2.4 percent in Indonesia to 36 percent in Saudi Arabia.

Additional data from WHO indicate a global problem with obesity and overweight. Among 194 nations, age-adjusted obesity estimates for 2005 for both males and females revealed a median prevalence of 14 percent for individuals 15 years of age or older.

One of the most problematic elements of the obesity pandemic is that those most at risk for long-term health outcomes specific to obesity are children and adolescents. Youth who are classified as either overweight or obese are much more likely to carry their weight problem into adulthood and to suffer more physical consequences of advanced size. A study by Janssen et al. (2005) outlined the scope of overweight and obese school-aged youth in 34 countries indicating that Malta had the highest prevalence of overweight and obese youth (aged 10–16); 25 percent and 8 percent, respectively, followed by the United States (25 percent and 7 percent, respectively). Lithuania and Latvia were identified as countries with the lowest prevalence of each (5 percent; 0.4 percent and 6 percent; 0.5 percent, respectively).

Without consistent international analysis and oversight, public health consequences of obesity will amplify the global burden of disease.

GLOBAL HEALTH CONSEQUENCES

Obesity is associated with numerous comorbidities such as diabetes, cancer, osteoporosis, and cardiovascular disease, and should therefore be viewed as a gateway to other, more problematic and costly health conditions. For example, a study by Goodarz et al. indicated that in higher-income countries, overweight and obesity, along with smoking and alcohol use, were the most prominent correlates of cancer. Additionally, Strum has equated the physical health of an individual classified as obese with that of a normal-weight individual 20 years senior.

The impact of obesity on the global community is great and far reaching. Negative outcomes include those related not only to quality and quantity of life, but also the accompanying economic burden of dealing with the threats to public health brought about by overweight and obesity. A number of such consequences have been identified. Increases in BMI above 25 may result in the emergence of a host of chronic diseases such as cardiovascular disease (currently the primary global, noninfectious killer), diabetes, osteoporosis, and some forms of cancer, each increasing a nation's social and economic burden of the disease. Faced with epidemics of both infectious (e.g., human immunodeficiency virus [HIV] and tuberculosis) and noninfectious diseases (e.g., heart disease and diabetes) many low- and middle-income nations are beginning to encounter what WHO refers to as

a "double burden" of disease. These public health challenges are especially problematic among nations where a dearth of medical access, civil conflict, and infectious diseases have already taken a great toll on the economic security of the region.

ESTIMATES OF COST

Although highly reliable, among-country comparative measures are unavailable, data from the United States indicate that the economic cost of overweight and obesity range from $52 billion to $79 billion per year with an estimated 12 percent of the U.S. healthcare budget being consumed by conditions related to obesity. Relatively high expenditures have been reported in Australia and the United Kingdom where recent costs of obesity have been reported ($290 million and $232 million, respectively). Regardless of data source, it is clear that obesity accounts for a growing percentage of healthcare expenditures across the globe, resulting in larger portions of national budgets being consumed by complications related to issues of weight.

GLOBAL ETIOLOGIC FACTORS

Along with diet, lack of exercise, and genetics, a number of factors serve as antecedents to the growing international threat of obesity such as globalization and subsequent Western enculturation, along with advances in technology and communication.

For example, economic growth and development has become a mainstay for many less-advantaged nations, with Western countries infusing skill sets, funding, and infrastructure in an effort to boost international productivity and a sense of independence. With this process also comes the contribution of less desirable health behaviors often thought to be circumscribed to the West. This process of enculturation is a natural one, but one that may lead to the spread of health outcomes such as cardiovascular diseases, diabetes, smoking, alcohol consumption, and poor dietary choices.

In essence, health behaviors normally thought of as Western in nature are being exported to other, more vulnerable parts of the globe simply as a result of economic growth and development. Similarly, the accumulation of technologies designed to improve life quality has also fostered passiveness in both work and living environments which now require much less effort to maintain, leading to the instillation of sedentary lifestyles, a major progenitor of obesity.

GLOBAL PREVENTION EFFORTS

Global obesity prevention efforts must employ a bifurcated approach to include efforts on both the individual and community levels. For individuals, methods of reducing or maintaining weight are fairly straightforward. Effective measures include (1) adopting balanced eating patterns, (2) regular physical activity, and (3) limitation of high-energy foods such as those offered by fast-food establishments.

Community-level initiatives tend to be more challenging given the potential scope of psychosocial and environmental change. Community-specific deliverables include availability of nutritious foods; changes in outlook with regard to prenatal care and education; increasing access to healthcare as well as fostering a greater level of clinical involvement; control of access to predisposing risk factors; and development of legal guidelines with regard to prevention and school-based public health education. A number of school-based prevention initiatives have been identified, primarily in elementary schools; however, emerging nations tend not to focus on youth-mediated prevention in lieu of more immediate national healthcare needs.

A number of international organizations have joined efforts to begin addressing the many concerns raised by the growing global overweight and obesity pandemic. As an example, the WHO Global Strategy on Diet, Physical Activity and Health was adopted in 2004 and describes specific actions necessary for members of the global community to reduce the prevalence of chronic diseases and their risk factors, primarily diet and lack of regular, vigorous exercise. The Strategy also outlines mechanisms useful at the local, regional, and global levels.

Additional elements necessary for a successful international drive toward controlling the global obesity epidemic include responsible development practices on the part of Western nations; availability of healthy foods (especially for young children); educational interventions designed to teach responsible food choices as well as overall healthy lifestyle options; standardization of monitoring practices designed to enhance our understanding of need; across-region labeling of food products; and international cooperation and collaboration among regional, national, and nongovernmental organizations.

Those most at risk of obesity-related health complications include children and adolescents. Public

health intervention must include an understanding of age-specific etiology when designing intervention opportunities. Specific to youth, initiatives must include elements of vigorous physical activity, limited intake of high-energy foods, and restrictions with regard to watching television or long-term use of technology such as the internet.

DATA QUALITY AND PREVALENCE ESTIMATION

Estimates of current obesity prevalence in the United States, as well as in other regions of the world, vary depending upon how these data are monitored and collected. Therefore, the utility of international comparisons must be tempered given unique methods of tabulation. First, weight measures tend to be solely based upon the BMI. Calculations using the formula to calculate BMI do not take into consideration important factors such as muscle mass or race and ethnicity, both of which serve to influence BMI estimates. Also, standard BMI intervals are not generally used to classify children (aged 0–12) as either overweight or obese. Instead, BMI measures specific to age are calculated. Children who fall above the 85th percentile are considered overweight and those above the 95th percentile are classified as obese. Although inexpensive and uncomplicated to formulate, the BMI should be used with caution.

Second, data collection methods differ among countries, with some methods being more standardized than others. Survey sampling, census data collection, and meta-analyses yield useful information about the scope of obesity worldwide, yet differences in data collection methodology may reduce the validity of statistical estimation. In addition, weight is a sensitive issue for many individuals, particularly females, making self-reported estimates of BMI less accurate than data collected through more rigorous means.

Finally, international data tend to be based upon different populations in terms of the age intervals on which estimates are based. Estimates from Saudi Arabia are based upon individuals 30 years of age and over, while data from France represent a much younger population (15 and over). Although these reporting concerns have perhaps a negligible influence on data quality and comparability given that weight tends to increase with age, the growing pandemic of overweight and obesity among children makes this concern one which should be addressed in an effort to produce more uniform data collection procedures.

CONCLUSION

Given contemporary statistics regarding the international scope of overweight and obesity, a number of concerns are clear. First, overweight and obesity are threats to public health in many nations regardless of level of development, population structure, or history. Second, although based upon limited international data, the obesity pandemic appears to be spreading, especially among children and adolescents. Finally, members of the global community must work together to find community- and individual-level solutions to the issue of weight in an effort to curtail comorbidities that accompany BMI measures greater than 25 such as diabetes, cancer, and cardiovascular disease. Successful broad-based interventions will preserve national integrity, improve financial stability, and enhance both the quality and quantity of life of members of the global community.

SEE ALSO: Body Mass Index; Economics of Obesity; Governmental Policy and Obesity; International Obesity Task Force.

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Zone, The

THE ZONE DIET was introduced in 1995 by Dr. Barry Sears. The original Zone Diet required the individual to maintain the 40:30:30 ratio, meaning that at every meal and snack, a person should consume 40 percent carbohydrate, 30 percent protein, and 30 percent fat. The Zone Diet is therefore neither high or low in either carbohydrates, protein, or fat, but represents what Sears considers to be the optimal balance of those three macronutrients.

This requirement has been somewhat relaxed and in more recent versions of the diet, meal compositions in the ranges of 35 to 50 percent carbohydrate, 25 to 35 percent protein, and 20 to 35 percent fat are acceptable. Sears claims that individuals who follow the Zone Diet will maintain stable blood sugar levels and hormonal balance, in particular to avoid insulin spikes which he claims results from high carbohydrate consumption. The Zone Diet has been popularized in Sears' books and several Web sites and through the sale of nutritional products such as snack bars.

The name *The Zone* refers to a concept used by athletes, who say they are "in the zone" when they are achieving their optimal level of performance. This concept is a cornerstone of Sears's philosophy: He claims that people who follows his dietary recommendations can put themselves into a state not only of optimal performance but of optimal health. He further argues that food should be thought of as a powerful drug, and eating as a kind of technology serving as a drug delivery, and thus that food should only be consumed in appropriate proportions and on a controlled schedule.

Sears includes a number of tools to help an individual plan a diet which will follow the principles of The Zone, using ordinary supermarket food selected and prepared by the individual. His system is based on defining "blocks" of each macronutrient: for instance, one block of protein contains about 7 g of protein and could be 1 oz of skinless chicken breast, 1 oz of tofu, 2 oz of low-fat cottage cheese, or two egg whites. Similarly, carbohydrates and fats are classified into blocks: for instance, a carbohydrate block contains about 9 grams of carbohydrate, the amount in one-half a medium apple or one-quarter cup lentils, and three olives or one-half teaspoon peanut butter constitute one fat block, defined as 1.5 grams of fat. After a person has determined his or her daily protein requirement (using a formula found in his books), he or she is advised to distribute his or her protein consumption throughout the day, over three meals and two snacks. The individual can then construct his or her daily diet so each meal and snack contains the appropriate number of protein blocks, matched by an equal number of carbohydrate and fat blocks. Because the carbohydrate blocks contain more calories, this keeps the diet in the proper balance to remain "in the zone."

Although the Zone Diet has some flexibility depending on a person's caloric and protein requirements, a typical plan calls for 1,200 calories per day for adult females and 1,500 for adult males. Sears recommends that the individual eat five to six small meals per day which are evenly spaced, and claims that individuals who adhere to the Zone Diet will not feel hungry despite consuming fewer calories than is typical in an American diet, because of what he calls "The Zone Paradox." This is another cornerstone of Sears's philosophy: He believes that not all calories are equal, and that if macronutrients are consumed in the specified ratios (in particular if sufficient protein is consumed every time a person eats), a person's insulin ratio will not spike and will not feel hungry despite eating fewer calories than he or she might consume on a high-carbohydrate diet.

It is possible to adhere to the Zone Diet wholly through selection and preparation of ordinary foods, although a variety of Zone products such as snack bars and shakes are also available for purchase, although not every product advertising that it maintains the 40:30:30 ratio is affiliated with or approved by Sears. The Zone Diet is not aimed primarily at weight loss, although it may be used for that purpose: Sears advocates it as a healthy diet for everyone. He also advocates the Zone Diet for athletes; Sears was a national-level volleyball player as a young man and has acted as a nutritional adviser to a number of elite athletes, including the swim team of Stanford University and the Los Angeles Rams football team.

Most research into the Zone Diet (or diets that are based on a similar ratio of carbohydrates, protein, and fat) were concerned with its effectiveness in weight loss and fat reduction among overweight individuals. For instance, D. Kalman and colleagues conducted a randomized study in which 29 overweight adults were randomized to either a 40/30/30 or 60/15/25 (American Heart Association Step 1) diet for six weeks. All subjects also participated in a supervised exercise program and met with a dietitian two to four times. Both groups lost a significant amount of weight, but the 40 percent carbohydrate (CHO) group lost more weight and body fat, and also achieved reduction in fatigue and increase in vigor, while no such change was observed in the 60 percent CHO group.

M.L. Dansinger and colleagues conducted a randomized trial of 160 overweight adults assigned to one of four popular diet groups: Atkins, The Zone, Weight Watchers, or Ornish. After two months, participants were in charge of their own level of adherence to the assigned diet. Sixty-five percent of those assigned to The Zone completed the two-year study, tied for highest among the four groups with Weight Watchers. Self-reported adherence to any of the diets was associated with weight loss and improvement in cholesterol and other heart disease risk factors, but there was not a difference between diet types.

Some studies have also looked at the efficacy of the Zone Diet in improving athletic performance and body composition, often focusing on comparison with high-carbohydrate diets which have typically been recommended particularly for endurance athletes. For instance, a 1990 study at Ohio State University compared the performance of college swimmers who consumed a diet of either 40 percent or 80 percent carbohydrates.

After a nine-day trial period, there was no difference between groups on performance. A similar study at Ohio State in 1993 came to the same conclusion. This time, the subjects were runners and cyclists, but again, the adoption of a high-carbohydrate diet produced no gains in performance.

SEE ALSO: Carbohydrate and Protein Intake; High Protein Diets; Insulin; Low Calorie Diets; Portion Control.

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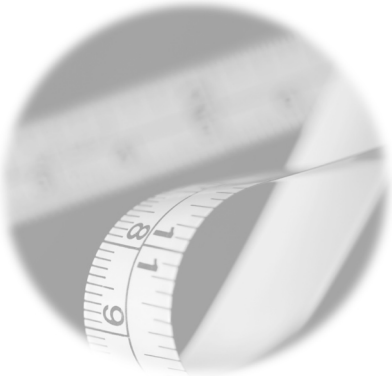

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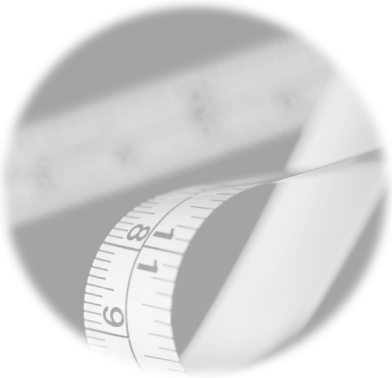


Appendix

Prevalence of Obesity in Males and Females by Country

World Health Organization

Obesity and overweight are defined as an accumulation of excess body fat, to an extent that may impair health. A crude population measure of excess fat is the body mass index (BMI), a person's weight (in kilograms) divided by the square of his or her height (in meters). WHO defines overweight as a BMI of 25 or more, and obesity as a BMI of 30 or more. These cut-off points can provide a reference for individual assessment.

There is also evidence that, on a population level, the risk of chronic disease increases progressively as average BMI increases above 21. A high body mass index is a major risk factor for a number of chronic diseases, including cardiovascular diseases, cancer and diabetes.

OBESITY – SORTED IN DESCENDING ORDER
2005 BMI ≥ 30 kg/m², Females, aged 15-100

Rank	Country	Prevalence (%)
1.	Nauru	78.8
2.	Tonga	76.1
3.	Micronesia, Federated States of	72.9
4.	Cook Islands	70.8
5.	Niue	61.0
6.	Samoa	57.3
7.	Palau	55.0
8.	Kuwait	52.9
9.	Barbados	50.8
10.	Trinidad and Tobago	46.1
11.	Dominica	46.0
12.	Egypt	45.5
13.	United States	41.8
14.	Jamaica	41.0
14.	Kiribati	41.0
15.	United Arab Emirates	39.4
16.	Seychelles	38.6
17.	Jordan	35.6
18.	Bahrain	35.2
18.	South Africa	35.2
19.	Malta	34.8
20.	Saint Lucia	34.7
21.	Lesotho	34.3
21.	Mexico	34.3
21.	Nicaragua	34.3
22.	Saudi Arabia	33.8
23.	Bolivia	33.1
24.	Fiji	32.5
24.	Turkey	32.5
25.	Belarus	32.2
26.	Dominican Republic	31.8
27.	Chile	31.6
28.	New Zealand	31.5
29.	Peru	31.1
30.	Argentina	31.0
31.	Tunisia	30.2
32.	Guatemala	29.7
33.	Qatar	29.3
34.	Mongolia	29.0
35.	Andorra	28.8
36.	Monaco	27.5
37.	Brunei Darussalam	27.4
38.	San Marino	27.2
39.	Bahamas	27.1
40.	Iran (Islamic Republic of)	27.0
41.	Vanuatu	26.3
42.	Venezuela (Bolivarian Republic of)	26.2
43.	Lebanon	25.2
43.	Slovenia	25.2
44.	Australia	24.9
44.	Azerbaijan	24.9
45.	Cuba	24.6
46.	Greece	24.5
47.	Israel	24.3
47.	The former Yugoslav Republic of Macedonia	24.3
48.	Costa Rica	24.2
48.	United Kingdom	24.2
49.	Albania	23.8
49.	Tuvalu	23.8
50.	Russian Federation	23.6
51.	Saint Kitts and Nevis	23.4
52.	Uruguay	23.3
53.	Canada	23.2
53.	Iceland	23.2
54.	Antigua and Barbuda	22.9
54.	Mauritania	22.9
55.	Slovakia	22.8
56.	Libyan Arab Jamahiriya	22.5
57.	Cyprus	22.2
57.	Syrian Arab Republic	22.2
58.	Maldives	22.0
59.	Bosnia and Herzegovina	21.5
60.	Grenada	21.2
61.	Czech Republic	20.7
62.	Montenegro	20.6
62.	Serbia	20.6
62.	The former state union of Serbia/Montenegro	20.6
63.	Morocco	20.5
64.	Germany	20.4
65.	Austria	20.3
66.	Colombia	19.9
67.	Armenia	19.8
67.	Panama	19.8
68.	Ukraine	19.4
69.	Saint Vincent and the Grenadines	19.2
70.	Bulgaria	19.0
71.	Switzerland	18.7
72.	Belize	18.6
73.	Brazil	18.3
73.	Mauritius	18.3
74.	Poland	18.0
75.	El Salvador	17.8
75.	Finland	17.8
76.	Uzbekistan	17.6
77.	Paraguay	17.2
77.	Suriname	17.2
78.	Guyana	17.0
79.	Iraq	16.8
80.	Ecuador	16.7
81.	Croatia	16.2

OBESITY – SORTED IN DESCENDING ORDER
2005 BMI ≥ 30 kg/m², Females, aged 15-100

Rank	Country	Prevalence (%)
82.	Hungary	16.1
82.	Marshall Islands	16.1
82.	Portugal	16.1
83.	Luxembourg	16.0
84.	Spain	15.8
85.	Gabon	15.5
86.	Democratic Republic of Timor-Leste	15.4
86.	Equatorial Guinea	15.4
87.	Zimbabwe	15.3
88.	Haiti	15.0
88.	Latvia	15.0
88.	Turkmenistan	15.0
89.	Oman	14.8
90.	Georgia	14.7
90.	Solomon Islands	14.7
91.	Botswana	14.6
92.	Honduras	14.4
93.	Bhutan	14.3
94.	Kyrgyzstan	14.2
95.	Lithuania	13.9
96.	Swaziland	13.5
97.	Algeria	13.4
98.	Sierra Leone	12.7
99.	Italy	12.6
100.	Cape Verde	12.5
100.	Republic of Moldova	12.5
101.	Romania	12.0
102.	Netherlands	11.5
103.	Kazakhstan	11.0
103.	Liberia	11.0
104.	Sweden	10.9
105.	Cameroon	10.8
106.	Democratic People's Republic of Korea	10.7
107.	Lao People's Democratic Republic	10.4
107.	Tajikistan	10.4
108.	Republic of Korea	10.1
109.	Belgium	9.5
110.	Benin	9.3
110.	Norway	9.3
111.	Senegal	9.2
112.	Ireland	9.1
112.	Myanmar	9.1
113.	Estonia	8.4
113.	Thailand	8.4
114.	Malaysia	8.2
115.	Comoros	7.1
115.	Denmark	7.1
116.	Angola	6.9
117.	France	6.6
118.	Mali	6.2
119.	Nigeria	6.0
120.	Djibouti	5.8
121.	Cote d'Ivoire	5.4
122.	Namibia	5.3
122.	Togo	5.3
123.	Guinea	5.2
124.	Sudan	5.1
124.	Yemen	5.1
125.	Sao Tome and Principe	4.4
126.	Ghana	4.2
126.	Papua New Guinea	4.2
127.	Philippines	3.7
128.	Pakistan	3.6
129.	United Republic of Tanzania	3.1
130.	Congo	3.0
130.	Mozambique	3.0
131.	Guinea-Bissau	2.8
132.	Indonesia	2.6
132.	Somalia	2.6
133.	Gambia	2.5
134.	Niger	2.3
135.	Malawi	2.0
136.	China	1.9
136.	Kenya	1.9
136.	Madagascar	1.9
137.	Singapore	1.8
138.	Chad	1.7
139.	Uganda	1.6
140.	Burundi	1.5
140.	Japan	1.5
141.	Afghanistan	1.4
141.	India	1.4
142.	Central African Republic	1.3
142.	Rwanda	1.3
142.	Zambia	1.3
143.	Burkina Faso	1.1
144.	Democratic Republic of the Congo	0.8
145.	Viet Nam	0.3
146.	Bangladesh	0.2
146.	Nepal	0.2
147.	Cambodia	0.1
147.	Eritrea	0.1
147.	Sri Lanka	0.1
148.	Ethiopia	0.0

OBESITY – SORTED IN DESCENDING ORDER
2005 BMI ≥ 30 kg/m², Males, aged 15-100

Rank	Country	Prevalence (%)
1.	Nauru	83.2
2.	Cook Islands	69.5
3.	Micronesia, Federated States of	66.2
4.	Tonga	60.7
5.	Samoa	38.4
6.	Niue	36.8
7.	United States	36.5
8.	Argentina	31.4
9.	Palau	31.2
10.	Kiribati	29.8
11.	Kuwait	29.6
12.	Greece	27.7
13.	Malta	25.9
14.	United Arab Emirates	24.5
15.	Mexico	24.0
16.	Australia	23.8
17.	Canada	23.7
18.	Venezuela (Bolivarian Republic of)	23.2
19.	New Zealand	23.0
19.	Saudi Arabia	23.0
20.	Egypt	22.0
21.	United Kingdom	21.6
22.	Austria	21.3
23.	Bahrain	21.2
24.	Germany	20.9
25.	Uruguay	20.1
26.	Dominica	20.0
27.	Jordan	19.6
28.	Chile	19.0
29.	Finland	18.9
30.	Albania	18.6
31.	Czech Republic	18.5
32.	Croatia	18.2
33.	Montenegro	17.7
33.	Serbia	17.7
33.	The former state union of Serbia/Montenegro	17.7
34.	Qatar	17.4
35.	Bulgaria	17.0
36.	Barbados	16.8
36.	Lithuania	16.8
37.	Iceland	16.7
37.	Seychelles	16.7
38.	Belarus	16.2
38.	Israel	16.2
39.	Andorra	15.8
39.	Hungary	15.8
40.	Guatemala	15.7
41.	Spain	15.6
42.	Azerbaijan	15.4
43.	Brunei Darussalam	15.2
44.	Colombia	14.9
44.	Cuba	14.9
44.	Lebanon	14.9
45.	Bahamas	14.7
45.	Bolivia	14.7
46.	Monaco	14.5
47.	San Marino	14.3
48.	Trinidad and Tobago	14.0
49.	Bosnia and Herzegovina	13.8
50.	Portugal	13.7
51.	Vanuatu	13.4
52.	Belgium	13.3
53.	Peru	13.2
54.	Costa Rica	13.0
55.	Italy	12.9
55.	Poland	12.9
56.	Slovenia	12.5
57.	Switzerland	12.4
58.	Armenia	12.1
58.	Luxembourg	12.1
59.	Tuvalu	11.9
60.	Sweden	11.8
61.	Saint Kitts and Nevis	11.6
62.	Nicaragua	11.5
63.	Libyan Arab Jamahiriya	11.4
64.	Norway	11.3
65.	Antigua and Barbuda	11.2
65.	Syrian Arab Republic	11.2
66.	Slovakia	10.8
66.	Turkey	10.8
67.	Denmark	10.6
68.	Netherlands	10.4
69.	Ireland	10.3
70.	Cyprus	10.1
71.	Iran (Islamic Republic of)	10.0
72.	Grenada	9.8
73.	Latvia	9.7
74.	Russian Federation	9.6
75.	Turkmenistan	9.3
76.	Panama	8.8
77.	Brazil	8.7
77.	Fiji	8.7
78.	Estonia	8.6
79.	Saint Vincent and the Grenadines	8.4
80.	Belize	7.9
80.	Kazakhstan	7.9
80.	Mongolia	7.9
81.	France	7.8
82.	Dominican Republic	7.7
82.	Oman	7.7

OBESITY – SORTED IN DESCENDING ORDER
2005 BMI ≥ 30 kg/m², Males, aged 15-100

Rank	Country	Prevalence (%)
82.	Tunisia	7.7
83.	Cameroon	7.5
84.	El Salvador	7.4
84.	Ukraine	7.4
85.	Iraq	7.2
86.	Uzbekistan	7.1
87.	Paraguay	7.0
87.	Suriname	7.0
88.	Guyana	6.8
89.	Ecuador	6.7
89.	South Africa	6.7
90.	Saint Lucia	6.6
91.	Democratic Republic of Timor-Leste	6.5
92.	Equatorial Guinea	6.4
93.	Marshall Islands	6.3
94.	The former Yugoslav Republic of Macedonia	5.9
95.	Bhutan	5.8
96.	Maldives	5.7
97.	Mauritius	5.6
98.	Romania	5.5
99.	Botswana	5.4
99.	Solomon Islands	5.4
100.	Algeria	5.2
100.	Georgia	5.2
100.	Honduras	5.2
101.	Jamaica	5.1
102.	Kyrgyzstan	5.0
103.	Swaziland	4.7
104.	Cape Verde	4.6
105.	Republic of Korea	4.1
106.	Republic of Moldova	4.0
107.	Liberia	3.8
108.	Mauritania	3.7
108.	Morocco	3.7
109.	Ghana	3.3
110.	Tajikistan	2.9
111.	Democratic People's Republic of Korea	2.7
112.	Lao People's Democratic Republic	2.6
113.	Papua New Guinea	2.5
113.	Thailand	2.5
114.	Sierra Leone	2.4
115.	Gabon	2.3
116.	Myanmar	2.1
117.	Nigeria	2.0
117.	Yemen	2.0
118.	Angola	1.9
118.	Lesotho	1.9
119.	Japan	1.8
120.	China	1.6
120.	Malaysia	1.6
121.	Djibouti	1.4
122.	Senegal	1.3
122.	Singapore	1.3
123.	Comoros	1.2
123.	Sudan	1.2
124.	India	1.1
124.	Philippines	1.1
125.	Benin	1.0
125.	Madagascar	1.0
125.	Pakistan	1.0
126.	Sao Tome and Principe	0.9
126.	Togo	0.9
127.	Guinea	0.8
128.	Haiti	0.7
128.	Malawi	0.7
128.	United Republic of Tanzania	0.7
129.	Mali	0.6
129.	Niger	0.6
129.	Zimbabwe	0.6
130.	Afghanistan	0.5
130.	Guinea-Bissau	0.5
131.	Burkina Faso	0.4
131.	Chad	0.4
131.	Congo	0.4
131.	Somalia	0.4
132.	Gambia	0.3
132.	Namibia	0.3
133.	Cambodia	0.2
133.	Cote d'Ivoire	0.2
133.	Ethiopia	0.2
133.	Indonesia	0.2
133.	Mozambique	0.2
133.	Nepal	0.2
133.	Sri Lanka	0.2
134.	Bangladesh	0.1
134.	Burundi	0.1
134.	Central African Republic	0.1
134.	Kenya	0.1
134.	Rwanda	0.1
134.	Uganda	0.1
134.	Zambia	0.1
135.	Viet Nam	0.0
135.	Democratic Republic of the Congo	0.0
135.	Eritrea	0.0

OVERWEIGHT – SORTED IN DESCENDING ORDER

2005 BMI ≥ 30 kg/m², Females, aged 15-100

Rank	Country	Prevalence (%)
1.	Nauru	92.4
2.	Tonga	91.4
3.	Micronesia, Federated States of	90.1
4.	Cook Islands	89.2
5.	Niue	85.0
6.	Palau	82.4
7.	Samoa	82.1
8.	Barbados	80.1
9.	Kuwait	79.0
10.	Dominica	77.1
11.	Trinidad and Tobago	77.0
12.	Jamaica	74.7
13.	Egypt	74.2
14.	Kiribati	73.9
15.	United States	72.6
16.	Seychelles	70.7
17.	Belarus	69.9
18.	United Arab Emirates	69.6
19.	Lesotho	69.5
20.	Mongolia	69.3
21.	Saint Lucia	69.1
22.	New Zealand	68.2
23.	Nicaragua	68.1
24.	Bolivia	68.0
24.	Chile	68.0
25.	Mexico	67.9
26.	Bahrain	67.3
27.	South Africa	67.2
28.	Andorra	66.8
29.	Dominican Republic	66.4
30.	Malta	66.1
31.	Argentina	65.7
31.	Turkey	65.7
32.	Fiji	65.6
32.	Monaco	65.6
33.	Guatemala	65.4
33.	San Marino	65.4
34.	Peru	64.7
35.	Qatar	64.1
36.	Bahamas	63.8
36.	Saudi Arabia	63.8
37.	Slovenia	63.5
38.	Jordan	63.4
39.	Brunei Darussalam	63.2
40.	Vanuatu	62.9
41.	Australia	62.7
42.	United Kingdom	61.9
43.	Iceland	61.7
44.	Venezuela (Bolivarian Republic of)	61.4
45.	Greece	61.3
46.	Cuba	61.1
47.	Tuvalu	60.7
48.	Cyprus	60.6
48.	Slovakia	60.6
49.	Saint Kitts and Nevis	60.3
50.	Antigua and Barbuda	59.8
51.	Tunisia	59.2
52.	Uruguay	58.1
53.	Grenada	58.0
54.	Costa Rica	57.8
54.	Iran (Islamic Republic of)	57.8
55.	Israel	57.5
55.	Libyan Arab Jamahiriya	57.5
56.	The former Yugoslav Republic of Macedonia	57.4
57.	Syrian Arab Republic	57.2
58.	Canada	57.1
59.	Azerbaijan	56.8
60.	Switzerland	56.7
61.	Panama	56.3
62.	Saint Vincent and the Grenadines	55.7
63.	Germany	55.1
64.	Belize	54.9
65.	Morocco	54.7
66.	Colombia	54.6
66.	Mauritania	54.6
67.	Lebanon	54.3
68.	El Salvador	54.0
68.	Luxembourg	54.0
69.	Brazil	53.5
70.	Austria	53.2
70.	Paraguay	53.2
70.	Suriname	53.2
71.	Guyana	52.9
72.	Armenia	52.8
73.	Ecuador	52.6
74.	Albania	52.5
75.	Finland	52.4
76.	Mauritius	52.3
77.	Marshall Islands	51.8
78.	Russian Federation	51.7
79.	Bosnia and Herzegovina	51.0
80.	Georgia	50.8
80.	Iraq	50.8
81.	Haiti	50.6
82.	Solomon Islands	49.9
82.	Uzbekistan	49.9
83.	Botswana	49.4
83.	Honduras	49.4
84.	Portugal	49.2
85.	Zimbabwe	48.9

OVERWEIGHT – SORTED IN DESCENDING ORDER

2005 BMI ≥ 30 kg/m², Females, aged 15-100

Rank	Country	Prevalence (%)
86.	Equatorial Guinea	48.5
86.	Montenegro	48.5
86.	Serbia	48.5
86.	The former state union of Serbia/Montenegro	48.5
86.	Ukraine	48.5
87.	Democratic Republic of Timor-Leste	48.2
88.	Czech Republic	47.8
88.	Oman	47.8
88.	Swaziland	47.8
89.	Gabon	47.7
89.	Spain	47.7
90.	Maldives	47.6
91.	Hungary	47.4
91.	Republic of Moldova	47.4
92.	Bhutan	46.5
93.	Croatia	46.4
94.	Democratic People's Republic of Korea	46.2
95.	Algeria	45.6
95.	Lao People's Democratic Republic	45.6
96.	Bulgaria	45.5
96.	Turkmenistan	45.5
97.	Sweden	44.9
98.	Latvia	44.7
99.	Sierra Leone	44.5
100.	Poland	44.3
101.	Cape Verde	44.1
102.	Netherlands	44.0
103.	Kyrgyzstan	43.9
103.	Lithuania	43.9
103.	Tajikistan	43.9
104.	Republic of Korea	43.8
105.	Norway	43.4
106.	Myanmar	43.3
107.	Ireland	41.7
108.	Liberia	41.6
109.	Cameroon	41.1
110.	Belgium	40.7
111.	Romania	40.6
112.	Benin	39.1
112.	Denmark	39.1
113.	Kazakhstan	38.9
114.	Italy	38.3
115.	Malaysia	37.2
116.	Senegal	36.7
117.	Comoros	35.9
118.	Thailand	35.2
119.	France	34.7
120.	Cote d'Ivoire	34.2
121.	Estonia	33.8
122.	Angola	33.6
122.	Mali	33.6
123.	Namibia	32.6
124.	Nigeria	32.2
125.	Djibouti	31.0
126.	Togo	30.9
127.	Guinea	30.4
128.	Yemen	29.4
129.	Sudan	29.1
130.	Papua New Guinea	29.0
131.	Philippines	28.5
132.	Ghana	28.1
133.	Sao Tome and Principe	27.2
134.	United Republic of Tanzania	27.0
135.	Pakistan	25.5
136.	Mozambique	25.3
137.	Congo	25.2
138.	China	24.7
139.	Malawi	23.5
140.	Gambia	22.8
141.	Indonesia	22.7
142.	Uganda	22.2
143.	Guinea-Bissau	22.1
144.	Singapore	22.0
145.	Kenya	21.7
146.	Niger	21.3
147.	Somalia	21.1
148.	Madagascar	20.2
149.	Rwanda	20.1
150.	Chad	19.2
151.	Zambia	18.6
152.	Central African Republic	18.5
153.	Burundi	18.1
153.	Japan	18.1
154.	Afghanistan	17.4
155.	Burkina Faso	16.0
156.	India	15.2
157.	Democratic Republic of the Congo	13.3
158.	Cambodia	9.3
159.	Viet Nam	8.7
160.	Nepal	8.0
161.	Sri Lanka	5.9
162.	Eritrea	5.7
163.	Bangladesh	5.4
164.	Ethiopia	3.3

OVERWEIGHT – SORTED IN DESCENDING ORDER

2005 BMI ≥ 30 kg/m², Males, aged 15-100

Rank	Country	Prevalence (%)
1.	Nauru	96.5
2.	Cook Islands	92.6
3.	Micronesia, Federated States of	92.1
4.	Tonga	90.3
5.	Samoa	78.7
6.	Niue	78.5
7.	Greece	75.7
8.	United States	75.6
9.	Palau	74.5
10.	Kiribati	73.2
11.	Argentina	73.1
12.	Australia	72.1
13.	Malta	71.4
14.	Kuwait	69.5
15.	Venezuela (Bolivarian Republic of)	69.1
16.	New Zealand	68.7
17.	Mexico	68.4
18.	United Arab Emirates	66.9
19.	United Kingdom	65.7
20.	Canada	65.1
20.	Dominica	65.1
20.	Germany	65.1
21.	Finland	64.9
22.	Egypt	64.5
23.	Belarus	63.7
24.	Uruguay	63.6
25.	Saudi Arabia	63.1
26.	Bulgaria	62.8
27.	Chile	62.6
28.	Lithuania	62.3
29.	Croatia	61.3
30.	Montenegro	61.2
30.	Serbia	61.2
30.	The former state union of Serbia/Montenegro	61.2
31.	Austria	61.0
32.	Andorra	60.9
32.	Bahrain	60.9
33.	Barbados	59.2
33.	Cuba	59.2
34.	Monaco	59.1
35.	Iceland	59.0
36.	Trinidad and Tobago	58.9
37.	San Marino	58.8
38.	Portugal	58.5
38.	Seychelles	58.5
39.	Czech Republic	58.1
40.	Qatar	57.9
41.	Jordan	57.5
42.	Azerbaijan	57.4
43.	Albania	57.2
43.	Israel	57.2
44.	Bahamas	57.0
45.	Guatemala	56.9
46.	Bosnia and Herzegovina	56.6
47.	Colombia	56.5
48.	Brunei Darussalam	56.4
49.	Bolivia	56.3
49.	Vanuatu	56.3
50.	Slovenia	56.0
51.	Hungary	55.9
52.	Spain	55.8
53.	Norway	54.8
54.	Peru	54.6
55.	Sweden	54.5
56.	Luxembourg	54.4
57.	Switzerland	54.1
58.	Armenia	53.9
58.	Costa Rica	53.9
59.	Mongolia	53.0
60.	Nicaragua	52.9
61.	Italy	52.7
62.	Denmark	52.5
62.	Tuvalu	52.5
63.	Saint Kitts and Nevis	52.0
63.	Slovakia	52.0
64.	Belgium	51.9
65.	Cyprus	51.7
65.	Lebanon	51.7
66.	Ireland	51.5
67.	Antigua and Barbuda	51.2
68.	Estonia	50.7
68.	Poland	50.7
69.	Latvia	49.9
70.	Libyan Arab Jamahiriya	48.8
71.	Grenada	48.7
72.	Iran (Islamic Republic of)	48.5
73.	Syrian Arab Republic	48.4
74.	Turkmenistan	48.1
75.	Netherlands	48.0
76.	Turkey	47.9
77.	Brazil	47.4
78.	Dominican Republic	46.6
79.	Panama	46.5
79.	Russian Federation	46.5
80.	France	45.6
80.	Saint Vincent and the Grenadines	45.6
81.	Saint Lucia	45.5
82.	Belize	44.7
83.	Fiji	43.9
83.	Kazakhstan	43.9

OVERWEIGHT – SORTED IN DESCENDING ORDER

2005 BMI ≥ 30 kg/m², Males, aged 15-100

Rank	Country	Prevalence (%)
84.	El Salvador	43.5
85.	Oman	43.4
86.	Tunisia	42.8
87.	Suriname	42.4
88.	Paraguay	42.3
89.	Guyana	42.1
90.	Uzbekistan	42.0
91.	Ecuador	41.7
92.	Ukraine	41.2
93.	Marshall Islands	40.6
94.	Republic of Korea	40.2
95.	Iraq	40.1
96.	Jamaica	40.0
97.	South Africa	39.3
98.	Mauritius	39.0
99.	Georgia	38.9
100.	Cameroon	38.7
101.	Solomon Islands	38.2
102.	Botswana	37.8
103.	Romania	37.7
104.	Honduras	37.6
105.	Equatorial Guinea	37.5
106.	Democratic Republic of Timor-Leste	37.2
107.	The former Yugoslav Republic of Macedonia	37.1
108.	Swaziland	35.8
109.	Bhutan	35.3
110.	Republic of Moldova	34.8
111.	Kyrgyzstan	34.5
112.	Algeria	34.1
113.	China	33.1
114.	Democratic People's Republic of Korea	32.7
115.	Cape Verde	32.4
116.	Maldives	32.3
117.	Lao People's Democratic Republic	32.1
118.	Papua New Guinea	31.5
119.	Morocco	31.1
120.	Tajikistan	30.8
121.	Mauritania	30.4
122.	Ghana	30.3
123.	Liberia	29.6
124.	Myanmar	29.4
125.	Thailand	27.9
126.	Lesotho	27.5
127.	Japan	27.0
128.	Gabon	25.4
129.	Yemen	24.6
130.	Singapore	23.8
131.	Malaysia	22.7
132.	Sierra Leone	22.4
133.	Nigeria	21.9
133.	Philippines	21.9
134.	Angola	21.3
135.	Comoros	20.0
136.	Djibouti	18.9
137.	Pakistan	18.8
138.	Benin	17.9
139.	Sudan	17.2
140.	Togo	17.1
141.	India	16.8
142.	Guinea	16.5
143.	Senegal	16.1
144.	Sao Tome and Principe	15.5
145.	United Republic of Tanzania	15.4
146.	Zimbabwe	15.3
147.	Haiti	15.1
147.	Malawi	15.1
148.	Mali	14.6
149.	Madagascar	14.5
150.	Niger	13.9
151.	Cambodia	13.3
152.	Afghanistan	12.7
152.	Congo	12.7
153.	Namibia	12.3
154.	Burkina Faso	12.1
155.	Chad	12.0
156.	Cote d'Ivoire	11.6
157.	Guinea-Bissau	11.4
158.	Somalia	10.6
159.	Gambia	10.3
160.	Indonesia	9.7
161.	Mozambique	9.3
162.	Sri Lanka	8.9
163.	Nepal	8.8
164.	Burundi	7.8
164.	Ethiopia	7.8
165.	Zambia	7.5
166.	Uganda	7.4
167.	Rwanda	7.3
168.	Central African Republic	7.2
169.	Kenya	6.9
170.	Bangladesh	6.7
171.	Democratic Republic of the Congo	4.8
172.	Viet Nam	4.1
173.	Eritrea	3.1

MEAN BMI – SORTED IN DESCENDING ORDER
2005 BMI ≥ 30 kg/m², Females, aged 15-100

Rank	Country	Prevalence (%)
1.	Nauru	36.5
2.	Tonga	35.7
3.	Micronesia, Federated States of	34.7
4.	Cook Islands	34.0
5.	Niue	32.1
6.	Samoa	31.8
7.	Kuwait	31.0
8.	Palau	30.9
9.	Barbados	30.3
10.	Dominica	29.6
10.	Egypt	29.6
10.	Trinidad and Tobago	29.6
11.	Jamaica	28.8
11.	Kiribati	28.8
11.	United States	28.8
12.	United Arab Emirates	28.6
13.	Seychelles	28.4
14.	Malta	28.1
15.	Bahrain	27.9
15.	Jordan	27.9
15.	Lesotho	27.9
15.	Mexico	27.9
15.	Nicaragua	27.9
15.	Saint Lucia	27.9
15.	South Africa	27.9
16.	Belarus	27.7
16.	Bolivia	27.7
17.	Chile	27.6
17.	Fiji	27.6
17.	New Zealand	27.6
17.	Saudi Arabia	27.6
17.	Turkey	27.6
18.	Argentina	27.5
18.	Dominican Republic	27.5
19.	Mongolia	27.4
19.	Peru	27.4
20.	Andorra	27.3
20.	Guatemala	27.3
21.	Monaco	27.1
21.	Qatar	27.1
21.	San Marino	27.1
22.	Bahamas	26.9
22.	Brunei Darussalam	26.9
22.	Tunisia	26.9
23.	Australia	26.8
23.	Slovenia	26.8
23.	Vanuatu	26.8
24.	Greece	26.7
24.	United Kingdom	26.7
24.	Venezuela (Bolivarian Republic of)	26.7
25.	Cuba	26.6
25.	Iceland	26.6
26.	Iran (Islamic Republic of)	26.5
26.	Israel	26.5
26.	Tuvalu	26.5
27.	Antigua and Barbuda	26.4
27.	Azerbaijan	26.4
27.	Costa Rica	26.4
27.	Cyprus	26.4
27.	Saint Kitts and Nevis	26.4
27.	Slovakia	26.4
27.	The former Yugoslav Republic of Macedonia	26.4
28.	Uruguay	26.3
29.	Lebanon	26.2
30.	Canada	26.1
30.	Grenada	26.1
30.	Libyan Arab Jamahiriya	26.1
30.	Syrian Arab Republic	26.1
31.	Germany	26.0
32.	Austria	25.9
32.	Mauritania	25.9
32.	Panama	25.9
32.	Russian Federation	25.9
32.	Switzerland	25.9
33.	Albania	25.8
33.	Colombia	25.8
33.	Morocco	25.8
33.	Saint Vincent and the Grenadines	25.8
34.	Armenia	25.7
34.	Belize	25.7
34.	Bosnia and Herzegovina	25.7
35.	Brazil	25.6
35.	El Salvador	25.6
35.	Finland	25.6
35.	Luxembourg	25.6
36.	Guyana	25.5
36.	Paraguay	25.5
36.	Suriname	25.5
37.	Ecuador	25.4
37.	Mauritius	25.4
37.	Montenegro	25.4
37.	Serbia	25.4
37.	The former state union of Serbia/Montenegro	25.4
37.	Ukraine	25.4
37.	Uzbekistan	25.4
38.	Czech Republic	25.3
38.	Maldives	25.3
38.	Marshall Islands	25.3
39.	Georgia	25.2
39.	Haiti	25.2

OVERWEIGHT – SORTED IN DESCENDING ORDER

2005 BMI ≥ 30 kg/m², Females, aged 15-100

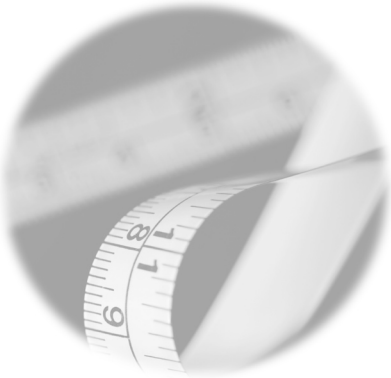
Rank	Country	Prevalence (%)
39.	Iraq	25.2
39.	Portugal	25.2
39.	Spain	25.2
40.	Botswana	25.1
40.	Honduras	25.1
40.	Hungary	25.1
40.	Solomon Islands	25.1
40.	Zimbabwe	25.1
41.	Bulgaria	25.0
41.	Croatia	25.0
42.	Democratic Republic of Timor-Leste	24.9
42.	Equatorial Guinea	24.9
42.	Gabon	24.9
42.	Oman	24.9
42.	Republic of Moldova	24.9
42.	Swaziland	24.9
42.	Turkmenistan	24.9
43.	Democratic People's Republic of Korea	24.8
43.	Latvia	24.8
43.	Poland	24.8
44.	Bhutan	24.7
44.	Kyrgyzstan	24.7
44.	Lao People's Democratic Republic	24.7
44.	Lithuania	24.7
45.	Algeria	24.6
45.	Netherlands	24.6
45.	Republic of Korea	24.6
45.	Sweden	24.6
46.	Myanmar	24.5
46.	Sierra Leone	24.5
46.	Tajikistan	24.5
47.	Cape Verde	24.4
47.	Norway	24.4
48.	Ireland	24.3
49.	Belgium	24.2
49.	Italy	24.2
49.	Romania	24.2
50.	Cameroon	24.1
50.	Liberia	24.1
51.	Denmark	24.0
51.	Kazakhstan	24.0
52.	Benin	23.8
53.	France	23.7
53.	Malaysia	23.7
54.	Senegal	23.6
55.	Comoros	23.5
55.	Estonia	23.5
55.	Thailand	23.5
56.	Cote d'Ivoire	23.4
57.	Angola	23.2
57.	Mali	23.2
57.	Namibia	23.2
58.	Nigeria	23.1
59.	Togo	23.0
60.	Djibouti	22.9
60.	Guinea	22.9
60.	Papua New Guinea	22.9
61.	China	22.8
61.	Philippines	22.8
61.	Yemen	22.8
62.	Sudan	22.7
62.	United Republic of Tanzania	22.7
63.	Ghana	22.6
64.	Congo	22.5
64.	Mozambique	22.5
64.	Sao Tome and Principe	22.5
65.	Malawi	22.4
66.	Pakistan	22.3
66.	Uganda	22.3
67.	Indonesia	22.2
67.	Kenya	22.2
67.	Singapore	22.2
68.	Gambia	22.1
68.	Rwanda	22.1
69.	Central African Republic	21.9
69.	Guinea-Bissau	21.9
69.	Japan	21.9
69.	Niger	21.9
69.	Zambia	21.9
70.	Madagascar	21.8
70.	Somalia	21.8
71.	Burundi	21.7
71.	Chad	21.7
72.	Afghanistan	21.5
73.	Burkina Faso	21.3
74.	Cambodia	21.2
75.	Democratic Republic of the Congo	21.1
75.	India	21.1
76.	Viet Nam	20.6
77.	Nepal	20.3
78.	Eritrea	20.2
78.	Sri Lanka	20.2
79.	Ethiopia	19.8
80.	Bangladesh	19.6

MEAN BMI – SORTED IN DESCENDING ORDER
2005 BMI ≥ 30 kg/m², Males, aged 15-100

Rank	Country	Prevalence (%)
1.	Nauru	35.8
2.	Cook Islands	32.8
3.	Micronesia, Federated States of	32.2
4.	Tonga	31.4
5.	Samoa	28.8
6.	Niue	28.6
7.	United States	28.4
8.	Palau	28.0
9.	Argentina	27.9
10.	Kiribati	27.8
11.	Greece	27.7
12.	Kuwait	27.5
13.	Malta	27.4
14.	Australia	27.3
15.	Mexico	27.1
15.	New Zealand	27.1
16.	United Arab Emirates	27.0
16.	Venezuela (Bolivarian Republic of)	27.0
17.	Canada	26.8
17.	United Kingdom	26.8
18.	Egypt	26.7
18.	Germany	26.7
19.	Dominica	26.6
19.	Finland	26.6
19.	Saudi Arabia	26.6
19.	Uruguay	26.6
20.	Austria	26.5
21.	Bahrain	26.4
21.	Chile	26.4
22.	Belarus	26.3
22.	Bulgaria	26.3
22.	Croatia	26.3
22.	Lithuania	26.3
22.	Montenegro	26.3
22.	Serbia	26.3
22.	The former state union of Serbia/Montenegro	26.3
23.	Andorra	26.2
24.	Barbados	26.1
24.	Czech Republic	26.1
24.	Iceland	26.1
24.	Jordan	26.1
25.	Albania	26.0
25.	Cuba	26.0
25.	Monaco	26.0
25.	Qatar	26.0
26.	Azerbaijan	25.9
26.	Guatemala	25.9
26.	Israel	25.9
26.	Portugal	25.9
26.	San Marino	25.9
26.	Seychelles	25.9
26.	Trinidad and Tobago	25.9
27.	Bahamas	25.8
27.	Bolivia	25.8
27.	Bosnia and Herzegovina	25.8
27.	Brunei Darussalam	25.8
27.	Colombia	25.8
27.	Hungary	25.8
27.	Spain	25.8
28.	Slovenia	25.7
28.	Vanuatu	25.7
29.	Luxembourg	25.6
29.	Peru	25.6
29.	Switzerland	25.6
30.	Armenia	25.5
30.	Costa Rica	25.5
30.	Italy	25.5
30.	Norway	25.5
30.	Sweden	25.5
31.	Belgium	25.4
31.	Nicaragua	25.4
32.	Cyprus	25.3
32.	Denmark	25.3
32.	Ireland	25.3
32.	Lebanon	25.3
32.	Mongolia	25.3
32.	Poland	25.3
32.	Saint Kitts and Nevis	25.3
32.	Slovakia	25.3
32.	Tuvalu	25.3
33.	Antigua and Barbuda	25.2
34.	Estonia	25.1
34.	Latvia	25.1
35.	Grenada	25.0
35.	Libyan Arab Jamahiriya	25.0
35.	Netherlands	25.0
35.	Turkey	25.0
35.	Turkmenistan	25.0
36.	Iran (Islamic Republic of)	24.9
36.	Russian Federation	24.9
36.	Syrian Arab Republic	24.9
37.	Brazil	24.8
37.	Dominican Republic	24.8
37.	Panama	24.8
38.	France	24.7
38.	Saint Vincent and the Grenadines	24.7
39.	Belize	24.6
39.	Kazakhstan	24.6
39.	Saint Lucia	24.6
40.	El Salvador	24.5

MEAN BMI – SORTED IN DESCENDING ORDER
2005 BMI ≥ 30 kg/m², Males, aged 15-100

Rank	Country	Prevalence (%)
40.	Fiji	24.5
41.	Guyana	24.4
41.	Oman	24.4
41.	Paraguay	24.4
41.	Suriname	24.4
41.	Tunisia	24.4
41.	Uzbekistan	24.4
42.	Ecuador	24.3
42.	Republic of Korea	24.3
42.	Ukraine	24.3
43.	Jamaica	24.2
43.	Marshall Islands	24.2
44.	Georgia	24.1
44.	Iraq	24.1
44.	South Africa	24.1
45.	Cameroon	24.0
45.	Mauritius	24.0
45.	Solomon Islands	24.0
46.	Botswana	23.9
46.	Honduras	23.9
46.	Romania	23.9
46.	The former Yugoslav Republic of Macedonia	23.9
47.	Democratic Republic of Timor-Leste	23.8
47.	Equatorial Guinea	23.8
47.	Swaziland	23.8
48.	China	23.7
48.	Republic of Moldova	23.7
49.	Bhutan	23.6
49.	Democratic People's Republic of Korea	23.6
49.	Kyrgyzstan	23.6
49.	Lao People's Democratic Republic	23.6
50.	Algeria	23.5
51.	Papua New Guinea	23.4
51.	Tajikistan	23.4
52.	Cape Verde	23.3
52.	Myanmar	23.3
53.	Maldives	23.2
53.	Morocco	23.2
54.	Ghana	23.1
54.	Japan	23.1
54.	Lesotho	23.1
54.	Mauritania	23.1
55.	Liberia	23.0
55.	Thailand	23.0
56.	Singapore	22.7
57.	Gabon	22.6
57.	Yemen	22.6
58.	Malaysia	22.5
58.	Philippines	22.5
59.	Nigeria	22.2
60.	Angola	22.1
60.	Cambodia	22.1
60.	Comoros	22.1
60.	Sierra Leone	22.1
61.	Pakistan	22.0
62.	Benin	21.9
63.	Djibouti	21.8
63.	Togo	21.8
63.	United Republic of Tanzania	21.8
63.	Zimbabwe	21.8
64.	Guinea	21.7
64.	Malawi	21.7
65.	Haiti	21.6
65.	India	21.6
65.	Sudan	21.6
66.	Cote d'Ivoire	21.5
66.	Mali	21.5
66.	Namibia	21.5
67.	Congo	21.4
67.	Niger	21.4
67.	Sao Tome and Principe	21.4
68.	Afghanistan	21.2
68.	Burkina Faso	21.2
68.	Chad	21.2
68.	Senegal	21.2
69.	Madagascar	21.1
69.	Mozambique	21.1
70.	Indonesia	21.0
71.	Central African Republic	20.9
71.	Rwanda	20.9
71.	Sri Lanka	20.9
71.	Uganda	20.9
71.	Zambia	20.9
72.	Gambia	20.8
72.	Guinea-Bissau	20.8
72.	Viet Nam	20.8
73.	Nepal	20.7
73.	Somalia	20.7
74.	Ethiopia	20.6
74.	Kenya	20.6
75.	Burundi	20.5
76.	Bangladesh	20.2
77.	Eritrea	20.0
78.	Democratic Republic of the Congo	19.9



Glossary

Abdominal fat: Fat (adipose tissue) that is centrally distributed between the thorax and pelvis and that induces greater health risk.

Absolute risk: The observed or calculated probability of an event in a population under study, as contrasted with the relative risk.

Aerobic exercise: A type of physical activity that includes walking, jogging, running, and dancing. Aerobic training improves the efficiency of the aerobic energy-producing systems that can improve cardiorespiratory endurance.

Age-adjusted: Summary measures of rates of morbidity or mortality in a population using statistical procedures to remove the effect of age differences in populations that are being compared. Age is probably the most important and the most common variable in determining the risk of morbidity and mortality.

Anorexiant: A drug, process, or event that leads to anorexia.

Anthropometric measurements: Measurements of human body height, weight, and size of component parts, including skinfold measurement. Used to study and compare the relative proportions under normal and abnormal conditions.

Atherogenic: Causing the formation of plaque in the lining of the arteries.

Behavior therapy: Behavior therapy constitutes those strategies, based on learning principles such as reinforcement, that provide tools for overcoming barriers to compliance with dietary therapy and/or increased physical activity.

Biliopancreatic diversion: A surgical procedure for weight loss that combines a modest amount of gastric restriction with intestinal malabsorption.

BMI: Body mass index; the body weight in kilograms divided by the height in meters squared (wt/ht2) used as a practical marker to assess obesity; often referred to as the Quetelet Index. An indicator of optimal weight for health and different from lean mass or percent body fat calculations because it only considers height and weight.

Body composition: The ratio of lean body mass (structural and functional elements in cells, body water, muscle, bone, heart, liver, kidneys, etc.) to body

fat (essential and storage) mass. Essential fat is necessary for normal physiological functioning (e.g., nerve conduction). Storage fat constitutes the body's fat reserves, the part that people try to lose.

BRL 26830A: An atypical B adrenoreceptor agonist drug that in obese rodents showed an increased metabolic rate and caused a reduction in weight by decreasing body lipid content. It is not approved as a weight loss drug by FDA.

Carbohydrates: A nutrient that supplies 4 calories/gram. They may be simple or complex. Simple carbohydrates are called sugars, and complex carbohydrates are called starch and fiber (cellulose). An organic compound—containing carbon, hydrogen, and oxygen—that is formed by photosynthesis in plants. Carbohydrates are heat producing and are classified as monosaccharides, disaccharides, or polysaccharides.

Cardiovascular disease (CVD): Any abnormal condition characterized by dysfunction of the heart and blood vessels. CVD includes atherosclerosis (especially coronary heart disease, which can lead to heart attacks), cerebrovascular disease (e.g., stroke), and hypertension (high blood pressure).

Central fat distribution: The waist circumference is an index of body fat distribution. Increasing waist circumference is accompanied by increasing frequencies of overt type 2 diabetes, dyslipidemia, hypertension, coronary heart disease, stroke, and early mortality. In the body fat patterns called android type (apple shaped) fat is deposited around the waist and upper abdominal area and appears most often in men. Abdominal body fat is thought to be associated with a rapid mobilization of fatty acids rather than resulting from other fat depots, although it remains a point of contention. If abdominal fat is indeed more active than other fat depots, it would then provide a mechanism by which we could explain (in part) the increase in blood lipid and glucose levels. The latter have been clearly associated with an increased risk for cardiovascular disease, hypertension, and type 2 diabetes. The gynoid type (pear-shaped) of body fat is usually seen in women. The fat is deposited around the hips, thighs, and buttocks, and presumably is used as energy reserve during pregnancy and lactation.

Cholecystectomy: Surgical removal of the gallbladder and gallstones, if present.

Cholecystitis: Inflammation of the gallbladder, caused primarily by gallstones. Gallbladder disease occurs most often in obese women older than 40.

Cholesterol: A soft, waxy substance manufactured by the body and used in the production of hormones, bile acid, and vitamin D and present in all parts of the body, including the nervous system, muscle, skin, liver, intestines, and heart. Blood cholesterol circulates in the bloodstream. Dietary cholesterol is found in foods of animal origin.

Cimetidine: A weight loss drug that is thought to work by suppression of gastric acid or suppression of hunger by blocking histamine H2 receptors. It is not approved by the FDA.

Cognitive behavior therapy: A system of psychotherapy based on the premise that distorted or dysfunctional thinking, which influences a person's mood or behavior, is common to all psychosocial problems. The focus of therapy is to identify the distorted thinking and to replace it with more rational, adaptive thoughts and beliefs.

Cognitive rehearsal: A technique used in cognitive behavior therapy. In a weight loss program, for example, individuals first imagine the situation that is causing temptation (such as eating a high fat food), describe the thoughts and feelings that accompany the imagined situation, and make positive self-statements about the situation (e.g., "I am feeling good about choosing a low calorie drink rather than the high fat cheese."). Then the next step is to follow the positive self-statement with an adaptive behavior (such as walking away from the buffet line to chat with a friend). Finally, individuals are encouraged to reward themselves for doing well in a difficult situation, with either positive statements or material rewards, or both. The idea is to rehearse one's thoughts and behaviors prior to experiencing the potentially difficult situation,

Cognitive restructuring: A method of identifying and replacing fear-promoting, irrational beliefs with more realistic and functional ones.

Comorbidity: Two or more diseases or conditions existing together in an individual.

Computed tomography (CT) scans: A radiographic technique for direct visualization and quantification

of fat that offers high image contrast and clear separation of fat from other soft tissues. CT can estimate total body adipose tissue volume and identify regional, subcutaneous, visceral, and other adipose tissue depots. Radiation exposure, expense, and unavailability restrict the epidemiologic use of CT.

Confounding: Extraneous variables resulting in outcome effects that obscure or exaggerate the "true" effect of an intervention.

Coronary heart disease (CHD): A type of heart disease caused by narrowing of the coronary arteries that feed the heart, which needs a constant supply of oxygen and nutrients carried by the blood in the coronary arteries. When the coronary arteries become narrowed or clogged by fat and cholesterol deposits and cannot supply enough blood to the heart, CHD results.

Cue avoidance: A stimulus control technique often used in weight loss programs in which individuals are asked to reduce their exposure to certain food cues by making a variety of changes in their habits. The rationale is to make it easier on oneself and reduce temptation by reducing contact with food cues. For example, coming home from work and feeling tired is a time when many people reach for the high fat foods if they are available. By not having the high fat foods within reach, one can avoid eating them.

Dexfenfluramine: A serotonin agonist drug used to treat obesity. FDA approval has been withdrawn.

Diabetes: A complex disorder of carbohydrate, fat, and protein metabolism that is primarily a result of relative or complete lack of insulin secretion by the beta cells of the pancreas or a result of defects of the insulin receptors.

Diastolic blood pressure: The minimum pressure that remains within the artery when the heart is at rest.

Diethylproprion: An appetite suppressant prescribed in the treatment of obesity.

Dopamine: A catecholamine neurotransmitter that is found primarily in the basal ganglia of the central nervous system. Major functions include the peripheral inhibition and excitation of certain muscles; cardiac excitation; and metabolic, endocrine and central nervous system actions.

Dual energy X-ray absortiometry (DEXA): A method used to estimate total body fat and percent of body fat. Potential disadvantages include whole body radiation and the long time required for scanning while the subject lies on a hard table.

Dyslipidemia: Disorders in the lipoprotein metabolism; classified as hypercholesterolemia, hypertriglyceridemia, combined hyperlipidemia, and low levels of high-density lipoprotein (HDL) cholesterol. All of the dyslipidemias can be primary or secondary. Both elevated levels of low-density lipoprotein (LDL) cholesterol and low levels of HDL cholesterol predispose to premature atherosclerosis.

Efficacy: The extent to which a specific intervention, procedure, regimen, or service produces a beneficial result under ideal conditions. Ideally, the determination of efficacy is based on the results of a randomized control trial.

Energy balance: Energy is the capacity of a body or a physical system for doing work. Energy balance is the state in which the total energy intake equals total energy needs.

Energy deficit: A state in which total energy intake is less than total energy need.

Ephedrine: A sympathomimetic drug that stimulates thermogenesis in laboratory animals and humans. Animal studies show that it may reduce fat content and, therefore, body weight by mechanisms that probably involve increased expenditure and reduced food intake.

Extreme obesity: A body mass index of 40 or above.

Femoxetine: A selective serotonin reuptake inhibitor drug used in obese patients for weight loss.

Fenfluramine: A serotonin agonist drug used in the treatment of obesity. FDA approval was withdrawn.

Fibrinogen: A plasma protein that is converted into fibrin by thrombin in the presence of calcium ions. Fibrin is responsible for the semisolid character of a blood clot.

Fluoxetine: An antidepressant drug used to promote weight loss whose action is mediated by highly specific

inhibition of serotonin reuptake into presynaptic neurons. Serotonin acts in the brain to alter feeding and satiety by decreasing carbohydrate intake, resulting in weight reduction.

Framingham Heart Study: Study begun in 1948 to identify constitutional, environmental, and behavioral influences on the development of cardiovascular disease. Framingham data show that increased relative weight and central obesity are associated with elevated levels of risk factors (e.g., cholesterol, blood pressure, blood glucose, uric acid), increased incidence of cardiovascular disease, and increased death rates for all causes combined.

Gallstones: Constituents in the gallbladder that are not reabsorbed, including bile salts and lipid substances such as cholesterol that become highly concentrated. They can cause severe pain (obstruction and cramps) as they move into the common bile duct. Risk factors for cholesterol gallstone formation include female gender, weight gain, overweight, high energy intake, ethnic factors (Pima Indians and Scandinavians), use of certain drugs (clofibrate, estrogens, and bile acid sequestrants), and presence of gastrointestinal disease. Gallstones sometimes develop during dieting for weight reduction. There is an increased risk for gallstones and acute gallbladder disease during severe caloric restriction.

Gastric banding: Surgery to limit the amount of food the stomach can hold by closing part of it off. A band made of special material is placed around the stomach near its upper end, creating a small pouch and a narrow passage into the larger remainder of the stomach. The small outlet delays the emptying of food from the pouch and causes a feeling of fullness.

Gastric bubble/balloon: A free-floating intragastric balloon used in the treatment of obesity.

Gastric bypass: A surgical procedure that combines the creation of small stomach pouches to restrict food intake and the construction of bypasses of the duodenum and other segments of the small intestine to cause food malabsorption. Patients generally lose two-thirds of their excess weight after two years.

Gastric exclusion: Same as gastric partitioning and Roux-en Y bypass. A small stomach pouch is created by stapling or by vertical banding to restrict food intake. A Y-shaped section of the small intestine is attached to the pouch to allow food to bypass the duodenum as well as the first portion of the jejunum.

Gastric partitioning: See gastric exclusion.

Gastroplasty: See also jejuno-ileostomy. A surgical procedure that limits the amount of food the stomach can hold by closing off part of the stomach. Food intake is restricted by creating a small pouch at the top of the stomach where the food enters from the esophagus. The pouch initially holds about 1 ounce of food and expands to 2-3 ounces with time. The pouch's lower outlet usually has a diameter of about 1/4 inch. The small outlet delays the emptying of food from the pouch and causes a feeling of fullness.

Genotype: The entire genetic makeup of an individual. The fundamental constitution of an organism in terms of its hereditary factors. A group of organisms in which each has the same hereditary characteristics.

Glucose tolerance: The power of the normal liver to absorb and store large quantities of glucose and the effectiveness of intestinal absorption of glucose. The glucose tolerance test is a metabolic test of carbohydrate tolerance that measures active insulin, a hepatic function based on the ability of the liver to absorb glucose. The test consists of ingesting 100 grams of glucose into a fasting stomach; blood sugar should return to normal in 2 to 21 hours after ingestion.

Hemoglobin: One of the fractions of glycosylated hemoglobin A1c. Glycosylated hemoglobin is formed when linkages of glucose and related monosaccharides bind to hemoglobin A and its concentration represents the average blood glucose level over the previous several weeks. HbA1c levels are used as a measure of long-term control of plasma glucose (normal, 4 to 6 percent). In controlled diabetes mellitus, the concentration of glycosylated hemoglobin A is within the normal range, but in uncontrolled cases the level may be 3 to 4 times the normal conentration. Generally, complications are substantially lower among patients with Hb levels of 7 percent or less than in patients with HbA1c levels of 9 percent or more.

Hemorrhagic stroke: A disorder involving bleeding within ischemic brain tissue. Hemorrhagic stroke occurs when blood vessels that are damaged or dead from lack of blood supply (infarcted), located within

an area of infarcted brain tissue, rupture and transform an "ischemic" stroke into a hemorrhagic stroke. Ischemia is inadequate tissue oxygenation caused by reduced blood flow; infarction is tissue death resulting from ischemia. Bleeding irritates the brain tissues, causing swelling (cerebral edema). Blood collects into a mass (hematoma). Both swelling and hematoma will compress and displace brain tissue.

Heritability: The proportion of observed variation in a particular trait that can be attributed to inherited genetic factors in contrast to environmental ones.

High-density lipoproteins (HDL): Lipoproteins that contain a small amount of cholesterol and carry cholesterol away from body cells and tissues to the liver for excretion from the body. Low-level HDL increases the risk of heart disease, so the higher the HDL level, the better. The HDL component normally contains 20 to 30 percent of total cholesterol, and HDL levels are inversely correlated with coronary heart disease risk.

Hirsutism: Presence of excessive body and facial hair, especially in women; may be present in normal adults as an expression of an ethnic characteristic or may develop in children or adults as the result of an endocrine disorder. Apert's hirsutism is caused by a virilizing disorder of adrenocortical origin. Constitutional hirsutism is mild-to-moderate hirsutism present in individuals exhibiting otherwise normal endocrine and reproductive functions; it appears to be an inheritable form of hirsutism and commonly is an expression of an ethnic characteristic. Idiopathic hirsutism is of uncertain origin in women, who may exhibit menstrual abnormalities and sterility. Some authorities believe the hirsutism reflects hypersecretion of adrenocortical androgens.

Hypercholesterolemia (high blood cholesterol): Cholesterol is the most abundant steroid in animal tissues, especially in bile and gallstones. The relationship between the intake of cholesterol and its manufacture by the body to its utilization, sequestration, or excretion from the body is called the cholesterol balance. When cholesterol accumulates, the balance is positive; when it declines, the balance is negative. In 1993, the NHLBI National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults issued an updated set of recommendations for monitoring and treatment of blood cholesterol levels. The NCEP guidelines recommended that total cholesterol levels and subfractions of high-density lipoprotein (HDL) cholesterol be measured beginning at age 20 in all adults, with subsequent periodic screenings as needed. Even in the group of patients at lowest risk for coronary heart disease (total cholesterol 200 mg/dL and HDL 35 mg/dL), the NCEP recommended that rescreening take place at least once every five years.

Hypertension: High blood pressure (i.e., abnormally high blood pressure tension involving systolic and/or diastolic levels). The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure defines hypertension as a systolic blood pressure of 140 mm Hg or greater, a diastolic blood pressure of 90 mm Hg or greater, or taking hypertensive medication. The cause may be adrenal, benign, essential, Goldblatt's, idiopathic, malignant PATE, portal, postpartum, primary, pulmonary, renal or renovascular.

Hypertriglyceridemia: An excess of triglycerides in the blood that is an autosomal dominant disorder with the phenotype of hyperlipoproteinemia, type IV. The National Cholesterol Education Program defines a high level of triglycerides as being between 400 and 1,000 mg/dL.

Incidence: The rate at which a certain event occurs (i.e., the number of new cases of a specific disease occurring during a certain period).

Insulin-dependent diabetes mellitus (type 1 diabetes): A disease characterized by high levels of blood glucose resulting from defects in insulin secretion, insulin action, or both. Autoimmune, genetic, and environmental factors are involved in the development of type I diabetes.

Ischemic stroke: A condition in which the blood supply to part of the brain is cut off. Also called "plug-type" strokes. Blocked arteries starve areas of the brain controlling sight, speech, sensation, and movement so that these functions are partially or completely lost. Ischemic stroke is the most common type of stroke, accounting for 80 percent of all strokes. Most ischemic strokes are caused by a blood clot called a thrombus, which blocks blood flow in the arteries feeding the brain, usually the carotid artery in the neck, the major vessel bringing blood

to the brain. When it becomes blocked, the risk of stroke is very high.

Jejuno-ileostomy: See gastroplasty.

J-shaped relationship: The relationship between body weight and mortality.

Lipoprotein: Protein-coated packages that carry fat and cholesterol throughout the bloodstream. There are four general classes: high-density, low-density, very low-density, and chylomicrons.

Locus/loci: A general anatomical term for a site in the body or the position of a gene on a chromosome.

Longitudinal study: Also referred to as a "cohort study" or "prospective study"; the analytic method of epidemiologic study in which subsets of a defined population can be identified who are, have been, or in the future may be exposed or not exposed, or exposed in different degrees, to a factor or factors hypothesized to influence the probability of occurrence of a given disease or other outcome. The main feature of this type of study is to observe large numbers of subjects over an extended time, with comparisons of incidence rates in groups that differ in exposure levels.

Low-calorie diet (LCD): Caloric restriction of about 800 to 1,500 calories (approximately 12 to 15 kcal/kg of body weight) per day.

Low-density lipoprotein (LDL): Lipoprotein that contains most of the cholesterol in the blood. LDL carries cholesterol to the tissues of the body, including the arteries. A high level of LDL increases the risk of heart disease. LDL typically contains 60 to 70 percent of the total serum cholesterol and both are directly correlated with CHD risk.

Lower-fat diet: An eating plan in which 30 percent or less of the day's total calories are from fat.

Macronutrients: Nutrients in the diet that are the key sources of energy, namely protein, fat, and carbohydrates.

Magnetic resonance imaging (MRI): Magnetic resonance imaging uses radio frequency waves to provide direct visualization and quantification of fat. The sharp image contrast of MRI allows clear separation of adipose tissue from surrounding nonlipid structures. Essentially the same information provided by CT is available from MRI, including total body and regional adipose tissue, subcutaneous adipose, and estimates of various visceral adipose tissue components. The advantage of MRI is its lack of ionizing radiation and hence its presumed safety in children, younger adults, and pregnant women. The minimal present use of MRI can be attributed to the expense, limited access to instrumentation, and long scanning time.

Menopause: The cessation of menstruation in the human female, which begins at about the age of 50.

Meta-analysis: Process of using statistical methods to combine the results of different studies. A frequent application is pooling the results from a set of randomized controlled trials, none of which alone is powerful enough to demonstrate statistical significance.

Mianserine: An antidepressant sometimes used in the pharmacotherapy of bulimia nervosa.

Midaxillary line: An imaginary vertical line that passes midway between the anterior and posterior axillary (armpit) folds.

Monounsaturated fat: An unsaturated fat that is found in plant foods, including olive and canola oils.

Myocardial infarction (MI): Gross necrosis of the myocardium as a result of interruption of the blood supply to the area; it is almost always caused by atherosclerosis of the coronary arteries, upon which coronary thrombosis is usually superimposed.

NHANES: National Health and Nutrition Examination Survey; conducted every 10 years by the National Center for Health Statistics to survey the dietary habits and health of U.S. residents.

Neural tube defects: These defects include problems stemming from fetal development of the spinal cord, spine, brain, and skull, and include birth defects such as spina bifida, anencephaly, and encephalocele. Neural tube defects occur early in pregnancy at about 4 to 6 weeks, usually before a woman knows she is pregnant. Many babies with neural tube de-

fects have difficulty walking and with bladder and bowel control.

Neuronal atrophy: Nerve cell death and function loss.

Obesity: The condition of having an abnormally high proportion of body fat. Defined as a body mass index (BMI) of greater than or equal to 30. Subjects are generally classified as obese when body fat content exceeds 30 percent in women and 25 percent in men. The operational definition of obesity in this document is a BMI 30.

Observational study: An epidemiologic study that does not involve any intervention, experimental or otherwise. Such a study may be one in which nature is allowed to take its course, with changes in one characteristic being studied in relation to changes in other characteristics. Analytical epidemiologic methods, such as case-control and cohort study designs, are properly called observational epidemiology because the investigator is observing without intervention other than to record, classify, count, and statistically analyze results.

Orlistat: A lipase inhibitor used for weight loss. Lipase is an enzyme found in the bowel that assists in lipid absorption by the body. Orlistat blocks this enzyme, reducing the amount of fat the body absorbs by about 30 percent. It is known colloquially as a "fat blocker." Because more oily fat is left in the bowel to be excreted, Orlistat can cause an oily anal leakage and fecal incontinence. Orlistat may not be suitable for people with bowel conditions such as irritable bowel syndrome or Crohn's disease.

Osteoarthritis: Noninflammatory degenerative joint disease occurring chiefly in older persons, characterized by degeneration of the articular cartilage, hypertrophy of bone at the margins, and changes in the synovial membrane. It is accompanied by pain and stiffness.

Overweight: An excess of body weight but not necessarily body fat; a body mass index of 25 to 29.9 kg/m2.

Peripheral regions: Other regions of the body besides the abdominal region (i.e., the gluteal-femoral area).

Pharmacotherapy: A regimen of using appetite suppressants to manage obesity by decreasing appetite or increasing the feeling of satiety. These medications decrease appetite by increasing serotonin or catecholamine—two brain chemicals that affect appetite.

Phenotype: The entire physical, biochemical, and physiological makeup of an individual as determined by his or her genes and by the environment in the broad sense.

Phentermine: An adrenergic isomeric with amphetamine, used as an anorexic; administered orally as a complex with an ion-exchange resin to produce a sustained action.

Polyunsaturated fat: An unsaturated fat found in greatest amounts in foods derived from plants, including safflower, sunflower, corn, and soybean oils.

Postprandial plasma blood glucose: Glucose tolerance test performed after ingesting food.

Prevalence: The number of events, e.g., instances of a given disease or other condition, in a given population at a designated time. When used without qualification, the term usually refers to the situation at specific point in time (point prevalence). Prevalence is a number, not a rate.

Prospective study: An epidemiologic study in which a group of individuals (a cohort), all free of a particular disease and varying in their exposure to a possible risk factor, is followed over a specific amount of time to determine the incidence rates of the disease in the exposed and unexposed groups.

Protein: A class of compounds composed of linked amino acids that contain carbon, hydrogen, nitrogen, oxygen, and sometimes other atoms in specific configurations.

Randomization: Also called random allocation. Is allocation of individuals to groups, e.g., for experimental and control regimens, by chance. Within the limits of chance variation, random allocation should make the control and experimental groups similar at the start of an investigation and ensure that personal judgment and prejudices of the investigator do not influence allocation.

Randomized clinical trial (RCT): An epidemiologic experiment in which subjects in a population are ran-

domly allocated into groups, usually called study and control groups, to receive or not to receive an experimental prevention or therapeutic product, maneuver, or intervention. The results are assessed by rigorous comparison of rates of disease, death recovery, or other appropriate outcome in the study and control groups, respectively. RCTs are generally regarded as the most scientifically rigorous method of hypothesis testing available in epidemiology.

Recessive gene: A gene that is phenotypically expressed only when homozygous.

Refractory obesity: Obesity resistant to treatment.

Relative risk: The ratio of the incidence rate of a disease among individuals exposed to a specific risk factor to the incidence rate among unexposed individuals; synonymous with risk ratio. Alternatively, the ratio of the cumulative incidence rate in the exposed to the cumulative incidence rate in the unexposed (cumulative incidence ratio). The term relative risk has also been used synonymously with odds ratio. This is because the odds ratio and relative risk approach each other if the disease is rare (5 percent of population) and the number of subjects is large.

Resting metabolic rate (RMR): RMR accounts for 65 to 75 percent of daily energy expenditure and represents the minimum energy needed to maintain all physiological cell functions in the resting state. The principal determinant of RMR is lean body mass (LBM). Obese subjects have a higher RMR in absolute terms than lean individuals, an equivalent RMR when corrected for LBM and per unit surface area, and a lower RMR when expressed per kilogram of body weight. Obese persons require more energy for any given activity because of a larger mass, but they tend to be more sedentary than lean subjects.

Risk: The probability that an event will occur. Also, a nontechnical term encompassing a variety of measures of the probability of a generally unfavorable outcome.

Roux-en-Y bypass: See gastric exclusion; the most common gastric bypass procedure.

Saturated fat: A type of fat found in greatest amounts in foods from animals, such as fatty cuts of meat, poultry with the skin, whole-milk dairy products, lard, and in some vegetable oils, including coconut, palm kernel, and palm oils. Saturated fat raises blood cholesterol more than anything else eaten. On a Step I Diet, no more than 8 to 10 percent of total calories should come from saturated fat, and in the Step II Diet, less than 7 percent of the day's total calories should come from saturated fat.

Secular trends: A relatively long-term trend in a community or country.

Serotonin: A monoamine vasoconstrictor, found in various animals from coelenterates to vertebrates, in bacteria, and in many plants. In humans, it is synthesized in the intestinal chromaffin cells or in the central or peripheral neurons and is found in high concentrations in many body tissues, including the intestinal mucosa, pineal body, and central nervous system. Produced enzymatically from tryptophan by hydroxylation and decarboxylation, serotonin has many physiologic properties (e.g., inhibits gastric secretion, stimulates smooth muscle, serves as central neurotransmitter, and is a precursor of melatonin).

Sibutramine: A drug used for the management of obesity that helps reduce food intake and is indicated for weight loss and maintenance of weight loss when used in conjunction with a reduced-calorie diet. It works to suppress the appetite primarily by inhibiting the reuptake of the neurotransmitters norepinephrine and serotonin. Side effects include dry mouth, headache, constipation, insomnia, and a slight increase in average blood pressure. In some patients it causes a higher blood pressure increase.

Sleep apnea: A serious, potentially life-threatening breathing disorder characterized by repeated cessation of breathing due to either collapse of the upper airway during sleep or absence of respiratory effort.

Social pressure: A strategy used in behavior therapy in which individuals are told that they possess the basic self-control ability to lose weight, but that coming to group meetings will strengthen their abilities. The group is asked to listen and give advice, similar to the way many self-help groups, based on social support, operate.

Stoma size: The size of a new opening created surgically between two body structures.

Stress incontinence: An involuntary loss of urine that occurs at the same time that internal abdominal

pressure is increased, such as with laughing, sneezing, coughing, or physical activity.

Stress management: A set of techniques used to help an individual cope more effectively with difficult situations in order to feel better emotionally, improve behavioral skills, and often to enhance feelings of control. Stress management may include relaxation exercises, assertiveness training, cognitive restructuring, time management, and social support. It can be delivered either on a one-to-one basis or in a group format.

Submaximal heart rate test: Used to determine the systematic use of physical activity. The submaximal work levels allow work to be increased in small increments until cardiac manifestations such as angina pain appear. This provides a more precise manipulation of workload and gives a reliable and quantitative index of a person's impairment if heart disease is detected.

Surgical procedures: See jejuno-ileostomy, gastroplasty, gastric bypass, gastric partitioning, gastric exclusion, Roux-en Y bypass and gastric bubble.

Systolic blood pressure: The maximum pressure in the artery produced as the heart contracts and blood begins to flow.

Triglyceride: A lipid carried through the blood stream to tissues. Most of the body's fat tissue is in the form of triglycerides, stored for use as energy. Triglycerides are obtained primarily from fat in foods.

Type 2 diabetes: Usually characterized by a gradual onset with minimal or no symptoms of metabolic disturbance and no requirement for exogenous insulin. The peak age of onset is 50 to 60 years. Obesity and possibly a genetic factor are usually present.

Validity: The degree to which the inferences drawn from study results, especially generalization extending beyond the study sample, are warranted when account is taken of the study methods, the representativeness of the study sample, and the nature of the population from which it is drawn.

Vertical banded gastroplasty: A surgical treatment for extreme obesity; an operation on the stomach that involves constructing a small pouch in the stomach that empties through a narrow opening into the distal stomach and duodenum.

Very low-calorie diet (VLCD): The VLCD of 800 (approximately 6-10 kcal/kg body weight) or fewer calories per day is conducted under physician supervision and monitoring and is restricted to severely obese persons.

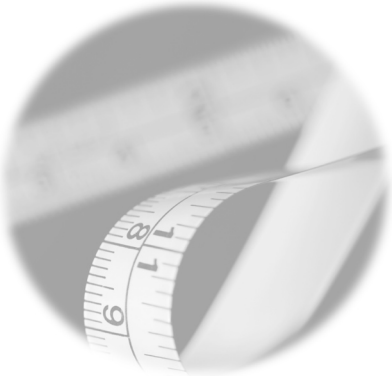
Very low-density lipoprotein (VLDL): The lipoprotein particles that initially leave the liver, carrying cholesterol and lipid. VLDLs contain 10 to 15 percent of the total serum cholesterol along with most of the triglycerides in the fasting serum; VLDLs are precursors of LDL, and some forms of VLDL, particularly VLDL remnants, appear to be atherogenic.

Visceral fat: One of the three compartments of abdominal fat. Retroperitoneal and subcutaneous are the other two compartments.

VO2 max: Maximal oxygen uptake is known as VO2 max and is the maximal capacity for oxygen consumption by the body during maximal exertion. It is used as an indicator of cardiorespiratory fitness.

Waist circumference: To define the level at which the waist circumference is measured, a bony landmark is first located and marked. The subject stands, and the technician, positioned to the right of the subject, palpates the upper hip bone to locate the right ileum. Just above the uppermost lateral border of the right ileum, a horizontal mark is drawn and then crossed with a vertical mark on the midaxillary line. The measuring tape is then placed around the trunk, at the level of the mark on the right side, making sure that it is on a level horizontal plane on all sides. The tape is then tightened slightly without compressing the skin and underlying subcutaneous tissues. The measure is recorded in centimeters to the nearest millimeter.

Waist-hip-ratio (WHR): The ratio of a person's waist circumference to hip circumference. WHR looks at the relationship between the differences in the measurements of waist and hips. Most people store body fat in two distinct ways, often called the "apple" and "pear" shapes, either the middle (apple) or the hips (pear). For most people, carrying extra weight around their middle increases health risks more than carrying extra weight around their hips or thighs. Overall obesity, however, is still of greater risk than body fat storage locations or WHR. A WHR 1.0 is in the danger zone, with risks of heart disease and other ailments connected with being overweight. For men, a ratio of .90 or less is considered safe, and for women, .80 or less.



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