THE PRAEGER INTERNATIONAL COLLECTION ON ADDICTIONS

Volume 1, 2, 3 and 4

Angela Browne-Miller

Greenwood
PUBLISHING GROUP

How to go to your page

This eBook contains four volumes. Each volume has its own page numbering scheme, consisting of a volume number and a page number, separated by a colon.

For example, to go to page 5 of Volume 1, type vol1:5 in the “page #” box at the top of the screen and click “Go.” To go to page 5 of Volume 2, type vol2:5 in the "page #" box… and so forth.

The Praeger International Collection on Addictions

Recent Titles in
Abnormal Psychology

Mental Disorders of the New Millennium, Volumes 1–3
Thomas G. Plante, editor

Bleeding to Ease the Pain: Cutting, Self-Injury, and the Adolescent
Lori G. Plante

Understanding and Treating Depression: Ways to Find Hope and Help
Rudy Nydegger

THE PRAEGER INTERNATIONAL COLLECTION ON ADDICTIONS

Volume 1
Faces of Addiction, Then and Now

Edited by Angela Browne-Miller

Praeger Perspectives

Abnormal Psychology

Thomas G. Plante, Series Editor

PRAEGER

Westport, Connecticut
London

Library of Congress Cataloging-in-Publication Data

The Praeger international collection on addictions / edited by Angela Browne-Miller.
v. cm. — (Abnormal psychology, ISSN 1554–2238)
Includes index.
Contents: v. 1. Faces of addiction, then and now—v. 2. Psychobiological profiles—v. 3. Characteristics and treatment perspectives—v. 4. Behavioral addictions from concept to compulsion.
ISBN 978–0–275–99605–5 (set : alk. paper)—ISBN 978–0–275–99607–9 (v.1 : alk. paper)—ISBN 978–0–275–99609–3 (v.2 : alk. paper)—ISBN 978–0–275–99611–6 (v.3 : alk. paper)—ISBN 978–0–275–99613–0 (v.4 : alk. paper) 1. Substance abuse. 2. Alcoholism. 3. Compulsive behavior. 4. Addicts. I. Browne Miller, Angela, 1952–
HV4998.P72 2009
616.86—dc22 2008051468

British Library Cataloguing in Publication Data is available.

Library of Congress Catalog Card Number: 2008051468
ISBN: 978–0–275–99605–5 (set)
978–0–275–99607–9 (Vol. 1)
978–0–275–99609–3 (Vol. 2)
978–0–275–99611–6 (Vol. 3)
978–0–275–99613–0 (Vol. 4)
ISSN: 1554–2238

First published in 2009

Praeger Publishers, 88 Post Road West, Westport, CT 06881
An imprint of Greenwood Publishing Group, Inc.
www.praeger.com

Printed in the United States of America

The paper used in this book complies with the Permanent Paper Standard issued by the National Information Standards Organization (Z39.48–1984).

10 9 8 7 6 5 4 3 2 1

Contents

PART II: DIMENSIONS

PART III: ASPECTS

This page intentionally left blank

Series Foreword

Tragically, most people across the globe have either struggled with a health- and relationship-damaging addiction or know someone who has. Addictions, broadly defined, have touched the lives of the majority of people in multiple cultures and locations. For centuries, numerous people have suffered with their addictions to alcohol and drugs as well as with other addictions, with often devastating outcomes. Sadly, important relationships, jobs and careers, and many lives have been lost due to the destructive power of addiction. These tragedies not only occur for those who suffer from addiction, but for their loved ones, coworkers, and community members, and for innocent victims who are perhaps in the wrong place at the wrong time when an addiction-related accident, crime, or violence occurs. The enormous cost of addiction in health care, traffic accidents, crime, violence, loss of workplace productivity, and broken families is too large to quantify. The global spread and success of organizations such as Alcoholics Anonymous (as well as related organizations such as Narcotics Anonymous, Sexoholics Anonymous, and Overeaters Anonymous) is a testament to the numerous people trying to recover from their addictions. Sadly, for every person seeking help for his addiction problem, there are likely to be many more people who never do. Clearly we need help to better understand, evaluate, treat, and cope with those who suffer from addictions.

In this remarkable four-volume set, *The Praeger International Collection on Addictions,* Angela Browne-Miller, PhD, DSW, MPH, has assembled an all-star and diverse team of leading experts from across the globe to provide a state-of-the-art understanding of the various facets of addiction. Each chapter

is written in a manner that is suitable for professionals working in the field as well as educated lay readers and those who either struggle with addictions or live or work with someone who does. What is especially remarkable about the four-volume set is its emphasis on addiction from around the globe, examining multicultural and international issues in addiction, as well as its coverage of so many multifaceted aspects of diverse addictions. For example, it certainly makes sense to cover fully addiction topics such as alcohol abuse and illegal drug use of, say, cocaine and heroin, yet chapters are also offered that examine addictions to caffeine, Internet pornography, work, television, intimate relationship abuse, and shopping. The chapters highlight biological, psychological, social, spiritual, and public health perspectives, with chapter authors who are psychologists, psychiatrists, other physicians, nurses, social workers, counselors, clergy, and other professionals. Dr. Browne-Miller is uniquely qualified to assemble this project as she is someone who has worked in the field of addiction for many years and has training in a unique blend of both the policy and the clinical sides of psychology, social work, education and public health.

The set is complete, state of the art, and highly informative and engaging. There is something for everyone interested in the field of addiction for professional or personal reasons. It is hoped that professionals and lay readers will greatly benefit from this important work and, in doing so, will find a way to improve the lives of those touched by addiction. It is my hope that both research and practice in the field of addiction will be greatly improved thanks to this set. The lives of those who either struggle with addiction or live with those who do will ultimately be improved thanks in part to this critical series. I am grateful to Dr. Browne-Miller and her assembled contributors for providing us all with such important and high-quality volumes that are now available to the public and professional communities. If only one life is saved or improved thanks to this set, it will be a great success in my view; yet I expect that many lives will ultimately be saved or greatly improved thanks to *The Praeger International Collection on Addictions*.

Thomas G. Plante, PhD, ABPP
Santa Clara University and Stanford University School of Medicine,
Praeger Series Editor, Abnormal Psychology

Preface

Angela Browne-Miller, PhD, DSW, MPH

Welcome to *The Praeger International Collection on Addictions,* addressing the insidious, pervasive, worldwide problem of human addiction. Addiction is clearly a global issue, touching every population, every nation, and every age group, people from all walks of life everywhere, directly or indirectly. Indeed, we are talking about an affliction of epic and epidemic proportions. We cannot look away. This is the health of the human species we are talking about.

The World Health Organization (WHO, 2008, p. 1) reports that "psychoactive substance use poses a significant threat to the health, social and economic fabric of families, communities and nations. The extent of worldwide psychoactive substance use is estimated at 2 billion alcohol users, 1.3 billion smokers and 185 million drug users" (p. 1). The WHO has estimated there to be at least 76.3 million persons with alcohol use disorders worldwide, and at least 15.3 million persons with drug use disorders worldwide. Alcohol use and abuse as well as the use and abuse of other psychoactive substances contributes to substantial individual and public health costs. Alcohol is but one substance playing a major role in this global addiction epidemic, but clearly there are many others, despite efforts to prevent new addictions and addicts, and to contain world drug markets (UN, 2008, p. 7).

For example, cocaine shares the stage with other abused drugs. Its prevalence is estimated to be up to 3 percent of the population in developing countries, with severe medical, psychological, social, and economic consequences including, but not limited to, the spread of infectious diseases (e.g., AIDS, hepatitis, and tuberculosis), plus crime, violence, and neonatal drug exposure.

Amphetamine-type stimulant (ATP) abuse is more widespread than cocaine abuse in at least 20 countries. Methamphetamine is presumed to lead in ATP addiction rates, with massive meth epidemics affecting several whole countries and entire regions of others. Social and public health costs of methamphetamine production and use via smoking, sniffing, inhaling, and injecting are staggering and growing in many regions. Additionally, there has been a global increase in the production, transportation, and use of opioids, especially heroin, with worldwide heroin production doubling or even tripling since the mid-1980s. Global estimates are that 13.5 million persons consume opioids, with 9.5 million of these being heroin users who face health risks including hepatitis, HIV, and death. And cocaine, meth, and heroin are just one piece of the picture.

The hotly debated drug cannabis—or the *Cannabis* family of drugs with the euphoric tetrahydrocannabinols, or THCs, including marijuana and hashish preparations—is said to be the most widely abused drug. Research is now suggesting the risk for acute health effects of long-term, chronic cannabis use, including potential impairment of cognitive development, learning, memory, recall, attention, and coordination. (Certainly the presence and extent of long-term effects of casual, of regular and of chronic use are as yet not entirely ascertained.) Both casual use of marijuana and medical use of forms of what is termed medical marijuana (e.g., dronabinol sold as Marinol, the cannabidiols, or CBDs) are subsets of all forms of cannabis use. There are legitimate therapeutic uses of this substance, and these uses make it all the more difficult to regulate marijuana drugs fairly and effectively.

We have here, and in the use of any psychoactive medication for therapeutic purposes, a gray area in which illicit and licit use overlap and can confuse many adult and youth consumers, researchers, and policy makers, among others. In the emergence (or reemergence in history, some will argue) of cannabis as medicine, we have a model for asking which, if any, abused substances may, and perhaps should, be repurposed for medicinal or treatment purposes, and how this is best done against the backdrop of the global addiction epidemic.

Regarding marijuana, we are confronted with the age-at-first-use issue, which suggests that early onset of regular cannabis use may affect not only the academic and social performance of children and teens, but also their future susceptibility to addictions. It was in the 1960s that the hotly debated label "gateway drug" was applied to marijuana, perhaps to scare off its use, and only in the decades since have we understood better what this might actually mean to us. It may not be that marijuana provides the training wheels for drug addiction, but rather that it may serve as an indicator of future use of the same or other drugs. Of course, today, with so many young people having access, and taking advantage of their access, to the whole range of psychoactive substances,

the question of which drug might be a gateway to which other drug dissolves into the fury of the countless addiction conundrums of our constantly changing times.

There is always a new, or rediscovery of an old, addiction on the horizon. There is also always a new (or rediscovered) psychoactive substance for exploratory, research, and perhaps even treatment purposes emerging (or re-emerging). Labeling all of these substances as addictive right out the gate may or may not serve science or even humanity itself. How can we be certain the approach we take will be a constructive one? With new legal (where licensed for development and experimentation) and illegal (where not being utilized under protection of law) so-called designer drugs emerging at a staggering rate, we must admit that we cannot know what is coming, nor whether the new compound will be addictive, or popular, or of medicinal value, or even accessible. We can only imagine what the brave new world of chemistry will continue to bring and whether any benefits can be made available without accompanying risks and detriments.

Moreover, the desire to explore and achieve various altered states of consciousness in religious, spiritual, ritual, and perhaps even treatment settings, is unfolding into debates about rights (Browne-Miller, 1989, pp. 258–260). When there is no demonstrated risk to self or others, we have to ask ourselves whether this right should be protected, especially in circumstances of traditional uses for religious purposes. Again, this dilemma arises against the backdrop of the global and runaway epidemic of substance addiction. How do we balance pressures from opposite directions (freedom protecting the right to use versus control to stop injury and costs of using), when these pressures are not balancing themselves?

Also against the backdrop of global addiction levels, is the massive level of addiction to legal drugs, many of which are heavily marketed to consumers. The legal drug tobacco is said to be the substance causing the most damage globally, with at least one-third of the global population smoking. While smoking rates may be dropping in some countries, the reverse is true globally. As just one of its effects, smoking accounts for some 90 percent of all lung cancer in men and 70 percent of all lung cancer in women. And yet tobacco use is overwhelmingly viewed as being "the single most avoidable cause of disease, disability and death" in the United States (CDC, 2008, p. 2).

And perhaps nothing here has touched so many lives as the regularly consumed, legal drug caffeine, perhaps because coffee drinking is considered so very normal and acceptable, even necessary, in everyday life. However, we must ask whether there is a level of caffeine use that is abuse—or perhaps self-abuse. Surely we do not want to throw caffeine use onto this list of substance abuses

and addictions. Still, a collection on addiction would not be complete without at least touching on this matter, and therefore we do address caffeine herein.

And then there are also the addictions to prescription drugs (such as Vicodin, Percocet, OxyContin, and Darvon), which we find increasing rapidly and already a worldwide phenomenon, with the most commonly abused prescription drugs being opiates. The U.S. National Institute of Mental Health characterizes prescription drug addiction as the second most common illegal use of drugs in the United States, second only to marijuana.

We must also note that unusual, virtually invisible psychoactive substances are working their way into our everyday lives. Household and workplace products contain many volatile substances, exposure to which can be not only damaging, but also intoxicating, and perhaps addicting. Although this domain of substance use and abuse is not specifically addressed herein, we must acknowledge the severe and perhaps largely unmeasured effects of this domain of even routine, legal substance use as well as unintentional and intentional abuse.

So as not to exclude nonsubstance addictions in this overview of addiction today, the fourth volume in this collection on addiction reminds us that work, television, shopping, food (with its particularly difficult-to-call-addiction nature), intimate partner relationship, gambling, Internet, and even pornography addictions make their marks in our lives, either indirectly or directly. These behavioral, nondrug addictions, which occur alone and co-occur with each other, also do co-occur with substance uses, abuses, and addictions. Every human being is in some way affected by the prevalence of behavioral addictions, either directly or indirectly. The study of behavioral addictions teaches us a great deal about addiction itself.

All this suggests the picture of an addiction-prone and largely chemically dependent human species. And this is just the tip of the iceberg. With this truly incomplete laundry list of human fallibilities—or better stated, perhaps, human *vulnerabilities*—this four-volume collection on addiction is truly that: a collection of perspectives, approaches, and findings. Each chapter is a snapshot of the work and thinking taking place in many fields of addiction. Contributors to this collection work with addiction on the various social, philosophical, psychological, spiritual, policy, political, economic, biological, and even cellular levels, all places where this thing we call "addiction" lives. Certainly this collection would have to comprise hundreds of volumes, rather than the four that it does, to address addiction in all its iterations.

Here we give voice to a diverse cross section of perspectives on addiction. This is in no way an exhaustive cross section (of either perspectives or addictions); rather, this collection suggests the diversity of perspectives, theories, practices, and types of addiction in the field—or better stated, *fields*—of addiction. The four volumes of this collection represent the voices of those who have

graciously and even bravely stepped forward from their numerous countries and arenas of work to contribute their ideas, research, and experiences. Certainly there are many others out there, many other aspects of addiction, and many other drugs and objects of addiction not addressed in these volumes.

This work is divided into four volumes, with the first three addressing addictions to substances and the fourth addressing behaviors that show characteristics of addiction. Volume 1, *Faces of Addiction, Then and Now,* offers a sampling of the depth and breadth of addiction today and in the past; volume 2, *Psychobiological Profiles,* surveys some of the interlinked psychological and biological aspects of addiction; volume 3, *Characteristics and Treatment Perspectives,* samples the range of addiction treatment perspectives and approaches; and volume 4, *Behavioral Addictions from Concept to Compulsion,* gives the reader a glimpse of behavioral addictions other than substance addictions.

Readers will observe that the content of these volumes is indeed diverse and in no way represents any one view or theory of addiction. There are many other voices out there who must also be heard, and only in the interest of time and space are we stopping here, at these volumes. The content of these volumes in no way expresses the opinion of this editor, nor of this publisher, regarding what is right, best proven, or even most en vogue in the addiction world; rather, this *International Collection on Addictions* seeks a display of, a sampling of, the diversity of effort to quell the detrimental effects of addiction on individuals, families, communities, societies, economies, and international relations; on ecologies; and in fact, on the human population of planet Earth.

REFERENCES

Browne-Miller, Angela. (2008, reprint of 1989). Synaptic rights. In *Working dazed: Why drugs pervade the workplace and what can be done about it* (pp. 258–260). Houston, Texas: Questia Media. Also available at http://www.questia.com/library/book/working-dazed-why-drugs-pervade-the-workplace-and-what-can-be-done-about-it-by-angela-browne-miller.jsp

Centers for Disease Control (CDC). (2008). *Targeting tobacco use: The nation's leading cause of preventable death* (March 2008 revision). Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

United Nations (UN). (2008). 2008 world drug report. UNODC (United Nations Office on Drug Control), Vienna, Austria: Vienna International Centre, Full report in English available at http://www.unodc.org/documents/wdr/WDR_2008/WDR_2008_eng_web.pdf

World Health Organization, Management of Substance Abuse, Facts and Figures. (2008). "The Global Burden," 2008, (Accessed 9-01-08), p. 1 http://www.who.int/substance_abuse/facts/global_burden/en/index.html. **See Also:** http://www.who.int/substance_abuse/facts/en/index.html

This page intentionally left blank

Introduction to Volume 1: The Many Faces of Addiction

Angela Browne-Miller, PhD, DSW, MPH

This is *Faces of Addiction, Then and Now,* volume 1 of *The Praeger International Collection on Addictions*. Here we bring together a collective understanding of addiction as we know it today. Studying this worldwide epidemic of addiction in all its forms, we see a troubling tide sweeping the planet and hope to turn it back.

We begin part 1 of this volume, "Perspectives," with the public health perspective on addiction as set forth by Elaine R. Feeney, PhD, RN, of the Foundation for Addictions Nursing in Baltimore, Maryland, United States, in chapter 1, "Addiction as a Public Health Issue." Feeney carefully establishes this public health perspective on addiction, one which we cannot deny and cannot afford to ignore. After all, your health is my health, and vice versa. Population and public health and individual health are inextricably linked. While perhaps dauntingly intricate to measure, and seemingly subtle, almost invisible, this linkage is profound. Feeney urges that we press for a strong public health approach to addiction: "By conceptualizing the substance abuse/dependence problem in this country as a public health issue, we might be able to identify possible solutions from the public health perspective as well. Clearly the individual focus has failed, and we cannot afford to continue to allocate resources to initiatives that are unsuccessful. It is my belief that where the problem can be viewed in a population context, the solutions can also be found."

We then turn to chapter 2, "Cross-Cultural Issues in Substance Addiction Research Including Black Americans and Latinos," contributed by Julia F. Hastings, PhD, MSW, from the School of Social Welfare, University of

California Berkeley, in Berkeley, California, United States. Hastings points out that social science researchers are under great pressure to address racial and cultural diversity; however, "culturally informed" research is not easily accomplished. Addressing outcomes according to race or ethnicity does not ensure cultural understanding of addiction. It remains to be seen whether social science research, and research on addiction, will fully recognize and define the differences between race and culture. Hastings notes that, while it is often said that "African Americans, Caribbean Blacks, and Latinos suffer more adverse health conditions from substance addictions than the White population," we must ask what this statement is actually telling us. Hastings comments, "Though identifying many health conditions and their racial differences is important, many other factors associated with substance addiction can impinge on higher substance addiction problems. True, other factors include poverty status, education, unemployment, families in crisis, psychiatric problems, little or no insurance coverage, and public program assistance limitations. Yet the one factor that many researchers and government documents focus on is race and ethnicity. Identifying with a group is important to all people. A group defines who we are and how we envision our place in the world. Above all, we all belong to *many* groups." In working to analyze and treat addiction, we cannot afford to overlook the limitations of a focus on race and ethnicity to the exclusion of an effective operationalization of *culture* itself—its norms, beliefs, and distributions of power and roles.

Next, we look at "Race, Ethnicity, and Early U.S. Drug Policy" in chapter 3, by Sean R. Hogan, PhD, MSW, of the Department of Social Work, California State University Fullerton, in Fullerton, California, United States. Perceptions and definitions of addictions have evolved over time, with dramatic shifts in theories and in biases occurring in the twentieth century (and even now in the early twenty-first century). Hogan provides important examples, one being that opium use was once associated with Chinese immigrants and that this prejudice was actually associated with the first antidrug legislation in the late 1800s. Long after, Hogan writes, "a significant shift in public perception and, consequently, the strategies designed to address substance use occurred when American society began to distinguish 'recreational,' or nonmedical, drug use from that of drug use initiated and prescribed by physicians." Here we see that public perception and public policy regarding substance use can be fueled by prejudice and bias, a tendency which may still to this day be having lasting effects on the formation of drug policy. This report (albeit perhaps inadvertently) brings to the table additional issues and questions: once recreational use is set apart from prescribed use, we are forced to decide whether any recreational use is acceptable, and, if so, what use by whom, and in what amount? Boundaries between categories of use are particularly difficult to establish and maintain. We are also compelled

to note that legal recreational use is perhaps immune to the moral imperative. And here we have it, the slippery matter of moral imperative—what forms this imperative always being the elephant in the room, the presence we hardly see.

In chapter 4, Martha Romero Mendoza, PhD, of the National Institute of Psychiatry, in Mexico D.F., Mexico, contributes her insights into "Gender Inequalities and Inequities among Women with Substance Abuse Problems." Romero Mendoza explains that "health inequalities" are avoidable and unnecessary. Yet, with the majority of addiction research being based on male experience, and based on a gendered society, we are in need of balancing perceptions and understandings of gender differences and similarities. We must be cautious as to the effects of our research paradigms and consequent findings, for, in Romero Mendoza's words, "social inequality and the bias that arises from it at the moment of the research are perpetuated through social hierarchy preservation, that is, power relationships." Here we have the added dimension of gender—the very necessary dimension of addiction research and treatment.

Following this brief look at matters of diversity, we step back in chapter 5 to look at "Ethical Theory and Addiction," by Ann N. Dapice, PhD, of T. K. Wolf Inc., a Native American–focused addiction treatment program in Tulsa, Oklahoma, United States. Dapice emphasizes that the higher levels of moral reasoning must be developed in both addiction treatment professionals and persons who are addicted once into their recoveries. (I emphasize here that Dapice includes professionals.). Dapice also notes that moral development is "hijacked" by addictive substances and behaviors, something we must pay closer attention to in our work. Connecting a few dots here, we might add that if moral development is impaired by addictive substance use, then how responsible is the user from a moral standpoint? Can sound moral judgments be made by an impaired moral function?

Chapter 6, "The Religious and Theological Roots of Alcoholics Anonymous," by Rev. Linda Mercadante, PhD, of the Methodist Theological School in Delaware, Ohio, United States, delves deeply into the theological roots of the Alcoholics Anonymous (AA) 12-Step Recovery Program. Mercadante unearths, via a colorful historical and analytical review, a key dichotomy in the disease versus sin or moral failing views of addiction to alcohol. Mercadante places the 12-Step AA approach in the historical context of the Christian evangelistic Oxford Group, which hit its peak in the 1920s and 1930s; AA founder Bill Wilson was once a member. These roots continue to inform the 12-Step method of addiction recovery, including the focus on sin and conversion in recovery from addiction. Clearly, recognizing what informs any approach to addiction, seeing the evolution of any philosophy of addiction and its recovery, is essential as we move forward in working to respond to addiction.

Casting another light on this matter of addiction, part 1 of this volume concludes through chapter 7 with a report by Jemel P. Aguilar, PhD, MSW, from the School of Social Work, University of Texas at Austin, in Austin, Texas, United States, titled "Adolescent Drug Sellers and Distributors." Aguilar urges us to be aware that, in his words, "with the growing numbers of youth entering into the juvenile justice system because of drug-related crimes, it is important that researchers and practitioners have a better understanding of the dynamics of youth drug selling and distribution so that more effective interventions can be developed to prevent youth from becoming active drug sellers and distributors and/or stem youth involvement once they enter into this economy." Only this awareness can open our eyes to truly understanding what is going on, and our arms to helping to do something about it.

Part 2, "Dimensions," begins with a look at "Historical Aspects of Alcohol Use in India: Role of Culture and Gender," chapter 8, contributed by Meera Vaswani, PhD, Atul Ambekar, MD, and Ramandeep Pattanayak, MD, all from the National Drug Dependence Treatment Centre, Department of Psychiatry, All India Institute of Medical Sciences, in New Delhi, Delhi, India. This chapter looks at the historical and cultural context of alcohol use in India, reaching as far back as 3300 B.C. and building through the eras into present-day alcohol use issues. The authors of this chapter note that alcohol use among women remains understudied in Indian literature; cultural attitudes toward women's alcohol use may be preventing them from reporting alcohol use and abuse. Again, the importance of gender-sensitive addiction research is highlighted.

We then, in chapter 9, turn to "Alcohol and Drug Abuse in Malaysia," contributed by See Ching Mey, PhD, MEd, from the School of Educational Studies, Universiti Sains Malaysia in Pulau Pinang, Malaysia, and Cecilia A. Essau, PhD, MA, HBA, of the School of Human and Life Sciences, Roehampton University, Whitelands College, in London, England. Mey and Essau report that the data they have reviewed indicate that Malaysia is the 10th largest consumer of alcohol in the world. Marketing approaches hard sell alcohol to Malaysians via several means not allowed in many Western nations, including particularly heavy forms of ethnic targeting loaded with health claims, such as the 1980 Guinness Stout campaign including the slogan "Guinness Stout is good for you." Additionally, Malaysia has identified drug use and trafficking as a security problem, one which is increasing, with an overwhelming majority of all drug addicts in Malaysia being in the 20- to 39-year-old age group.

Next, in chapter 10, we look at "Alcohol Use and Binge Drinking in Cyprus," by Xenia Anastassiou-Hadjicharalambous, PhD, of the Department of Psychology, University of Nicosia, in Nicosia, Cyprus, and Cecilia A. Essau, PhD, MA, HBA, and George Georgiou, PhD, both at the School of Human and Life Sciences, Roehampton University, London. Archaeological evidence

suggests that the grapevine has been cultivated and wine has been made in Cyprus for at least 6,000 years. The authors of this chapter report that their ongoing study indicates that today, about one-third of young adults in Cyprus binge drink on a regular basis, with a wide range of social and personal problems attending this behavior. Directions for future research are suggested.

From here, Anna Bokszczanin, PhD, from the University of Opole (Uniwersytet Opolski), in Opole, Poland, and Cecilia A. Essau, PhD, MA, HBA, and Jean O'Callaghan, PhD, from the School of Human and Life Sciences at Roehampton University, London, share with us "Alcohol Use and Abuse in Poland" as chapter 11. In recent decades, alcohol use and abuse have increased in Poland, parallel to the privatization of the market there, after years of markedly low alcohol consumption. Directions for treatment and prevention are reviewed and hope is offered. Questions come into focus—troubling questions. Can we say that the privatization of the market is largely responsible for what has happened in Poland? If yes, is this effect consistent across cultures around the world? If not, what other factors were present and are relevant? For example, do specific characteristics of the regulation of alcohol advertising and distribution make a difference?

Chapter 12, "Alcohol Consumption and Binge Drinking in the United Kingdom," is contributed by Cecilia A. Essau, PhD, MA, HBA, Changiz Mohiyeddini, MD, PhD, and Diane Bray, PhD, of the School of Human and Life Sciences at Roehampton University, London. As in many societies, alcohol use is found to be an acceptable part of social interaction in the United Kingdom. Of great concern is the high cost of prevalent binge drinking in the United Kingdom. The authors of this chapter note that there is a need to balance policies that promote public health with those that promote economic interests. Is this a tension that cannot be worked through to support both sides of this dilemma?

"Alcohol Use and Abuse in Germany," by Changiz Mohiyeddini, MD, PhD, Cecilia A. Essau, PhD, MA, HBA, and Regina Pauli, BSc, PhD, all of the School of Human and Life Sciences at Roehampton University, London, is chapter 13. Here we read more about modern-day alcohol use in developed countries, seeing the great cost of this use and abuse, and at the same time the great investment the alcoholic beverage industry has in the consumption of alcohol. For example, in Germany, where the legal age for alcohol consumption is 16 (typical of many European and other nations, and unlike the United States' general legal age of 21), television advertising aimed at young people utilizes bright colors, youthful music, and high action to attract young people and new consumers of alcohol. This is taking place even though alcohol advertising targeting young people in the European Union (EU) is controlled by means of rules *suggested* in the European Council Recommendation that was adopted on June 5, 2001, by the EU Health Ministry.

We conclude part 2 with chapter 14, "From British India to the Taliban: Lessons from the History of the Heroin Market," by Kathryn Meyer, PhD, from Wright State University's Department of History in Dayton, Ohio, United States. Meyer reports that the plan of Pino Arlacchi (who became head of the United Nations International Drug Control Program in 1997) was to eradicate narcotics worldwide. Arlacchi traveled to Afghanistan as part of a global plan to replace drug crops with alternative industries, yet poppy production continued in Taliban-controlled areas. Meyer weaves the story of a complex political and economic scenario surrounding heroin. Never is the problem of addiction free of politics and economics.

Part 3, "Aspects," includes a collection of chapters sampling aspects of addiction and some of its many faces, beginning with chapter 15, "Youth Tobacco Use: Its Health Effects, Trends in Smoking Rates, and Reasons Why Kids Use Tobacco," by Clete Snell, PhD, at the School of Juvenile Justice and Psychology, Prairie View A&M University in College Station, Texas, United States. Snell reports that the life stage of adolescents is a strong predictor of cigarette and tobacco use, with the transition between elementary and high school being the period of greatest risk for the onset of youth tobacco use. What may drive youth tobacco use is the young person's risk-taking behavior, desire for stress reduction, desire to look older, longing to relieve boredom, and peer pressure.

Next we hear from Va Nee L. Van Vleck, PhD, of the Department of Economics in the College of Social Sciences at California State University Fresno, in Fresno, California, United States, and Thomas K. Greenfield, PhD, at the Alcohol Research Group, in Emeryville California, United States, who report on their study in chapter 16, "Brewing Trouble: Some Employment Implications of Regular Malt Liquor Beer Consumption." Van Vleck and Greenfield examine the association between regular use of malt liquor and poor employment conditions, including unemployment. Van Vleck and Greenfield also note that patterns of alcohol dependence are similar to those of chronic diseases, the latter of which they remind us are "value-neutral." The so-called blame for the physical illness is far less than the blame for the addiction, although addiction can also be a physical illness and surely is a physical—where *physical* includes the brain and other parts of the body—illness.

We then hear, in chapter 17, from David T. Courtwright, PhD, from the University of North Florida, in Jacksonville, Florida, United States, in his chapter titled "The Roads to H: The Emergence of the American Heroin Complex, 1898–1956." Here Courtwright reviews the history of heroin in the United States as a model of the impact of novel psychoactive drugs. A new drug promises a range of therapeutic usages and then is debated per these claims. Eventually, the drug slips out of control and into the hands of the general

population. And then, predictably, government must intervene to control the drug. This is the way heroin emerged into modern times, as a product of Bayer Pharmaceutical Company in the late 1800s. In the decades that followed, a significant underground and underworld interest in heroin developed. By the 1920s, the U.S. federal government had outlawed the manufacture of heroin, listing it as the first official Schedule I drug, meaning that it was totally prohibited, except for research purposes. Having "escaped the realm of healing for that of self-indulgence," heroin has made its way through time and into the veins of far too many, leading the way for other novel drugs.

A consequent, or perhaps parallel, history emerges in the history of the treatment drug for heroin addiction: methadone. Herbert D. Kleber, MD, of the Department of Psychiatry and the Division on Substance Abuse, Columbia University, in New York, New York, United States, reviews "Methadone: The Drug, the Treatment, the Controversy" in chapter 18. Weaving the history and politics of methadone, we see that some things work perfectly but not at all well. Here methadone maintenance as the antidote to heroin addiction is both lauded and revealed for its shortcomings and attending controversies. And of course, here the administration of one drug is done to curb response to another. Is one addiction purposefully replacing another?

Next we consider another addictive drug, methamphetamine, as no sampling of work on addiction can overlook this drug in our times. Chapter 19 considers this drug in "Prevalence of Use and Manufacture of Methamphetamine in the United States: Is the Sky Falling, or Is It Not Really a Problem?" by Herbert C. Covey, PhD, of the Adams County Social Services Department and the College of Continuing Education, University of Colorado at Boulder, Boulder, Colorado, United States. Covey reminds us that unlike other illicit drugs, meth does not have to be imported into the country and can actually be homemade. We have all heard of the meth lab, and this is where it all happens.

Mary F. Holley, MD, of the national organization Mothers against Methamphetamine in Arab, Alabama, and the Alabama State Attorney General's Methamphetamine Task Force, Alabama, United States, adds to this discussion of methamphetamine (also known as "crystal meth" and "ice") use, offering chapter 20, "Interaction between Methamphetamine Use and HIV Infection." Holley points out that methamphetamine use is associated with higher rates of sexually transmitted diseases (STDs), specifically the most deadly STD, HIV. Where we go from here, once we see what meth can do and is doing to many cultures of the world, is perhaps the question of the hour.

The 20 fascinating and illuminating chapters following this introduction open *The Praeger International Collection on Addictions* and compose volume 1, *Faces of Addiction, Then and Now*. These chapters offer a striking and disturbing cross section of addiction today and set the stage for the volumes that follow.

Of great concern is the degree to which human addiction—a condition, an affliction, a malady—is a read on the state of the species. There is hope, however. We are responding as a species, around the globe, on all levels, in all professions, from all religions, world views, from all philosophical standpoints, from all walks of life. We have called ourselves to action and have heard the call. We know that no single theory, policy, philosophy, or religious approach alone can solve this problem, let alone define it.

To this end, the International Substance Abuse and Addiction Coalition (ISAAC) authored its October 2006 *Red Sea Declaration*, which ISAAC has been generous enough to share within this publication. We include this declaration here, in full recognition that there are other statements by numerous other organizations that at some future point we may also have the opportunity to share.

RED SEA DECLARATION

International Substance Abuse & Addiction Coalition (ISAAC)—October 2006

Introduction

The scourge of addiction to alcohol and other drugs is one of the most serious and urgent issues facing humanity today. Tens of millions of men, women and children are either directly or indirectly affected by addiction. The health, social, criminal justice and economic costs of addiction are colossal and well documented. Addiction causes millions of deaths worldwide each year, destroying human potential for millions of others. Drug misuse is one of the main driving factors behind the HIV/AIDS pandemic.

ISAAC's International Network

We represent more than 500 service providers, such as rehabilitation centres, prevention programmes, training providers and street agencies in over 55 countries around the world. Our programmes have been providing help and support to millions of drug addicts and their families over many decades. We have substantial experience in all fields of addiction.

Call to Governments

We call upon the Governments of the world to:

1. **Urgently recognise and address the physical, emotional, social and spiritual roots of addiction.** As faith-based/Christian organisations we recognise the significance of spirituality, in addiction prevention and rehabilitation. This has been demonstrated by scientific studies and we encourage governments to acknowledge this.
2. **Urgently develop, implement and support policies aimed at preventing drug misuse, recognising that demand reduction is more effective than supply reduction.** Communities have the right to be protected from drugs, including the right to information concerning drugs. Prevention goes hand-in-hand with rehabilitation and indeed prevention without rehabilitation is ineffective. Education, life skills development, participatory activities and care and support for young people with the aim of encouraging a drug-free lifestyle are crucial. Criminal justice systems can support, but will never replace, such policies.
3. **Recognise that drug misuse adversely affects all members of the community and vulnerable members, including women and children.** We stress the need for increased access to treatment for women as well as adequate care for their children and to stop all forms of violence and abuse in this context. We also confirm that key populations in the context of drug misuse, including sex workers and street children, should have specially

designated outreach programmes. We emphasise the importance of treating them with compassion and dignity.

4. **Resist all attempts to weaken, or even abolish, the United Nations Conventions on Drugs, which aim to control and prevent drug misuse.** Addicts have the right to rehabilitation and to be treated as patients. However, legalising currently illicit drugs will cause major damage to health, communities and society in general. Legalisation will greatly increase the burden to public health.
5. **Ensure that harm-reduction is never understood as merely reducing the harm to society.** These interventions should put the client at the centre of the care and support systems. It should always offer abstinence as the desirable and realistic option, and provide all means to achieve this for those who choose this option.

Conclusion

- Misuse of alcohol and other drugs is already of epidemic scale in many parts of the world. If we fail to fight the scourge of addiction and the drug trade with all available means, drug misuse will soon be out of control worldwide and the fight against drugs lost. Lack of radical action now will result in devastation for generations to come.
- In our programmes we have seen hope restored, freedom found, and transformation in the lives of addicts, their families and communities worldwide. We want to share this hope with you. Together, we can create societies able to meet the challenges of drug abuse.

Source: This ISAAC Red Sea Declaration is included here with the permission of David Partington, General Secretary, ISAAC. The ISAAC Red Sea Declaration was formulated and unanimously agreed by 160 delegates from over 30 countries at the ISAAC Congress in Egypt, in 2006.

Part I

PERSPECTIVES

This page intentionally left blank

Chapter **1**

Addiction as a Public Health Issue

Elaine R. Feeney, PhD, RN

Addictions and public health: on the surface, they seem only tangentially and linearly related. Certainly if substance dependence is prevalent, one can anticipate consequences for public health such as HIV/AIDS, tuberculosis, sexually transmitted diseases (STDs), other infectious diseases, and so on. However, a much closer parallel and a much stronger relationship exists between addictions and public health. In fact, a case can be made that substance dependence is primarily a public health issue.

For the purposes of this discussion, addictions will be referred to by the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (American Psychiatric Association [APA], 2000), category of *substance dependence*, the continued use of substances of abuse (alcohol, tobacco, and other drugs) despite negative physical, mental, or social consequences of such use, accompanied by signs and symptoms of tolerance (progressive increase of the amount of the substance needed to achieve the desired effect) or withdrawal (symptoms of withdrawal are substance-specific). In standard conceptualizations of substance dependence, the individual is the client/patient, and most of the interventions are directed toward that individual. Adjunctive treatment may also be given to the family, usually in response to the disruption caused by the individual client's substance dependence and other behavior. Public health chooses to look at disease state, treatment, and prevention in another way.

WHAT IS PUBLIC HEALTH?

According to the American Public Health Association (2008),

> Public Health is the practice of preventing disease and promoting good health within groups of people, from small communities to entire countries. Public Health professionals rely on policy and research strategies to understand issues such as infant mortality and chronic disease in particular populations. (para. 5)

Public health is a science dedicated to the study and treatment of populations. In public health, interventions are directed toward groups such as neighborhoods, communities, and populations. Therefore the major difference between public health and standard health/medical care is that in public health, the individual client/patient is not the issue of concern. Despite the seeming focus on the individual, as can be seen in public health clinics such as infant immunization clinics, tuberculosis clinics, HIV/AIDS clinics, and so on, it is the general public that is the beneficiary. In these instances, the public good is achieved by ministering to the individual. In immunizing children, or treating people with tuberculosis or HIV/AIDS, the aim is to prevent the spread of the disease and improve the health of the entire community. Therefore public health professionals view the community, not the individual, as the client.

WHAT DOES PUBLIC HEALTH DO?

The activities of public health are best described by the 10 Essential Services of Public Health:

1. Monitor health status to identify community health problems
2. Diagnose and investigate health problems and health hazards in the community
3. Inform, educate, and empower people about health issues
4. Mobilize community partnerships to identify and solve health problems
5. Develop policies and plans that support individual and community health efforts
6. Enforce laws and regulations that protect health and ensure safety
7. Link people to needed personal health services and assure the provision of health care when otherwise unavailable
8. Assure a competent public health and personal health care workforce
9. Evaluate effectiveness, accessibility, and quality of personal and population-based health services
10. Research for new insights and innovative solutions to health problems. (Centers for Disease Control and Prevention [CDC], 2008b, para. 2)

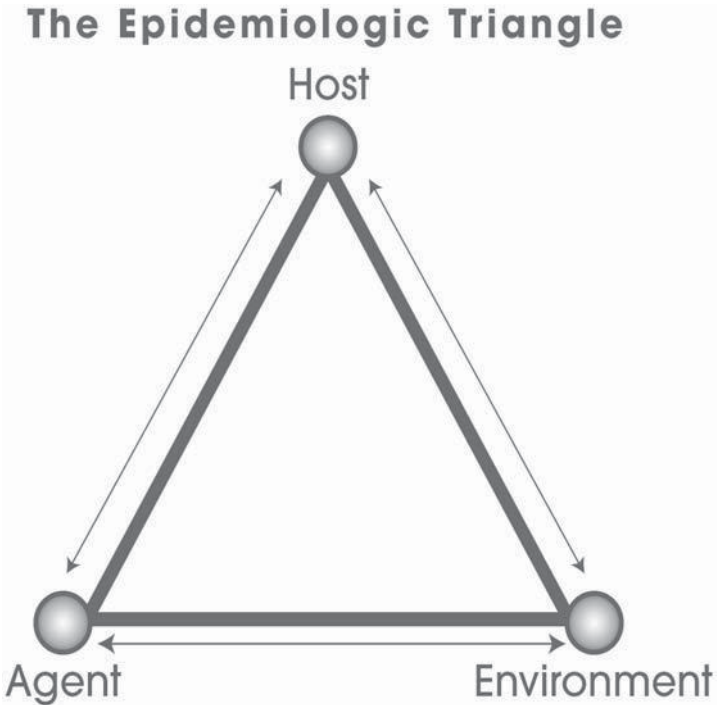

FIGURE 1.1 The Public Health Model prevention diagram. (From Centers for Disease Control and Prevention [2008a].)

As in all of health care, it seems those in need of support, empowerment, and education are those populations that are least informed and least able to advocate for themselves, and those groups that have been underserved by the traditional health care system. These populations are traditionally made up of minority racial and ethnic groups such as Asians, African Americans, Hispanics, and Native Americans. Because of economic and social disadvantages, as a group, these minorities have poorer health status, experience more difficult access to care, comprise a larger proportion of the uninsured, and receive poorer quality of care (Agency for Healthcare Research and Quality, 2005; Institute of Medicine, 2002). These are just the groups targeted by public health initiatives and policies.

THE PUBLIC HEALTH MODEL

The Public Health Model consists of three elements. In this model, all three elements must be present and interacting for disease to occur. The elements of

the Public Health Model (also known as the *epidemiological triangle*; see Figure 1.1) are the host, the agent, and the environment. The original purpose of the epidemiological triangle was to explain the necessary factors for infectious diseases to occur. However, it can also be used to understand noninfectious diseases, including substance dependence.

The Agent

This is the element that causes the disease and is frequently called the *causative factor* or *causative agent*. In the case of substance dependence, the agent refers to the substance (alcohol, cocaine, heroin, etc.). In substance use disorders, some agents facilitate dependence more quickly than others. For example, many people who use nicotine, crack cocaine, or methamphetamine quickly become addicted. Alcohol, on the other hand, is not, in itself, universally addicting. Only 10 percent of the adults in the United States who drink alcohol demonstrate signs of alcohol dependence; 90 percent of the adults in the United States who drink alcohol have no difficulty.

The Host

The host is the individual harboring the disease; in the case of substance dependence, he is the alcoholic or addict. Factors impacting the host include age, sex, gender, marital status, occupation, place in the family, race, genetic predisposition, age of first exposure to substances of abuse, health behaviors (such as sedentary lifestyle), personality factors (which influence the course of illness and tendency to seek care), social status, and educational status.

The Environment

The environment comprises those external factors that cause or allow disease to occur. Environmental factors have been studied more than host or agent factors and are frequently the targets of public health interventions. They include socioeconomic status, educational environment, economic and political organization, social values, customs, receptivity to new ideas, degree of social integration of social classes (mobility, isolation, alienation), natural disasters, presence of substances of abuse in proximity to the individual, ease of obtaining substances, prevalence of substance abuse in the community, law enforcement, and so on.

The host, agent, and environment must have certain factors present to allow for the interaction of the elements and the development of a pathological state.

HOW DO PUBLIC HEALTH PROFESSIONALS DECIDE WHAT NEEDS TO BE DONE?

Since it is the purpose of public health to intervene at the population level, these interventions are conceptualized differently from standard interventions with the individual client/patient. The choice of which of these interventions is conducted depends on the level of risk or impairment of the group in question. The types of interventions are categorized as levels of prevention.

Levels of Prevention

There are three levels of prevention: primary, secondary, and tertiary. These categories are separate and distinct, addressing separate and distinct health concerns with specific interventions.

Primary Prevention

Primary prevention is the lowest, most basic level of prevention in public health. Its aim is the total prevention of disease or health problems in a population that is free from a disease, but at risk for it. This is accomplished by making the individuals or groups less susceptible to disease, or reducing exposure to the disease through health promotion and protective measures. This conceptualization can be used for any disease. For example, clients who do not have measles can benefit from the primary prevention intervention of measles vaccine administration; clients without osteoporosis can benefit from the primary prevention measure of taking calcium and vitamin D supplements and engaging in weight-bearing exercise. Hand washing and prohibition of sharing eating utensils can prevent spread of the influenza virus.

Clients who are not substance-dependent, but are at risk, can benefit from a number of primary prevention measures, including alcohol, tobacco, and other drug education; provision of appropriate health care; job training; counseling; violence prevention; recreational facilities; opportunities to explore cultural arts; adequate child care; after-school/summer programs; community/economic development; employment assistance/job training; family support; mentoring; school-based initiatives; and youth development for disadvantaged children, youth, and families.

Secondary Prevention

This middle level of prevention involves the early detection and prompt treatment of a disease or health problem. The intervention is designed to cure, slow the progression, or prevent serious complications of the disease or

health problem. When infectious diseases are addressed, secondary prevention activities reverse communicability. Examples of secondary prevention include administration of antibiotics for communicable diseases, wearing of orthotics for plantar faciitis, aspirin and exercise for arthritis, ingesting electrolyte drinks for severe diarrhea, and so on. In terms of substance dependence, the choice between secondary and tertiary prevention activities might be a tough call. In some cases, it may be a matter of the difference between substance abuse and dependence (see APA, 2000). Before substance abuse becomes full-blown dependence, it may be possible to disrupt the pattern of substance use to a degree that prevents progression to dependence through educational sessions addressing potential substance dependence and a technique called *screening brief intervention, referral, and treatment* (SBIRT; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001; Fleming & Manwell, 1999; Substance Abuse and Mental Health Services Administration [SAMHSA], 2008c).

Tertiary Prevention

Tertiary prevention refers to activities designed to limit disability and promote rehabilitation by maximizing use of person's residual capacity. This category refers to the most sophisticated, labor-intensive, and individualized interventions: cardiac surgery and rehabilitation for clients with heart disease; chemotherapy for cancer patients; physical medicine and rehabilitation for stroke victims; and drug detoxification, rehabilitation, and use of medication deterrents to alcohol or drug use, and so on, for substance-dependent clients.

Formulation of Policy

Public health professionals have a strong responsibility to help formulate policy that guides primary, secondary, and tertiary prevention. The policies developed and implemented dictate everything from the administration of infant vaccinations to the treatment of drug-resistant tuberculosis and HIV/AIDS. Policies governing substances of abuse fall under this rubric as well. Policy formation usually requires collaborative efforts on the part of a number of community advocacy groups. Public health frequently partners with other groups (Mothers against Drinking and Driving, Gay and Lesbian Alliance, etc.) to achieve common goals.

An example of collaborative efforts working to formulate public health policy is occurring in the state of Maryland. The state legislature has voted to tax fruity alcohol drinks (called *alcopops*) in the same manner in which beer is taxed, as opposed to taxing them as hard liquor. In addition to the tax issue, the controversy seems to center on where the product will be sold.

If the alcopops are taxed like beer, they can be sold in convenience stores, rather than having their sale restricted to liquor stores (like hard liquor). If sold in convenience stores, the alcopops would be more available to underage drinkers, those who prefer the taste of sweet drinks to begin with (Coalition on Alcopops & Youth, 2006). A coalition of advocates consisting of the American Public Health Association, the Maryland Association of County Health Officials, representatives of Advocates for Highway and Auto Safety, and the Maryland Emergency Nurses Association brought pressure on the governor to delay signing the bill (Olson, 2008). While it appeared for a time that the governor would veto the bill, other pressures were brought to bear, resulting in the passage of the bill and allowing alcopops to be taxed (and sold) in a manner similar to beer.

Advocates working in other states have had better success: Utah, Maine, and, most recently, California have passed legislation that will ensure that alcopops are taxed in the same manner as other distilled spirits, thus making them less available to potential underage drinkers (Marin Institute, 2008).

Another example of collaborative efforts working to formulate public health policy is noted in a recent press release from SAMHSA (2008d). It is reported that over 15 percent of people operating a motor vehicle did so under the influence of alcohol at least once in the past year. The report indicated that this prevalence differs from state to state, with the lowest (9.5%) in Utah and the highest (26.4%) in Wisconsin. North Dakota and Minnesota also reported that close to 25 percent of drivers age 18 and up were impaired while driving at least once in the past year. This information may assist public health administrators and legislators to focus primary prevention of impaired driving initiatives on the geographical areas where the problem is most prevalent.

An Exemplar of a Challenge to Public Health Initiatives and Policies: Methamphetamine

The manufacture and use of methamphetamine is a public health problem that challenges every aspect of public health science. In addition to the elements of the epidemiological triangle interacting to cause disease when meth is the agent, the user is the host, and the environment is conducive to such use, the meth has profound detrimental effects on the social, physical, and mental health of the environment in which it is manufactured and used. This threatens the health of the user and others in proximity to the site of the manufacture. Because of the nature of the agent, communities are in very real danger of experiencing serious social and environmental harm.

Meth, also known as crank, crystal, glass, ice, rock, and by other names, is a strong, long-lasting stimulant that is administered by injection, smoking, snorting, or oral preparations. Although the effect of meth lasts longer than that of cocaine, the crash that follows use is as or more uncomfortable for the user. Therefore more meth is ingested, leading to rapid development of tolerance and addiction. The impact of meth on the body is documented in the literature (for thorough treatment, see Society for Public Health Education, 2002).

The aggregate focus of public health is challenged by the use and manufacture of methamphetamine. Because methamphetamine has been relatively easy to manufacture, many small labs have been found in the homes of the users and sellers of this substance. Aside from the detrimental impact on the user, there is an even more serious impact that the manufacturing process has on the environment.

Some of the caustic chemicals required to manufacture meth include pool acid/muratic acid, lye, acetone, brake fluid, brake cleaner, iodine crystals, lithium metal/lithium batteries, lighter fluid, drain cleaners (e.g., Drano or Liquid Fire), cold medicine containing pseudoephedrine or ephedrine, ethyl ether (in engine starting fluid), anhydrous ammonia (stored in propane tanks or coolers), sodium metal, and red phosphorous. When authorities find a meth lab, they often find children who have been neglected and abused. These children have also been exposed to meth, often have detectable meth levels in their blood, and have been exposed to the chemicals required for the drug's manufacture and the toxic substances produced during the manufacture of the drug. In addition, a meth laboratory generates five to six pounds of hazardous waste for each pound of meth produced. Since some so-called superlabs are capable of producing 10 pounds of meth during a single manufacturing cycle (National Drug Intelligence Center [NDIC], 2006), 50 to 60 pounds of hazardous waste are generated as well. Because those who manufacture meth are usually not aware of or concerned with the hazardous nature of the by-products, they frequently discard the toxic substances into the public sewer systems, streams, rivers, backyards, or public places. In addition, the hazardous by-products are frequently released into the immediate environment by the process of manufacture, with fumes permeating furnishings, walls, carpeting, floors, and so forth. Decontamination of the environment is a costly, lengthy, and complicated procedure that may or may not be effective in returning the location of the lab to a safe, inhabitable environment (Illinois Department of Public Health, 2008). Therefore exposure to caustic and toxic chemicals becomes a threat to those living in the home where the substance is made; those entering the home (such as guests, first responders, firefighters, etc.); the environment around the site of manufacture; and the areas in proximity to polluted ground, water, and air.

Reports from 22 geographical areas in the United States deemed by the Department of Justice to be representative of meth use indicate that the overall use of the drug has declined but remains a serious threat to public health across the country (NDIC, 2006). The report indicates that the number of methamphetamine labs west of the Mississippi has decreased sharply, but the number increased dramatically in Florida, Ohio, Michigan, and Pennsylvania. In addition, there is an increase of imported purer methamphetamine from Mexico and a more than expected increase in the use of the drug by youth, women, and Hispanics.

Because of the success of the collaborative relationship between the public health departments, Department of Justice, and other advocates and organizations in decreasing the overall prevalence of meth manufacture and use, there are plans to reduce the federal funding for antimeth activities. This plan is creating fear among officials in the states to be most affected by this decrease in funding. The fear is that as services are no longer available to control manufacture and use of meth, there will be a resurgence of these and related problems such as an increase in the numbers of addicts, children in foster care, prisoners, and crimes (Scharnberg, 2008).

WHAT ABOUT THE MONEY?

Economic Impact of Substance Abuse and Dependence

The Office of National Drug Control Policy (ONDCP; 2006) reports that in 2002, the economic burden to the United States of substance abuse/dependence was $180.8 billion. This figure represents loss in productivity due to industrial and occupational error, absenteeism, criminal justice, health care costs, and so on. In addition, according to the NDIC (2006), a division of the U.S. Department of Justice, the average cost to clean up a methamphetamine production laboratory is $1,900. Since an average of 9,777 methamphetamine laboratory seizures were reported to NCLSS *each year* between and including 2002 and 2004, the economic impact of this activity alone is an additional $37 million (NDIC, 2006).

Problems with all substances of abuse persist to this degree, despite the 2008 national budget of $13.7 billion in support of prevention, treatment, and international economic activities directed toward eradication of the drug problem in this country (ONDCP, 2008). The question is not whether enough money is being spent to correct the problem; rather, one must question whether the funds are being allocated properly. When all the expense is considered, perhaps less individual focus and more aggregate focus is required; the individual focus is not working.

The primary, secondary, and tertiary levels of prevention of disease range from the least expensive to the most costly, with primary prevention being the least expensive. There are funds available in the form of grants to develop programs mostly for underserved populations to increase the level of health for these communities of need. These grants can be categorized as funds available depending on the planned level of prevention.

Grants for Primary Prevention Strategies

There are a number of grants available for community leaders and researchers to conduct primary prevention interventions and study their effectiveness. Some are nonresearch grants that fund implementation of programs that have proven to be effective.

The Harry and Jeanette Weinberg Foundation has made available to organizations that provide direct services to the poor grants of up to $100,000 over two years (Harry & Jeanette Weinberg Foundation, 2008). Some grants have a more specific focus. Safe and Drug-Free Schools and Communities Programs for Native Hawaiians are two grants with specific populations in mind. The Programs for Native Hawaiians consist of two U.S. Department of Education awards totaling $579,518 designed to go to organizations serving and benefiting Native Hawaiians. The funds are to be used for the purpose of creating, developing, and managing interventions geared toward (among other things) primary prevention of use of and dependence on illegal substances by the indigenous Hawaiian population. For the purpose of this grant, a native Hawaiian is defined as any individual whose ancestors were natives, prior to 1778, of the area that now composes the state of Hawaii ("Safe and Drug-Free," 2003). This specificity of target population addresses the important cultural influences that must be considered to achieve the purpose of any specific-population-based intervention.

Grants for Secondary Prevention Strategies

Because 29 percent of those with substance abuse problems will not contact a substance abuse treatment professional directly, perhaps because they do not realize they have a problem, the U.S. Substance Abuse and Mental Health Services Administration (SAMHSA) is providing $3.75 million to medical schools to develop and implement training programs to teach medical residents the techniques of SBIRT. It is hoped that by utilizing the techniques of SBIRT, many of those 29 percent will be identified and treated in time to prevent long-term disability or greater social problems (SAMHSA, 2008b).

Grants for Tertiary Prevention Strategies

In an attempt to limit the long-term disability of alcohol and tobacco use on the fetuses by pregnant women, and make for better outcomes of pregnancy, the Centers for Disease Control and Prevention (CDC/P) has set aside funds to initiate six networks that demonstrate effectiveness and leadership with smoking abstinence and cessation, and abstinence from ingesting alcohol among women of childbearing age (National Networks for Tobacco Control and Prevention, 2006). It is through these interventions directed toward the host that the population of female alcohol consumers of child-bearing age, among others, and their children will benefit.

One population targeted for tertiary prevention interventions has been identified as the underage drinker. The surgeon general of the United States asserted that a comprehensive effort on the part of citizens and the community geared toward prevention of underage drinking will be required to eliminate this problem. A cultural change will be required to realize this goal, and an unusual degree of cooperation and consistency of approach will be needed among community organizations such as local and state government, the criminal justice system, education, business, religious and/or fraternal organizations, civic or volunteer organizations, health care professionals, media, parents, and youth (U.S. Public Health Service, 2007). To this end, the U.S. Public Health Service is awarding 80 grants for up to $50,000 each year as part of the Sober Truth on Preventing Underage Drinking Act Grants (SAMHSA, 2008b). These grants are awarded to communities to strengthen the environmental support of efforts to prevent underage drinking (environment element in the epidemiological triangle). Recipients of these grants are encouraged to solicit funds from other sources for interventions that target the host element in the epidemiological triangle. It is understood that the agent in question here is alcohol.

"Reclaiming Futures: Communities Helping Teens Overcome Drugs, Alcohol and Crime" (Robert Wood Johnson Foundation, 2008) is another grant to be awarded for tertiary prevention activities. These grants will be awarded to communities to help them implement a new program that has proven to be effective in working with substance-abusing or substance-dependent adolescents who have had trouble with the criminal justice system. This program has changed the approach taken when targeting juveniles who are substance abusers or substance-dependent in 10 locations throughout the country. The purpose of this grant is to disseminate the findings of research that has been conducted supporting the effectiveness of these new approaches. It is hoped that the techniques will be utilized in communities in addition to the

10 already engaged in the program. This will be carried out through sharing resources, improving data collection and use, and continuing development and promotion of new standards of practice.

Tertiary prevention is not limited to adolescents. The Center for Substance Abuse Prevention (CSAP) has issued a request for applications for "Grants to Expand Substance Abuse Treatment in Adult Criminal Justice Populations" (SAMHSA, 2008a). Adults who are substance abusers or substance-dependent and involved with the criminal justice system are clearly a specific population with specific needs. Seven grants of up to $400,000 each will be awarded for a period of up to three years to provide treatment for substance-dependent inmates with the hope that recidivism will diminish.

Written documents such as this frequently identify more problems than they solve, but by conceptualizing the substance abuse/dependence problem in this country as a public health issue, we might be able to identify possible solutions from the public health perspective as well. Clearly the individual focus has failed, and we cannot afford to continue to allocate resources to initiatives that are unsuccessful. It is my belief that where the problem can be viewed in a population context, solutions can also be found.

REFERENCES

Agency for Healthcare Research and Quality. (2005). *National healthcare disparities report* (AHRQ Publication No. 06-0017). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Arlington, VA: Author.

American Public Health Association. (2008). *Fact sheet: What is public health?* Retrieved March 18, 2008, from http://www.apha.org/NR/rdonlyres/C57478B8-8682-4347-8DDF-A1E24E82B919/0/what_is_PH_May1_Final.pdf

Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., & Monteiro, M. G. (2001). *AUDIT, the alcohol use disorders identification test: Guidelines for use in primary care* (2nd ed.). Geneva, Switzerland: World Health Organization.

Centers for Disease Control and Prevention. (2008a). *The public health model prevention diagram.* Retrieved April 15, 2008, from Centers for Disease Control and Prevention Web site: http://www.cdc.gov/nceh/ehs/EHSNet/images/EpiTriangle.jpg

Centers for Disease Control and Prevention. (2008b). *Ten essential services of public health.* Retrieved March 30, 2008, from Centers for Disease Control and Prevention Web site: http://www.cdc.gov/od/ocphp/nphpsp/EssentialPHServices.htm

Coalition on Alcopops & Youth. (2006, March 10). *Media advisory: California teens see drinking alcopops as fun, easy, cool.* Retrieved June 20, 2008, from http://www.marininstitute.org/take_action/alcopops/03–10NewsRelease.org

Fleming, M. F., & Maxwell, L. B. (1999). Brief intervention in primary care settings: A primary treatment method for at-risk, problem, and dependent drinkers. *Alcohol Research and Health, 23*(2), 128–138.

Harry and Jeanette Weinberg Foundation. (2008). *Grants.* Retrieved April 17, 2008, from http://www.hjweinbergfoundation.org

Illinois Department of Public Health. (2008). *Guidelines for cleaning up former methamphetamine labs.* Retrieved April 29, 2008, from http://www.idph.state.il.us/envhealth/factsheets/meth-cleanup.htm

Institute of Medicine. (2002). *Unequal treatment: Confronting racial and ethnic disparities in health care.* Retrieved April 2, 2008, from http://www.iom.edu/CMS/3740/4475.aspx

Marin Institute. (2008, May 23). *Maryland governor backs big alcohol at the expense of youth.* Retrieved June 20, 2008, from http://www.marininstitute.org/alcopops/maryland_press_release.htm

National Drug Intelligence Center. (2006). *National drug threat assessment 2006.* Retrieved April 14, 2008, from http://www.usdoj.gov/ndic/pubs11/18862/meth.htm

National Networks for Tobacco Control and Prevention. (2006). Retrieved April 20, 2008, from http://www.tobaccopreventionnetworks.org/site/c.ksJPKXPFJpH/b.2613539/k.9C6A/National_Networks.htm.

Office of National Drug Control Policy. (2006). *National drug control strategy FY 2009 budget summary, February 2008.* Retrieved March 15, 2008, from http://www.whitehousedrugpolicy.gov/publications/policy/09budget/index.html

Olson, B. (2008, April 25). O'Malley delays "alcopops" bill. *Baltimore Sun.* Retrieved April 26, 2008, from http://www.baltimoresun.com

Robert Wood Johnson Foundation. (2008). *Reclaiming futures: Communities helping teens overcome drugs, alcohol and crime.* Retrieved April 1, 2008, from http://www.reclaimingfutures.org

"Safe and drug-free schools programs for native Hawaiians grant." (2003). *Federal Register, 68*, 40257–40259.

Scharnberg, K. (2008, March 4). Officials fear meth surge as funding dries up. *Chicago Tribune.* Retrieved November 18, 2008, from http://pqasb.pqarchiver.com/chicagotribune/access/1438759581.html?dids=1438759581:1438759581&FMT=ABS&FMTS=ABS:FT&type=current&date=Mar+4%2C+2008&author=Kirsten+Scharnberg&pub=Chicago+Tribune&edition=&startpage=4&desc=Officials+fear+surge+in+meth+as+funding+dries+up

Society for Public Health Education. (2002). *Helping communities combat clandestine methamphetamine laboratories.* Washington, DC: Author.

Substance Abuse and Mental Health Services Administration. (2008a). *Grants to expand substance abuse treatment in adult criminal justice populations.* Retrieved March 23, 2008, from http://www.samhsa.gov/Grants/2008/ti_08_012.aspx

Substance Abuse and Mental Health Services Administration. (2008b, March 10). SAMHSA accepting applications for FY 2008 sober truth on preventing underage drinking act (STOP Act) grants. *SAMHSA Bulletin.* Retrieved

April 22, 2008, from http://www.samhsa.gov/newsroom/advisories/0803105601.aspx

Substance Abuse and Mental Health Services Administration. (2008c, March 5). SAMHSA to provide $3.75 million to train medical residents in SBIRT (screening, brief intervention, referral and treatment) medical residency program techniques. *SAMHSA Bulletin*. Retrieved March 12, 2008, from http://www.samhsa.gov/newsroom/advisories/0803041505.aspx

Substance Abuse and Mental Health Services Administration. (2008d). *State estimates of persons aged 18 or older driving under the influence of alcohol or illicit drugs*. Retrieved May 1, 2008, from http://oas.samhsa.gov/2k8/stateDUI/stateDUI.htm

U.S. Public Health Service. (2007). *The surgeon general's call to action to prevent and reduce underage drinking*. Retrieved March 1, 2008, from U.S. Department of Health and Human Services Web site: http://www.surgeongeneral.gov/topics/underagedrinking/programs.html

Chapter **2**

Cross-Cultural Issues in Substance Addiction Research Including Black Americans and Latinos

Julia F. Hastings, PhD, MSW

Social science researchers face growing pressure to address racial and cultural diversity issues more explicitly as communities expand in population and clients enter social service agencies with increasingly complex substance addictions. Starting from the position that most researchers want to address the complex issues associated with culturally informed research, outcomes often reflect lists of dos and don'ts based on race or ethnic group. While recording outcomes in this fashion may attend to diversity issues, the practice of identifying issues by racial or ethnic group, unfortunately, rarely enhances the cultural understanding of addictions or advances knowledge. However, understanding the difference between race and culture remains an objective for researchers.

Substance abuse refers to a pattern of substance use leading to significant impairment in functioning (American Psychiatric Association [APA], 1994). The *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (*DSM-IV*; APA, 1994) further defines *dependence* as a person with a substance use history that includes substance abuse, continuation of use despite related problems, increase in tolerance, and withdrawal symptoms. Obvious, unpleasant physical symptoms are easier to define and recognize than psychological or behavioral manifestations. Under constant debate is the issue of divergent thresholds because a clinical diagnosis of an alcohol and/or drug problem is usually based on moral judgment. Though researchers and clinicians may argue on the definitional accuracy and reliability of the definitions to measure differences in use, abuse, and dependency, neither professional group can ignore that disparities in substance addiction dominate the American public.

The critical issue for contemporary social scientists is to examine whether symptoms can be reliably recognized not only as part of research collaboration to address the World Health Organization's (WHO) focus on universality of psychiatric problems and the reliability of structured diagnostic instruments. The current diagnosis system is far from perfect, yet perhaps, with the development of *DSM-V*, a more useful section dealing with culture will be included. By relegating the cultural component of diagnosis to an appendix, the major aspect of what makes persons different from each other in their presentation of addiction remains omitted from general diagnostic research and arguments. To understand the existing disparities in addiction, researchers must be able to understand more clearly how race, ethnicity, culture, and the meaning attributed to various addictions are valued and recognized among the increasing diverse populations.

It is clear that population growth among racial and ethnic groups in the United States is rapidly increasing. The Census Bureau's Web site extols the significant growth of the population such that by 2050, the majority population is projected to comprise persons from racial and ethnic groups. Research findings, proposed studies, and public health interventions will need to appropriately adjust to the changes naturally occurring. The former director of the National Institute on Drug Abuse (as quoted in Primm, Brown, Primm, & Friedman, 2006) pointed out that "significant gaps in knowledge about the effects of drug abuse and addiction in minority populations" exist (p. 322). These gaps in knowledge will be reflected in this chapter, and many will find frustration with existing data collected on substance abuse where primary efforts are to determine use in the last year, past month, or more recent time periods. For epidemiologists, the information remains quite useful. For social researchers and practitioners, the derived information holds little clinical utility, especially when race and ethnicity, combined with culture and other demographic characteristics, are studied.

The purpose of this chapter is to identify for substance addiction researchers some of the cultural dimensions that may be relevant to understanding cross-cultural patterns of use, misuse, and abstinence for black Americans and Latinos residing in the United States. Both populations are growing due to immigration and birth; however, substance addictions are operationally defined in research—use, misuse, or abstinence—and understanding the manifestation of different patterns, trajectories, and consequences across different cultures and ethnicities remains an important goal (De La Rosa, Holleran, Rugh, & MacMaster, 2005). Researchers need to pay attention to the type of methodology chosen to evaluate the potential impact of culture (for a contemporary discussion of the use of culture, see Johnson, 2007). Though Johnson's

work discusses culture in more international terms, this chapter focuses on the black diaspora (both African American and Caribbean) and Latinos in the United States because these groups traditionally have been researched very little in substance addiction studies and have been portrayed in the literature as having alcohol and drug use patterns that show contradictions. The chapter discusses the black and Latino populations, moves on to the prevalence of substance addictions for both groups, and then covers the dimensions of culture as defined in the literature. Afterward, the chapter examines how research enterprises using a mixed-method approach might improve our understanding of the etiology of substance addiction, and finally, the chapter concludes with understudied research areas related to African Americans and Latinos.

THE U.S. BLACK AMERICAN POPULATION

The 2000 Census reported that of the entire U.S. population, blacks represented 36.4 million, or 12.9 percent. Of this number, 12.3 percent, or 34.7 million people, reported themselves as being black or African American (McKinnon, 2001; U.S. Census Bureau, 2000a); the remaining 1.7 million reported their race as being West Indian. According to the U.S. Census Bureau (2000b), the countries that represent the West Indies are the Bahamas, Barbados, Belize, the British West Indies, the Dutch West Indies, Haiti, Jamaica, Trinidad, and Tobago. In this chapter, the black population includes persons of Caribbean descent. Latinos who reported their race as black, either alone or in combination with one or more other races, are not included in the numbers for African Americans. The 2000 Census reported that an overwhelming majority of the African American population is non-Hispanic, at 98 percent American-born. Those who are foreign-born constitute the remaining percentage of the black population in the United States (McKinnon, 2001; U.S. Census Bureau, 2000a). McKinnon (2001) also reported that between 1990 and 2000, the total African American population increased by 21.5 percent (or 6.4 million).

The majority of African Americans (54%) live in the southern part of the United States, and 19% live in the Midwest, 18% in the Northeast, and 10% in the West. New York City and Chicago are the cities where the largest black populations are located (McKinnon, 2001). One-third (31.3%) of blacks are under 18 years of age, whereas a higher percentage, 38.8 percent, of Caribbean Blacks are under 18 years of age (McKinnon, 2001). Caribbean Blacks have larger family sizes, with four persons, than African Americans, whose families average three persons. Along with the increasing diversity of the black population in the United States, research exploring economic and psychosocial factors

is needed to understand true prevalence rates and how the transitioning population affects substance addiction rates, treatment programs, and prevention strategies.

THE U.S. LATINO POPULATION

Data from the 2000 Census indicate that 35.5 million Latinos, or 12 percent of the U.S. population, live in the United States. The 2000 Census also indicates that during the 1990s, the Latino population increased by 58 percent, from 22.4 million in 1990 to 35.3 million (Therrien & Ramirez, 2000). Guzmán (2001) reports that the Latino population increases at a rate five times faster than any other ethnic group. People of Mexican decent compose the largest Latino population subgroup (Therrien & Ramirez, 2000), followed by persons from South and Central America. Immigrants increased the United States population by 13.2 percent, the majority immigrating from South and Central American countries.

California, Florida, New York, and Texas are states where the majority of all Latinos live (Guzmán, 2001). Almost 39 percent of the Latino population is foreign-born, and 35 percent are under the age of 18 (Guzmán, 2001). Over one-quarter of the Latino population lives below the poverty line (Therrien & Ramirez, 2000). Despite the diversity within the Latino population in the United States, much of the research exploring the economic and psychosocial factors for Latino substance addiction rates has not accounted for inconsistent rates between groups or within ethnic groups. Therefore research findings on Latinos might lead to misunderstandings about the severity of and factors influencing substance addiction among this population.

PREVALENCE OF SUBSTANCE ADDICTION AMONG AFRICAN AMERICANS, CARIBBEAN BLACKS, AND LATINOS

According to the National Comorbidity Survey, from 1992, and the National Comorbidity Survey–Replication (NCS-R), from 2003, few differences in prevalence rates exist for adult alcohol use, tobacco smoking, use of psychoactive drugs outside the prescribed purpose, marijuana, and cocaine (Degenhardt, Chiu, Sampson, Kessler, & Anthony, 2007; Kessler et al., 2004; Kessler et al., 1994). Modest decreases were reported for tobacco smoking, from 76 percent to 74 percent, and for marijuana use, from 46 percent to 43 percent. On the basis of the published work, it is difficult to ascertain racial and ethnic differ-

ences. The national data from the National Survey on Drug Use and Health (NSDUH), however, tell a different story.

Research studies using the NSDUH data document substance addiction problems within various Latino and black populations. In the last decade, rates of dependency on alcohol and illicit drugs increased (Substance Abuse and Mental Health Services Administration, 2007) for Latinos and African Americans. No data were available on Caribbean Blacks or subgroups of the Latino population. Variations within these groups likely exist. To date, the data suggest that African Americans abuse illicit drugs at the same or lower prevalence rates than whites. Illicit drugs refer to marijuana/hashish, cocaine (including crack), inhalants, hallucinogens, heroin, or prescription-type drugs used nonmedically. Marijuana use and nonmedical use of prescription-type drugs are highlighted because these were the most common illicit drug use behaviors reported in the NSDUH for combined data in 2004 and 2005. What is unknown are the methodological issues associated with using samples nonrepresentative to the current population to quantify disparities. Nevertheless, emerging from this research are the findings displayed in Table 2.1.

Overall, respondents 12 or older (an estimated 19.4 million persons) used an illicit drug in the past month, with 6.0 percent (14.6 million persons) using marijuana and 2.6 percent (6.2 million persons) using a prescription-type drug nonmedically. When looking at specific racial groups, African Americans report using illicit drugs and marijuana at higher rates than Caribbean Blacks and Latinos. However, regarding using a prescription-type drug nonmedically, Caribbean Blacks report a higher prevalence rate compared to African Americans and Latinos.

Table 2.1
Prevalence of Past Month Substance Addictions Among African Americans, Caribbean Blacks, and Latinos

Substance Addiction	African American	Caribbean Black[a]	Latino (Mexican)
Illicit drugs	9.2	1.6	7.4
Marijuana	7.3	5.2	5.0
Prescription drugs	1.7	5.5	2.4

Source: Substance Abuse and Mental Health Services Administration (2007).
[a]Alegria, Jackson, Kessler, and Takeuchi (2007).

Overall, research on the drug-using behaviors of Latino populations indicates that Latinos have high prevalence rates for substance abuse, the highest reported symptoms of dependency, and the highest need for alcohol and drug treatment compared to whites. On the basis of the presented prevalence rates, Caribbean Blacks also show a need for focused and culturally appropriate treatment.

Race and Ethnicity Issues in the United States

African American, Caribbean Blacks, and Latinos suffer more adverse health conditions from substance addictions than the white population (Alegría et al., 2006; De La Rosa et al., 2005; Escobar & Vega, 2006; Paniagua, 2000; Primm et al., 2006; Williams, Neighbors, & Jackson, 2003). Though identifying many health conditions and their racial differences is important, many other factors associated with substance addition can create greater substance addiction problems, including poverty status, education, unemployment, families in crisis, psychiatric problems, low or no insurance coverage, and public program assistance limitations. Yet the one factor that many researchers and government documents focus on is race and ethnicity. Identifying with a group is important to all people. A group defines who we are and how we envision our place in the world. Above all, we all belong to *many* groups. We try to identify groups by appearance, culture, nationality, and so on, but the most common (and awkward) means of identification is by a set of categories that often do not capture human variation well.

Researchers in the United States continue their long-standing preoccupation with race and ethnicity issues, particularly the need to categorize groups for descriptive reasons such as accounting for population differences, health disparities, and social problem significance. Interestingly, in 1997, the Office of Management and Budget, the agency that determines the National Institutes of Health's population taxonomy, established standards for the collection of data on race and ethnicity, in which the only recognized ethnic group was "Hispanic" or "Latino" (Escobar & Vega, 2006). Though much research has argued against categorizing people (Fullilove, 1998; Stolley, 1999), and the discussion would extend beyond the purpose of this chapter, five racial groups continue to be recognized, namely, Asian American, American Indian or Alaska Native, native Hawaiian or other Pacific Islander, black or African American, and white. For states that increasingly grow due to immigrant populations, such as California and New York, the ethnic and race categories are increasingly inaccurate and less useful when comparing with other nations. International studies have shown that U.S. ethnic categories hold little meaning in other countries that have their own immigrant sets and minority groups. It is important to allow for a variety of racial and ethnic groups in research *beyond* the standard five

commonly used so that substance addiction research can discover the impact of racial identity on particular substance addictions.

Culture and Its Meaning to Substance Addiction Research

Although the term *culture* is widely used, and most appreciate what the term brings to the research environment, few agree on its meaning or definition, and even less on how we operationalize culture in research. Even fewer use similar operational definitions within the context of addiction research. Part of the problem in gaining a solid grasp on a unifying definition of culture is that its use varies with norms, beliefs, power (including issues of oppression), organizations, and institutions such as *academic culture* or *prison culture*. Culture does not always refer to heritage or ethnicity. Culture, as supported by social science researchers, encompasses all of life's patterns, socialization practices, religious ideals, habits in thinking, and organizations, which groups of persons pass on from one generation to the next. Lum (2003) notes that cultural pluralism significantly affects our current society. *Cultural pluralism* involves the coexistence of many group associations and values that beg to be tolerated and acknowledged in research. Respondent substance addictions can no longer conceptually exist outside of the multiple influences impinging on expressed behavior. The culture in which research on addiction takes place must be carefully identified, and the parts applicable to the specific research project clearly defined.

Effective cross-cultural social science research accurately describes conditions of the research participants' lives by discovering pertinent cultural information about the defined group (Lum, 2004). How might researchers gain a better sense of cultural information when budgets are limited? One way is to develop places throughout the research study to support interactive exchanges of information (data collection between researchers and participants). The exchange of information throughout the research process can assist substance-addicted people in negotiating their environment, finding networks of support, and exploring culturally relevant ways of reducing harm. For addiction researchers, the information exchanges help with interpreting research findings and transform researchers into the *culturally formed*. The exchange of cultural information offers researchers an insider's view of addiction because the position of the researcher is transposed into a position of being an avid learner. Many researchers would agree that publishing research that communicates *new* knowledge would be improved if posed research questions were developed with a cultural consciousness.

Initializing research with an overall definition of *population culture* may be misguided. Instead, starting by identifying a group and then deciphering its

culture would serve as a more effective manner to advance research knowledge. In addition to referring to the addiction literature and exchanging information with other researchers, there are other valuable means of learning about a group's culture. One is through the emic approach of interviewing members of the target population to glean an understanding of the values, behaviors, norms, and symbols of that group that provide coherence for its members. Another means is through ethnographic accounts like those of Philippe Bourgois in his 2003 book *In Search of Respect: Selling Crack in El Barrio,* about the culture of urban Puerto Rican addicts and dealers. Ethnographies provide insight into culture and the intersections of race, ethnicity, and geographic or physical environments. By having the group reveal its culture, researchers can examine the mechanisms in the culture that create or sustain addiction and problems in locating help. Including culture in the research enterprise also includes the realization of situations in which cultural practices both constrain and sanction those who use substances. For example, women of Mexican descent drink alcohol, but where these persons and their social groups follow customs, they are also ridiculed for participating in the alcohol drinking behavior (Vega et al., 1998).

Dimensions of Culture

Culture is a key factor in the helping process. Dawson (1998) offers three reasons why using race to symbolize culture will not advance knowledge: (1) racial or ethnic labeling reinforces notions that racial identity operates as a determining factor in substance addiction and ignores other characteristics known to vary in alcohol use and its associated problems (Collins, 1996); (2) stating racial categories from three racial groups (mainly, African American, Latino, and white) ignores other minority populations, such as the rapidly growing category of Latino immigrants from South America and Mexico and black immigrants from the Caribbean, either by omitting them altogether or lumping them into an "other" category; and (3) listing race ignores heterogeneity within groups, the "other" category included. The task, therefore, charged to contemporary cross-cultural researchers is to conduct research studies that record cultural interpretations of addictive behaviors and treatment practices from both the researcher and research participant perspective.

Beyond the challenges of using race as an organizing category, there is the matter of culture, which is—when it is attended to—treated as a static construct. Culture is organic, dynamic, and fluid. It interacts heavily with the environment and with both race and ethnicity. Along with what Dawson (1998) has pointed out, Bentancourt and Lopez (1993) note that researchers are apt to point to so-called culture when they discover differences among racial and eth-

nic groups. Left out is the fundamental question of what cultural *mechanisms* account for the difference.. To ascribe differences to culture without attention to its operationalization at the individual or group level leaves open a wide gap in our understanding of addictions.

To avoid the traps that Dawson (1998) and Betancourt and Lopez (1993) caution researchers about, this chapter focuses on the research context to advance addiction research. The research context must take into account differences among people, situations, and treatment implementations. This refocusing calls for diversity-inclusive research thinking, planning, and conducting. To begin, a brief argument is presented for utilizing mixed-method research as its tenets may offer a means to generate new knowledge about addiction within racially diverse communities.

Mixed Methodology: An Argument for Addiction Research

Although we know unequivocally that addiction is a disease like any other medical disease and that disparities exist among racial demographic characteristics, the literature is not clear on how to move beyond current findings. This reluctance among researchers is partly due to researchers' adherence to choosing either quantitative or qualitative methods. While addiction in communities of color remains a stigmatized disease, the stigma spills over to all aspects of addiction research, experiences of alcohol consumption, drug use, prevention, and treatment (e.g., obtaining measures of use, safety and legal concerns) and what to do about denial.

To perhaps enjoy greater depth in exploring the new frontier and the dramatic changes in addiction among racially diverse communities (e.g., gambling, prescription drugs, etc.), researchers might want to utilize mixed research methods. Quantitative and qualitative methods are often depicted as dichotomous, separate approaches to conducting research. Each differs in underlying assumptions, purpose, approach, and the appropriate role of the researcher. For example, quantitative methodologies purport that researchers can objectively investigate addictions, while qualitative methodologies suggest that addictions are socially constructed. When quantitative and qualitative methods are viewed as ends of a continuum, one logical argument put forth is that mixing these methods can improve our understanding of addiction. Ercikan and Roth (2006) explain the difficulties to adequately answer any social research questions without both approaches.

Mixed-method research designs, proper notation, terminology, and strengths and weaknesses are still being discussed and fine-tuned within the emerging field of mixed-method research (Creswell & Plano-Clark, 2007). The last 20 years witnessed a growing number of social science and public health research-

ers accepting mixed methods as a legitimate form of research. While some scholars continue to debate the wisdom of mixing methods traditionally associated with the postpositivist or constructivist paradigms, many researchers are beginning to adopt a pragmatic perspective by allowing community-informed research questions to drive the scientific enterprise. It is acknowledged in the addiction research field that racial and ethnic populations are adversely affected by the stigma of addiction; however, unknown are the cultural interpretations of addiction. Also depicted in the literature are the deleterious effects of addiction, which lead to misperceptions about communities in a manner that affects how prevention and treatment programs are delivered. What mixed-method research offers to social scientists is a means with which to produce new knowledge. The contemporary literature points to many areas of disparity for diverse communities, yet available data often lack information on how populations ascribe meaning to addiction. Enhancing data to better depict populations in depth will lead researchers to a greater level of understanding of health service needs. Perhaps a mixed-method approach might provide an avenue to filling in the gaps.

Limited Data

Understanding why addiction disparities exist continues to challenge researchers. It appears that on nearly every index measured, African Americans suffer more severely in relation to white Americans, and often in relation to other racial and ethnic groups. While the causes of and correlations for addiction are immense, researchers are offered a great opportunity to learn more about understudied populations, especially how the concepts of health and disease hold social and cultural meaning. There are deeply entrenched ideologies for all arguments. Though it is clear to many researchers that race is a culturally constructed notion, agreement on the study of racial and ethnic health disparities requires data to illuminate contemporary issues. National data sets are often inadequate to support addiction research because in some cases, the sample is insufficiently diverse, and in other circumstances, the sample includes small numbers of minority respondents compared to whites (LaVeist, 1995). Also, data sets with sufficient sample size frequently lack the range of psychosocial variables that are often of interest in understanding social and behavioral aspects of minority health. For example, most national data sets (such as the National Health Interview Survey, the National Health and Nutrition Examination Survey, or the Behavioral Risk Factor Surveillance System) have limited psychosocial variables. Because these data sets are designed to produce prevalence rates of morbidity and other health-related characteristics of the population, they have broad coverage across a variety of health domains but are

limited in their ability to test novel hypotheses related to the etiology of health disparities.

As one response to the lack of data examining racial and ethnic health disparities, and addiction disparities in particular, the National Institute of Mental Health Collaborative supported the completion of four psychiatric epidemiology surveys that included three national surveys: the National Survey of American Life (NSAL), the NCS-R, and the National Latino and Asian American Study (NLAAS; Pennell et al., 2004). Each of the studies provides a rich opportunity to focus on culture-specific aspects of mental health and substance addiction among U.S. communities of color. Though each study collected data independently, all can be combined with unifying diagnostic interview questions. A slightly modified version of the World Mental Health Composite International Diagnostic Interview was used to ascertain mental disorders as defined by the *DSM-IV*. This instrument was developed for the World Mental Health Project and was also used in the NLAAS and the NCS-R. To explain, a description of the NSAL is provided.

The primary goal of NSAL was to utilize contemporary methodological advancements to gather the best data possible about the physical and mental health as well as the structural and economic conditions of diverse groups of Americans, with a particular focus on African Americans and Caribbean Blacks. The survey instrument included specific assessments of mental disorders, stress, coping, neighborhood characteristics, religion, social support, and work. As background, the NSAL is a national area probability sample based on a race-stratified screening procedure to determine where the sample would be obtained (Jackson et al., 2004). Trained interviewers completed face-to-face interviews with 6,082 adults (3,570 African Americans; 1,621 Caribbean Blacks; 891 non-Hispanic whites) and 1,170 adolescents (810 African Americans; 360 Caribbean Blacks) aged 13–17 years, for a total of 7,252 participants from across the nation.

Barriers to Addressing Differences in Addiction Research

The public health and social work literature in the last 10 years illuminates numerous barriers to addressing diversity in broadening the addiction knowledge base through cross-cultural studies. Recognized barriers vary at the individual, family, systemic, and organizational levels. The barriers challenging researchers infusing diversity issues into their research at the individual level include language, style and content of nonverbal communication, acknowledging the existence of racism and prejudice, stereotypes, and stress. At the systemic level, many of the same barriers exist, and many are reoccurring because nonverbalized norms, policies, procedures, and programs clearly undermine any

efforts to address difference in an effective manner (Loue, 2003). Recruitment may fail because of distrust of the researchers or historical exclusion of specific populations such as women, persons of color, non-English speakers, and persons actively involved in drug or alcohol use. The recruitment issues associated with these populations include increasing costs of translating research materials, liability considerations with women who might be pregnant and using drugs, and unknown health consequences. Other barriers to recruitment are fear of community ostracism or confidentiality in the community regarding substance addiction, poverty status (high transportation costs or in need of monetary incentive), child care, and adverse legal consequences if substance use becomes known. Though a researcher may apply for a certificate of confidentiality to have the research records protected, the mere fact that a participant must return to a community of people who will have knowledge of her substance addiction is an adverse effect that researchers cannot control. Finally, the organization may present barriers to data collection efforts, including a lack of training of staff on recordable service practices; application forms for services that ask lengthy, complex, and indistinguishable diversity questions; service operational hours that do not coincide with the availability of diverse clientele; no stated policies regarding cross-cultural practices; and fiscal constraints toward implementing innovative services. The impact of organizational practices on conducting cross-cultural research is not surprising; however, for addiction researchers to promote and advance cross-cultural knowledge, organizational structure and capacity need to be addressed for addiction populations.

Researchers engaged in building empathy also show a direct path toward moving beyond the barriers listed. Building empathy within the structure of the research process suggests that participants take on a more informed role of community process than the researcher. For example, participants can suggest when it is appropriate and inappropriate to return to communities after participating in research projects. By allowing the participants to share their knowledge of community living, researchers integrate the value of research participants' knowledge into the research process by asking for assistance when needed. This mode of inquiry would allow cross-cultural researchers to shift from understanding racial groups to understanding the experience of diverse people with a cultural identity. Cultural identity plays a central role in one's willingness to participate in research or the ability of so-called treatment to succeed. In the research context, some individuals may be reluctant to volunteer their participation in research due to hearing about adverse problems of past research endeavors or personally experiencing misrepresentation of shared information which led to harm, for example, the Public Health Service–led Tuskegee syphilis study with African American men (Jones, 1981); the use of

alcohol to gain unfair advantage in negotiating Native American homelands (Mancall, 1995); or the Tearoom Trade experiment, identifying the characteristics of men who sought quick and impersonal sexual gratification from other men (Humphreys, 1970).

The tools we use to reach and help people can be culturally appropriate and enhanced. Ultimately, the goal of any research is to create a workable frame of reference for discussion, to advance the knowledge base, and to achieve better understanding of persons in their environments. Rather than focusing solely at the micro level of the individual or solely at the macro level of the neighborhood, breaking down barriers in the future will require a more holistic approach that includes the addiction culture at all levels. To truly address the role social science addiction researchers can play in investigations, the voices of research participants must be heard and understood.

MISSION OF SOCIAL SCIENCE FOR SOCIAL WORK RESEARCHERS IN ADDICTION

Part of what defines a profession is a shared vision, typically referred to as a *mission*. The mission of social work research in addiction is quite similar to the missions of other national professional organizations in that we want "to enhance human well-being and helpmeet the basic human needs of all people, with particular attention to the needs and empowerment of people who are vulnerable, oppressed, and living in poverty" (National Association of Social Workers, 1977, p. 1). The aspects that make the mission of addiction research unique are its focus on the integration of culture, the language used to describe addictive behaviors, and the meaning respondents bring to the research arena from the social context. Social work researchers, not any different from other social scientists, realize that they must pay attention to the environment in which people live and work to change the environment to improve interventions. In doing so, researchers must *listen for meaning*.

Our Mission as Researchers

A culturally informed research agenda focuses on stages, issues, informed findings, and relevant recommendations. The contact phase of research involves establishing a relationship between the researcher and the respondent. As researchers gain a preliminary sense of the populations in their research, such as becoming acquainted with the neighborhoods, leaders, and residents who compose the area in which persons with addictions live, the diverse community, or commonly, the ethnic communities, becomes acquainted with the research

process. The development of the relationship between the community and the researcher fosters rapport and trust. The research process then becomes a working task in which the researcher, the research team, and the respondents collaborate on an accurate description and understanding of the social problem and culturally appropriate responses. The process just described is called *culturally congruent*, which means that no matter the stage of the research process, all participants in the research remain in tune with what is congruent with community, research, and methodological expectations.

Although both researchers and practitioners place high value on informativeness of cross-cultural research, much attention still remains focused on how we conceptualize culture and the role it plays in addiction research. Although many would agree that active listening, translating research materials, using the services of an interpreter, and asking appropriate questions are ways to begin, much more attention needs to focus on the meaning behind words and actions (e.g., Green, 1995; Perlman, 1979). A pathway to discovering new knowledge is attending to word choice and understood meaning. According to Spradley (1979), "language is more than a means of communicating about reality: it is a tool for constructing reality" (p. 17). For all people, and especially for people of color, language serves as more than a tool for transmitting information; it establishes the meanings speakers assign to their experiences. In this regard, a key to understanding differently requires the addition of a question or two about meaning, for example, "Would you share with me what you mean when you use the word *nip*?" By attending to the way that words are used in phrases and terms in ordinary language, greater clarity, understanding, and accurate interpretation are achieved.

The mission of cross-cultural research is to advance knowledge using culturally appropriate methods. In this manner, the goal is to promote social justice and equity, promote a better understanding of what works within the community, enhance researchers' ability to work across systems (micro/mezzo/macro), and translate knowledge into meaningful explanations.

FUTURE RESEARCH AGENDA FOR SOCIAL SCIENTISTS IN CROSS-CULTURAL RESEARCH

What Actions Do We Need to Take?

Though most researchers recognize many pathways to including culture in research, few truly deal with all the issues that contain both subjective and objective content. Advancing social science research requires that researchers use culturally congruent interventions to inform practice. The researcher

must become more involved in the federal funding aspect by participating in the grant proposal review process and demanding that sponsored research delineate the process for diverse subject recruitment. If the research sample is not diverse, challenge the principal investigators to explain how the proposed research could enhance scientific understanding of addiction. All researchers should try to design interventions that practitioners can use and disseminate findings to local interested parties that will enhance practice directly.

Future research also needs to include studies that document what practitioners are doing in service delivery so that researchers know what might represent effective and ineffective practices and also more informed useful practices. Ultimately, practice-oriented researchers need to maintain and develop collaborative relationships with other disciplines and increase connections to communities.

We need to discuss issues that practitioners bring to the research environment in the context of working in the trenches.

Interpersonal Sensitivity

For example, when developing prevention programs for young women of color, researchers should move beyond gaining parental or guardian consent; researchers should include parents or guardians in the data collection process. Not only would this action show researchers to be persons connected to the lives of community members, but also, the potential intervention might be understood as beneficial to the identified group and maintained after the research project is completed. Since the movement among agencies is to use only so-called best practices, or model programs, then attention to cultural concerns will place addiction researchers in the forefront of advancing knowledge.

Assets That Social Science Researchers Bring to Addiction Research

Few careers rival social work in the diversity of opportunities offered; however, all social scientists want to understand social settings that include community centers, courts, prisons, hospitals, nursing homes, and addiction treatment centers, where addiction problems exist at multiple levels—micro (individual), mezzo (community), and macro (policies). The desire to help others and change social conditions represent only two characteristics that allow practitioners and social scientists to show their uniqueness. Social scientists, and social workers, in particular, encompass a large labor force and are located in a wide range of service areas. They possess unique knowledge about diverse populations, skills informed by an interdisciplinary knowledge base, a systems

perspective, community connections, and an unwavering commitment to social justice and equity issues, from a strengths perspective.

CONCLUSIONS

From this chapter, it is clear that complex conceptual and scientific issues need focused attention. A long-term commitment to federal, state, and local funding as well as funding from private foundations is needed to address the multiple gaps in our knowledge. It is important to support new ideas for conducting addiction research so that the next generation of scientists can seek the input of key stakeholders. For instance, the primary stakeholders are the research participants, the social science researchers, and the funders; support from any of these three bodies will help in advancing knowledge. Research studies of the twenty-first century must move forward with the permission of individuals from diverse communities who share their life stories and thoughts. New and provocative questions need to be posed, without social scientists' investment in local and national communities, that allow outsiders to learn something new. Clearly, if the discussion of culture accomplishes little else, advancing the scientific enterprise by better understanding the addictions attributed to African Americans, Caribbean Blacks, and Latinos requires interdependency of all systems to reduce or even eliminate challenges.

ACKNOWLEDGMENTS

Thank you to Dr. Arlene Stiffman and Dr. Luis Zayas of Washington University's George Warren Brown's School of Social Work for the opportunity to develop a conceptual basis for the manuscript. I especially appreciate the guidance and support of the National Survey of American Life principal investigators, namely, Drs. James S. Jackson and Harold W. Neighbors of the University of Michigan Program for Research on Black Americans.

REFERENCES

Alegría, M., Cao, Z., McGuire, T. G., Ojeda, V. D., Sribney, B., Woo, M., et al. (2006). Health insurance coverage for vulnerable populations: Contrasting Asian Americans and Latinos in the United States. *Inquiry—Excellus Health Plan, 43*, 231–254.

Alegria, M., Jackson, J. S., Kessler, R. C., & Takeuchi, D. (2007). *National Survey of American Life (NSAL), Collaborative Psychiatric Epidemiology Surveys (CPES),*

2001–2003 [UNITED STATES] (Vol. 2008-06-19) [Computer File No. ICPSR20240-v5]. Ann Arbor, MI: Institute for Social Research, Survey Research Center / Interuniversity Consortium for Political and Social Research.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Bentancourt, H., & Lopez, S. R. (1993). The study of culture, ethnicity, and race in American psychology. *American Psychologist, 48*, 629–637.

Bourgois, P. (2003). *In search of respect: Selling crack in el barrio*. New York: Cambridge University Press.

Collins, R. L. (1996). The role of ethnic versus nonethnic sociocultural factors in substance use and misuse. *Substance Use and Misuse, 31*(1), pp. 95–101.

Creswell, J. W., & Plano-Clark, V. L. (2007). *Designing and conducting mixed methods research*. Thousand Oaks, CA: Sage.

Dawson, D. A. (1998). Beyond black, white and Hispanic: Race, ethnic origin and drinking patterns in the United States. *Journal of Substance Abuse, 10*, 321–339.

Degenhardt, L., Chiu, W. T., Sampson, N., Kessler, R. C., & Anthony, J. C. (2007). Epidemiological patterns of extra-medical drug use in the United States: Evidence from the National Comorbidity Survey Replication, 2001–2003. *Drug and Alcohol Dependence, 90*, 210–223.

De La Rosa, M. R., Holleran, L. K., Rugh, D., & MacMaster, S. A. (2005). Substance abuse among U.S. Latinos: A review of the literature. In M. R. De La Rosa, L. K. Holleran, & S.L.A. Straussner (Eds.), *Substance abusing Latinos: Current research on epidemiology, prevention, and treatment* (Vol. 5, pp. 1–20). Binghamton, NY: Haworth Press.

Ercikan, K., & Roth, W. (2006). What good is polarizing research into qualitative and quantitative? *Ageing and Society, 20*, 369–375.

Escobar, J. I., & Vega, W. A. (2006). Cultural issues and psychiatric diagnosis: Providing a general background for considering substance use diagnoses. *Addiction, 101*(Suppl. 1), 40–47.

Fullilove, M. T. (1998). Abandoning "race" as a variable in public health research: An idea whose time has come. *American Journal of Public Health, 88*, 1297–1298.

Guzmán, B. (2001). *The Hispanic population: Census 2000 brief*. Retrieved January 10, 2008, from http://www.census.gov/prod/2001pubs/c2kbr01-3.pdf

Humphreys, L. (1970). *Tearoom trade: Impersonal sex in public places*. Chicago: Aldine.

Jackson, J. S., Torres, M., Caldwell, C. H., Neighbors, H. W., Nesse, R., Taylor, R. J., et al. (2004). The National Survey of American Life: A study of racial, ethnic, and cultural influences on mental disorders and mental health. *International Journal of Methods in Psychiatric Research, 13*, 196–207.

Johnson, T. P. (2007). Cultural-level influences on substance use and misuse. *Substance Use & Misuse, 42*, 305–316.

Jones, J. (1981). *Bad blood: The Tuskegee syphilis experiment—A tragedy of race and medicine*. New York: Free Press.

Kessler, R. C., Berglund, P., Chiu, W. T., Demler, O., Heeringa, S., Hiripi, E., et al. (2004). The US National Comorbidity Survey Replication (NCS-R) design and field procedures. *International Journal of Methods in Psychiatric Research, 13*(2), 69–92.

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., et al. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Results from the National Comorbidity Survey. *Archives of General Psychiatry, 51*(1), 8–19.

LaVeist, T. A. (1995). Data sources for aging research on racial and ethnic groups. *Gerontologist, 35*, 328–339.

Loue, S. (2003). *Diversity issues in substance abuse treatment and research*. New York: Kluwer.

Lum, D. (2003). Culturally competent practice. In D. Lum (Ed.), *Culturally competent practice: A framework for understanding diverse groups and justice issues* (2nd ed., pp. 3–14). Belmont, CA: Thomson Brooks / Cole.

Lum, D. (2004). *Social work practice with people of color*. Monterey, CA: Thomson Brooks / Cole.

Mancall, P. C. (1995). *Deadly medicine: Indians and alcohol in early America*. Ithaca, NY: Cornell University Press.

McKinnon, J. (2001). *The black population: 2000*. Retrieved January 10, 2008, from http://www.census.gov/population/www/cen2000/briefs.html

National Association of Social Workers. (2008). *Code of ethics of the National Association of Social Workers*. Retrieved September 14, 2008, from http://www.socialworkers.org/pubs/code/code.asp

Paniagua, F. (2000). Culture-bound syndromes, cultural variations, and psychopathology. In I. Cuèllar & F. A. Paniagua (Eds.), *Handbook of multicultural mental health: Assessment and treatment of diverse populations* (pp. 139–169). San Diego, CA: Academic Press.

Pennell, B. E., Bowers, A., Carr, D., Chardoul, S., Cheung, G. Q., Dinkelmann, K., et al. (2004). The development and implementation of the National Comorbidity Survey Replication, the National Survey of American Life, and the National Latino and Asian American Survey. *International Journal of Methods in Psychiatric Research, 13*(4), 241–269.

Perlman, H. H. (1979). *Relationship: The heart of helping people*. Chicago: University of Chicago Press.

Primm, B. J., Brown, L. S., Primm, A. B., & Friedman, J. S. (2006). Tobacco, alcohol, and drugs. In D. Satcher, R. J. Pamies, & N. N. Woelfl (Eds.), *Multicultural medicine and health disparities* (pp. 321–330). New York: McGraw-Hill.

Spradley, J. P. (1979). *The ethnographic interview*. New York: Holt, Rinehart & Winston.

Stolley, P. D. (1999). Race in epidemiology. *International Journal of Health Services, 29*, 905–909.

Substance Abuse and Mental Health Services Administration. (2007, June 21). *The NSDUH report: Illicit drug use, by race/ethnicity, in metropolitan and non-metropolitan counties: 2004 and 2005*. Rockville, MD: Office of Applied Studies.

Therrien, M., & Ramirez, R. R. (2000). *The Hispanic population in the United States: March 2000 brief*. Washington, DC: U.S. Census Bureau.

U.S. Census Bureau. (2000a). *Census 2000 demographic profile highlights: African American alone*. Retrieved January 10, 2008, from http://facfinder.census.gov, and http://www.factfinder.census.gov/servlet/SAFFIteratedFacts?_event=&geo_id=01000US&_geoContext=01000US&_street=&_county=&_cityTown=&_state=&_zip=&_lang=en&_sse=on&ActiveGeoDiv=&_useEV=&pctxt=fph&pgsl=010&_submenuId=factsheet_2&ds_name=DEC_2000_SAFF&_ci_nbr=004&qr_name=DEC_2000_SAFF_R1010&reg=DEC_2000_SAFF_R1010%3A004&_keyword=&_industry=

Vega, W. A., Kolody, B., Aguilar-Gaxiola, S., Alderete, E., Catalano, R., & Caraveo-Anduaga, J. (1998). Lifetime prevalence of DSM-III-R psychiatric disorders among urban and rural Mexican Americans in California. *Archives of General Psychiatry, 55*, 771–778.

Williams, D. R., Neighbors, H. W., & Jackson, J. S. (2003). Racial/ethnic discrimination and health: Findings from community studies. *American Journal of Public Health, 93*, 200–208.

This page intentionally left blank

Chapter **3**

Race, Ethnicity, and Early U.S. Drug Policy

Sean R. Hogan, PhD, MSW

Prior to the twentieth century, the perception of drug use and addiction in the United States was very different than it is today. Drug use was not a criminal activity, nor was it regarded as inherently evil. Drug addiction was generally looked on as a bad habit or some type of personal misfortune (Booth, 1996). One reason for the naïveté around substance abuse during this period was simply a lack of information and understanding by the medical establishment as to the consequences of drug use and addiction. For example, early physicians thought morphine could be used as a cure for opium addiction. Ironically, heroin, developed commercially in 1898, was then thought to be a cure for morphine addiction. Cocaine, with its stimulating effects, was first thought to be a cure for alcoholism (Musto, 1999). The American public and its political representatives were equally uninformed. With such a lack of knowledge surrounding drugs, insightful policy making and practices were virtually impossible.

A significant shift in public perception, and, consequently, the strategies designed to address substance use, occurred when American society began to distinguish so-called recreational, or nonmedical, drug use from that of drug use initiated and prescribed by physicians. During the nineteenth century, the typical drug addict was a middle- to upper-class white woman with a dependence on morphine. This dependence was generally satisfied by a prescription from a family physician (Aldrich, 1988) or overindulgence in patent medicines containing opium or morphine (Lindesmith, 1957). The relationship between middle- and upper-class white female opiate addicts, the medical establishment, and a certain social acceptability of opiate use during the nineteenth century

was no coincidence (Courtwright, 1982). As long as drug use and addiction were limited to the dominant and upper-class members of society, addiction was not going to be regarded as a serious social problem; however, once attention for substance use began to shift toward less socially desirable members of society (e.g., minorities and immigrants), the public's perception and reaction to drug use also began to change (Musto, 1999). Drug use and addiction were no longer seen as a consequence of illness or the result of some type of upper-class personal misfortune. Drug use and addiction began to be interpreted as the self-indulgent and irresponsible behavior of lower-class and socially inferior members of an ever diverging American social landscape.

The relationship between race, ethnicity, and early U.S. drug policy is important to U.S. drug policy historians and analysts as it represents the foundation of antidrug legislation in that country. For historians of U.S. drug policy, the years between 1875 and 1937 are significant as it was during this period that antidrug policies specific to narcotics (e.g., opium, morphine, and heroin, cocaine, and marijuana) were first established. Policy analysts, particularly those examining social policy from a constructionist perspective, see social dynamics related to racism and xenophobia as critical to understanding the creation of substance abuse policy in the United States. It is the insidious relationship early U.S. drug policies have with minority and nonnativist groups that defines and contextualizes early antidrug policy making in the United States. Understanding this fundamental characteristic of early U.S. antidrug policy making has helped analysts interpret the construction of substance abuse policy throughout the history of the United States.

In this examination of the relationship between race, ethnicity, and early U.S. drug policy, three examples of race and specific substance associations are provided as models for the genesis of early antidrug legislation. First, the association between the Chinese and opium is examined by looking at some of the details that led to the first antidrug policies in the United States. Despite a more serious social problem with morphine use and dependence, opium became singled out as America's first nonalcoholic substance of abuse. Second, the relationship between African Americans and cocaine is described. Early racist and discriminatory attitudes in the southern United States aided in creating drug-crazed perceptions of an already vilified population, perpetuating the United States' first comprehensive federal antidrug legislation (Musto, 1999). Third, the association between Mexicans and marijuana in the southwestern United States is examined. Economics and the fear of losing jobs to nonnativist laborers would contribute to the passage of America's first antimarijuana policies. Finally, this chapter concludes with a brief discussion of the symbolic association between race, ethnicity, and early U.S. drug policy and its subsequent effects on minority drug users in the United States.

OPIUM AND THE CHINESE

During the 1800s, opium was brought to the United States by Chinese immigrants. In many parts of Asia, opium smoking had become quite commonplace, particularly among the Chinese (Musto, 1999). Consequently, Chinese immigrants who came to the United States during this period also brought with them the custom of smoking opium. Most of these immigrants came to the West Coast of the United States as cheap labor to work in the mining and railroad construction industries. By the late 1800s, it was very common to see opium-smoking establishments, known as *opium dens*, in most cities with any appreciable Chinese population (Lindesmith, 1957). Besides opium, these dens offered other iniquitous activities to their patrons such as gambling and prostitution. Many opium dens were run and organized by secret Chinese criminal societies, known as *tongs* (Booth, 1996). Chinese laborers fell victim not only to the addictive properties of opium, but also to the domination of these oppressive criminal elements. The majority of the Chinese who came to the United States had their transportation paid for and provided by other, more powerful interests (Sandmeyer, 1939). The Chinese laborer would then bind himself to service to repay his debt. Practically all Chinese laborers who came to the United States were single men intent on achieving economic gain (Sandmeyer, 1939). In most cases, the Chinese laborer did not intend to settle permanently in the United States, but to accumulate a sufficient amount of income to enable him to return to China and live in comparative comfort. The dreams and hopes of Chinese immigrants to acquire an economic reserve in America and return home to some type of increased socioeconomic well-being were often compromised by the demands of opium addiction and the opium dens. One author has suggested that some Chinese laborers would spend as much as 50 percent of their daily income in the opium dens (about 50 cents, on average; Booth, 1996). The actual amount of money spent by Chinese immigrants on opium during this period is impossible to determine; however, some insight into the magnitude of the problem may be seen in the importation figures of the time.

Statistics on opium importation during the last half of the nineteenth century are available and are considered by many authors to be accurate and indicative of narcotic use in the United States during that period (Lindesmith, 1957; Terry & Pellens, 1928). According to Lindesmith (1957), between 1860 and 1900, the consumption of opiates increased enormously, far greater than the growth of the population. By the 1890s, the average domestic consumption of crude opium was over 500,000 pounds each year, and that of morphine and its salts over 20,000 ounces annually (Musto, 1999). These figures represent the total consumption of opium and morphine in the United States, includ-

ing opiates used by the medical and patent medicine industries, and are not indicative of opium used solely among the Chinese. While the largest amount of opium ever legally imported into the United States came in 1897 (1,073,999 pounds), opium smoking reached its zenith in 1883 with the importation of 208,152 pounds of smoking opium, mostly through San Francisco, California (Terry & Pellens, 1928); in the 1880s, approximately 20 percent of the entire Chinese population of the United States resided in San Francisco (Helmer, 1975; Sandmeyer, 1939). The relationship between opium and Chinese communities in the western United States during this time period was undoubtedly quite substantial.

Eventually, opium smoking and opium dens would begin to attract individuals from society who were not Chinese. These individuals included gamblers, prostitutes, and underworld figures (Musto, 1999). The isolation of the opium dens within the Chinese communities provided some criminals a safe haven from otherwise inquisitive law enforcement officials. In San Francisco's Chinatown, in 1885, there were 26 opium dens, most of which "catered for twenty-four smokers at a time" (Booth, 1996, p. 195). As opium smoking began to attract members of the non-Asian community, attitudes toward opium smoking also began to change. Although opium smoking was not nearly as widespread as morphine use, opium was the first drug in the United States to receive negative public attention. Many historians of early U.S. drug policy attribute the social opprobrium toward opium and opium smoking to its negative association with the Chinese (Booth, 1996; Helmer, 1975; Lindesmith, 1957). The negativity toward opium and its use had nothing to do with the drug's addictive qualities. According to Booth (1996), public opinion toward opium was primarily based on "racial hatred" (p. 195) by mainstream society toward Chinese immigrants. To support, or validate, this negativity, newspapers of the time carried lurid tales of crime and debauchery in opium dens. All these accounts portrayed the Chinese in a negative light (Musto, 1999). Anti-Chinese sentiments among the general public produced a type of antiopium hysteria. Images of aggressive and depraved opium smokers became indelibly etched in the minds of concerned public officials. A common concern of people was "that many women and young girls, and young men from respectable families, were being induced to visit opium dens, where they were ruined morally and otherwise" (Kane, 1882, p. 1). The issue of interracial smoking was of great concern among prominent members of U.S. society. A San Francisco doctor, Winslow Anderson, expressed his concerns when he claimed to have seen the "sickening sight of young white girls from sixteen to twenty years of age lying half-undressed on the floor or couches, smoking with their lovers. Men and women, Chinese and white people, mix[ing] in Chinatown smoking houses"

(Booth, 1996, p. 195). The antiopium movement was one of the first antidrug movements in which critics used the welfare of young people as a major rationale for social indictment (H. W. Morgan, 1981). It would not be the last.

The hysteria related to the Chinese and opium smoking, the majority of it unfounded, prompted the first antiopium legislation in the United States. It came in the form of a city ordinance. In 1875, the San Francisco municipal authority passed a city ordinance forbidding the smoking of opium (Brecher, 1972). This ordinance also forbade any individual from possessing opium and the paraphernalia necessary to smoke it, and the creation of an opium-smoking establishment (Booth, 1996; Bureau of Justice Statistics, 1992). However, the actual importing and selling of the drug were not included in the ordinance as criminal acts. Significantly, the importers and sellers of opium were usually wealthy San Francisco merchants (Austin, 1978). They did not fall subject to the penalties of the law. This policy never attempted to stop the importation of opium, but merely served to harass and intimidate the Chinese opium den operators and patrons (Helmer, 1975).

Enforcement of the new ordinance was inconsistent. According to Booth (1996), "dens to which whites went were closed down, on racial grounds, but smaller establishments catering primarily for Chinese were ignored" (p. 196). The result of the ordinance was not the cessation of opium smoking, but rather the relocation of non-Chinese (i.e., white) opium smokers from opium dens in Chinatown to "boarding houses owned by Caucasians on the fringes of Chinese residential areas" (Booth, 1996, p. 196). Supplies of opium did not lessen. In 1881, the state of California passed its own statute prohibiting the selling of opium or the visiting of any place for smoking opium (Sandmeyer, 1939). This still did not have much effect on opium smoking. Consequently, in 1882, a bill was passed restricting the immigration of Chinese laborers into the country. While passage of this bill had strong economic underpinnings, opium smoking was also a significant contributing factor to its passage. Regardless of these legislative measures, as noted previously, the greatest amount of smoking opium ever imported into the United States came in 1883, the year after this bill was passed (Terry & Pellens, 1928). The fact that opium importation peaked following the passage of anti-Chinese legislation indicates that the use of opiates in the United States at that period of time was much more than simply a Chinese phenomenon.

Further evidence of the direct relationship between the Chinese race and U.S. drug policy comes from 1887. As the city and state laws enacted against opium smoking between 1875 and 1887 were proving to be rather ineffective, the federal government finally passed its own legislation related to the importation of opium for smoking. The significance of the Opium Import Act (1887)

was in its limitations: the law explicitly stated that "the importation of opium into any of the ports of the United States by any *subject of the Emperor of China* [italics added] is hereby prohibited" (Belenko, 2000, p. 15). This law forbade the importation of opium by Chinese nationals only; Americans were not affected. In 1890, a similar law was passed prohibiting nonnativist Americans from engaging in opium manufacturing, that is, the processing of raw opium into smoking opium. Using antidrug legislation as a device for differentially affecting nonnativist Americans was a relatively new political stratagem at this point in American history. Unfortunately, it would become quite commonplace. In 1904, in an attempt to further discriminate between American nativists and the Chinese Other, and presumably based in part on their negative association with opium use, all Chinese laborers were prohibited from coming into the United States and its territories (Sandmeyer, 1939).

Following the 1904 ban on the immigration of Chinese laborers to the United States, relations between the two countries became very strained. Chinese who had immigrated to the United States prior to the ban had been treated horribly by their American hosts. Chinese travelers visiting the United States for purely recreational reasons were also treated with great disdain. In an attempt to temper Chinese resentment toward America, President Roosevelt proposed a humanitarian act intended to help the Chinese government deal with its own endemic opium problem (Musto, 1999). This proposal came at the bequest of his political and drug advisory representatives in the Far East. The reconciliatory effort would come in the form of an international conference on opium control. On February 1, 1909, the first International Opium Commission convened in Shanghai, China. The conference was initiated by the United States and was attended by 12 countries: Austria-Hungary, China, France, Germany, Great Britain, Italy, Japan, the Netherlands, Persia, Portugal, Russia, and Siam (Terry & Pellens, 1928). Turkey, a prominent opium-producing country of the time, was conspicuous in its absence.

No antiopium policy changes were implemented in any country as a result of this convention. At the request of Great Britain and the Netherlands, the Shanghai conference on international opium control was officially ranked as a commission. This relegated the proceedings to a "fact-finding body which could make only recommendations and not commitments" (Musto, 1999, p. 35). The first universal drug-specific federal narcotic control law in U.S. history, the 1909 Smoking Opium Exclusion Act, was passed as a face-saving measure by the U.S. government. Prior to the convention, the United States did not have any universal federal legislation directed toward the control of opium. Hamilton Wright, the primary U.S. representative for the conference, believed that, as the convening nation of the Shanghai conference, the United States had a respon-

sibility to model its conviction and determination toward antiopium legislation (Musto, 1999). Consequently, on February 9, 1909, in the midst of the conference, the United States proudly announced the passage of the Smoking Opium Exclusion Act. This act banned the importation and use of opium by all members of U.S. society, barring prescription from a physician (Helmer, 1975). The Chinese were no longer singled out with regard to opium prohibition. The circumstance under which this law was borne, however, may call into question its legitimacy. Had the United States not been under such extreme pressure from the international community prior to the Shanghai conference of 1909 to enact some type of national opium control policy, the Smoking Opium Exclusion Act would have been inevitably delayed.

In the final analysis, hostility toward the Chinese and their negative association with opium were predicated more on the economic conditions and market dynamics of the period than the effects opium actually had on American society (Austin, 1978; H. W. Morgan, 1981). Racial and cultural differences, an apparent lack of concern for American political and social institutions, and a perceived clannishness among Chinese immigrants also served to foster intense anti-Chinese sentiment among the American people (Sandmeyer, 1939). However, as many scholars of the subject submit (Austin, 1978; Helmer, 1975; Sandmeyer, 1939), economics was the primary basis for ethnic antagonisms during the last half of the nineteenth century. Hostilities toward Chinese laborers in California began early on their arrival. In the mining communities of the 1850s and 1860s, Chinese laborers were already being regarded as a source of economic trouble by white mine workers. Chinese laborers willing to work for wages deemed unacceptable by white workers were seen as undermining and splitting the early labor market (Helmer, 1975). Rather than direct their economic dissatisfactions toward their employers, white workers attacked the Chinese and drove them from the quickly disintegrating mining industry. Forced from the mines, Chinese laborers relocated to larger urban centers such as San Francisco.

In the 1870s, the economy in California began to suffer from competition with the eastern United States. Access to Californian markets by eastern businesses "had been increased by the completion of the railroads and the resulting drop in freight rates" (Helmer, 1975, p. 21). Severe economic depression ensued, particularly in the city of San Francisco. Chinese laborers were once again identified by white workers as a cause of their own economic hardships. With the increase in unemployment, small manufacturers and white laborers aligned themselves to unfairly identify cheap Chinese competition as the cause of their problem. Cheap Chinese laborers, who were much more tolerable during prosperous times, were now a threat and inconvenience to the

economic well-being of the unemployed nativist workers of the dominant society. According to Austin (1978), it was then that opium became negatively associated with the Chinese. As mentioned previously, a significant result of this negative association was the first antidrug legislation in the nation. Unfortunately, the construction of drug policies in the early United States based on racial and ethnic distinctions would not end with opium and the Chinese.

COCAINE AND AFRICAN AMERICANS

Concurrent with the developing public hysteria over opium use and Chinese immigrants in the West was an even more vicious campaign targeting African Americans and the drug cocaine in the southern United States (Pittman & Staudenmeier, 1994). During the end of the nineteenth century, the United States experienced what some historians refer to as a cocaine explosion (Musto, 1999). The use of cocaine, much like the use of opium and morphine, became very popular. Besides cocaine's euphoric and anesthetic effects, it was well known for its abilities to fend off fatigue and allow its users to be more energetic and productive. As a result, cocaine became popular among some African American laborers in the South (Austin, 1978). Some southern labor bosses were even known to provide their black laborers with cocaine to increase their endurance and productivity (H. W. Morgan, 1981). Cocaine provided by whites to blacks under the pretense of increased economic productivity was seen as forgivable; however, cocaine used by blacks for recreational purposes was an indication of moral depravity. This double standard would ultimately serve to stigmatize southern blacks and cocaine use beyond the already existent racial and social discriminations of the southern United States.

During this historical period in the South, a more energetic and active African American was also perceived as being more aggressive and threatening (Austin, 1978). This perception among the members of the dominant white society only served to perpetuate already existing fears and prejudices. Following the turn of the nineteenth century, increasing reports of cocaine use among African Americans aroused all the malevolent stereotypes and fears of American racism (H. W. Morgan, 1981). As a result, cocaine and African Americans became negatively associated, spurring some fantastic and incredibly lurid tales of cocaine-related offenses committed by African Americans. Anecdotal accounts often told of superhuman strength, violent criminal acts, and ghastly sexual offenses perpetrated by blacks against their white counterparts. Cocaine became associated with aggression, murder, rape, and African American males (Booth, 1996). The myth that cocaine increased the physical

powers of criminals became common knowledge. According to Musto (1999), "one of the most terrifying beliefs about cocaine was that it actually improved pistol marksmanship" (p. 7). Some police departments insisted that ordinary police revolvers lacked the firepower to kill blacks who were under the influence of cocaine. This prompted some police officers to increase the strength of their weapons from .32 to .38 caliber (Musto, 1999).

The fear that cocaine would lessen African American inhibitions and increase their sexual impulses permeated social and political discussions of the time (H. W. Morgan, 1981). Official concerns were expressed by the likes of the aforementioned antiopium crusader Hamilton Wright. Wright (as cited in Austin, 1978) reported that the use of cocaine by southern blacks was "one of the most elusive and troublesome questions which confront the enforcement of the law" and was "often the direct incentive to the crime of rape" (p. 230). Wright went on to emphasize his belief that the misuse of cocaine, particularly by blacks, was the most threatening habit in the country. In a 1914 testimony before Congress, Christopher Koch (as cited in Musto, 1999), a physician with the Pennsylvania State Pharmacy Board, was quoted as saying, "Most of the attacks upon white women of the South are the direct result of a cocaine-crazed Negro brain" (p. 305). In Atlanta, Georgia, the anticocaine discourse was particularly prejudiced. By 1914, the Atlanta chief of police was attributing exorbitant amounts of crime to cocaine use (Musto, 1999). The sensationalizing of cocaine use and crime in Atlanta also led to legal action against the Coca-Cola Company. The Atlanta-based soda fountain drink manufacturer included cocaine in its original formula. After 1903, cocaine was replaced in the soft drink with caffeine (Office of Technology Assessment, 1994).

The imagery and rhetoric of the cocaine-crazed southern black man also spuriously validated an increase in violence against blacks in the South during this period. In the minds of white law enforcers and townspeople, cocaine-related crimes warranted increased physical force and harsher penalties toward African Americans. Unlawful lynching of black men also increased during this period (Helmer, 1975). The relationship between perception and reality, relevant to African Americans and cocaine during this period, appears to be spurious because, although 80 percent of the nation's African American population were concentrated in the South (Timberlake, 1963), it is estimated that fewer than 10 percent of the nation's addict population were African American (Duster, 1970). Additional evidence indicating a lack of support for the relationship between cocaine, African Americans, and crime was provided by E. M. Green, clinical director of the Georgia State Sanitarium. Between 1909 and 1914, Green examined 2,119 blacks admitted to his Georgia institution. In his study, Green found only three cases of narcotic addiction among black patients,

in contrast to 142 drug-related disorders among the white clientele (Helmer, 1975). Furthermore, of the three African American narcotics users, only two used cocaine, while the third was an opium addict (Musto, 1999). Commission reports conducted from 1900 to 1908 of institutionalized inmates and patients in the Washington, D.C., area also failed to indicate any appreciable regard for the use of cocaine among blacks, or otherwise (Helmer, 1975). The reality of the hysteria related to cocaine and African Americans was that it was an invention of white society, a myth created in an environment of fear and prejudice by mainstream society with the intent of maintaining a dominant and oppressive status quo. Depicting African Americans as cocaine-crazed sexual deviants, and a threat to the purity of white women and children, served this end.

Vilifying African Americans in the South was not a new concept at the turn of the twentieth century. Cocaine was just another method for justifying and validating the already existing social disorder in that predominantly racist part of the country. According to Musto (1999), the South has always feared that African Americans "might become oblivious to their prescribed bounds and attack white society" (p. 6). Cocaine use by blacks simply amplified those fears. It was within this context and environment that anticocaine policy was first developed. The fact that the drug was highly addictive, much like opium and morphine, was not a consideration. The synchronicity of the hysteria between opium smoking and the Chinese and cocaine use and African Americans resulted in both drugs being included in the United States' first comprehensive federal antidrug law: the Harrison Narcotic Act of 1914.

MARIJUANA AND MEXICANS

Marijuana smoking, much like opium smoking, was brought to the United States by a foreign population as a norm of their indigenous culture. During the early 1900s, Mexican immigrants, coming to the southwestern United States as cheap laborers, brought with them their native cultural practice of smoking marijuana (Helmer, 1975). It would not be unusual during this period of time to observe a rural Mexican farmworker smoking marijuana at the end of a hard workday, similar to having an alcoholic beverage after work in other cultural settings. Ultimately, the relationship between marijuana and Mexican immigrants during this time period would lead to the construction of America's first antimarijuana laws.

Interestingly, the exact impact that marijuana smoking among Mexican immigrants during the early 1900s had on the United States and the creation of antimarijuana drug policy remains a point of scholarly debate (see Galliher & Walker, 1977, for an early example of this discussion). According

to Musto (1999), during the 1920s, most of the nation had very little concern about marijuana and the individuals who used it. However, Musto goes on to state that "in areas with concentrations of Mexican immigrants, who tended to use marihuana as a drug of entertainment or relaxation, the fear of marihuana was intense" (p. 219). Specifically, these areas were Texas, California, Arizona, and New Mexico; between 1920 and 1930, nearly 90 percent of all the Mexicans in the United States lived in these four states. The peak year of Mexican immigration into the United States during this period was 1924. In this year, 105,000 Mexicans entered the United States, and only 3,572 had reason to leave (Helmer, 1975). However, these figures only reflect the official record; the actual number of Mexicans who entered the country is impossible to determine accurately.

The prohibition of alcohol between 1919 and 1933 increased the popularity of marijuana smoking among drug users. An influx of marijuana smuggling from Mexico and Cuba into the southern United States began to arouse much concern among law enforcement and government officials (Brecher, 1972). The increased popularity in the smoking of marijuana sparked media and government antimarijuana campaigns (Bonnie & Whitebread, 1974). In a remarkably similar course to that of opium and the Chinese, and cocaine and African Americans, marijuana became negatively associated with Mexican immigrants. Public campaigns against the smoking of marijuana began to "center on its use by Mexican laborers and the fears that they would spread the drug to youth and other vulnerable populations" (Brecher, 1972, p. 409). Stereotypical perceptions of disfavored Mexican laborers were created that included marijuana smoking and intoxication (Helmer, 1975). According to Musto (1999), marijuana came to be perceived as the impetus of criminal, sexual, and violent behavior by Mexican immigrants in the southwestern United States. Like opium and cocaine, it was propagandized that marijuana caused Mexican laborers to commit crimes, rape women, and become mentally unstable (Musto, 1999). Both Musto and Brecher (1972) feel that it was this type of discourse that drove the creation of antimarijuana policy.

Evidence to support the validity of these assertions is hard to find. P. A. Morgan (1990) states that "in general, there was simply no basis whatsoever for assuming that Southwestern Anglos were the least bit disturbed about Mexican marijuana use" (p. 235) in the early 1900s. In the principal Californian newspapers of that time, there was very little mention of Mexicans and marijuana (P. A. Morgan, 1990). This obvious lack of media attention seems unlikely for a social problem of such reported repute as drug-crazed Mexicans ravaging women and children in the southwestern United States. Another point that Morgan emphasizes is that "among the Mexican population, low rates

of arrests for marijuana existed during the time of the alleged scare" (p. 236). If the Southwest was so overcome with intense fear, and marijuana-smoking Mexicans were committing so many crimes, arrest records of the time should have been more supportive. A 1930 survey of crime and delinquency among Mexicans in Texas, the state with the greatest Mexican population, concluded that Mexican immigrants "show delinquent tendencies less than their proportion of the population would entitle them to show" (Helmer, 1975, p. 58). The construction of marijuana-smoking immigrants as a menace to society appears to have been more of an invention perpetrated by ulterior social, economic, and political interests of the time, rather than a real social problem.

During the early 1900s, the immigration of Mexican laborers into the southwestern United States was not seen as particularly problematic. World War I had limited labor that would have been provided by European immigrants. Furthermore, according to Helmer (1975), "farmers and planters in the West were unable to compete with the North for black labor" (p. 57). Rather than allowing wage rates to rise, Mexican labor was actually imported by agricultural interests in the West. The surplus of cheap Mexican laborers kept wages low, profits high, and most important, the price of land low for the large agricultural companies (Helmer, 1975). It was not until the beginning of the Great Depression that Mexican labor became a major issue in the United States. As the country became poorer and more frustrated, hostilities toward outsiders and immigrants grew. Rationales to restrict Mexican immigration began to develop among the dominant society. According to Helmer, the actual ostracism of the Mexican laborer "differed from place to place, depending not so much on how prevalent marijuana use was, but on how the economic crisis affected the local population" (p. 57).

Some Americans condemned Mexican immigration for other reasons. According to P. A. Morgan (1990), three primary rationales, based mostly on nativistic and moralistic arguments, were being proffered during this time to keep or restrict Mexicans from coming into the country. First, some American social leaders questioned the civic competence of Mexicans to become proper American citizens. These opponents felt that the Mexican immigrants were not supportive of established American social and political institutions, nor were they capable of contributing to any progressive democratic ideals (P. A. Morgan, 1990). This argument was similar to detractions made about the Chinese during their years of social degradation in the late 1800s. Second, there existed "a strong biological nativist campaign against the growing Mexican population" (P. A. Morgan, 1990, p. 240). This should not be surprising given the United States' history of racism and xenophobia. One prominent member of an apparent eugenic group known as the American Coalition, C. M. Goethe (as cited

in Musto, 1999), was quoted in the *New York Times*, on September 15, 1935, as saying,

> Marijuana, perhaps now the most insidious of our narcotics, is a direct by-product of unrestricted Mexican immigration. Early grown, it has been asserted that it has recently been planted between rows in a California penitentiary garden. Mexican peddlers have been caught distributing sample marijuana cigarets to school children. Bills for our quota against Mexico have been blocked mysteriously in every Congress since the 1924 Quota Act. Our nation has more than enough laborers. (p. 220)

Third, there was a fear that Mexican immigrants would not remain isolated in the rural farming regions of the United States, but would ultimately end up in the populated urban areas (P. A. Morgan, 1990). Urban blight was an ever increasing problem for big cities, and the addition of more poor, nonnative immigrants to the streets of newly developing urban centers, such as Los Angeles, was not a welcome proposition for many Americans. Job competition in the big cities was yet another issue (P. A. Morgan, 1990). It was one thing for Mexicans to work for low wages out on the farm, but to come into the city and compromise the economic saliency of white workers was another matter altogether. It was this perception of the Mexican laborer as a threat to the economic and social welfare of the American nativist, in addition to his negative association with marijuana, that contributed to the passage of the first federal antimarijuana law in U.S. history: the Marijuana Tax Act of 1937.

SYMBOLIC ASSOCIATION

The genesis of the first federal antimarijuana policies in the United States was remarkably similar to that of opium and cocaine policy formation a generation earlier. The relationship between race, ethnicity, and the creation of early U.S. drug policy was quite significant. While other sociopolitical and economic determinants were also instrumental in the construction of these early policies, it appears that the symbolic association between drugs and specific racial and ethnic groups had a significant influence on public opinion and new policy formation. Through the symbolic association of a specific minority group and a negatively recognized drug, powerful groups in society could effectively demonize and vilify less desirable social and economic counterparts (Helmer, 1975). The negatively sanctioned substance simply came to symbolize that specific group. The negative association between a substance and a group proved to be politically and legislatively powerful. In conjunction with the symbolic association of a specific group with a drug came the rationale that these specific

drug-using groups also represented a threat to the welfare of American society (Austin, 1978). Musto (1999) posits that the legal prohibition of certain drugs occurred because these drugs and their use "seemed to undermine essential social restrictions" (p. 294) that were explicitly designed to protect dominant society and maintain a privileged status quo. Musto (1999) further states,

> The occasion for legal prohibition of drugs . . . appears to come at a time of social crisis between the drug-linked group and the rest of American society. Customary use of a certain drug came to symbolize the difference between that group and the rest of society; eliminating the drug might alleviate social disharmony and preserve [the] old order. (p. 295)

A lack of understanding among U.S. society relevant to drugs and drug use made drug use a viable scapegoat for the causes of many social ills (Musto, 1999).

Public response to minority-linked drugs (i.e., opium, cocaine, and marijuana) differed radically from attitudes toward other drugs with similar potential for harm such as morphine or barbiturates. The main differences between opium use and morphine use in the nineteenth century were how and by whom each drug was used. Opium, while available in many patent medicines, was primarily smoked and associated with Chinese immigrants and lower-class opiate users; morphine, on the other hand, was primarily swallowed or injected by middle- and upper-class white women (Courtwright, 1982). Early U.S. antidrug legislation focused primarily on opium used for smoking; morphine was not included. This situation is strikingly parallel to the sociolegal dichotomy that exists between crack cocaine and powder cocaine use in the United States. Crack cocaine is smoked and primarily associated with minority and lower-class users; powdered cocaine is inhaled through the nostrils (i.e., snorted) and primarily associated with white upper-class users. Legislation and policy, while encompassing both variations of the drug, are differentially written and implemented, with more severe and harsher penalties applied to crack dealers and users (Reinarman & Levine, 1997).

Fear and ignorance among mainstream policy makers and the American public played an enormous role in the symbolic associations that contributed to the creation of early U.S. drug policy. The public's fear of addicts and minority-group drug users supplied the powerful motive force behind early antidrug legislation (Musto, 1999). Courtwright (1982) believes that what people think about drug use and addiction depends to a great extent on who is using the substances at any particular time in history. The association between racial groups and drug use served to reinforce the dominant position of white American nativists. Drug-using racial groups became symbolically associated

with backward, premodern, unproductive elements of the ever progressive American ideal (H. W. Morgan, 1981). Groups that represented a threat to, or interfered with, the economic prosperity of the dominant culture became vilified (Helmer, 1975). Drug use was the agent, and symbolic association the device, by which this vilification process took place. Eventually, villains would become criminals, and drugs would continue to be symbolically associated with the less desirable members of society. Law enforcement and the criminal justice system would come to dominate the facilitation of substance abuse policy in the United States. The relationship between race, ethnicity, and early U.S. drug policy formation would provide the foundation for subsequent social and political antidrug responses.

One result of this dynamic has been that many disadvantaged and minority groups in the United States have had to suffer differentially for their drug use. The United States' distinct record of creating substance abuse policy predicated on which individuals in society are using drugs, rather than on the specific characteristics of the drugs or their effects on society, has perpetuated discriminatory social and political responses toward low-income and minority substance abusers. The U.S. predilection for punitive and zero-tolerance substance abuse policies has further compromised the health and welfare of these groups by neglecting their special needs for substance abuse prevention and treatment. Minority groups in the United States that have been historically and unjustly vilified as a result of fear, ignorance, and public misperceptions around substance abuse will continue to suffer inordinate social injustices until drug use and its consequences are more fully understood by U.S. policy makers and the American public.

REFERENCES

Aldrich, M. R. (1988). Historical notes on women addicts. In *Youth Environment Study* (pp. 1–6). San Francisco: Project MENU.

Austin, G. A. (1978). *Perspectives on the history of psychoactive substance use*. Rockville, MD: National Institute on Drug Abuse.

Belenko, S. R. (Ed.). (2000). *Drugs and drug policy in America: A documentary history*. Westport, CT: Greenwood Press.

Bonnie, R. J., & Whitebread, C. H. (1974). *The marihuana conviction: A history of marihuana prohibition in the United States*. Charlottesville: University Press of Virginia.

Booth, M. (1996). *Opium: A history*. New York: St. Martin's Press.

Brecher, E. M. (1972). *Licit and illicit drugs*. Boston: Little, Brown.

Bureau of Justice Statistics. (1992). *Drugs, crime, and the justice system: A national report* (Report No. NCJ-133652). Washington, DC: U.S. Department of Justice.

Courtwright, D. T. (1982). *Dark paradise: Opiate addiction in America before 1940.* Cambridge, MA: Harvard University Press.

Duster, T. (1970). *The legislation of morality*. New York: Free Press.

Galliher, J. F., & Walker, A. (1977). The puzzle of the social origins of the Marihuana Tax Act of 1937. *Social Problems, 24*, 367–376.

Helmer, J. (1975). *Drugs and minority oppression*. New York: Seabury.

Kane, H. H. (1882). *Opium-smoking in America and China*. New York: G. P. Putnam's.

Lindesmith, A. R. (1957). *Opiate addiction*. Evanston, IL: Principia Press.

Morgan, H. W. (1981). *Drugs in America: A social history, 1800–1980*. Syracuse, NY: Syracuse University Press.

Morgan, P. A. (1990). The making of a public problem: Mexican labor in California and the marijuana law of 1937. In R. Glick & J. Moore (Eds.), *Drugs in Hispanic communities* (pp. 233–252). New Brunswick, NJ: Rutgers University Press.

Musto, D. F. (1999). *The American disease: Origins of narcotic drug control* (3rd ed.). Oxford: Oxford University Press.

Office of Technology Assessment. (1994). *Technologies for understanding and preventing substance abuse and addiction*. Washington, DC: U.S. Government Printing Office.

Pittman, D. J., & Staudenmeier, W. J. (1994). Twentieth century wars on alcohol and other drugs in the United States. In P. J. Venturelli (Ed.), *Drug use in America: Social, cultural, and political perspectives* (pp. 149–156). New York: Jones and Bartlett.

Reinarman, C., & Levine, H. G. (Eds.). (1997). *Crack in America: Demon drugs and social justice*. Berkeley: University of California Press.

Sandmeyer, E. C. (1939). *The anti-Chinese movement in California*. Urbana: University of Illinois Press.

Terry, C. E., & Pellens, M. (1928). *The opium problem*. New York: Bureau of Social Hygiene.

Timberlake, J. H. (1963). *Prohibition and the progressive movement, 1900–1920*. Cambridge, MA: Harvard University Press.

Chapter 4

Gender Inequalities and Inequities among Women with Substance Abuse Problems

Martha Romero Mendoza, PhD

Gender constitutes a social life–organizing principle, and as with socioeconomic, demographic, and geographic factors, gender is essential to understanding health matters. Evans and Brown (2003) have gathered variables that might define public health policies, resource distribution, power, morbidity, mortality, and development, represented in the acronym PROGRESS. These variables are place of residence (urban/rural), race, occupation, gender, religion, education, socioeconomic status, and social capital/resources. Interaction between these elements could give rise to gender inequalities in well-being.

The significance of gender inequities for women's health has been underscored at two United Nations conferences: at the 1994 International Conference on Population and Development, held in Cairo, and at the 1995 World Conference on Women, carried out in Beijing. These conferences recognized women's physical, emotional, and social well-being as a human right and as an essential element of sustainable development.

Women's health problems related to use, abuse, and dependence on psychoactive substances are not an exception and deserve to be examined from this standpoint.

The aim of this chapter is to discuss, from a gender perspective theoretical framework, the burden of substance abuse disease in women. Inequalities in health with which these women have to cope will be treated here, as will relevant health issues in their treatment and the gaps in policies to deal with their special needs.

WHAT IS A GENDER PERSPECTIVE?

The growing implementation of a gender perspective in the design of interpretations, diagnoses, and public policies has allowed greater visibility of women and their problems. But this has also brought about a certain decline with respect to a reductionist tendency within the realm of this perspective; that is to say, many researchers talk about gender only because they write about women without taking into consideration the theoretical implications, and this constitutes the source of many distortions. Mainly, the theory of gender concerns historical and cultural relationships. It is useful to analyze and understand the female condition and women's situations, but it also serves to analyze the male condition and vital situations of men.

Gender analysis implies a reference to the social and economic contexts in which differences are constructed. Thus a gender perspective is a construction of theoretical, categorical, hypothetical, and interpretative articulations that, once assembled, allow an account of the complexity of decisions, behaviors, and beliefs of social subjects, men and women, as well as of the whole magnitude of social structure and the realm in which each one reproduces itself (Lagarde, 1996).

Gender is the cultural symbolic structure derived from biological sex. It is constructed with the whole of social, cultural, political, psychological, judiciary, and economic characteristics assigned differentially to both sexes. It is also the constitutive element of social relationships based on the differences that characterize sex and is a primary form of significant power relationships (Scott, 1996).

Among the most important gender elements are the following:

1. The available cultural symbols and myths that evoke multiple representations
2. The ruling concepts that express multiple interpretations of the meanings of symbols and behavior; these concepts are expressed in religious, educational, scientific, legal, and political tenets that affirm categorically and in an univocal way the meaning of man and woman
3. The social institutions and organizations of gender relationships: the kinship system, the family, the labor market segregated by gender, and the educational, health, and political institutions
4. Identity

Gender implies a distinct control over material and symbolic resources, and consequently, it is also implied in the concept and structure of power.

Gender structure can become such an overpowering social factor that it may even be thought of as *natural;* this is also true about certain supposed biological

capacities or abilities that are socially and culturally constructed and promoted. It is important to analyze the *articulation* of the biological matter with the social one and to *avoid the denial* of the unquestionable biological difference between men and women (Wilsnack, Vogeltanz, Wilsnack, & Harris, 2000).

HEALTH GENDER INEQUALITIES AND INEQUITIES

There are gender differences in most societies, and as a consequence, there are also health inequalities and inequities that are inherent to social definitions of femininity and masculinity, in terms of resources, opportunities, and problems.

Within this sphere, *equality* and *inequality* are dimensional concepts that refer to measurable quantities.[1] On the other hand, *inequity* and *equity* are political concepts that express a moral commitment to social justice.

In the words of Kawachi, Subramanian, and Almeida-Filho (2008), the difference can be expressed as follows:

> *Health inequality* is the generic term used to designate differences, variations, and disparities in the health achievements of individuals and groups. *Health inequity* refers to those inequalities in health that are deemed to be unfair or stemming from some form of injustice. They are avoidable and unnecessary. (p. 647)

It is more frequent to find research information on health issues linked to men than linked to women's problems (Doyal, 2000). Inequalities have a significant effect on health, and they are not given randomly. Instead, we can state that health troubles are selectively distributed among a population; some people and some sectors do undergo a higher number of troubles than others. This systematic variability is virtually hidden and/or masked in the whole of the population's health, except for some epidemiological data (Starfield, 2006).

Social inequality, and the bias that arose from it at the moment of the research, is perpetuated through social hierarchy preservation, that is, power relationships.

In accordance with Burke and Eichler's (2006) proposal, the existence of social hierarchies gives rise to a problem that has three sides: (1) the problem of preserving them, (2) the problem of failing when examining the generated differences, and (3) the problem of using double standards. What do we mean by this?

1. *Hierarchy preservation*. Every society stratifies people according to gender, abilities, or social classes. These variables are joined to other factors like age, religion, geographic location, sexual preference, or physical appear-

ance. Stratifying people into categories determines their access to all kinds of resources such as power and decision making, education, and income, among other things. There are several mechanisms by which these hierarchies are evident. As an example, we can mention that, in general terms, around the world, women have less social power than their male counterparts, and they have less educational attainment, lower economic income, and limited participation in political decision making.

2. *Failure when examining differences.* The lack of sensitivity when naming differences and when treating as equal people who must be treated in a different way is the prime form by which this problem becomes evident. Treating equally people who are not the same increases inequality, that is, denying social differences, not taking into account biological differences among sexes, or denial of the differences in the expression of multiple health problems.
3. *Using double standards.* Using double standards implies the action of treating groups differently to strengthen their subordinate status. Reputation may be a good example of this. Within the male population, reputation is linked to honesty, so if a man has a bad reputation, he is considered dishonest; in the case of women, reputation is tied to sexuality, so when a woman has a bad reputation, she is regarded as promiscuous. Therefore women's sexuality is subordinated with regard to men's. This constitutes a double social valuation.

Recently, a solid body of working knowledge has been built; this body demonstrates the close relation between gender inequalities and inequities and mental health (Outlook, 1999, Afifi, 2007). Other research has focused on poverty (Medina-Mora & Rojas, 2003; Reidpath & Allotey, 2007), life expectancy, education, employment, reproductive health, access to health services (PAHO, 2007), and other more qualitative dimensions such as well-being.

Results show that health problems suffered by women are not only linked to their specific biological characteristics, such as reproductive health and motherhood (Murphy, 2003); but they also reflect discrimination, and that women are at a disadvantage just by the act of performing their daily activities. This can be shown in cases of substance abuse.

Particularly, when selecting a gender perspective to understand alcohol consumption by men and women, an interpretative aspect is opened that allows one to disclose means of consumption, roles, attitudes, beliefs, restrictions, social consequences, rules, and opportunities.

From a sociocultural perspective, differences in how men and women are allowed or encouraged to drink or use drugs, and to behave when drinking or using drugs, help to define and symbolize the cultural and social structural distinctions between being a man and being a woman. Understanding how and

to what extent women and men drink or use drugs differently should also help to understand better how societies divide both sexes. Gender differences in alcohol use encourage costly biases in how societies try to control or reduce alcohol-related problems (Wilsnack & Wilsnack, 1997). (These differences do vary from country to country and from culture to culture.)

ALCOHOL, DRUGS, AND MENTAL HEALTH

Mental and neurological problems are among the most significant contributors to the worldwide burden of disease. Increasingly sophisticated methods to measure health and its burden, in particular, the *disability-adjusted life year lost* (DALY), have helped to provide a more balanced conception of needs and priorities in the area of health for both developing and developed countries. By 1990, the total percentage of DALY attributed to mental and neurological problems was 10.5 percent, including problems such as unipolar and bipolar affective disorders, psychosis, epilepsy, dementia, Parkinson's disease, multiple sclerosis, drug and alcohol dependence, posttraumatic stress disorder, obsessive compulsive disorder, panic disorder, and other problems (Murray & Lopez, 1996).

Alcohol-related problems account for 288 million cases (World Health Organization [WHO], 1999), and they represent a different DALY for men and for women. Nonetheless, some authors have considered violence, mental health, and substance abuse together to ascertain the burden of disease due to the fact that these factors represent overlapping clusters of problems, especially for women (Wyshak, 2000).

Traditionally, women were excluded from most of the early alcohol and drug research; reasons for this early neglect most likely include: the higher (perceived and actual) prevalence of drinking and drug abuse, and of drinking and drug abuse problems, among men than among women; the fact that men's problems with alcohol (e.g., alcohol-related crime, accidents, or work problems) tended to be more visible than those of women; and finally, the greater accessibility of men with alcohol dependence as research subjects (WHO, 1992). Most of the focus on women's bodies is centered on women's reproductive role in society; as a result, much of the available information about the effects of alcohol on women's bodies is limited to issues related to women's reproductive health.

The greater social stigma of alcohol abuse for women may reflect the common and deep-seated cultural fears that point out that if women drink in excess, this will threaten their successful performance of traditional wife-mother roles and will result in unrestrained sexual activity that will upset the traditional male-dominated sexual balance of power. Whatever its source, the stigmatization of women's drinking has clearly contributed to the greater invisibility of women's

alcohol abuse, by causing both women and their significant others to deny or minimize signs of women's alcohol abuse and by discouraging women abusers from seeking help for alcohol problems they do acknowledge (Wilsnack, 1995). (The degree to which this stigmatization has this effect likely varies from country to country and from culture to culture.)

Biological Gender Differences in Alcohol and Drug Use

Biomedical sciences integrate biological, psychological, and social factors. Hence the distinction between sex differences and gender differences in the biomedical sciences is often arbitrary. The use of the expression *biological gender differences* acknowledges that sex and gender are overlapping, potent, and underrecognized mediators of human health and disease.

Alcohol consumption is not an exception. Inborn differences shape each individual's response to alcohol, including the risk of developing complications from alcohol abuse. Some of these inborn differences may be related to gender: women seem to be more vulnerable than men to alcohol-related liver disease; cardiovascular disease, especially, cardiomyopathy; and brain damage ("Are Women More Vulnerable," 1999; Hommer et al., 1996; National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2000).

An overview of alcohol and women (NIAAA, 2000) concludes that the reasons for this vulnerability are not well understood. Differences in metabolism at a finer level and hormonal and cellular differences may lie behind differences in health consequences. One uniformly observed difference between women and men is that women attain higher peak blood alcohol levels than men when ingesting the same dose per kilogram of body weight. The higher peak blood levels are mainly related to the distribution of alcohol—a water-soluble substance—in the woman's body, with a smaller body water content, as compared to the man's body.

Studies have also differed on whether alcohol elimination rates—a reflection of how quickly the enzyme alcohol dehydrogenase (ADH) in the liver processes alcohol—are different for women and men. No consistent changes in rates of alcohol elimination have been found during the various phases of the menstrual cycle (Gil, 1997); however, higher levels of acetaldehyde—a toxic by-product of alcohol metabolism—were evident after alcohol ingestion in women during high estradiol phases of the menstrual cycle or in women taking oral contraceptives (Erickson, Fukunaga, Sarkola, Lindholm, & Ahola, 1996).

Recent studies indicate that women have larger liver volumes per unit of body weight than men, which could result in higher rates of alcohol elimination in women when expressed per kilogram of body weight (Kwo et al., 1997). The

consequence is that a larger liver will have more ADH available to metabolize alcohol.

In summary, women have a lower body water content than men of similar body weight so that women achieve higher concentrations of alcohol in the blood after drinking equivalent amounts of alcohol. In addition, women appear to eliminate alcohol more quickly from the blood than men. This finding may be explained by women's higher liver volume per unit of lean body mass, because alcohol is metabolized almost entirely in the liver ("Are Women More Vulnerable," 1999). Additional research is needed to clarify the relative alcohol metabolic rates in women and men and the mechanisms behind gender-related differences.

The existence of a genetic vulnerability to alcoholism continues to be debated in the alcoholism research field, in spite of the fact that some studies support the notion that some genetic mediation occurs and that alcoholism is as strongly genetically influenced in women as it is in men. Since alcoholism does not follow the simple rules of Mendelian inheritance in multigeneration pedigrees, it is clear that alcoholism is a genetically complex disorder, influenced by multiple genes that interact in an unknown fashion with each other and with similarly unknown environmental factors to produce the disease (NIAAA, 2000; Svikis, Velez, & Pickens, 1994).

Another example is the effect of combinations of gender differences in drinking styles and gender differences in reactions to stress. For instance, alcohol may play a direct role in cardiovascular disease mortality related to the physiological effects of binge drinking (more common in men) on the cardiovascular system, including the precipitation of sudden death. It may also play an indirect role via the methods that men and women use to cope with stress and depression (Holmila & Raitasalo, 2005).

Regarding reproductive health, alcohol abuse and alcohol dependency are associated with disorders in both men and women. Alcohol-dependent women may have several dysfunctions such as disorders of the menstrual cycle, including amenorrhea, dysfunctional luteal phase, anovulation, and in some cases, early menopause. Most available information about alcohol effects on reproductive function has been derived from clinical studies of alcohol-dependent women during sobriety (Mello, 1988).

Women with alcohol problems are at higher risk of getting sexually transmitted infections such as HIV, leading to AIDS; this is especially important if they or their partners have multiple sexual partners. Furthermore, alcohol suppresses the body's immune system and influences individuals' reasoning and decision-making skills concerning sex. In addition, a woman with an alcohol problems is more likely than other women to be in a violent relationship, and she may be forced to have sex against her will (WHO, 1992).

Pregnancy

Available research information linked to maternal alcohol use is based on data collected in the United States, and there may be a number of biases when such information is applied to other populations, including issues like polydrug use, environmental confounds, and cultural and ethnic facts that affect both alcohol use and drug use and outcomes in mothers and children (Boyd, 1999; Coles, Russell, & Schuetze, 1997); however, in other countries, this information is scarce.

According to Burd, Cotsonas-Hassler, Martsolf, and Kerbeshian (2003), fetal alcohol syndrome (FAS) is a common cause of developmental disability, neuropsychiatric impairment, and birth defects. FAS is identified by the presence of a growth impairment, central nervous system dysfunction, and a characteristic pattern of craniofacial features. The reported prevalence of the disorder varies widely, and recent estimates approach 1 percent of live births. A newborn baby with congenital FAS can develop higher rates of comorbid conditions: attention deficit–hyperactivity disorder (40%), mental retardation (15% to 20%), learning disorders (25%), speech and language disorders (30%), sensory impairment (30%), cerebral palsy (4%), and epilepsy (8% to 10%). Birth defects are common. In the United States alone, the annual birth cohort of persons with FAS could be as high as 39,000 cases yearly.

The overall incidence of FAS in advanced industrial societies is 1 in every 750 children. In South Africa, FAS is thought to be by far the most common cause of mental disability: a 1985 study found that the incidence of FAS in Cape Town is 1 per 281 live births. When examining disadvantaged communities, the incidence of fetal alcohol effects is likely to be much higher (Parry & Bennetts, 1999).

Drug Abuse

Drug abuse research has concluded that there are differences in genetic, physiological, psychosocial, and environmental dimensions of drug abuse and addiction between men and women (Hanson, 2002). The neurobiological basis for drug abuse and addiction is essentially the same, regardless of the drug taken or the person who is taking it. But men and women may differ in their biological and behavioral responses to drugs. Women typically progress to dependence from first use of cocaine, heroin, or marijuana faster than men. Additionally, cocaine-induced cognitive impairments and risk for stroke have been found to be more severe in men than in women. Among the risk factors for using drugs are depression and conduct disorders, which are more pronounced in women than in men.

Women who use illicit drugs also are more likely to experience adverse obstetric and perinatal outcomes than women in the general population. Differences are seen depending on the type of illicit drug used. The most studied case is that of cocaine. Following some recent research, the size of babies born from mothers using cocaine is smaller in all parameters (size, weight, volume), their mothers' pregnancies are likely not to reach full term, the size of the babies is smaller than their gestational ages suggest.

Despite the evident need for adequate care of pregnant alcohol- and drug-abusing women, there is a strong consensus among health professionals concerned with this topic: they affirm that criminalization of alcohol and drug use in pregnancy is misguided and counterproductive (Feinman, 1992).

Adequate prevention requires that obstetrical personnel identify those women who have drinking problems early during their pregnancies or, preferably, before pregnancy (Blume, 1988). The challenge for future policies is to reduce the prevailing punitive and stigmatizing stances concerning pregnant substance-abusing women, while also working to increase the availability of appropriate and sensitive treatment services for this population (Wilsnack, 1995).

SOCIAL ASPECTS OF FEMALE ALCOHOL AND DRUG ABUSE

Attitudes toward drinking and abstention seem to be shaped at an early age by external influences and role models. Especially among women, family influence plays an important role in outlining viewpoints on drinking and abstention, and abstainers are more likely to come from abstaining families. Reasons for abstaining from alcohol are numerous and complex; according to each culture, abstention can be a matter of personal taste, a question of religious faith, or a health issue.

In many societies, the use of alcohol by women is strongly disapproved or prohibited; this tends to restrict alcohol consumption, for example, limiting women's drinking to their homes or forcing them to do it in private. Furthermore, given the differences in drug response between men and women, some authors have emphasized the need to have different measures for male and female patterns of drinking. Though countries vary in their traditions of measuring alcohol patterns, it is unusual to find these different measures.

Gender differences in the use and effects of alcohol may be strongly influenced by sociocultural factors. In the developing countries, these factors can range from traditionalism to more recent social transitions derived from development and the influence of globalization. Although some progress has been

observed in the developed world, full gender equity has not been attained. In developing societies, feminism has reached, at most, middle-class educated women, but other transitions have greatly influenced the situation of poor women, among whom migration is outstanding.

Men's national migration to urban locations and foreign migration have resulted in a familiar reshaping, where women who are left behind must take on some other roles, such as outside-household work, and they must accept the change in their traditional functions (Salgado de Snyder, 1993). The strengthening of women's social and economic positions has increased their ability to cope with problems, but at the same time, it has increased tensions between men and women (Szas, 1998). This acculturation has also created a gender impact on drinking practices for those who migrate.

Caetano and Medina-Mora (1988) describe the way in which, when Mexican men cross the border to the United States, they modify their drinking patterns, and after living in the United States for at least five years, they increase their frequency of consumption and maintain the high quantities of alcohol associated with drinking occasions common in Mexico. In the case of women, length of stay or a woman's generation within the United States has less impact on levels of consumption; rather, the increase in consumption is explained by the degree of acculturation to the American way of life.

Substantial changes in the position of women in society have recently raised the question, known as the *convergence hypothesis*, of whether women, when they become more like men in many respects, will also adopt negative behaviors that used to be more typical for men.

Around the world, men drink more frequently than women, and the latter are subject to more social rejection when they develop drink-related problems. Nevertheless, drinking among women is increasing, and women's share in alcohol-related problems is also growing. Women tend to start abusive drinking much later in life than men, and it is likely that they also develop alcohol-related problems later. However, once alcohol dependence is established in women, the harmful effects tend to progress faster. This phenomenon has been called *telescopic*, and it suggests that when men and women consume the same quantities of alcohol and/or drugs, women will develop health problems earlier, and they will have greater problems related to its consumption.

LIFE CYCLE AND ROLES

The roles people have are historical, and as a consequence of that, they change. They are related to the life cycle of men and women and to their social status and geographical location. *Life cycle* refers to a series of prospects and tasks

that, in an ideal way, women must carry out as they arrive at a certain point of the life cycle; these stages are a construction based on biological changes as well as on social and cultural issues. Roles also vary between rural and urban areas and within generations.

Typically, in all cultures, alcoholic beverages are used more by men than by women and more by young adults than by preadolescents or elderly people. Hence, in any society, the major consumers of alcoholic drinks are more likely to be young men, between their mid-teens and their mid-thirties. Men's drunken comportment usually is more exaggerated and potentially more explosive than that of women, regardless of relative ethanol consumption (Marshall, 1979).

Gender differences in drinking behavior have been observed and explained using the *role theory*, according to which each man and each woman can have at least two types of roles: status and positional.

Status roles involve social expectations based on characteristics that the individual cannot easily influence. From this point of view, gender is considered a status role, along with age, religion, and social class. Status roles determine the appropriate "wetness" of situations. Thus the amount and style of drinking considered appropriate in certain situations may be different for women and men (Knibbe, n.d.).

Positional roles are expectations related to positions in social networks such as work and family. Positional roles structure the everyday life of individuals, and they determine into what kind of situation an individual may enter. From a sociological point of view, the life span can be viewed as a sequence of positional roles. Marked differences in the life courses of men and women may be expected to lead to differences in positional roles and their effects on drinking behavior (Neve, Lemmens, & Drop, 1997).

Traditional Roles

Roles and stereotypes are interconnected. Negative stereotypes about women who drink heavily come from the double standards for women and men that are prevalent in any society, and from social and cultural expectations that state that women are responsible for certain roles within the family.

According to Wilsnack and Wilsnack (1997), Cazes (1994), and Guttman (1997), drinking has often served men as an indication of manliness: by their behavior when drinking heavily, men can or may be forced to demonstrate endurance, self-control, nonconformity, and willingness to take risks. Drinking by male groups, typically in all-male settings, has often been an essential way for men to escape from the control exerted by others, to ignore social differences, to gain social support, and to form strong personal ties between them;

male drinking groups constitute a space of equality. Consumption is socially tolerated and used as a coping strategy to solve problems. Especially among men, employment has a positive influence on alcohol consumption.

Women's drinking is more often discouraged or inhibited because of the fear that it will adversely affect their behavior and their social role of *being for others*. In many cultures, it is often feared that women's intoxication will reduce social control of their sexuality by making women either more sexually disinhibited or more vulnerable to sexual insinuations (Leigh, 1995). Women's drinking has also been restricted or hidden because intoxication was thought to be a signal of a dangerous failure of social control over women's family obligations and public behavior, a fact that threatens the social order. Another reason for condemning women's drinking is motherhood, which is viewed as an overrated and sometimes unique role: "The hand that rocks the cradle shouldn't be tipsy."

A man's sex role is affronted by having an alcoholic wife who has asked for help. How can a man maintain his image of male dominance if his wife is out of control because of alcoholism? The high desertion rate of husbands of alcohol-abusing women may be partly attributable to these common beliefs. Unnecessary or unrestrained drinking, which might be allowed or even encouraged in the case of men, has often led to condemnation, punishment, and ostracism for women, perhaps as a reminder of the social power imbalance between men and women (Wilsnack & Wilsnack, 1997).

Common traditional sex role stereotypes prescribe that women are mostly valued for their nurturing functions and their roles as wives and mothers. Consequently, an alcoholic woman will tenaciously try to hide her alcoholism to keep her status based on those roles (Downing, 1991). As a result of these stereotypes, there is a stigma against women who use alcohol. This makes it more difficult for a woman to admit to herself and to others that she has a problem with alcohol, and she may try to hide her alcohol use or to downplay it. On the other hand, women internalize these negative stereotypes: they are more likely to feel ashamed or guilty about their substance use (they may feel that they are failing in their roles as partners and mothers). The aforementioned has a negative effect on their self-esteem and can lead to increased consumption (Addiction Research Foundation, 1996).

In some cultures, women can also have the role of being producers and distributors of alcohol, but availability of beer and wine does not constitute a stimulus for women to drink heavily or have alcohol problems. Researchers who have occasionally raised the question seem invariably to meet a typical woman's response: "The only trouble we have with alcohol is what some of our men do when drunk" (Heath, 1991, pp. 180–181).

Modern Social Roles

At least since the beginning of the 1970s, a growing number of studies have researched the influence of social roles on alcohol consumption. Research results remain controversial. For example, some of them maintain that multiple roles, such as paid employment, marriage, and having children (*role overload*), can lead to increased alcohol consumption in women. In contrast, another theory proposes that *role deprivation*, the lack of social roles such as marriage, work, and having children, has the effect of leading to less social contact and to isolation, further leading to increased alcohol consumption (Addiction Research Foundation, 1996; Bloomfield, 1999). These results are valuable and interesting but do not necessarily reflect the research needs of other countries.

It is necessary to think in terms of the dual roles many women play in monoparental families, one typical characteristic of many family units living in poverty (Salles, 1994), and, increasingly, above the poverty level as well. Men can be absent for different reasons: migration, extensive labor journeys, abandonment, death, or simply because they have never been present. In these families, women might have both role overload (being single women, earning low salaries in paid employment and domestic work, having children out of school) and role deprivation (fewer social contacts, poverty, and isolation). Insofar as developing countries go, this is the reality of many households. This reality points to the need to rethink words such as *family* and *sex roles*, terms that sometimes tend to be used in an uncritical way, as taken-for-granted concepts. Undoubtedly, historical, geographical, economic, generational, and cultural factors have a profound influence on the roles people play. It is necessary to link these concepts with notions such as the sex-gender system; sexual discrimination; and social inequalities of sex, race, and class (Ettore, 1992). An in-depth reflection on these topics could promote research projects without gender biases.

VIOLENCE

Violence against women is a structural phenomenon that has been deeply rooted—for a thousand years or more—in patriarchal culture. Oppression and inequity have been predominant elements in women's lives for centuries. Power, control, and supremacy have been common features of the relations men have traditionally maintained with women.

Now days, domestic violence is considered a serious problem in most societies, so much so that domestic violence is almost universally ranked as a major public health problem having deep economic and social implications. In recent

years, the international community has acknowledged the urgency of responding to this phenomenon by means of several international mechanisms that regulate and punish violence against women, taking it as an attack on and a violation of their human rights. The Inter-American Convention to Prevent, Sanction, and Eradicate Violence against Women (Convención Interamericana para Prevenir, Sancionar y Erradicar la Violencia contra la Mujer, Belém do Pará, Brazil, 1994) acknowledged domestic violence as a problem of human rights, as a crime that governments must face through multiple actions.

This convention defined violence against women as "an offense to human dignity and an expression of historically unequal power relationships between men and women, which has pervaded to all sectors of society, regardless their class, age and religion, and its eradication constitutes a *sine qua non* for development" (Convecion Interamericana, p. 1)

Violence against women is expressed in several forms, among which intrafamily violence is only one. However, at the social level, it includes values, customs, language, symbols, and behaviors, which are accepted and transmitted from generation to generation, establishing and legitimizing as natural unfair behaviors and actions. Clinical studies have found elevated rates of both physical abuse (domestic violence) and childhood sexual abuse in the histories of alcoholic women in treatment as well as elevated rates of alcohol abuse and dependence in mental health clients with histories of childhood sexual abuse. Long-term effects of child abuse, in addition to substance use disorders, can include self-destructive behavior, anxiety, depression, poor self-esteem, difficulty trusting in others, and hostility (Beitchman et al., 1992; Fox & Gilbert, 1994).

Widom, Ireland, and Glynn (1995) consulted court records to identify cases of childhood physical or sexual abuse. These researchers found that for the case of women, a history of childhood neglect, but not abuse, significantly predicted the number of alcohol-related symptoms experienced, independent of parental alcohol or other drug problems, childhood poverty, race, and age. Wilsnack, Vogeltanz, Klassen, and Harris (1997) found that after controlling for the effects of age, ethnicity, and parental education, women with histories of child sexual abuse were significantly more likely to report recent alcohol use, intoxication, drinking-related problems and alcohol dependence symptoms, depression and anxiety, pain that prevented intercourse, and consensual intercourse before the age of 15 than women without these experiences. Alcoholism cannot be attributed to child sexual abuse or child physical abuse per se without taking into account other possible contributing factors. However, there is a higher likelihood of alcohol problems in women if they were sexually or physically abused as children (Langeland & Hartgers, 1998).

Many studies show a high rate of alcohol abuse among men who batter their female partners. Yet no evidence supports a cause-and-effect relationship between the two problems. Battering is a learned behavior and is not the result of substance abuse or mental illness (Browne-Miller, 2007, pp. 14 and 168). Men who batter frequently tend to use alcohol as an excuse for violence. However, there are many abusive men who do not drink heavily, and conversely, many alcoholics do not beat their partners.

Alcohol is the main drug used by the aggressor/partner at the time of the aggression. The aggressor/partner is often unemployed or has no fixed income, while the woman is often economically dependent on him and fears his threats to kill her if she leaves him. When women are pregnant, aggression is frequent. Women often report men changing because of "aversion" to the "pregnant body," because they are jealous of the future baby, or because they have suspicions about paternity (Monteiro & Oliva, 1993).

DIAGNOSIS

Specific gender-related factors for the diagnostic evaluation of alcohol and drug abuse (or dependence) have not been recommended. There is emerging evidence that the lack of specific gender-related diagnostic criteria may compromise the quality of psychiatric evaluations (Mendelson & Mello, 1998). Women may underreport alcohol consumption while pregnant, and direct questions about drinking may provoke denial (Ernhart, Morrow, & Sokol, 1988). Also, medical biases are established against those women who will never be mothers and who are older, although epidemiological data point out that in some countries, such as Mexico, women begin to drink precisely in their fourth decade of life, when they feel they have moved beyond their period of fertility.

On the other hand, health professionals tend to deny women's alcohol consumption as well as the occurrence of other problems such as violence or HIV. As Morse, Gehshan, and Hutchins (1997) state, "everyone agrees that these problems exist—but not in their practice" (p. 3). As a result, inquiring about drug and alcohol use is often neglected, but especially when providing prenatal care.

Brief instruments, such as the TWEAK (an acronym for *Tolerance* [number of drinks to feel high, number of drinks one can hold], *Worry* about drinking, *Eye-opener* [morning drinking], *Amnesia* [blackouts], and *Cut* down on drinking [K/C]), which incorporates questions about tolerance to alcohol, can be more sensitive than other instruments to a woman's risk of drinking during pregnancy (Russell, 1994).

BARRIERS TO TREATMENT AND TREATMENT MODALITIES

Recovery is a long-term process that involves every aspect of a person's life, including economics, relationships to others, and a redefinition of personhood. Gender is a major element in the way the recovery story develops, the way shame impacts recovery, and how social support materializes and can be—and is—accessed differently by women and men (Davis, 1997). Designing strategies to cope with family, spouses, and/or friends who act as barriers to recovery may be an important skill to develop.

Women face many gender-specific barriers that may limit their access to alcoholism treatment. These barriers can be internal and external. The latter can be further differentiated into interpersonal and structural barriers. In general, there seem to be differences between men and women in how they find their way to treatment. Problems in the workplace often push men to treatment, while in the case of women, family-related issues are often the most prominent (Makela et al., 1996). Also note that alcoholic women are more likely to get divorced when they enter treatment, or to get married to or live with an alcoholic significant other, than men (Gundmundsdottir, 1999; Romero & Durand, 2007). They are more likely than alcoholic men to link the onset of pathological drinking to a particularly stressful event (Blume, 1988).

Women are more likely to have histories both of suicide attempts and of previous psychiatric treatment. Their main reasons for entering treatment and the problems they perceive as related to alcohol are more likely to be health and family problems, whereas in the case of men, job problems and troubles with the law, particularly arrests for driving while intoxicated, are more prevalent.

Various studies have shown that women have histories of other drug dependencies along with their alcoholism—particularly dependence on tranquilizers, sedatives, and amphetamine (Graham & Wilsnack, 2000; Romero, Medina-Mora, Villatoro, & Durand, 2005). Female alcoholics are also more likely to have symptoms of psychological distress, such as anxiety and depression, and to have lower self-esteem than their male counterparts (Gundmundsdottir, 1994), but men are diagnosed more frequently with antisocial personality (Hesselbrock, 1991).

Women used to be underrepresented in alcoholism treatment (Dawson, 1996; Weisner, Greenfield, & Room, 1995; Weisner & Schmidt, 1992). However, according to Green (2006), recent studies suggest that gender either has no effect on treatment initiation or, if it has an effect, women are more likely than men to initiate treatment. Now women appear at least as likely as men to engage in and complete a treatment. On the other hand, men and women are

equally likely to complete treatment, but women who complete are nine times more likely to keep themselves abstinent than women who do not; men who complete treatment are only three times more likely to keep themselves abstinent than men who do not. Current research suggests that women's treatment outcomes are as good as, or better than, men's.

Women in substance abuse treatment are less likely to suffer a relapse than men in treatment. When women relapse, their reasons for going back to substance abuse differ from men's. According to Walitzer and Dearing (2006), relapse rates are similar across genders. Although negative mood, childhood sexual abuse, alcohol-related self-efficacy, and poorer coping strategies predicted alcohol relapse, gender did not moderate these effects. Gender did moderate the association between marriage and alcohol relapse. For women, marriage and marital stress were risk factors for alcohol relapse; among men, marriage lowered this risk. This gender difference in the influence of marriage on relapse may be a result of partner differences in drinking problems.

Alcoholic women are more likely to get married to heavy-drinking partners than alcoholic men (Romero, Mondragón, Cherpitel, Medina-Mora, & Borges, 2001); thus alcoholic women may be put at risk of relapse by marriage, and alcoholic men may be protected by marriage. Few studies document gender differences in substance abuse relapse, and consequently conclusions, are limited and tentative. In contrast to the lack of gender differences in alcohol relapse rates, women appear less likely to experience relapse to substance use compared to men. Women relapsing to substance use appear to be more sensitive to negative affect and interpersonal problems, and they are more likely to relapse when their romantic partners are substance users (Covington, 2002).

Pregnant, Alcohol-Abusing Women

Pregnant, alcohol-abusing women must be provided with comprehensive addictive and obstetrical care and psychosocial counseling. Care must be given in a supportive, proactive, and nonjudgmental way in order that women can know that sharing confidential information with health care providers will not render them liable to criminal prosecution. They should be evaluated in a hospital setting, where a complete history and physical examination should be performed, and target laboratory tests should be carried out to evaluate the woman's overall health status. Maternal-infant attachment should be fostered both prenatally and postpartum; emphasis should be placed on enhancing parenting skills; and the ability of the mother to care for the infant after discharge from the hospital should be assessed by frequent observation at home and in clinical settings (Boyd, 1999; Finkelstein, 1993; Finnegan & Kandall, 1992).

CONCLUSIONS

The majority of addiction research, literature, treatment, and clinical practice is still based on male experience. Unfortunately, stereotypical views of women, lack of acknowledgment of gender differences and gender-based needs, and gender inequalities and inequities are present and based on gendered societies that, with frequency, exclude, or at least downgrade in significance, women's lives and experiences (Covington, 2002). Health staff, legal professionals, and policy makers need to be fully sensitized to a gender framework to have alternative and more realistic points of view of alcohol and drug use among women, and among men as they relate to women.

NOTE

1. The Gender Development Index is a good example; this index is a measure of human development that adjusts the Human Development Index to reflect inequalities between men and women along three different dimensions: a long and healthy life, knowledge, and a decent standard of living (PAHO, 2007).

REFERENCES

Addiction Research Foundation. (1996). *The hidden majority: A guidebook on alcohol and other drug issues for counselors who work with women*. Toronto: Author.

Afifi, M. (2007). Gender differences in mental health. *Singapore Medical Journal, 48*, 385–391.

Are women more vulnerable to alcohol's effects? (1999, December). *Alcohol Alert, 46*. Retrieved May 13, 2007 from http://pubs.niaaa.nih.gov/publications/aa46.htm

Beitchman, J. H., Zucker, K. J., Hood, J. E., Dacosta, G. A., Akman, D., & Cassavia, E. (1992). A review of long term effects of child sexual abuse. *Child Abuse & Neglect, 16*, 101–118.

Bloomfield, K. (1999, March 24–26). *Alcohol consumption and alcohol problems among women in European countries*. Paper presented at the European Conference on Alcohol and Gender in Europe, Berlin, Germany.

Blume, S. (1988). *Alcohol/drug dependent women: New insights into their special problems, treatment and recovery*. Minneapolis, MN: Johnson Institute.

Boyd, S. (1999). *Mothers and illicit drugs: Transcending the myths*. Toronto: University of Toronto Press.

Browne-Miller, A. (2007). *To have and to hurt: Seeing, changing or escaping patterns of abuse in relationships*. Westport, CT: Praeger, pp. 14 & 168.

Burd, L., Cotsonas-Hassler, T. M., Martsolf, J., & Kerbeshian, J. (2003). Recognition and management of fetal alcohol syndrome. *Neurotoxicology and Teratology, 25*, 681–688.

Burke, M. A., & Eichler, M. (2006). *The bias free framework: A practical tool for identifying and eliminating social biases in health research.* Geneva, Switzerland: Global Forum for Health Research.

Caetano, R., & Medina-Mora, M. E. (1988). Acculturation and drinking among people of Mexican descent in Mexico and the United States. *Journal of Studies on Alcohol, 49*, 462–471.

Cazes, D. (1994). La dimensión social del género: Posibilidades de vida para mujeres y hombres en el patriarcado. In M. A. Porrua (Ed.), *Antología de la sexualidad humana* (pp. 335–388). Mexico City, Mexico: Consejo Nacional de Población.

Coles, C., Russell, C., & Schuetze, P. (1997). Maternal substance use: Epidemiology, treatment outcome, and developmental effects: An annotated bibliography, 1995. *Substance Use & Misuse, 32*, 149–168.

Convington, S. (2002). Helping women recover: Creating gender-responsive treatment. In S. L. Straussner & S. Brown (Eds.), *The handbook of addiction treatment for women: Theory and practice* (pp. 1–17). San Francisco: Jossey-Bass.

Convención Interamericana para Prevenir, Sancionar y Erradicar la Violencia contra la Mujer "Convención de Belém do Pará", Brasil, 1994. p. 1. Available at http://www.inmujeres.gob.mx/dgaai/belem_do_para/index.html

Davis, D. (1997). Women healing for alcoholism: A qualitative study. *Contemporary Drug Problems, 24*, 147–177.

Dawson, D. (1996). Gender differences in the probability of alcohol treatment. *Journal of Substance Abuse, 8*, 211–225.

Downing, C. (1991). Sex role setups and alcoholism. In Van Der Bergh (Ed.), *Feminist perspectives on addictions* (pp. 47–60). New York: Springer Publishing.

Doyal, L. (2000). Gender equity in health: Debates and dilemmas. *Social Science & Medicine, 51*, 931–939.

Erickson, C. J., Fukunaga, T., Sarkola, T., Lindholm, H., & Ahola, L. (1996). Estrogen-related acetaldehyde elevation in women during alcohol intoxication. *Alcoholism: Clinical and Experimental Research, 20*, 1192–1195.

Ernhart, C. B., Morrow, M., & Sokol, R. J. (1988). Underreporting of alcohol use in pregnancy. *Alcoholism: Clinical and Experimental Research, 12*, 506–511.

Ettore, E. (1992). *Women and substance use*. New Brunswick, NJ: Rutgers University Press.

Evans, T., & Brown, H. (2003). Road traffic crashes: Operationalizing equity in the context of health sector reform. *International Journal of Injury Control and Safety Promotion, 10*(1), 11–12.

Feinman, C. (1992). *The criminalization of a woman's body*. New York: Harrington Park Press / Haworth Press.

Finkelstein, N. (1993). Treatment programming for alcohol and drug-dependent pregnant women. *International Journal of the Addict, 28*, 1275–1309.

Finnegan, L., & Kandall, S. (1992). Maternal and neonatal effects of alcohol and drugs. In J. Lowinson, P. Ruiz, & R. Millman (Eds.), *Substance abuse: A comprehensive textbook* (2nd ed.). Baltimore: Williams & Wilkins.

Fox, K., & Gilbert, B. (1994). The interpersonal and psychological functioning of women who experienced childhood physical abuse, incest and parental alcoholism. *Child Abuse & Neglect, 18,* 849–858.

Gil, J. (1997). Women, alcohol and the menstrual cycle. *Alcohol and Alcoholism, 32,* 435–441.

Graham, K., & Wilsnack, S. (2000). The relationship between alcohol problems and use of tranquilizing drugs: Longitudinal patterns among American women. *Addictive Behaviors, 25,* 13–28.

Green, C. (2006). Gender and use of substance treatment services. *Alcohol Research & Health, 29*(1), 55–62.

Gudmundsdottir, A. (1994, June 4–8). *Women seeking treatment, alcohol abuse and psychological problems.* Paper presented at the 20th annual Alcohol Epidemiology Symposium, Ruschlikon, Switzerland.

Gudmundsdottir, A. (1999, May 3–June 4). *Social and emotional problems of women in alcoholism treatment.* Paper presented at the 25th annual Alcohol Epidemiology Symposium, Montreal.

Guttman, M. (1997). Traficando con hombres. *La Ventana, 8,* 47–99.

Hanson, G. (2002). In drug abuse, gender matters. *NIDA Notes, 17*(2).

Heath, D. (1991). Women and alcohol: Cross-cultural perspectives. *Journal of Substance Abuse, 3*(2), 175–185.

Hesselbrock, M. (1991). Gender comparison of antisocial personality disorder and depression in alcoholism. *Journal of Substance Abuse, 3*(2), 205–220.

Holmila, M., & Raitasalo, K. (2005). Gender differences in drinking: Why do they still exist? *Addiction, 100,* 1763–1769.

Hommer, D., Momenan, R., Rawlings, R., Ragan, P., Williams, W., Rio, D., et al. (1996). Decreased corpus callosum size among alcoholic women. *Archives of Neurology, 53,* 359–363.

Kawachi, S. V., Subramanian, N., & Almeida-Filho, A. (2008). *Glossary for health inequalities.* Retrieved March 13, 2008, from http://jech.bmj.com/cgi/content/full/56/9/647

Knibbe, R. (n.d.). The comparability of alcohol consumption units and alcohol measures in European countries and possibilities for standardization. In *Women and alcohol: A biomed project.* Unpublished manuscript.

Kwo, P. Y., Ramchandani, V. A., Amann, D., Carr, L. G., Sandrasegaran, K., Kopecky, K., et al. (1997). Gender differences in alcohol metabolism are explained in part by computer liver volumen and lean body mass. *Alcoholism: Clinical and Experimental Research, 21,* 51A.

Lagarde, M. (1996). *Género y feminismo: Desarrollo humano y democracia* (Cuadernos Inacabados No. 25). Madrid: Horas y Horas.

Langeland, W., & Hartgers, C. (1998). Child sexual and physical abuse and alcoholism: A review. *Journal of Studies on Alcohol, 59,* 336–348.

Leigh, B. (1995). A thing so fallen and so vile: Images of drinking and sexuality in women. *Contemporary Drug Problems, 22*, 415–434.

Makela, K., Arminen, I., Boomfield, K., Eisenbach-Stangl, I., Helmersson, K., Kurube, N., et al. (1996). *Alcoholics Anonymous as a mutual-help movement: A study in eight societies*. Madison: University of Wisconsin Press.

Marshall, M. (1979). *Weekend warriors: Alcohol in a Micronesian culture*. Mountain View, CA: Mayfield.

Medina-Mora, M. E., & Rojas, E. (2003). Mujeres, pobreza y adicciones. *Perinatología y reproducción humana, 17*(4), 230–244.

Mello, N. (1988). Effects of alcohol abuse on reproductive function in women. In M. Galanter (Ed.), *Recent development in alcoholism* (Vol. 6, pp. 253–276). New York: Plenum Press.

Mendelson, J., & Mello, N. (1998). Diagnostic evaluation of alcohol and drug abuse problems in women. *Psychopharmacology Bulletin, 34*, 279–281.

Monteiro, M., & Oliva, A. P. (1993). Report on women and substance use, HIV/AIDS in Brazil. In *Women and substance abuse: 1993 country assessment report* (pp.59–80). Geneva, Switzerland: World Health Organization.

Morse, B., Gehshan, S., & Hutchins, E. (1997). *Screening for substance abuse during pregnancy: Improving care, improving health*. Arlington, VA: National Center for Education in Maternal and Child Health.

Murphy, E. (2003). Being born female is dangerous to your health. *American Psychologist, 58*(3), 205–210.

Murray, C., & Lopez, A. (1996). *The global burden of disease: Summary*. Cambridge, MA: Harvard University Press.

National Institute on Alcohol Abuse and Alcoholism. (2000). *Alcohol and health: 10th special report to the U.S. Congress*. Retrieved May 13, 2007, from http://silk.gov/silk/niaaa1/publication/10report/10-order.htm

Neve, R., Lemmens, P., & Drop, M. (1997). Gender differences in alcohol use and alcohol problems: Mediation by social roles and gender-role attitude. *Substance Use & Misuse, 32*, 1438–1459.

Pan American Health Organization. (2007). *Gender, health and development in the Americas: Basic indicators*. Washington, DC: Organización Panamericana de la Salud.

Parry, C., & Bennetts, A. (1999). Country profile on alcohol in South Africa. In L. Riley & M. Marshall (Eds.), *Alcohol and public health in 8 developing countries* (Report No. WHO/HSC/SAB/99.9, pp. 139–160). Geneva, Switzerland: Substance Abuse Department, Social Change and Mental Health, World Health Organization.

Reidpath, D., & Allotey, P. (2007). Measuring global health inequity. *International Journal for Equity on Health, 6*(16). Retrieved May 13, 2007, from http://www.equityhealthj.com/content/6/1/16

Romero, M., & Durand, A. (2007). *Mujeres, prisión y drogas: Intervención desde la perspectiva de género y reducción del daño*. México City, Mexico: Instituto Nacional de Psiquiatría.

Romero, M., Medina-Mora, M. E., Villatoro, J., & Durand, A. (2005). Alcohol consumption in Mexican women: Implications in a syncretic culture. In I. Obot & R. Room (Eds.), *Alcohol, gender and drinking problems: Perspectives of low and middle income countries* (pp. 125–142). Geneva, Switzerland: World Health Organization.

Romero, M., Mondragón, L., Cherpitel, C., Medina-Mora, E., & Borges, G. (2001). Characteristics of Mexican women admitted to emergency care units: Alcohol consumption and related problems. *Salud Pública Mexico, 43*, 537–543.

Russell, M. (1994). New assessment tools for risk drinking during pregnancy: T-ACE, TWEAK, and others. *Alcohol Health & Research World, 18*, 55–61.

Salgado de Snyder, V. N. (1993). Family life across the border: Mexican wives left behind. *Hispanic Journal of Behavioral Sciences, 15*, 391–401.

Salles, V. (1994). *Las mujeres en la pobreza*. Mexico City, Mexico: El Colegio de México.

Scott, J. (1996). El género: Una categoría útil para el análisis histórico. In M. Lamas (Ed.), *El género: La construcción cultural de la diferencia sexual* (pp. 265–302). Mexico City, Mexico: Miguel Angel Porrua.

Starfield, B. (2006). State of the art in research on equity in health. *Journal of Health Politics, Policy and Law, 31*(1), 11–32.

Svikis, D., Velez, M., & Pickens, R. (1994). Genetic aspects of alcohol use and alcoholism in women. *Alcohol Health & Research World, 18*, 192–196.

Szasz, I. (1998). Los hombres y la sexualidad: aportes de la perspectiva feminista y primeros acercamientos a su estudio en México. In (Ed.) Lerner S. Varones, *Sexualidad y reproducción* (pp. 137–160). El Colegio de México, México.

Outlook. (1999). Gender inequities and women's health. UNFPA. PATH (Program for appropriate technology in health) *16*(4), 1–8.

Walitzer, K. S., & Dearing, R. L. (2006). Gender differences in alcohol and substance use relapse. *Clinical Psychology Review, 26*, 128–148.

Weisner, C., Greenfield, T., & Room, R. (1995). Trends in the treatment of alcohol problems in the US general population, 1979 through 1990. *American Journal of Public Health, 85*, 55–60.

Weisner, C., & Schmidt, L. (1992). Gender disparities in treatment for alcohol problems. *Journal of the American Medical Association, 268*(14), 1872–1876.

Widom, C. S., Ireland, T., & Glynn, P. (1995). Alcohol abuse in abused and neglected children followed-up: Are they at increased risk? *Journal of Studies on Alcohol, 56*, 202–217.

Wilsnack, S. (1995). Alcohol use and alcohol problems in women. In A. Stanton & S. Gallant (Eds.), *The psychology of women's health: Progress and challenges in research and application* (pp. 51–84). Washington, DC: American Psychological Association.

Wilsnack, S., Vogeltanz, N., Klassen, A., & Harris, T. (1997). Childhood sexual abuse and women's substance abuse: National survey findings. *Journal of Studies on Alcohol, 58*, 264–271.

Wilsnack, R., Vogeltanz, N., Wilsnack, S., & Harris, T. (2000). Gender differences in alcohol consumption and adverse drinking consequences: Cross-cultural patterns. *Addiction, 95,* 251–265.

Wilsnack, R., & Wilsnack, S. (1997). *Gender and alcohol: Individual and social perspectives*. New Brunswick, NJ: Rutgers Center of Alcohol Studies.

World Health Organization. (1992). Programme on substance abuse. In *Women and substance abuse*: *Interim report* WHO/PSA/92.9, (p. 89). Geneva, Switzerland: Author.

World Health Organization. (1999). The newly defined burden of mental problems. Fact sheet No. 217, April, p.3. Geneva, Switzerland.

Wyshak, C. (2000). Violence, mental health and substance abuse—problems for women worldwide. *Health Care of Women International, 21,* 631–639.

This page intentionally left blank

Chapter **5**

Ethical Theory and Addiction

Ann N. Dapice, PhD

The more we learn, through brain imaging and genetics, about the variety of addictions and the numbers of addicted worldwide, the more we realize the moral and ethical challenges we face. We are presented with an illness that results from nature, nurture, or toxins from the physical environment—or a combination of these factors. The use of moral development, value analysis, psychological type, and ethical theories is critical to address the challenge of addiction, whether to tobacco, alcohol, cocaine or other drugs, food, gambling, online pornography, or shopping. The consequences of addiction vary greatly—from chronic disease to sudden death. The social and financial costs of these addictions eventually affect us all.

The addiction illness appears related to once adaptive responses gone astray (Dapice, 2006; Fisher, 2004; Popkin, 2007). The illness causes people to act in ways that can result in danger to themselves and others. What are the ethical responses? If we cannot, or do not, stop addictive behaviors that are dangerous to us and society, whose responsibility and obligation is it to protect all who are endangered? Our jails are full of the addicted as well as the addicted mentally ill, and our statistics from treatment and interventions remain dismal. When the consequences of addiction lead to child abuse and neglect that result in continuing generations of addiction, the question again arises, Whose responsibility is it to intervene? These questions are at the heart of ethical and moral examination.

Until recently, the so-called medical model has generally not placed blame on individuals who suffer from a disease. In the classical model, there has been

an expectation that individuals who have an illness will attempt to obtain treatment and, whether treatments are successful or not, those who are ill will not be held responsible for the success or failure of the treatments. For example, if individuals are diagnosed with cancers not related to known behaviors, and they undergo treatment for the cancers, relapses of the cancer will be seen to be either the result of ineffective treatment, or no effective treatments yet available. This is especially true in that cancer treatment is mainly carried out by physicians and nurses and is not left to cancer victims to treat by themselves. In recent decades, the use of nicotine has been an exception as research came to show clearly the relationship of cancer as well as other illnesses to tobacco use.

Increasingly, however, it is understood that certain lifestyle activities, such as overeating, lack of exercise, and heavy use of alcohol, lead to disease. People are beginning to be held accountable accordingly. Food cravings and even lack of sleep (University of Bristol, 2004) lead to obesity, type 2 diabetes, and cardiovascular disease. We learn how obesity is linked to cancer, and we are warned by Laura Beil (2008) that "among all preventable cancer risk factors, only smoking claims more lives" than obesity (p. 104). We begin to examine questions of obligation and responsibility. Research tells us that use of tobacco affects not only the user but also others through secondhand smoke, and a study at Duke University (2004) documents that prenatal nicotine causes lasting damage, leaving the infant brain vulnerable to injury and addiction upon later use of the drug.

Using values, moral development, and psychological-type research, along with classical and modern ethical theories, this chapter responds to issues of addiction by examining the challenges we face with an ever growing and increasingly diverse body of knowledge on the nature of addiction. Prevention, intervention, and treatment for addiction as well as punishment for illegal addiction also vary greatly and on an increasing basis. The study of moral development and values among humans is required to begin to respond to this complex, multifaceted dilemma. With that knowledge, ethical theory can then be used to analyze the ethical dilemma of addiction that we find ourselves confronting.

MORAL DEVELOPMENT

Building on the work of Piaget, Kohlberg (1984) developed and researched a six-stage model for moral reasoning. Each stage is seen to be qualitatively better:

Preconventional Level (Egocentric)

Stage 1. Avoidance of punishment and deference to power: "eye for eye and tooth for tooth"
Stage 2. Satisfy self and occasionally others (ethical egoism)

Conventional Level (Ethnocentric)

Stage 3. Behave for approval of others; manners and peer pressure
Stage 4. Orientation toward authority and fixed rules: the 10 Commandments

Postconventional Level (Universalizing)

Stage 5. Right action defined after examination; social contract: the U.S. Constitution
Stage 6. Individual conscience and self-chosen ethical principle: the Golden Rule

The addicted, their families, counselors, researchers, policy makers, and lawmakers can all be seen to be at one of three levels and within one of the six stages of moral development. Kohlberg's research showed most people to be at stage 3 of moral reasoning—behaving for the approval of others—with fewer individuals at the lower stages and fewer still at higher stages. In a sense, stage 3 is an ego-ethnocentric stage since it is the approval of one's family and known groups that is sought, not that of outside groups.

Applied to addiction, a person at stage 1 would be dissuaded from activities of addiction out of avoidance of punishment and deference to power. Family members, counselors, researchers, and policy/lawmakers could all reason at this, the punishment/revenge, stage. At stage 2, people will act just to satisfy themselves. An interesting thing happens, however, at whatever stage the addicted individual *can reason:* acting on the addiction moves the individual to stage 2—in spite of possible punishment (stage 1), in spite of lack of approval from others (stage 3) or laws (stage 4)—and even if the individual can reason at a higher stage 5 or 6. In a similar way, families, counselors, researchers, and policy/lawmakers, depending on their moral development, could make moral decisions based on punishment or revenge (stage 1), to satisfy themselves (stage 2), for approval of others (stage 3), because of fixed rules and laws (stage 4), or by determining what is right based on examination (stage 5). Finally, they could make decisions based on conscience and ethical principle (stage 6). True moral and ethical decision making requires development to stage 5 or 6—least represented in Kohlberg's research.

Not only did Kohlberg utilize Jean Piaget's understanding of cognitive development in his theory and research, but later in his life, Kohlberg related moral behavior to Piaget's theories of socialization for autonomy. In studying the relationship of cognitive judgment to action, Kohlberg and Candee (1981) observed that there are two decisions to be made in moral action: (1) the decision of what is morally right (deontic judgment) and (2) the commitment to act (judgment of responsibility) on one's decision of what is right. Kohlberg and Candee found that people who had been socialized for heteronomy (obedience to authority) would make the deontic choice, but might not follow through on the commitment to act. They would assume that the responsibility to act (or ensure action) lies with those in authority. People who had been socialized for autonomy acted on behavior more similarly to stage 5 individuals, even though they could not reason at that stage. They intuited what they could not consciously reason or understand. In general, people at higher stages of reasoning were more likely to carry through on moral action. From this research, we can say that attempts to respond to the problems of addiction will only be taken by those at higher stages of moral reasoning or those who have been socialized to be autonomous and take responsibility. Others may admit there is a problem with the present, largely ineffective response to addiction, but not feel any obligation to take action.

VALUE RESEARCH

Kohlberg's work was based on conscious reasoning in response to a situational dilemma. Dapice (1997) studied more than 6,000 individuals using the Values Unfolding Instrument (VUI) developed to allow access to less conscious values. These individuals came from a variety of backgrounds and included men and women from 27 countries, all races, and six religions, ranging from 14 to 96 years of age. Continuing results from analyzing responses to the VUI are as follows:

1. Initial values stated by individuals unfold to deeper values only 6 percent of the time. As values unfold in response to the question "why is that important?," quite different values and themes develop. Some of the themes may be in direct conflict with each other.
2. While people's values vary tremendously, nearly all values first listed show concern for others (e.g., love, compassion, fairness, etc.—*ideal* values) but unfold to values that are concerned with self (*real* values).
3. One's gender, race, occupation, geographical region, national origin, and religion make no difference to whether one unfolds values that are concerned with others.

4. While people most often first state values with an orientation toward others, when there is a conflict in values, people's behavior matches their unfolded values focused on self (e.g., feeling good, success, status, feeling loved and appreciated).

Therefore the research shows that the large majority of adults (93%) are self-oriented at the less conscious level but consciously believe themselves to be otherwise. They may act in ways that help others, but only if doing so accomplishes values important to them—feeling good, success, feeling loved, and so on. This knowledge is critical to the understanding of how we respond to the challenge of addiction.

The value unfolding research corresponds to what Paul Tillich (1957), a noted theologian, described as our *god values:* the core or central values that exercise power in our lives. These are the values that we give all our time and energy to. Tillich said a god value could be career, power, or prestige, or it could be a love relationship, one's family, an institution, or a larger cause. In the case of addiction, the substance or behavior can become the god value that exercises enormous power in people's lives, allowing people to behave in ways that otherwise they might not.

PSYCHOLOGICAL TYPE

Four different continua compose the Myers-Briggs Type Inventory (MBTI):

Extroverts (E) focus on the outer world of people and things, while introverts (I) prefer an inner world of ideas and impressions.

Sensing (S) types focus on the present, with a preference for concrete information gained through the senses, while intuitive (N) types prefer the future with a perspective of patterns and possibilities.

Thinking (T) types prefer to base their decisions on logic and objective analysis of cause and effect, while feeling (F) types focus on values and subjective information of person-considered concerns.

Judging (J) types want a planned and organized life with decisions made, while perceiving (P) types like a flexible and spontaneous approach to life and prefer to leave their options open.

These four preferences yield 16 different types, for example, extroverted, sensing, feeling, and perceiving. In many ways, the different types experience quite different worlds and realities. In terms of moral and ethical responses, Myers (1980) wrote that each type has positives and negatives, but different

types are "likely to go wrong from different angles" (p. 25). When an introvert breaks a moral principle, "it may be knowingly and with bitterness"; extroverted intuitives and judging extroverts "may consider that the ends justify the means"; extroverted sensing types are most likely to "drift into wrongdoing" (p. 181).

A comparison of people who completed the VUI, the MBTI, and written journals that described their behaviors (all anonymously coded) yielded the following observations (Dapice, 1997):

1. Extroverts have more difficulty taking moral stands if it means a lone decision. Introverts may not sufficiently attend moral issues in the external environment and may avoid moral actions that require interaction with people.
2. Sensing types have more difficulty going beyond their more concrete and fact-bound reality to take on the role of the Other—an ability necessary for mature moral action. Intuitives may play with moral ideas but not act on them.
3. Thinking types often are insufficiently aware of hurting others and thus may have difficulty with empathy. Feeling types may use insufficient reason to project the consequences of their actions.
4. Judging types too often make moral decisions with inadequate information. Perceiving types often do not make moral commitments (decisions) and have difficulty completing tasks related to commitments of a moral nature.

More specific to the data, scores for intuitives are slightly higher on altruistic values, and intuitives are more likely to be universalizing in perspective (Dapice, 1997). Sensing types, as documented by some of the literature on Myers-Briggs types (Keirsey & Bates, 1978), are more likely to be bound to their known reality of family and group. Carl Jung (1923/1971) wrote that each type needs to develop the less preferred side for wholeness and goodness.

MORAL EPISTEMOLOGY—IS, OUGHT, AND IMPERATIVE

Religions have often referred to addictive behaviors in terms of sin: gluttony, drunkenness, lust, sloth, greed, and so on. Accordingly, religions have prescriptives against many so-called sins. However, on the whole, such age-old lists of sins seem not to have been slowed much by the prescriptives. When viewed as once adaptive behaviors instead of sins, what help do we find from ethical theories that can assist in this complex situation we call addiction?

In moral epistemology, there are three levels of understanding (Dapice, 1997):

1. The *descriptive*, which, while once the territory of philosophy and religion, is now generally seen to be grounded in science. Its tools are description,

correlation, and inference. It can pertain to both a particular group and culture. It can also describe research that applies to a universal or global setting.

2. The *prescriptive*, which is the area of philosophy, religion, and law. It is the language of should and ought. Generally, these shoulds and oughts are particular to a certain group, culture, religion, and nation. Examples of this level are prescriptives such as the following: children ought to obey their parents, elders should be honored, and conditions for taking human life (just versus unjust war, capital punishment, self-defense, conditions for divorce, etc.).
3. The *imperative*, which is the area of philosophy, religion, and international (versus national) law. Examples of this include cross-cultural commands such as the Golden Rule, held as an ideal across religions over thousands of years; its philosophical cousin, the Kant Imperative; and international agreements about treatment of prisoners of war and the Nuremberg Code. Imperatives *must* be capable of being generalized to *all* and reflect the essence of *being* (e.g., dignity, justice, love, treatment of other beings, etc.).

Discussion to this point has involved the descriptive science about moral development, value research, and research related to Myers-Briggs types. It has included worldwide or universal findings. In ethical language, the preceding discussion has been what "is" the case about addiction, not what "should" be the case, or what imperatives require in response to this worldwide challenge. Science can tell what is, and it can point to likely consequences, but it takes another layer of analysis to determine what ought to be.

ETHICAL THEORIES

Three questions require response and are critical to the area of addiction:

1. Do we humans have free will, or are we and our actions determined for us?
2. A related question is, Are we to intervene in the world as we find it; in other words, are we to try to make it different or better, or are we to accept it as it is?
3. Knowing what a situation is, how do we decide what it ought to be?

The answers to all three questions are important to the understanding of ethical reasoning.

What is the impact of understandings of free will and determinism? In scientific terms, addiction appears to be overdetermined by both nature and nurture. For society as well as all individuals directly affected, questions of free will and determinism are not simple or frivolous. These understandings have major implications for everyday life as well as for large-scale social policy. If we believe,

for example, that behavior is genetic and thus determined, we will respond differently than if we believe that a person has chosen to behave badly. Either argument can have a variety of responses. In one case, it may be decided that the behavior is genetic and people with those genetic characteristics should not have children—a decision similar to deciding that people who carry genes for severe physical disease should not bear children because of the potential suffering of the children. Or, as scientific consensus grows that the nature of addiction is not solely based on free will or an individual's conscious choice (Saah, 2005), a different response would be that addiction needs to be perceived and treated as a disease with appropriate intervention and treatment.

If, however, it is believed that the behavior is a choice and not predetermined, the addicted are seen as responsible for their actions, and either incarceration or other compensatory action is required. Psychiatrist Amen (2005), after completing many scans on human brains, came to the conclusion that the amount of free will anyone has is determined by the health of the brain—that people with healthy brains have a great deal of free will, and that those with damaged brains (e.g., obsessive-compulsive disorders or drug and behavioral addictions) have significantly less, and those with Alzheimer's disease have almost none.

Increasing numbers of conditions are being classified as genetic and/or physiological in origin. Conditions once thought to be bad behavior are now termed disease. Decisions about responsibility, about who is responsible—and thus societal policy and law—are changing accordingly. In the field of mental illness, we already have ethical questions about whether people with a mental illness have a right to make free-choice decisions to refuse medication that makes their behaviors more socially acceptable—when such medication often causes uncomfortable side effects. Does someone who is mentally ill have the ability to make such a choice if that is part of the nature of the illness? Freedom and responsibility are therefore issues that are at the center of ethics. They are also at the center of how we respond to people who are addicted—and those who may suffer because of the addictive behaviors of others. For this reason, it is critical to use the best science that exists to address the problem of addiction. At this point, many in the United States, but not all, will say that addiction is a disease, yet still blame relapse on the addicted because "they didn't work their program" or "they weren't spiritual enough." With prisons full of people whose only crime is use of drugs or alcohol, it is clear that at this point in time, it is the individual who is held responsible for the addiction.

In the understanding of *psychological egoism*, all human actions are seen to be motivated by selfish desires. This view agrees that people may do things for others—but only as a means to their own happiness. In the description given

previously, psychological egoism is descriptive, not prescriptive. It not only said that all people are selfish, but that they are incapable of being otherwise (Feinberg, 1987). In the Dapice (1997) research discussed previously, it was demonstrated that at the less conscious level, the large majority is self-oriented, *but not everyone is,* and for nearly all persons, the initial conscious values indicate an orientation to others. In other words, people think that they should be concerned for others, or should at least appear to be, even when they are not.

The theory of *ethical egoism* said that people *should* act according to their own self-interests—thus it is prescriptive, not descriptive. Furthermore, ethical egoism holds that this principle must be universalized to all. People should serve their own interests. The "good" that makes up people's interests may be defined as happiness or pleasure, or it may be of a more "enlightened" nature. For example, one might help others to bring benefit to oneself because not doing so would have negative consequences on long-term self-interest (Frankena, 1973). This is considered to be the business ethic—although not all in business use this ethic. Because professionals are expected to work at a higher level of moral reasoning and action, problems occur when professionals begin working for profit-making institutions. Since professionals in the United States are increasingly employed by for-profit corporations, this has become an area of ethical concern.

According to utilitarian theory, we should choose the alternative that will result in the greatest total good for everyone, regardless of the impact—positive or negative—on those making the decision (Hospers, 1982). As in egoism, the good may be considered happiness or pleasure, or it may be of a higher order, such as helping others (Sher, 1987). John Stuart Mill (1987) stressed that the moral agent is required by utilitarianism to be strictly impartial as a disinterested and benevolent spectator. He gave the examples of the Golden Rule and "loving your neighbor as yourself" as ideal utilitarian morality. A special problem with utilitarianism concerns its use to exclude minorities in a democracy. In the name of maximizing the good for the greatest number of people, theologian George Tinker (1996) has written, "Utilitarianism continues to exert its powerful influence on political practice to such an extent that abject racism can thrive, rationalized as being in the best interests of the state" (p. 167).

Utilitarian theory is the ethic of science. Scientific study often necessarily involves actions that may result in good for many, but not for all. A typical example is a study that involves a research control group. Other variables can affect outcomes. If a control group is used and the method shows positive results, those in the control group will not benefit or will have to wait until a later time to benefit—which, in cases of disease, may cause longer suffering or be too late.

For the scientist, there may be a number of motivations. Research has long been a step toward promotion and tenure in the university setting, so self-oriented motivation cannot be ruled out. For the university, it may bring in grants and money. If the scientist is also a physician, for example, the physician's obligation to the individual patient is different from the obligation of the scientist to research, so there is often a legitimate conflict. In addition to the conflict between the ethic of the physician and that of the researcher, the utilitarian perspective can conflict with that of business. Again, as some research and medical facilities have become for-profit concerns, a conflict may exist between the requirements of quality research and those of the business bottom line. In general, scientific studies must be examined, including those reported previously, for possible conflicts of interest.

Deontological theory is the professional ethic. The Golden Rule and the Kant Imperative are the most common examples of deontology. (Also seen by some philosophers as an example of utilitarian theory.) A frequent misinterpretation of the Golden Rule by less mature individuals is that immature people—and societies—often understand and act on "do unto others as they have done unto you" (Kohlberg stage 1, revenge) or "do unto others so they will do for you" (Kohlberg stage 2). Another difficulty of egocentric individuals is that they make behavioral decisions based on their own preferences, that is, "I like chocolate, so everyone should." In addiction treatment, it would be, "it worked for me, so everyone should have this treatment."

Immanuel Kant (1724–1804) attempted to address this problem. His *Categorical Imperative* stated, "Act as if the maxim of thy action were to become by thy will a Universal Law of Nature" (Hospers, 1982, p. 186). In doing so, he clearly universalized the principle (Hospers, 1982, pp. 186–188). Furthermore, Kant stated that all human beings are to be treated as ends in themselves and never merely as means to ends (Albert, Denise, & Peterfreund, 1988).

The deontological understanding that people are to be treated as ends and not means is the nature of the professional agreement. Patients who assume they are being treated according to their own best interests should not be limited to the interventions of a research project in which their physicians may be engaged—at least not without the consent and desire of the patient. Similar conditions apply to therapists, lawyers, ministers, and professors. It is the interest of the client that is the focus. The frequent vulnerability of clients' situations, especially in relationship to the power of the professionals' status and extensive knowledge, requires a special level of trust and confidentiality and an assumption by clients that advantage will not be taken of them. When people go to physicians due to illness, for example, it is important that they know that their physicians will determine the medication or treatment or surgery that is

best for them and not one that their physicians happen to use based on their own convenience or benefit. In addiction, the goal needs to be what will best help the client's problem with addiction, not the preference of the professional, or a one-size-fits-all treatment, as so often happens in addiction treatment. The needs of clients, not professionals, should be reflected in professional codes of ethical conduct.

How big is "all"? The problem of the size of the universe to be universalized is an old one. In historical Greek times, the universalizing perspective excluded women, non–property owners, and slaves in the same way that earlier in U.S. history, voting in the democracy excluded women, blacks, American Indians, and other groups. Part of the ethnocentric problem is that we may not perceive some people as fully human. Sometimes exclusion is purposeful, but often, others unlike oneself just are not seen.

There is yet another understanding of the inclusivity of "all." Tinker (1996) notes that the "Greek word *cosmos*, translated 'world,' is usually interpreted . . . as referring only to the world of human beings." He continues, "The danger of such privileging of human beings should be obvious. It runs the risk of generating human arrogance, which too easily sees the world in terms of hierarchies of existence, all of which are ultimately subservient to the needs and whims of humans" (p. 156). The Lakota phrase *mitakuoye oyasin*, translated as a "prayer for all my relations," is "inclusive not only of immediate family or even extended family, but of the whole of a tribe or nation; of all the nations of two-leggeds in the world; and particularly of all the nations other than two-leggeds—the four-leggeds, the wingeds, and the living-moving things." Humans in this view are seen as part of a whole, who have particular responsibilities to that whole—as do all the "createds" (p. 158). In this sense of Otherness is a special understanding. Violence cannot be done, even through harvesting and hunting for survival, without an act of reciprocation through prayers and ceremonies to restore the balance of existence. Such a perspective gives the understanding of the "do unto others" of the Golden Rule a much larger interpretation. The way in which animals are used in much of scientific research is important to consider here. Humans, of course, are also used in research, but for four-legggeds and two-leggeds, research requires humane treatment. This has too often not been the case. Since plants are also among the living things that make up the universe, we can say that they should not be used abusively or addictively either.

MODERN ETHICAL THEORIES

Rawls's (1971) *distributive justice* has two principles. The first principle is that each person has an equal right to the most extensive basic liberty com-

patible with the liberty of others. What does this mean, then, regarding the addicted and those who may become their victims? The second principle is that social and economic inequalities are to be arranged so that they are both expected to be to the advantage of all and attached to positions and offices open to all (Albert et al., 1988). What does this mean, again, in relationship to professional and legal responses to those who are addicted and their potential victims? How are inequalities arranged in the situation of addictions—their access to research-based treatment, for example?

Frankena's (1973) *theory of obligation* teaches us that we must do good—the results must be good, not just that we want or intend to do good (Frankena, 1973). We ought to do the following:

1. *Not to inflict evil or harm*. Inflicting harm can be seen to be the case when substitutes are given to addicted clients—such as nicotine gum, methadone, or benzodiazepines (Amen, 2005)—which are in and of themselves known to be addictive and can be toxic to the brain. Inflicting harm can also be prescribing addictive pain medications over a long time.
2. *To prevent harm*. The frequent practice of telling addicts that relapse can be expected, although perhaps well intended, can become self-fulfilling prophecy and does not prevent harm to them, their families, or innocent others.
3. *To remove evil or harm*. It is critical that addicted people not engage in activities that are harmful to themselves and others, for example, driving vehicles or abusing others while under the influence or committing crimes to pay for illegal drugs, gambling debts, and so on.
4. *To do or promote good*. It is critical that professionals update their practice related to ongoing addiction research and related treatment and that professionals not just continue old methods of "doing the same thing, expecting different results" in intervention and treatment.

Firth's (1970) *ideal observer theory* has five characteristics of moral decision making. The first, *disinterestedness or dispassionateness*, means being impartial, *not* that one does not care. It means that one has no egocentric or ethnocentric interests at stake. The second, *omniscience*, means that one obtains all the information one can about a situation. The third, *omniprecipience*, means seeing implications and consequences of actions as if one is experiencing them oneself. The fourth, *consistency*, means reacting in the same manner to the same given act. And the fifth, *normality*, means that we are not sick, hungry, tired, cold, overly stressed, and so on, when making a moral decision. This has major implications in jobs and professions where many errors occur due to regressive conditions. It is also a factor in much family and violent crime.

With regard to the first characteristic, being *disinterested*, it is easy to assume that because we are professionals or researchers, we are, of course, not operating

out of self or group interest. That often requires further examination. Do we counsel using our personal likes or dislikes, what our peers say or think, what the professional norm requires in treatment if not based on recent research—as opposed to what is in the client's best interest? The second characteristic, *omniscience*, requires not that we can be all knowing, but that we obtain all the information and knowledge we can to best serve others—that includes ongoing updating of our scientific knowledge and skills so we can better respond to the problem. While most professionals are required to obtain continuing education credits, there is rarely testing of this knowledge to determine the professional's actual understanding of new research needed to more successfully treat clients. The *omniprecipient* characteristic is often difficult for professionals in dealing with clients. Attempts to accept consequences of actions as if we were experiencing them ourselves is where much burnout occurs. There are few resources to support professionals with the exhausting impact of taking on the role of the suffering Other. Often, in misplaced attempts to "not get too close to the client," we use professional distancing techniques that are both well taught and well learned. These serve to protect us from the often painful reality presented to us. How do we manage our own emotions at the same time we use compassion in serving our clients? Another challenge is the human problem that we too often have difficulty understanding experiences until they happen to us. On the other hand, in addiction treatment, there are many professionals who are in recovery themselves. The temptation here is to believe that their experiences are identical to those of the client or that treatment should be the same as theirs was. The *consistency* question arises especially in how we treat people with differing status and power. Do we react in the same manner with each individual regardless of race, religion, socioeconomic status, and so on? Do we treat the addicted differently if they are in powerful or moneyed positions? Are they placed in jail (punished), as many addicted are, or given a choice to check in to a treatment center that they can afford? It would appear, on the other hand, that when people in power are arrested for drunk driving, they may be more likely to make the news. Regarding *normality*, as professionals and humans, we know that sometimes we are too tired or sick or overly stressed to make an ethical decision or help a client make a decision. Outpatient centers are often understaffed and underfunded. In life experiences, people have often been taught that they are to accomplish the work given to them without question. Most employee handbooks do not give time to think about and consider a client problem. This is a pragmatic, but overlooked, consideration.

Each of these theories shows that practicing in a professionally ethical way is not as simple as following a set of rules set by one's profession. These ethical theories provide practical ethical guidelines to remind us what being an ethical person actually requires. And these principles are not limited to professionals. Most of

us were taught the Golden Rule as a standard. These modern ethical guidelines help us to do the Golden Rule better—to do unto others as we would have them do unto us, not as we have too often been done unto. As we learn more about the science of any problem, we are challenged to develop better responses through treatment, policies, and laws to meet our ethical obligations better.

It becomes clear that conscious choice and free will are markedly reduced. On the basis of scientific research of moral development, most, as described previously, are at an unconscious developmental stage 2—seeking to satisfy themselves and, occasionally, others. Even if the addicted can reason at higher levels of development, addictive substances and behaviors, having hijacked their brains (Amen, 2005), cause their victims to act at stage 2. While most people now call addiction a disease, the most frequent intervention and response to addiction is stage 1 punishment and use of power—either in jails, or programs that tend to shame the addicted—for "not working the program." Since clients operating at stage 2 choose the reward of the substance or behavior over fear of punishment or ill effects, this neither works as intelligent operational policy nor as a way to treat a disease. The number of persons who, at a less conscious level, operate with altruism toward others is about 7 percent, making ethical decision and action dangerously low.

Who are the beneficiaries of addiction? There are, of course, many—whether food, tobacco, alcohol, cocaine, prescription drugs, gambling, video games, or pornography. The expectation of business, and distributors of street drugs, is profit. They are at stage 2 business ethic. Similarly, services, whether treatment facilities or prisons, provide employment.

Who suffers? The obvious victims are the addicted, their families, and the innocents affected by child abuse or death on the highway. Less obvious at first are those who suffer from our two largest and most costly addictions—food and tobacco. This suffering includes type 2 diabetes, cardiovascular disease, and cancer. But it also includes the results of a mother's smoking in pregnancy—children with low birth weight, likelihood to addiction, antisocial behavior, and attention deficit–hyperactivity disorder. In behavioral addictions, we see a range of results, from children victimized by Internet pornography, to loss of families and incomes from gambling, to death, to the victims of stalkers. Clearly the addicted meet the longtime standard of being "dangerous to themselves and others"—whether through chronic disease, secondhand smoke, driving under the influence, suicide, or crime to pay for illegal drugs. Whether from insurance rates, taxes, or low productivity of workers, the financial costs to society are staggering.

While law enforcement will generally use punitive measures with those who drive under the influence and who possess illegal drugs, this is unevenly

enforced with women and minorities, who are more likely to be arrested for similar crimes, while others go to treatment. Yet in stalking, there is a curious reversal where the most lethal and violent of criminals are rarely arrested or convicted, even when protective orders have been put in place by the victims (Meloy, 1998). While stage 1 punishments rarely stop the addicted, there is a need is to stop negative and dangerous behaviors while protecting victims from suffering—and even death. Stalkers, like others who are addicted, will say that they cannot stop. Stopping the behavior becomes important for all those who are addicted—as well as for their victims.

Science gives clear evidence from brain scans that the addicted have limited or little free will. Rawls's (1971) distributive justice says that people should have "extensive basic liberty compatible with the liberty of others" (p. 302). The addicted, especially those who believe that their addictions do not harm others (e.g., food and tobacco), most often claim their rights to eat and smoke as they wish. Obesity is a special problem, for which the argument is often given that weight is a matter of choice and society should not cave in to so-called anorexic norms. However, unlike tobacco, it is necessary to eat to live. People cannot stop eating in the same way that they can stop smoking or drinking alcohol or abusing prescription drugs. Obesity is often a delicate matter because people feel embarrassed when weight is mentioned. Meanwhile, the costs of obesity continue to rise, from health costs that we all share to airline reservations where seats are narrower and people are, literally, wider. Whose rights? Whose responsibilities? Beyond economic realities, do people have a right to cause the suffering of others, whether through disease, abuse, or death? If, due to the nature of the disease, they are unable to meet their responsibilities, then what?

The good news is that in only a few years, we have taken a sharp turn in our consciousness about the problems of obesity. There is new understanding of the kinds of foods and nutrients that are needed by the body and brain. Emphasis on obesity has gone public and is now part of public policy—removing soda machines from schools, adding exercise at school, incentives in the workplace, and so on. Schools and entire cities are changing their diets, led by their elected officials. Perhaps the one addiction that we cannot do totally without will lead the way to success in addressing all of the others.

CONCLUSION

The state of addiction is that we have tremendous numbers of addicted and a larger array of addictions worldwide. Suffering and costs are beyond calculation. Brain scans show the amount of damage in the brains of the addicted. This damage can be clearly seen by all. It does not take an expert to tell which is

a normal brain and which is a damaged brain when the pictures are placed side by side. This is especially important to sensing types for whom seeing is believing. Scans show the functional limits of a damaged brain and thus the limits of free will. If choice is beyond the ability of the addicted to make, whose obligation is it to stop the behavior? With a majority of the population at lower stages of moral development, addicted or not, there is little likelihood that decisions will be made based on higher ethical principles. What are the choices? The ethical question is, What *ought* we to do, or what is it *imperative* that we do?

There are options. There are teaching and learning strategies that can be used to encourage professionals—and the addicted, once they are healthier—to move to higher levels of moral reasoning and responsibility (Dapice, Cobb, Hutchins, & Siegel, 1988). Autonomy as a moral agent can also be taught and learned. Meanwhile, because of the great cost of addiction to us all, it is in the self-interest of all of us to take action. Therefore the concept of enlightened self-interest can be taught and used when people are not able to understand or respond to the suffering of others. Research already tells us much that can be done to help treat the addicted that is generally not being done now. These measures cost far less than most addiction treatment does now. And more is learned about addiction with every new study published. It will be necessary that the conflicts of interest of corporations and organizations be set aside. Knowledge must direct decisions about what treatments are used, not financial benefit, not refusal to change, and certainly not those in the world who gain from the weakness, illness, and death of others.

In addiction, the world's biggest killers interact with each other and are at stake: obesity, type 2 diabetes, cardiovascular disease, cancer, family violence, suicide, so-called accidental death, and murder. It is not a disease about "them." We are all affected. We all pay the price. An addiction deontology is imperative. Ethical theory teaches us that we must do good—the results must be good, not just that we want or intend to do good. We ought not to inflict evil or harm. We ought to prevent and remove evil or harm. We ought to do or promote good. Much work remains to be done, but we are not without a map to guide us.

REFERENCES

Albert, E., Denise, T., & Peterfreund, S. (1988). *Great traditions in ethics* (6th ed.). Belmont, CA: Wadsworth.

Amen, D. G. (2005). *Making a good brain great*. New York: Harmony Books.

Beil, L. (2008). Weighty evidence: The link between obesity, metabolic hormones, and tumors brings the promise of new targets for cancer therapies. *Science News, 173*(7), 104–106.

Dapice, A. (1997). *To thine own self . . . Values and ethics in a complicated world.* Skiatook, OK: Institute of Values Inquiry.

Dapice, A. (2006). The medicine wheel. *Journal of Transcultural Nursing, 17*(3): 251–260.

Dapice, A., Cobb, L., Hutchins, E. B., & Siegel, G. (1988). Teaching and learning values. *Educational Horizons, 66*(3), 107–110.

Duke University. (2004). *Prenatal nicotine primes adolescent brain for addiction.* Retrieved March 5, 2008, from http://www.sciencedaily.com/releases/2004/04/040420214434.htm

Feinberg, J. (1987). Psychological egoism. In G. Sher (Ed.), *Moral philosophy* (pp. 5–15). San Diego, CA: Harcourt, Brace, Jovanovich.

Firth, R. (1970). Ethical absolutism and the ideal observer. In W. Sellers & J. Hospers (Eds.), *Readings in ethical theory* (pp. 212–214). Englewood Cliffs, NJ: Prentice Hall.

Frankena, W. (1973). *Ethics* (2nd ed.). Englewood Cliffs, NJ: Prentice Hall.

Hospers, J. (1982). *Human conduct* (2nd ed.). San Diego, CA: Harcourt, Brace, Jovanovich.

Jung, C. G. (1971). *Psychological types* (H. G. Baynes, Trans., & R.F.C. Hull, Rev.). Princeton, NJ: Princeton University Press. (Original work published 1923)

Keirsey, D., & Bates, M. (1978). *Please understand me* (3rd ed.). Del Mar, CA: Prometheus Nemesis.

Kohlberg, L. (1984). *The psychology of moral development: Essays on moral development* (Vol. 2). San Francisco: Harper and Row.

Kohlberg, L., & Candee, D. (1981, December 17–19). *The relationship of moral judgment to moral action.* Paper presented at the Florida International University conference on Morality and Moral Development, Miami Beach.

Meloy, J. R. (1998). *The psychology of stalking: Clinical and forensic perspectives.* San Diego, CA: Academic Press.

Mill, J. S. (1987). *Utilitarianism.* London: Longmans, Green.

Myers, I. B. (1980). *Gifts differing.* Palo Alto, CA: Consulting Psychologists Press.

Popkin, B. (2007, September). The world is fat. *Scientific American, 297*(3), 91–93.

Rawls, J. (1971). *A theory of justice.* Cambridge, MA: Harvard University Press.

Saah, T. (2005). The evolutionary origins and significance of drug addiction. *Harm Reduction Journal, 2*(8). Retrieved March 5, 2008, from http://bmc.ub.uni-potsdam.de/1477-7517-2-8/

Sher, G. (1987). *Moral philosophy.* San Diego, CA: Hartcourt, Brace, Jovanovich.

Tillich, P. (1957). *Dynamics of faith.* New York: Harper and Brothers.

Tinker, G. E. (1996). An American Indian theological response to ecojustice. In J. Weaver (Ed.), *Defending mother earth: Native American perspectives on environmental justice* (pp. 153–176). Mary Knoll, New York: Orbis Books.

University of Bristol. (2004, December 13). Does the lack of sleep make you fat? *ScienceDaily.* Retrieved March 31, 2008, from http://www.sciencedaily.com/releases/2004/12/041206204702.htm

This page intentionally left blank

Chapter **6**

The Religious and Theological Roots of Alcoholics Anonymous

Rev. Linda Mercadante, PhD

Alcoholics Anonymous (AA) prides itself on being "spiritual but not religious." Its ethos asserts that AA takes no position on religion, proclaims no theological doctrine, and promotes no religious adherence. Many members would be surprised to learn, therefore, that the AA 12 Step program is deeply embedded in some quite distinctive religious and theological roots.

Certainly many scholars, and a few AA members, are aware that founder Bill Wilson was for a time involved in the Christian evangelistic Oxford Group. But they assume that because Wilson broke away from it, the Oxford Group had only minimal influence on what would become AA. This chapter claims instead that by examining the beliefs, practices, and assertions of the Oxford Group, one can see direct and long-lasting, yet hidden, theological and practical links between this evangelistic group and AA. This is not to take a stand, pro or con, about this connection, but simply to note how these roots continue to inform the 12 Step method of addiction recovery.

THE OXFORD GROUP HISTORY

The Oxford Group arose in the early 1900s as an independent religious organization meant to revitalize the American church. It hit its peak in the 1920s and 1930s, when it was popular and widely known. It attracted the mighty, the wealthy, and the influential of American society to its program of spiritual revival. The Oxford Group, in many ways, was a throwback to older ideas of sin and conversion. It privatized morality, insisting that private

behavior, rather than negative social conditions, was responsible for human unhappiness and wrongdoing. In fact, the Oxford Group was a reaction against the liberal *social gospel*, which contended that society as a whole needed reform (Mercadante 1996, 189 n. 5).

The Oxford Group was begun by an ordained Lutheran minister, Frank Buchman. From central Pennsylvania, he had received a conservative pietistic German Lutheran upbringing and an education for the ministry. After a formative religious conversion experience in 1908 at a Keswick conference in England, Buchman's charismatic personality began to attract attention. He had tried various forms of ministry—running a settlement house, working as a YMCA evangelist at colleges, serving as an adjunct professor at Hartford Seminary. But in modeling himself after evangelist Dwight L. Moody (Clark 1951, 117–22), eventually, Buchman hit on what would work best for him. Striking out on his own in a ministry of personal evangelism, Buchman soon gathered a group of loyal young people around him. The group was first called "First Century Christian Fellowship" (1908) but soon took the more prestigious name "Oxford Group" (not to be confused with the Oxford movement) since Buchman had tried ministering at that college for a time (Woolverton 1983).

Buchman's message was essentially simple, but in the theological mode of traditional Protestant Christianity mixed with a nineteenth-century revivalist emphasis. Buchman's concise and spare pivotal concepts can be summarized this way: (1) all people are sinners; (2) all sinners can be changed; (3) sincere and thorough confession is essential to any change; (4) once changed, persons can access God directly; (5) miracles are possible; (6) the duty of changed persons is to change others. The group claimed that this was just basic Christianity put into practice. Astute observers of AA should already perceive some similarities between the foundational concepts of the Oxford Group and those of AA's 12 Steps, but this will be elaborated later (Mercadante 1996).

Bill Wilson, who would later go on to found AA, was first exposed to the Oxford Group in 1934 through his friend and former drinking buddy Ebby T. Ebby had become sober through the group and wanted to share his discovery with Wilson. Wilson, in fact, soon became involved in the group when he responded to an altar call at Calvary Episcopal Church and Mission in New York. This was a headquarters of the Oxford Group, led by group leader and Episcopal priest Samuel M. Shoemaker. Shoemaker had a distinct, long-lasting influence on Wilson, and they remained in contact for many years, even after Wilson had moved on to form AA. At AA's twentieth anniversary celebration in 1955, Wilson invited Shoemaker to give one of the major speeches. Also a devoted member of the Oxford Group was Dr. Robert Smith ("Dr. Bob"), who

would go on to cofound AA with Wilson. He had been involved in the group for several years before meeting Wilson. In 1935, Wilson was on a business trip to Ohio when he felt tempted to resume drinking. Through a series of connections, Wilson met Dr. Bob, another struggling alcoholic. The two men found that by helping each other, they helped themselves (Kurtz 1991).

Even though this one-alcoholic-helping-another method would eventually become the heart of AA, it is important to note that the first link between Wilson and Smith was their appreciation and use of conversionist and evangelistic Oxford Group principles. Both men had been committed Oxford Group members for several years. Their breakaway from the group to form what would eventually become AA took place gradually, the final resolution not becoming completely clear until around 1939.

TROUBLESOME CONNECTIONS

Revivalism

Even though many historians of AA are aware of the prior Oxford Group membership of Wilson and Smith, the Oxford Group roots of AA are often played down or only lightly treated when AA history is discussed (Mercadante 1996, 189 n. 3). Before going on to elaborate the connections between the two groups, we should pause to explore some of the reasons this connection is minimized.

The Oxford Group's pietistic Christian roots and revivalist attitude have long been an embarrassment to many people, including those in the church. Even in the group's heyday, revivalism was considered by many to be a nineteenth-century phenomenon, more connected with frontier than with city. It was not seen as a method or emphasis relevant to modern, rapidly industrializing America, with its increasingly apparent and large social problems. Such roots can seem equally problematic today. An organization such as AA, which stoutly insists it is "spiritual but not religious," would not want to be linked to a distinctly religious approach. Instead, it wants to appeal to a more diverse constituency. In a world of increasing heterogeneity, AA would not want to be seen as a derivative Christian program.

Sin and Conversion

Also troublesome to many is the clear-cut Oxford Group stress on sin and conversion. This was a problem from the beginnings of the Oxford Group. As one grouper noted (Grensted 1933, 19), "the very word [sin] is out of date;

it smacks of street-corner salvation meetings; they think only people who are behind the times believe in Sin these days."

Today the concept of sin poses an additional problem for the recovery movement. Science has made us aware of the many unconscious, biological, and inherited factors in human behavior. Addiction has become a scientific concept, linked with theories of genetics, biochemistry, neurobiology, and the like. Although Bill Wilson avoided the term *disease*, he did refer to addiction as a "malady," or illness (Kurtz 1991, 22). Today, this ethos has progressed further, and addiction recovery in general is based on the principle that addiction is a disease, not a sin. In other words, it is a medical, not a moral, problem.

There are some problematic assumptions in this dichotomy, however. The largest is the misperception that sin is primarily about right behavior—that it is moralistic. The Christian doctrine of sin is not about morality, but about relationality. Sin, as a diagnosis of human dysfunction, focuses on alienation first, with behavior following. Thus sin refers to one's orientation toward or away from God, self, and others. Turning away from sin means turning away from destruction and toward human flourishing. The idea of sin also recognizes that humans are in many ways in a bondage they may not always recognize. They need God's grace not only for their release, but even for the true recognition of their plight (Mercadante 1996, 27–39). In many ways, this refined understanding of sin makes it sound much like addiction.

Nevertheless, this refined theological understanding does not change the popular connotation of the word *sin*. When people reject the concept of sin, especially in relationship with addiction, they are most often linking the theological concept with guilt, blame, and free choice. When sin is seen as moralistic, the human will is assumed to be neutral, aware, and fully functional, able not only to clearly distinguish between good and evil, but able to choose one over the other. Yet ever since St. Augustine, this view has been rejected by Christian theology and labeled "Pelagian." Having a Pelagian understanding of sin denies the need for God's grace and credits humans for being totally free and undetermined. Under this mistaken view of sin, alcoholics have chosen their plight and are fully guilty for their condition.

Clearly this Pelagian view presents problems for AA since the program stresses the out-of-control nature of alcoholism. To even suggest that human control over addiction is possible seems to undercut its primary message (Mercadante 1996). We cannot conclude, however, that the Oxford Group held a fully Pelagian view of sin, even though it did emphasize morality. The group did not expect changed behavior until a person changed his or her orientation and turned toward God. Nevertheless, the group was functioning in a

culture that had already begun to distance itself from such Christian concepts as sin, however they might be understood.

Public Perceptions

Another problematic area is the actual history and public perception of the Oxford Group. Over time, this organization fell out of favor with the public (Mercadante 1996, 189–90 n. 7, 8). Not only was it sometimes ridiculed, but important church leaders, such as Reinhold Niebuhr, protested the Oxford Group's methods and principles. This became acute when—before the outbreak of World War II—founder Frank Buchman endorsed Adolph Hitler as a great leader, saying (Niebuhr 1936, p. 1315), "I thank heaven for a man like Adolph Hitler, who built a front line of defense against the anti-Christ of communism. . . . Think what it would mean to the world if Hitler surrendered to the control of God. . . . Through such a man God could control a nation overnight." The Oxford Group tried to shed the bad press by renaming itself Moral Re-Armament in 1938 and by working on issues of political reconciliation (Luttwak 1994). Although this did give it a new lease on life for several decades after the war, many continued to see the Oxford Group as a retrograde organization. AA's departure, right around this time, was just one more aspect of the public distancing.

These and other factors about the Oxford Group–AA connection keep the theological and religious roots of AA buried. Yet the Oxford Group heritage continues to deeply inform the program, principles, and practices of AA. In fact, at least one person who attended Oxford Group meetings with Bill Wilson insists that (Houck 1995, p. 10)

> to leave out the Oxford Group is like saying that the Ford Motor Company began in 1927 with the Model A. . . . The two people who really gave Wilson and Smith their tools were Frank Buchman . . . and . . . Samuel Shoemaker. The latter was Bill Wilson's spiritual mentor, and Bill always referred to him as his chief source. It was from the O.G. that Wilson and Smith discovered the life-changing mechanisms they brilliantly codified into the Twelve Steps and marketed for the benefit of millions.

COMPARISONS OF METHODS AND PRACTICES

Although its message was not new, the Oxford Group prided itself on its contemporary methods and practices. It presented itself as an active group, not mired down in religious controversy. The group stoutly denied that its program

was theological or its practices anything like church. To engage newcomers, they used small groups, personal invitation, one-on-one interviews, interesting entertainment, and "house parties," rather than anything remotely like religious ritual or services. Members' stories of dramatic change were publicized, and members were available to help newcomers and each other at any time. As one scholar notes (Grensted 1933, 5), "'It Works!' was a phrase more frequently heard in the testimonials and endorsements of the Group program and practices." Members grew quite close to each other, doing much more than just meeting together a few times a week. They formed tight social bonds and sometimes even gave up other jobs to join cooperatively in group work.

Unlike traditional evangelism, which often sought out the marginalized or deprived, the Oxford Group focused especially on the well off and well connected. Since they focused on the up and out rather than the down and out, this made some caricature the group as a "Salvation Army for snobs" (Pound 1976/1985). Yet the Oxford Group had this driving force in common with old-time Christian revivalism: they both wanted to awaken a deep sense of individual sin, they both wanted to prompt dramatic conversion, and they both wanted to reform behavior. No matter what a person's background, sin was the common denominator. Whether you "live on Park Avenue or a park bench," they liked to say, the Oxford Group message and practices were relevant.

Group literature assured readers that no one was too far gone to be saved and changed (Grensted 1933, 5):

> Witnesses will go anywhere at any time to help any one whose life has become a burden of Sin, and will stand by until that person has been set free from trouble and brought to a true understanding of Christ and life. No soul has sunk too far, no case for spiritual cure can go beyond their scope. The knowledge that we are in need and have a need for change in our lives is all that is necessary for these life changes to work on.

Those who "changed" through Oxford Group ministrations were expected to remain faithful small group members and then reach out to other hurting people. Volunteers made up the bulk of the helpers, much self-discipline was expected from them, and if they had once harmed others, they were expected to make restitution. Buchman believed that change happened from inside out, starting and ending with individual change. From early on, the Oxford Group took no official stand on social issues. This only changed after 1938, once the Oxford Group reformed to become Moral Re-Armament. Wilson and Smith had split off by that time and were working to create what would become AA.

AA uses many methods similar in spirit to those of the Oxford Group. Meetings are marked by informality, friendliness, and little that seems like reli-

gious ritual (even the traditional AA closing with the Lord's Prayer is, in some places, coming under question). Newcomers are drawn by personal invitation. Testimony is often a core element, and *lead meetings*, where a recovering alcoholic shares his or her story, are both entertaining and interesting. Small group intimacy is a core aspect of the program, and members are encouraged to talk about their problems. Personal support is a primary benefit of commitment, and members are urged to select a sponsor. Members often become friends and form tight social bonds. They are expected to remain loyal attendees of their home group, be ready to help fellow members, take their message to other suffering alcoholics, make restitution, and increasingly exercise self-discipline. AA, like the Oxford Group, keeps its focus on the individual's problem. It takes no organizational stance on social change, but starts and ends with helping alcoholics.

In AA, "working the program" is explained as hard work, but slips and faults are expected. Intense conversation happens before meetings, and members often socialize with each other afterward. Goals, too, are similar to the Oxford Group, including a deep sense of one's plight, conversion to one's Higher Power, and clear-cut behavioral change. AA meetings are often closed with the affirmation "keep coming back; it works!" All three—Christian revivalism, the Oxford Group, and AA—use narratives of dramatic conversion to gain the attention of newcomers and solidify the faith of adherents. And all three see a common problem uniting disparate people (sin for the first two, alcoholism for AA).

Although few writers analyze the theological similarities between the Oxford Group and AA, not everyone plays down the connections. A minority of contemporary writers stress the commonalities as evidence that AA is compatible with traditional Christianity (Mercadante 1996, 188 n. 2). Taking another approach is historian Ernest Kurtz (1991, 49), who insists that early AAs were unaware of the Oxford Group's evangelistic background. Nevertheless, he says, they deliberately borrowed many practices and principles, including submission to God, listening for guidance, checking with others, restitution, confessing one's sins, witnessing to one's conversion, the unpaid status of helpers, and the obligation to help others change.

Is it possible that founders Wilson and Smith could have been unaware of Buchman's Christian orientation? They, and some other early members, had spent formative time in the group when Buchman was dominant, and Wilson kept a close, nearly lifelong connection to Oxford Group principal Reverend Shoemaker. The borrowings that Kurtz mentions are underlaid with many theological assumptions. Therefore it is more likely that the early AAs had a struggle on their hands when they broke with the group. They were disassociating with an organization that had helped them in many ways. They had to

decide how many of the Oxford Group beliefs to explicitly retain, how many to reconfigure, and how many to discard.

It is often thought that Wilson and Smith left the Oxford Group because the group did not make alcoholism a priority or that Wilson's special interest in alcoholics was not appreciated. But a close look at the Oxford Group shows it did not ignore the problem of heavy drinking. Buchman had begun his ministry at Penn State, a hard-drinking school, and he met with much success there. Ever after, he continued to show interest in helping people caught in the snares of alcohol addiction. In fact, the story of the reformed drinker was a favorite one in Oxford Group lore. Ebby T., Wilson, and Dr. Bob, among others, were drawn to the Oxford Group because of members' concern for their alcoholic plight. The Oxford Group expected that miracles and dramatic conversion were possible for anyone. They expected nothing more from a person than a simple desire to change, no matter what his or her particular problems.

COMPARISONS OF BELIEFS

Buchman was generally uninterested in formal theology, and his message was not only simple, but age-old. Groupers refrained from theological argument with prospects, and they insisted they did not care what a person's religious beliefs were. They insisted they "read men, not books" (Clark 1951, 108–10). This anti-intellectual approach is a common American ethos, but observers might note how it is replicated in AA. A similar tendency can be seen in various addiction recovery groups where new members' questions can be dismissed as "intellectualizing." Sometimes recovering persons are told, "Your best thinking got you here," meaning to their addictive plight.

Sin and Disease

Nevertheless, the Oxford Group did focus on several key concepts. Most important was that individual sin was the core problem. Interestingly, especially when compared to the addiction recovery ethos, the Oxford Group often used medical analogies for this. They sometimes referred to new prospects as "patients" (Eisler 1950, 173); spoke about making a "spiritual diagnosis," about confession and conversion as "soul surgery" (Walter 1919); and likened sin to disease. AA carried on this medical terminology from the Oxford Group, but increasingly used it less analogously and more literally over the years as it gained credibility from science. While at first, AA played with the idea that alcoholism was almost like an "allergy" (Alcoholics Anonymous World Services 1976a, xxiv, xxvi, xxviii), later recovery groups began talking about addiction as a potential genetic defect or neurochemical problem.

Yet the content used to define the disease of alcoholism was still very similar to that used for the problem of sin by the Oxford Group. Sin, for Buchman, was anything that stands between the individual and God. It frustrated God's plan for self and other. Sin was primarily against the self and the self's optimum development. Sin would include such problems as hate, greed, fear, and personal shortcomings. Most important, self-centeredness was the heart of sin. They made a big point of showing how "I" stands at the center of the word *sin*. The group's focus on confession and brutal honesty were part of the soul surgery (Mercadante 1996).

AA and modern addiction recovery groups have a very similar understanding. Addiction is primarily a violation of the self. Alcoholics have a large problem with self-centeredness, it is said. The heart of the problem, "an extreme example of self-will run riot" (Alcoholics Anonymous World Services 1976a, 62), blocks alcoholics off from their Higher Power. Not only that, but they are in "denial" about it. Honesty and "deflation at depth" (Wilson 1957, 65) are part of the solution. Defects of character remain after the alcoholic stops drinking and they become part of one's ongoing program.

The Oxford Group, like AA, believed that conversion happens in stages, rather than all at once. Some suggest that the "five Cs" of the Oxford Group—confidence, conviction, confession, conversion, and continuance (Clark 1951, 28)—were enlarged by Wilson to become the famous 12 Steps of AA. In the Oxford Group, all problems got the same treatment. Members were to stay honest, locate their shortcomings, respond to group influence, confess their sins, and continue to submit to "God control." AA is similar, except that Wilson noticed alcoholics could not take group pressure nor perfectionist standards, and he warned against them. In AA, individuals are repeatedly urged to admit their powerlessness and turn their wills and lives over to their Higher Power.

God and Christ

Since the Oxford Group was unashamedly, though generically, Christian, it had views regarding God and Christ. The group believed, for instance, that God can and does communicate with people and desires to guide their every activity. They affirmed that God is loving but does not condone sin. They mentioned the Spirit. Most important, to be changed, they insisted that a person must submit to this guiding God. As for Christ, his place in their theology was conventional. Although there was no worked-out doctrine of the Trinity, they did believe that the finished work of Christ releases humans from sin. This principle undergirds their expectation of miracles and healing. Even with all this Christian doctrine, however, Buchman was not opposed to bracketing the work of Christ if a more generic spirituality would appeal to his listeners.

AA's views echo some of this group theology but pick up Buchman's strategy and move beyond it. The focus is more on a generic spirituality, with no mention of Christ at all. AA is similar to the Oxford Group in expecting a release from human bondage, but unlike its parent organization, AA does not tie this to any once-for-all work of God or Christ. Thus AA retains the diagnosis, expectation of conversion, need for God-control, optimistic hope, and ongoing healing, but loses the theological foundation in Christ's work that secured all this for the Oxford Group.

Church and Religion

Both the Oxford Group and AA are ambivalent regarding church and organized religion. The Oxford Group sometimes cooperated with the church and worked with pastors, but generally, it kept the institution at arm's length. Implicitly there was—as there often is in other para-church organizations—a critique of the church as too rule-bound, mired in controversy, or inactive. Although Buchman was ordained, he did not regularly officiate in any church or submit to the authority of or commit himself to any denominational structure. The church might be used when expedient, but Oxford Group members often had a poorly concealed contempt for conventional religion (Clark 1951, 56).

AA and other recovery groups exhibit a similar attitude toward the church, although officially, they are careful not to criticize religion. Integral to AA is a resistance to organizational ties and an implicit belief that those best equipped to help alcoholics are fellow alcoholics. Although Wilson claimed that AA was only a "spiritual kindergarten" (Alcoholics Anonymous World Services 1976b, 95), AA makes no claim to revitalize church or religion. In fact, many members treat AA as though it is an adequate or superior spiritual replacement for organized religion. And some church leaders have been so impressed by AA's success that they use its principles in their ministries.

Some AA members eventually make their way into organized religion once their recovery is in progress. But for many, the implicit judgment on the church as ineffective in truly helping alcoholics—as well as the sin-versus-disease dichotomy so crucial to the recovery ethos—more often works to keep people away from religion. Instead, members often find themselves tied to the recovery movement as the main way to fulfill their spiritual needs. This is ironic, given the Oxford Group roots of AA. Yet it could also have been predicted, given the trajectory so early set. The irony is enhanced when one realizes how similar are some basic methods and concepts between the Oxford Group and AA. Most striking is that the diagnosis of addiction and the prescription for recovery have so much in common with the Oxford Group's understanding of sin and conversion.

REFERENCES

Alcoholics Anonymous World Services. 1976a. *Alcoholics Anonymous: The story of how many thousands of men and women have recovered from alcoholism*. 3rd ed. New York: Alcoholics Anonymous World Services.

Alcoholics Anonymous World Services. 1976b. *As Bill Sees It: The A.A. Way of Life*. New York: Alcoholics Anonymous World Services.

Clark, Walter Houston. 1951. *The Oxford Group: Its history and significance*. New York: Bookman Associates.

Eisler, Allan W. 1950. *Drawing-room conversion: A sociological account of the Oxford Group movement*. Durham, NC: Duke University Press.

Grensted, Laurence William. 1933. *What is the Oxford Group?* London: Oxford University Press.

Houck, James W. 1995. In the mail. *New Yorker*, May 1, p. 10.

Kurtz, Ernest. 1991. *Not-God: A history of Alcoholics Anonymous*. Exp. ed. Center City, MN: Hazelden.

Luttwak, Edward. 1994. Franco-German reconciliation: The overlooked role of the moral re-armament movement. In *Religion: The missing dimension of statecraft*, ed. Douglas Johnston and Cynthia Sampson (37–57). New York: Oxford University Press.

Mercadante, Linda. 1996. *Victims and sinners: Spiritual roots of addiction and recovery*. Westminster, UK: John Knox.

Niebuhr, Reinhold. 1936. Hitler and Buchman. *Christian Century*, October 7, 1315–16.

Pound, Reginald. 1976/1985. *A. P. Herbert*. In *Frank Buchman: A Life*, ed. Garth Lean. London: Constable.

Walter, H. A. 1919. *Soul-surgery: Some thoughts on incisive personal work*. Calcutta: Association Press.

Wilson, William Griffith, et al. 1957. *Alcoholics Anonymous comes of age*. New York: AA Publishing.

Woolverton, John F. 1983. Evangelical Protestantism and alcoholism 1933–1962: Episcopalian Samuel Shoemaker, the Oxford Group, and Alcoholics Anonymous. *Historical Magazine of the Protestant Episcopal Church* 52: 53–65.

This page intentionally left blank

Chapter **7**

Adolescent Drug Sellers and Distributors

Jemel P. Aguilar, PhD, MSW

Young Americans, between the ages of 12 and 18, are actively involved in the selling and distribution of illicit drugs such as marijuana, heroin, crack cocaine, and methamphetamine (E. Anderson, 1999; Black & Ricardo, 1994; Bouchard & Tremblay, 2005; Centers & Weist, 1998; Hohman, Oliver, & Wright, 2004). Although youths are involved in drug selling and distributing, little is known about this population's motivation for and involvement in drug selling and distribution. This chapter offers an overview of the burgeoning literature on youth involvement in drug selling and distribution. Consequently, this chapter includes the current research on young drug sellers and distributors, the outcomes associated with drug selling, and what researchers have identified as a part of the lives of young drug sellers.

As will be discussed, the research on the drug economy encompasses different roles, positions within the overall hierarchy of organizations, and levels of risk to a participant's welfare. Discussing all these structures and roles within the drug economy is beyond the purpose of this chapter; therefore it is important to note that the research included in this chapter focuses on drug selling and distribution. Drug selling refers to the selling of marijuana, heroin, crack cocaine, and methamphetamine in small amounts for another's personal consumption. Distribution describes the activities of individuals that both sell small amounts of illegal drugs and provide other drug sellers with illegal drugs that these sellers can peddle for profit. Although other roles exist, the following section outlines what is known about the selling and distribution of marijuana, heroin, crack cocaine, and methamphetamine.

WHO ARE THE DRUG SELLERS AND DISTRIBUTORS?

Few studies examine the backgrounds of adolescent drug sellers. Of the available studies, it is clear that both young men and women are involved in the selling and distribution of crack, heroin, marijuana, methamphetamine, cocaine, and other illegal substances (Centers & Weist, 1998). Researchers are less clear about the ways in which gender may operate as a factor in a person's decision to enter into drug sales (J. F. Anderson, Brooks, Langsam, & Dyson, 2002). For example, some researchers suggest that young men are more often involved in drug selling and distribution than young women because of the opportunities for young men to garner social and economic prestige. Comparatively, researchers find that young women enter into drug selling and distribution as a result of their sexual relationships.

Regardless of the gender of a drug seller, researchers find that youths become involved in drug sales because they perceive limited opportunities for social and economic advancement through traditional avenues. Some of these findings may be reflective of the populations recruited in that many of the youths surveyed live in low-income and poor neighborhoods in which few economic opportunities exist. For example, in Centers and Weist's (1998) review of drug selling, they report that 1 of the 15 studies the authors reviewed indicated a young population other than low-income youths.

In addition to youths' attempts to garner a positive social standing, youths may enter into drug selling and distribution because they are substance users themselves (Black & Ricardo, 1994). According to this body of research, youths may enter into the drug culture as recreational or episodic users. As users' drug habits evolve and their social relationships become impaired or overwrought by the economic and social costs of drug use, obtaining funds to support a drug habit becomes equally strained. Brunelle, Brochu, and Cousineau (2000) write the following about the youth's drug use to selling evolution:

> To the extent that dependency takes it roots in the lives of some youths, their need for drugs intensifies to the point of becoming their unique and central interest. At this stage of drug dependency, the adolescent consumer does not have the legitimate financial means to meet this compulsive consumption. This therefore leads to involvement in various sorts of money-oriented crimes. This economic-compulsive relation is particularly accentuated among youths who develop a cocaine or heroin dependency, because such ill.icit drugs are more expensive and more addictive than cannabis. (p. 849)

Brunelle and colleagues highlight cocaine and heroin in their economic explanation for the drug-crime connection; however, similar assertions can be made

about other highly addictive substances such as methamphetamine. Overall, researchers examining the user-seller phenomenon report that some drug sellers or distributors will market a portion of their drugs to offset the cost of using illegal drugs.

Aside from drug selling, several options are available for adolescent substance users to obtain drugs or money for drugs: these are prostitution, having a relationship with another drug user that can provide him or her with an adequate supply of drugs, or robbery (Brunelle et al., 2005; Fagan, 1994). For some, drug use is a coping mechanism for dealing with prostituting (Brunelle et al., 2005), while others prostitute and sell drugs to earn enough money for a steady supply of drugs for their own use. For example, in a phenomenological study of drug-using youths, Brunelle and colleagues (2000) write that "drug consumption sometimes turns out to be indispensable for activities such as prostitution, because drugs allow prostitutes to be more at ease in this activity" (p. 844). Alternatively, a youth will have a relationship with another drug user so that he or she may access drugs; however, these relationships are typically short-lived. The last, and more lucrative of the options, is drug selling (Fagan, 1994). Interestingly, the advent of crack and spread of cocaine created avenues for young women to sell drugs with little initial start-up funds, presenting lesser risk than prostitution or relationships with other drug users (Fagan, 1994).

Gossop, Marsden, and Stewart's (2000) study of youth drug sellers, for example, indicates that regular use of benzodiazepines increases the likelihood that a person will engage in drug selling. Similarly, Black and Ricardo (1994) report that some of the youth respondents in their study used drugs and engaged in drug distribution activities as well as other risky behaviors. Some youths will become drug distributors through their participation in drug use, while others may engage in drug selling as a means to increase their social standing.

Most studies of youth drug sellers and distributors indicate that these individuals also engage in criminal behavior to maintain their position within the drug economy or obtain material goods that can be traded or sold. Gossop and colleagues (2000), for example, report that 71 offenders they surveyed were involved in over 900 arrests in the three months before entering into drug treatment. This same group of offenders reported over 39,000 drug offences prior to intake. Consequently, similar to criminological research that posits that a few juvenile offenders engage in a significant number of criminal offenses, this study suggests that a few drug offenders are involved in committing a majority of drug-related crimes.

The research literature on drug selling and distribution reveals another avenue for engaging in drug selling and distribution: through gangs. Valdez and

Sifaneck's (2004) research on Mexican American gang members reveals four drug-related roles that youths engage in that link them to the drug economy. According to these authors, *homeboys* are gang members who are user-sellers and who are a part of street gangs, yet the gangs are not focused on drug selling and distribution. Homeboys will buy drugs for their own consumption and sell a portion of the drugs to support their drug use. Many times, the drug-related activities of homeboys are apart from the activities of the gang. *Hustlers,* on the other hand, are gang members who deal illegal substances from within gang structures, even though the gang is not an organized drug selling and distribution entity. By contrast, hustlers are integrated into the gang network, yet the profits from drug sales are solely for the hustler and do not support the gang. Hustlers may benefit from the protection of the gang and make use of the social connections of the gang members to sell drugs. *Slangers* are a part of gangs that are drug selling and distribution networks, are drug users themselves, and are actively selling and distributing drugs from within the gang. Similar to hustlers, slangers benefit from the protection of the gang, and unlike hustlers, the profits from drug sales go back to benefit the gang enterprise. The final group of gang-related drug sellers are *ballers*. Ballers are members of gang enterprises that sell and distribute illegal drugs, the profits of which are returned to the gang for the benefit of the whole enterprise. Similar to criminal organizations, the purpose of the drug selling and distributing is not for individual profit, but for the profit of the organization. As a result, a gang member's introduction to drug selling and distribution may stem from his or her entrance into a gang network as well as the opportunity for social prestige that leads youths to participate in gang activities and drug selling. The research on youths' entrance into drug selling and distribution provides some ideas about why youths may engage in drug selling and distribution; however, additional questions remain about the interactions of these factors. For example, researchers have yet to examine the dynamic relationship between social prestige and drug use that may encourage a youths to enter into drug selling. Until these relationships are examined, it is clear that social prestige, drug use, and gang affiliation are factors in youths entering into drug selling and distribution.

THE DRUG ECONOMY AND ORGANIZATIONAL ROLES

Current knowledge about the drug economy and organizational roles within that economy primarily comes from research on adult drug sellers and distributors. Thus this section includes research from adult and youth studies of the drug economy. Adler's (1993) ethnographic study is a prominent study in the area of drug selling and distributing. Although Adler's work does not discuss

youth drug selling and distributing, this quintessential work deserves mention because it sets the foundation for studies of drug selling and distributing and highlights the complex nature of social interactions within the adult drug economy that may also apply to studies of youth drug selling. In her study, Adler conducted in-depth observations and interviews with members of a southwest drug distribution cartel that trafficked marijuana and, at times, cocaine from Central American countries into the United States. What her work reveals is the complex nature of the drug-distributing organizations and the tenuous relationships that produce the widespread drug economy. Adler and other researchers (Natarajan & Belanger, 1998; Zaitch, 2005) report that organizations selling drugs are loosely connected, encompass tenuous relationships, and include many isolated individuals. These findings are supported by other research as well as qualitative studies of drug sellers such as Valdez and Sifaneck's (2004) study of drug-selling gang members.

Natarajan and Belanger (1998) conducted a study of drug-distributing organizations and delineate several roles, and associated responsibilities, that individuals occupy within these organizations. These roles include those individuals who trade illicit drugs on the streets, called *retailers*, to those individuals who break down a larger quantity into smaller portions that can be sold on the streets. Moreover, their analysis also revealed that *brokers* are the representatives who link sellers and retailers and/or sellers with other sellers.

Additional research into youths' perceptions of the drug economy and the youth-led drug selling and distribution enterprises is needed to understand the dynamics of these organizations as well as the ways in which youths enter into, operate, and leave drug selling. As previously stated, most of the studies of the drug economy stem from research on adult participants in the drug economy, and research has yet to compare and contrast youth roles within the drug economy against adult participation in the same economy.

DRUGS AND VIOLENCE

Some researchers are examining the relationship between drug selling and violence to determine the best ways to stem the violence that consumes drug-infested communities. In an attempt to understand the theoretical linkages between substance use and violent behavior patterns, Goldstein (1985) developed what he describes as a tripartite model of the drug and violence nexus. In this conceptual framework, Goldstein outlines three models, or avenues, that link violence and drug use. In each of the models, the substance user can be either a victim or offender, and at times, the offender's motives can cross these separate models.

Goldstein (1985) begins his conceptualization with a pharmacological model of substance use and violence. The pharmacological model focuses on biological changes that occur as a result of ingesting substances or during withdrawal from substance use. For example, in Brunelle and colleague's (2005) research on substance-abusing juvenile offenders, the authors apply Goldstein's (1985) model to a sample of 38 juvenile substance abusers between the ages of 16 and 18. Brunelle and colleagues found that a majority of the crimes committed by these youths were classified as serious and violent. Youths in this study described using substances to commit some crimes or avoid negative feelings and enhance other feelings associated with the crimes (Brunelle et al., 2000). In their review of substance abuse and violence literature, Boles and Miotto (2003) confirm Brunelle and colleague's (2005) findings and describe several studies that examined the role of substance use in the commission of crimes. In these studies, 73 percent of state prisoners and 65 percent of federal prisoners confirmed their drug use during the commission of a crime, and 60 percent of arrestees tested positive for illicit drug use at the time of their arrest. An alternative relation between substance use and violence occurs during the withdrawal period. In this scenario, harsh withdrawal symptoms, such as irritability or excitability, may result in aggression and/or violence (Boles & Miotto, 2003; Goldstein, 1985). As Boles and Miotto (2003) note, "intranasal cocaine and crack use have both been found to be associated with pharmacological violence" (p. 167; see also Fagan, 1994). Thus, for many offenders, substance use is a part of their process for committing crimes.

The economic-compulsive model, alluded to earlier, links the economics of drug use with violence (Boles & Miotto, 2003; Goldstein, 1985). Goldstein defines the economic-compulsive model as the quelling of a person's aggressive and violent tendencies as his or her need to obtain money for a supply of drugs increases. Several research studies on the drug economy and substance users support the notion that substance users will commit violent or aggressive acts to obtain the needed money for further drug use. In Fagan's (1994) study of women in the drug economy, he found that sex workers would "vic" (slang for "steal") money from their customers to purchase drugs. Sometimes these robberies would become violent, as the johns would attempt to stop the robbery (Fagan, 1994). In Goldstein's (1985) economic-compulsive model, the victims of these violent crimes are people who have valuables or money such as drug dealers, other drug users going to "cop" (i.e., buy drugs), and strangers. The last model in Goldstein's framework is the systemic model. This model considers the violence embedded within the drug culture that occurs because of a drug user's attempt to obtain drugs or a dealer's management of his or her sellers. Obviously there are other mechanisms leading to violent behavior, but these

two examples are usually presented in the research literature. Fagan's (1994) research on women in the drug economy is again instructive of the dynamics of drug selling and using partnerships. In his study, he found that women selling drugs in groups were more likely to use violence as a means to protect their "turf" from competing dealers, as retribution against users who attempt to steal drugs, and as a means for a managing dealer to discipline her street sellers. The extent of systemic violence is difficult to determine because the victims are less likely to report such incidents to police or hospital personnel. However, respondents continually discuss acts of systemic violence in many qualitative studies of drug dealers (Boles & Miotto, 2003; Fagan, 1994; Feigelman, Stanton, & Ricardo, 1993; Li, Priu, & MacKenzie, 2000). There is additional evidence that youths are entering into drug selling for social prestige instead of the primarily economic motives that are typically attributed to adolescent drug sellers and distributors. For instance, most adolescent drug sellers and distributors act in these roles for short periods of time. Gossop and colleagues (2000) illustrate the short duration of drug selling in their study of adolescent drug sellers.

Drug use and/or selling are also associated with serious and violent crime (Boles & Miotto, 2003; Brunelle et al., 2005; Goldstein, 1985; Okundaye, 2004; Stanton & Galbraith, 1994). Reviews of delinquency and adult crime consistently reveal that drug-using youths and adults are more likely to commit serious and violent crimes to support their drug-using habits and, for sellers, as a customary dispute-resolution mechanism. For example, Fagan's (1994) study of women in the drug trade found that women who sold crack or cocaine in groups were regularly involved in violence that was directed toward regulating interactions between sellers and buyers or other sellers. As Fagan describes, "group sellers have generally higher rates of both nondrug crimes and systemic violence. This association suggests that, as with males, these two dimensions of violence tend to coincide and be a part of a generalized pattern of violence among both male and female drug sellers" (p. 220). Scholars of juvenile delinquency also agree that drug selling is a part of a larger pattern of problematic behaviors among both female and male juvenile offenders (Boles & Miotto, 2003; Brunelle et al., 2005; Huizinga, Loeber, Thornberry, & Cothern, 2000; Lane, 2003).

CONCLUSIONS

The research into youth drug sellers and distributors is just beginning to explain some of the roles and processes that encompass youths' position within the overall drug economy. With the growing numbers of youths entering into the juvenile justice system because of drug-related crimes, it is important that

researchers and practitioners have a better understanding of the dynamics of youth drug selling and distribution so that more effective interventions can be developed to prevent youths from becoming active drug sellers and distributors and/or to stem youth involvement once they enter into this economy.

REFERENCES

Adler, P. A. (1993). *Wheeling and dealing: An ethnography of an upper-level drug dealing and smuggling community*. New York: Columbia University Press.

Anderson, E. (1999). *Code of the street: Decency, violence and the moral life of the inner city*. New York: W. W. Norton.

Anderson, J. F., Brooks, W., Jr., Langsam, A., & Dyson, L. (2002). The "new" female gang member: Anomaly or evolution? *Journal of Gang Research, 10*(1), 47–65.

Black, M. M., & Ricardo, I. B. (1994). Drug use, drug trafficking, and weapon carrying among low-income, African-American, early adolescent boys. *Pediatrics, 93*, 1065–1073.

Boles, S. M., & Miotto, K. (2003). Substance abuse and violence: A review of the literature. *Aggression and Violent Behavior, 8*, 155–174.

Bouchard, M., & Tremblay, P. (2005). Risks of arrest across drug markets: A capture-recapture analysis of "hidden" dealer and user populations. *Journal of Drug Issues, 35*, 733–754.

Brunelle, N., Brochu, S., & Cousineau, M.-M. (2000). Drug-crime relations among drug-consuming juvenile delinquents: A tripartite model and more. *Contemporary Drug Problems, 27*, 835–866.

Brunelle, N., Cousineau, M.-M., & Brochu, S. (2005). Juvenile drug use and delinquency: Youths' accounts of their trajectories. *Substance Use & Misuse, 40*, 721–734.

Centers, N. L., & Weist, M. D. (1998). Inner city youth and drug dealing: A review of the problem. *Journal of Youth and Adolescence, 27*, 395–411.

Fagan, J. A. (1994). Women and drugs revisited: Female participation in the cocaine economy. *Journal of Drug Issues, 24*, 179–225.

Feigelman, S., Stanton, B. F., & Ricardo, I. (1993). Perceptions of drug selling and drug use among urban youths. *Journal of Early Adolescence, 13*, 267–284.

Goldstein, P. J. (1985). The drugs/violence nexus: A tripartite conceptual framework. *Journal of Drug Issues, 39*, 143–174.

Gossop, M., Marsden, J., & Stewart, D. (2000). Drug selling among drug misusers before intake to treatment and at 1-year follow-up: Results from the National Treatment Outcome Research Study (NTORS). *Drug and Alcohol Review, 19*, 143–151.

Hohman, M., Oliver, R., & Wright, W. (2004). Methamphetamine abuse and manufacture: The child welfare response. *Social Work, 49*, 373–381.

Huizinga, D., Loeber, R., Thornberry, T.P., & Cothern, L. (2000). Cooccurrence of delinquency and other problem behaviors. *Juvenile Justice Bulletin* (November, pp. 1–8). Washington, DC: OJJDP.

Li, S. D., Priu, H. D., & MacKenzie, D. L. (2000). Drug involvement, lifestyles, and criminal activities among probationers. *Journal of Drug Issues, 30,* 593–620.

Natarajan, M., & Belanger, M. (1998). Varieties of drug trafficking organizations: A typology of cases prosecuted in New York City. *Journal of Drug Issues, 28,* 1005–1025.

Okundaye, J. N. (2004). Drug trafficking and urban African American youth: Risk factors for PTSD. *Child and Adolescent Social Work Journal, 21,* 285–302.

Stanton, B., & Galbraith, J. (1994). Drug trafficking among African-American early adolescents: Prevalence, consequences, and associated behaviors and beliefs. *Pediatrics, 93,* 1039–1043.

Valdez, A., & Sifaneck, S. J. (2004). "Getting high and getting by": Dimension of drug selling behaviors among American Mexican gang members in South Texas. *Journal of Research in Crime and Delinquency, 41*(1), 82–105.

Zaitch, D. (2005). The ambiguity of violence, secrecy, and trust among Colombian drug entrepreneurs. *Journal of Drug Issues, 35,* 201–227.

This page intentionally left blank

Part II

DIMENSIONS

This page intentionally left blank

Chapter **8**

Historical Aspects of Alcohol Use in India: Role of Culture and Gender

Meera Vaswani, PhD, Atul Ambekar, MD,
and Ramandeep Pattanayak, MD

India, as a civilization, has a rich cultural history marked by enormous diversity and deep-rooted traditional value systems. There have been contemporary influences of industrialization and globalization in recent times. The attitudes, belief systems, and patterns of alcohol use have been modified with the changing religious, cultural, and sociopolitical influences. Some of these influences are reflected in alcohol use patterns in contemporary India. Alcohol use has been largely prohibited for women since historical times and was seen as a marker of bad character. A slow but gradual change has been observed in societal attitudes toward women consequent on their social empowerment.

This chapter begins with a review of the history and cultural context pertaining to alcohol use in India. This is followed by a discussion of alcohol use in women and the role of cultural norms in shaping alcohol use in India. A few important statistics regarding India are provided in Table 8.1.

ALCOHOL USE IN INDIA: HISTORY AND CULTURAL CONTEXT

Alcohol use in India can be traced back to antiquity. The degree of cultural permissiveness to alcohol has varied according to the changing historical periods. In this section, perspectives from Indian history and culture will be discussed. The issues pertaining to gender are also highlighted.

Table 8.1
Country Background Information

Total population	1,027,015,247
Adult population (aged 15–59 years; %)	56.9
Sex ratio (no. of women/1,000 men)	933
Population distribution (%)	
Urban	28
Rural	72
Literacy rate (%)	64.8
Life expectancy at birth (years)	
Boys	60.1
Girls	62.0
Gross national income per capita (in USD for year 2006)	820
Percentage below poverty line (less than USD 1 per day)	36
Human Development Index rank	115

Sources: Census of India (2001), World Health Organization (2004), and World Bank World Development Indicators database.

Alcohol Use in Antiquity (Bronze Age, 3300–1700 B.C.)

Alcohol distillation possibly originated in the Indus Valley civilization as early as 2000 B.C. Needham (1980) has examined the origins of the distillation of alcohol to India in volume 5(4) of *Science and Civilisation in China*. There have been unearthings of distillation assemblies made of clay at Taxila and at Shaikhan Dheri (now in Pakistan). These were referred to as the Gandhara Stills and were thought to be the origin of all kinds of distillation stills. The alcohol produced from this primitive distillation apparatus might have been only a diluted sort. *Soma* and *sura* were the two earliest beverages, traceable to 2000 B.C. (Chopra & Chopra, 1965). *Soma* came to be identified with the mythical tree of life and immortality (Chand, 1972). The use of beverages derived from the *soma* plant was confined to the nobles and saints, and the rituals of preparation were often kept a secret. Experiences of intoxication on *soma* have been described as transcendentally blissful, enlightening, and tranquilizing, creating euphoric states and stimulating both the mind and body (Chand, 1972). *Sura* was mostly derived from fermented rice, barley, jaggery, or *mahuwa* (flowers of the *madhyaka* tree) and was relatively stronger than *soma*. It was

used by warriors, peasants, and common people. Its effects have been described as invoking moderate euphoria to intoxication (Dikshitar, 1951).

Alcohol Use in the Vedic and Epic Period (Iron Age, 1700–500 B.C.)

In this period, Hinduism was seen not only as a religion, but as a way of life. Drinking was totally prohibited by the *Rig-Veda*.[1] The Sutras,[2] however, mentioned that liquor could be served on special occasions such as on festivals and to guests as they enter a new house (Praskara, 1956). Women, students, and Brahmins were prohibited from using alcohol or any other intoxicant. Members of other castes (Kshatriyas and Vaishyas[3]) were allowed to take milder liquors but were forbidden the spirits distilled from fermented grain (Prakash, 1961). The themes and images associated with alcohol depicted warriors and courtiers. The lowest section of Shudras, the Dalits, were not considered part of mainstream society and therefore were not governed by the religious scriptures. Non-Brahmins were never considered to be able to attain a sense of high religious purity and therefore were allowed alcohol use. The great Indian epic *Ramayana*, in its description of society, generally followed a bad-good or black-white dichotomy. The bad factions were consumers of alcohol and meat, while the good were abstinent vegetarians. Another epic, *Mahabharata*, portrayed the characters in a gray shade and mentioned alcohol consumption by them. It is important to note that in the Vedic and post-Vedic period, alcohol use was never seen as a daily phenomenon, and it was limited mainly to ritual drinking. Alcohol was never considered a part of daily nutrition. Inebriation was severely censured, and abstinence was held in high esteem (Chand, 1972).

Alcohol Use in the Ancient Period (500 B.C. to A.D. 550)

A balanced view of drinking was promoted in this period, and moderation was counseled. The Ayurvedic[4] texts described the beneficial effects of alcoholic beverages and also the consequences of intoxication and alcoholic diseases. Alcohol was thought to increase the mental principle (*pitta*), while diminishing the physical and vitality principles (*kapha* and *vata*). There were prevalent beliefs such as that alcohol mixed with fruit juices could be enjoyed in winter but should be avoided in summer. Kautilya, the prime minister of India's first great emperor, Chandragupta Maurya, in his treatise on statecraft and economic policy, called *Arthashastra*, codified the rules and regulations for alcohol, pointing to its common usage among people in the regime. With the origin of Buddhism, which was spread by Emperor Ashoka (r. ca. 273–232 B.C.) to several neighboring Asian countries, a movement for general abstention

from alcohol was begun. Buddhism prohibited drinking by monks and in monasteries and influenced its followers to stop alcohol use. Alcohol use continued to be forbidden for women. The Laws of Manu,[5] in A.D. 200, mentioned that "a wife who drinks spirituous liquor, is of bad conduct, rebellious, diseased, mischievous, or wasteful, may at any time be superseded (by another wife)" and warned that drinking liquor is one of the major causes of the ruin of women (Doniger, 1992).

Alcohol, however, continued to be used by men belonging to warrior, trader, and peasant classes. Alcoholic beverages were made from fermentation and distillation of grain (barley and rice), flowers (the *mahuwa* flower and jasmine), and fruits (mangos, dates, *kadamba* fruit, and grapes). The sweet exudates from the coconut palm were fermented into a drink called *thari* or *toddy*, and then were distilled to yield *arrack*. The practice remains popular today in southern parts of India. At the height of its trade with Rome in the early Christian era, India also imported amphorae of wine for use by the nobility. But the common drinks were *toddy*, *arrack*, and a variety of highly flavored rice wines.

Alcohol Use in the Medieval (A.D. 550–1500) and Postmedieval (A.D. 1500–1800) Periods

Brewing was practiced all over the country in this period. Alcohol consumption became prevalent in a considerable section of Hindu society, especially home-brewed, low-ethanol beers (Prakash, 1961). Distillation of wine was described in detail in the texts as an art, and a relatively permissive attitude to alcohol consumption continued, especially in the warrior groups. Kshatriyas drank liquor brewed from the grape and sugarcane, Vaishyas preferred strong distilled liquor, and Brahmins drank only fruit juices. Some tribal and low-caste peasants (Shudras) also abstained from drinking in the belief that this would raise their status in society as food is considered an important identity symbol in Indian culture. The anthropologist Srinivas (1997) has defined this process as *Sanskritization*, that is, the process by which a person belonging to a lower Hindu caste changes his customs, rituals, and way of life in the direction of a higher caste, thereby adopting vegetarianism and alcohol abstinence as a marker of higher social class.

The postmedieval period was dominated by the Mughal regime. The Koran prohibited the use of alcohol, referring to it as "great sin and harm." However, there are references to the emperors Babur and Jahangir being regular heavy alcohol users (Rahman, 1984). Alcoholic beverages were consumed by courtesans and nobles alike. Hindu society, in the face of the external Mughal regime,

became somewhat more introverted and rigid in following preexisting codes of behavior and restrictive attitudes toward alcohol.

Alcohol Use in the Colonial Era (A.D. 1800–1947)

The British administration noted the widespread production of *toddy*, *arrack*, and other traditional beverages all over India. In addition, the Portuguese monks in Goa had also developed a distilled liquor called *fenny* from the cashew. Distilled products made from rice and *mahuwa* flowers were also common and mostly were locally produced on a small scale in villages. The Indian rulers of the time neither expected any revenues nor encouraged the commercialization of liquor. The British colonial administration, however, felt the alcoholic beverages to be an excisable commodity (Achaya, 1998). The first distillery in the country was set up for the British army at Cawnpore (now Kanpur) for the manufacture of rum in the year 1805. The British East India Company banned the previously village-based manufacturing system and made strong efforts to increase revenue generation and profits from alcohol. The right to set up distilleries was offered through auction, resulting in an appreciable increase in overall consumption. The production of locally made, low-strength alcoholic beverages was replaced by factory-produced, high-strength spirits.

Voices were raised against drinking as well as against the British educational system, which was seen to be promoting drinking culture. There were national movements promoting temperance in the nineteenth century. Among the nationalist leaders, there were two schools of thought on the subject. One school believed that colonial policy was distorting the existing healthy drinking culture of the people, in particular, causing the mild and harmless drink *toddy* to be replaced by bad spirits, and demanded change in these policies. The other school of nationalist leaders—the predominant school—demanded complete prohibition in India. Many of the political leaders, such as G. K. Gokhale, held the belief that an independent India had to be a pure India, and legal prohibition was a step toward national purity. With the emergence of Gandhi, several attempts were made to encourage temperance among the masses. This also included the "scheduled," or "lower," castes, who traditionally were "permitted" to drink, and women, who otherwise would have been left out from the freedom struggle. Picketing of alcohol sellers was a major activity of women's groups. Sales of drink decreased significantly in many areas, and in the Bombay Presidency alone, liquor consumption fell by approximately 20 percent as a result of picketing during the noncooperation movement (Brown, 1972).

Though this appears to be quite in harmony with the ancient traditions, in fact, it was a significant deviation. The ancient Hindu tradition did not rule out alcohol for everybody in society. In fact, there was a relatively relaxed attitude toward alcohol, barring the elitist Brahmins and the spiritually elevated (Mandelbaum, 1965).

DRINKING CULTURE IN INDIA

India essentially has never accepted alcohol as a part of normal social discourse or eating behavior, and there is a virtual absence of normative patterns of drinking alcohol and attitudes toward it. An exception is seen in the tribal population and a few Hindu religious (*Tantric*) sects. Alcohol is seen by the tribal people of India as a gift of god, as an antidote to witchcraft and sorcery (Rao & Rao, 1976). It has gotten sanction to be consumed on all social occasions from cradle to grave, and as a gesture of friendship and reconciliation after quarrels, though the alcohol used is generally low strength, at 3 to 5 percent alcohol by volume. Tribal people are generally habituated to alcohol, and a tribal man (*chenchu*) may even promise to give away his daughter for marriage in exchange for alcohol (Reddy, 1971). Some Hindu *Tantric* sects also use alcohol rampantly to achieve a different level of consciousness and indulge in casting magic spells under its influence. Such followers are, however, few, and have been generally unsuccessful in having any significant impact on Indian norms and attitudes (Chand, 1972).

In the main, India does not neatly fit into any of the conceptualizations of *abstinent* (condemnation of alcohol use in any form, e.g., by Muslims and some Protestants), *ambivalent* (negative attitudes coexist with attitudes idealizing intoxication, e.g., English and Scandinavian countries), *permissive* (moderate consumption accepted, e.g., Jews and Italians), or *overpermissive* (alcohol use widely accepted and drunkenness permitted in certain contexts, e.g., French and Japanese) cultures, as outlined by Pittman (1964). India displays the culture of purity and abstinence on one hand and permissiveness toward drinking on the other. At best, it can be described as being closest to an *ambivalent* drinking culture, in which prohibitive attitudes coexist with attitudes idealizing intoxication. Drinking is a generally an activity in male-predominated areas away from home. Unlike many other cultures, the aim, while drinking in India, is to get intoxicated. The pattern of drinking in India has undergone a change from occasional and ritualistic use to being a social event with a common purpose to get drunk (Mohan, Chopra, Ray, & Sethi, 2001). The adult per capita per year consumption in India has been found to be 0.8 liters for recorded and 1.7 liters for unrecorded consumption (World Health Organization, 2004), which is on the lower side. However, only around 20 percent of the male popu-

lation have been found to be current drinkers (Ray, Mondal, Gupta, Chatterjee, & Bajaj, 2004); in a nationwide survey, these numbers reflect the fact that those who drink alcohol tend to do so in heavy amounts.

Undersocialized, solitary drinking of spirits, drinking to intoxication, and expectancies of drink-related disinhibition and violence add to the hazardous patterns (Gupta, Saxena, Pednekar, & Maulik, 2003; Saxena, 1999). Despite the appearance of public bars and pubs in some parts of India, pub drinking, as understood in the West, has not yet been institutionalized and norms are only beginning to evolve. These developments have raised concerns about the health and the social consequences of excessive drinking (Saxena, 1999).

ALCOHOL USE IN CONTEMPORARY INDIA

The changes brought by the colonial era are reflected in the Post Independence period and present India. The traditional drinks, such as *toddy* or cottage-made *arrack* (Table 8.2), have been largely replaced by more expensive factory-made alcoholic beverages with greater alcohol content. This, in turn, has led to organized and unorganized production of illicit alcoholic beverages in most parts of the country.

Most authentic figures for prevalence of alcohol use in the general population of India are available from the National Survey on Extent Pattern and Trends of Drug Use in India (Ray et al., 2004). A general population survey (National Household Survey; Ray et al., 2004) was conducted in which a nationally representative sample of over 40,000 males was interviewed. The prevalence of current alcohol use (defined as "use in the preceding 30 days") was

Table 8.2
Traditional Alcoholic Beverages in India

toddy	fermentation of the sap of a coconut palm; alcohol content, 3% to 6%
rice beer	low-strength alcohol beverage used in the tribal belts; alcohol content, 3% to 6%
jack-fruit wine	alcohol content, 7% to 8%
arrack	distillation of fermented molasses, raw brown sugar, palm wine, rice, or palm sugar, common in southern India; alcohol content, 20% to 40%
palm wine	produced from coconut or other palm trees
daru	from the flowers of the *mahuwa* plant, common in northwestern India; alcohol content, 20% to 40%

Source: Global Status Report 2004, World Health Organization (2004).

found to be 21.4 percent. Projecting this figure to the absolute number, it can be safely estimated that there are at least 62.5 million current users of alcohol in the country. The prevalence ranged from 7 percent in the western state of Gujarat (officially under complete prohibition) to 75 percent in the northeastern state of Arunachal Pradesh. Alcohol was found to be the primary drug of abuse in the clinical population (i.e., patients attending drug and substance abuse treatment centers) as well, with as many as 43.9 percent of patients seeking treatment for alcohol-related problems (Drug Abuse Monitoring System; Ray et al., 2004).

The complexity of Indian culture is reflected in the fact that there is a wide variation among various regions, urban-rural settings, socioeconomic statuses, caste groups, and religious beliefs. An earlier study covering both urban and rural areas of eastern, northern, southern, and central India showed that alcohol use was higher among specific groups such as industrial workers, tea plantation workers, and tribals compared to the general population (Mohan, Sharma, Sundaram, & Advani, 1981). A significantly higher use has also been recorded among tribal, rural, and lower socioeconomic urban sections (Ray & Sharma, 1994; Thimmaiah, 1979). Choice of beverages also differed, with home-brewed beverages being more popular among tribal populations and in economically less developed areas. The urban and industrial workers favored factory-made spirits (whiskey, rum, brandy, or locally made liquor). In the rural areas, certain factors, such as age more than 20 years, married, male, Hindu religion, lower educational status, and agriculture as occupation, were found to be significantly associated with alcohol use.

Additional risk factors were having a good harvest, additional farm income, easy availability of local alcoholic beverages, and formation of a subcultural group of friends and relatives (Sundaram, Mohan, Advani, Sharma, & Bajaj, 1984). Caste affiliation was observed to be a major influence on drinking patterns (Mohan et al., 1981), with the Rajputs, ex-rulers and landlords, having a significant effect on the landless laborers and other social groups. Even with affluence as a result of agricultural revolution in the state of Punjab, it was observed that concomitant economic or developmental assets did not emerge; rather, there was a breakdown of the traditional social value systems in the villages, which led to increased alcohol consumption, especially among the young (Deb & Jindal, 1974). The demand for Indian-made foreign-style liquors, local liquors, and the home-distilled illicit liquors has increased dramatically.

Alcohol in contemporary India, thus, reflects the same paradox as in other areas of development of the country, where travel by modern airlines is juxtaposed with the continuing use of the oxcart. Along similar lines, drinking in India also exhibits widely divergent patterns (Mohan & Sharma, 1995).

POLICY AND PUBLIC HEALTH ISSUES IN INDIA

Under the federal structure of India, the country is divided into numerous states, currently 28 in number. Prohibition is a state subject, with alcohol legislation, excise, production, and sale under full state control. The complete prohibition of alcohol was much debated during the framing of the Constitution of India after independence but eventually was not made a mandatory and legally enforceable goal of the state (Government of India, 1996). Article 47 of the Constitution directs that "the State shall endeavor to bring about prohibition of the consumption, of intoxicating drinks and of drugs which are injurious to health." There are three main types of prohibition policies seen in practice: *complete prohibition* of production and consumption (only in the state of Gujarat); *partial prohibition*, where one or more types of liquor (usually *arrack*) are prohibited (in Tamil Nadu, Kerala, and Andhra Pradesh); and *dry days*, where consumption is prohibited only for certain days of the week or month. Regulatory laws pertaining to hours of sale, sale to minors, and drunken driving are in place all over India (Table 8.3).

The alcohol industry, however, continues to grow at a steady pace in India, with an estimated overall growth rate of 7 percent per annum and the cheaper beverages like rum growing at 22 percent per annum, per industrial estimates (Benegal, Gururaj, & Murthy, 2003). The volume of alcoholic beverages sold increased by 44.9 percent from 1,100.3 to 1,594.6 million liters between 1999 and 2004, while the value of alcoholic drinks rose by 75.6 percent from 368,122.2 to 646,528.2 million rupees (Euromonitor, 2005). The recent economic liberalization and relaxation of regulations for multinational companies are benefiting the alcohol industry (Isaac, 1998). The declining trend of alcohol consumption and saturation in many parts of the world, coupled with a huge untargeted potential market in India, could be contributing to this growth.

Table 8.3
Regulatory Measures for Alcohol in India

Prohibition of sales of alcoholic beverages to children below 18 years of age
Restriction of sales at retail shops between 9:00 P.M. and 9:00 A.M.
Prohibition of sales of alcoholic beverages in pubs and hotels between 2:30 P.M and 5:30 P.M. and after 10:00 P.M.
Prohibition of retail outlets within a one-kilometer radius of schools and places of worship
Attempts to regulate sales and consumption of alcohol along the state highways
Restrictions against drinking and driving or operating heavy machinery, including a breath alcohol limit of 30 milligrams %
Advertising of alcoholic beverages and tobacco products is banned

The practical challenges faced in today's India are those of inadequate implementation of policies and lack of political will for restriction of alcohol use. A recent study in Bangalore City across a calendar month found that 40 percent of drivers were over the legal limit (Gururaj & Benegal, 2002). In addition to alcohol-induced physical diseases, alcohol consumption has a disproportionately high association with injuries reported to emergency rooms (Benegal, Gururaj, & Murthy, 2002), domestic violence (Bhatt, 1998), deliberate self-harm and suicide (Gururaj & Isaac, 2001), and high-risk sexual behavior and HIV infection (Chandra, Krishna, Benegal, & Ramakrishna, 2003). Deaths as a result of consumption of adulterated illicit liquor remain by and large underreported. Alcohol use, therefore, inflicts a high cost on society.

Most states derive up to 10 percent of their revenue from taxation on alcohol (Saxena, 1999) and prohibition is seen to be associated with considerable loss of revenues. A little appreciated fact is that the direct and indirect costs attributable to people with alcohol dependence alone far exceed the profits from alcohol taxation (Benegal, Velayudhan, & Jain, 2000). There is an inadequate reappraisal of the tremendous public health impacts of alcohol (Table 8.4). The governmental response remains centered—that, too, in a limited way—on treatment provision to clinical populations instead of on a public health approach directed at communities. Identification of increasing alcohol use in women, as discussed subsequently, has also not attracted any specific intervention from governmental agencies.

Table 8.4
Global Burden of Disease and Disability Attributable to Alcoholic Beverages, by World Bank Region, 1990

Deaths (in thousands)	As percentage of total deaths	YLL(in thousands)	As percentage of total YLL	YLD (in thousands)	As percentage of total YLD	DALY (in thousands)	As percentage of total DALY
112.9	1.2	2,723	1.4	1,974	2.3	4,697	1.6

Source: Adapted from Murray and Lopez (1996).
Note: Abbreviations are as follows: DALY, disability-adjusted life years; YLD, years of life disabled; YLL, years of life lost.

ALCOHOL USE BY INDIAN WOMEN

Several aspects about alcohol use by women remain understudied in Indian literature. It is only recently that alcohol use in Indian women has begun to generate research interest. In the following section, we provide an overview of the status of women in India, followed by a review of the available literature, and we discuss some pertinent gender issues.

Status of Women in India

The problem of alcohol use among women cannot be separated from other sociocultural aspects (P. Murthy, 2002). The male child is favored over the female in India and is believed to carry the family lineage forward. This is reflected in the adverse sex ratio of the country, with 93 women for every 100 men (Census of India, 2001). With the exception of Kerala, every state has fewer women than men. Only 54 percent of women are literate, as compared to 76 percent of men. The average Indian woman bears her first child before she is 22 years old and has little control over her own fertility and reproductive health. In many states, more than half of women have been found to suffer from anemia (International Institute of Population Sciences and Macro International, 2007). Most women remain at home and do not have autonomy in decision making in their personal lives. Women are generally underrepresented in governance and decision-making positions in the country. The traditional roles and situation have been changing in recent times, and India has had a woman as both prime minister and president. More women are going for higher education and jobs and are economically independent. However, there are wide variations in the progress of women across different regions and settings. Alcohol use in women should be seen in the context of this prevailing social scenario.

Gender Perspectives and Cultural Background

The Indian sociocultural ethos, based largely on Hinduism, has always perceived alcohol use by women as a deviant behavior, and consequently, it has been heavily stigmatized. The tribal populations, sex workers, and low-caste groups living on the fringes of the dominant society were seen as exceptions and not bound by societal rules. The dominant stereotype associated alcohol use with the primitive and/or poor, the immoral, and the privileged (Musgrave & Stern, 1988; Thimmaiah, 1979). There is also a widespread notion that alcohol is limited to the upper crust of the rich and not favored by women from the

middle or upper socioeconomic classes (Kumar & Parthasarathy, 1994; Ray & Sharma, 1994). By and large, until recently, women have been seen as the victims of alcohol-related social and familial problems. The media have highlighted the role of women at the forefront of antialcohol agitations, perpetuating the image of women as champions of temperance (Pande, 2000). The fact that women can be alcohol users has not been adequately recognized and addressed.

In recent times, however, there are indications that the gender gap may be slowly bridging, with an emergence of liberal sociocultural values and increasing economic independence of women. There is identification of alcohol use in women. Cultural attitudes in India, being intensely guilt provoking and discriminatory about the use of alcohol by women, may prevent women from spontaneously reporting alcohol use. The woman who uses alcohol is triply stigmatized (Blume, 1991). First, there is the same stigma as for alcohol-dependent males, which is an attribution of alcohol use to either a moral deficiency or a self-inflicted illness. Second, the woman is held to a higher moral standard than the man, and consequently, the shame involved in her fall from grace is more intense. The third is the sexual stigma related to the myth that alcohol use makes women promiscuous. These stigmatizing beliefs may have implications for the acknowledgment of alcohol use and help seeking by women.

Gender Differences in the Prevalence of Alcohol Use

Gender is a strong and universal predictor of drinking behavior. Research on drinking patterns among women has generally shown that women drink less than men across the developing (Assanangkornchai, Pinkaew, & Apakupakul, 2003; Wei, Derson, Xiao, Li, & Zhang, 1999) and the developed world (Kraus, Bloomfield, Augustin, & Reese, 2000). Reports from India on women's drinking are sparse. The nationwide household survey on drug use (Ray et al., 2004) did not include women and was directed at a representative male population. However, in the same multimodality survey, in a focused thematic study, drug use in women was evaluated qualitatively. Among the surveyed women, alcohol was commonly used in addition to opioids and tranquilizers. The recently concluded National Family Health Survey (NFHS-3; International Institute for Population Sciences and Macro International, 2007) conducted on more than 230,000 men and women, revealed that only two percent of women drink alcohol in contrast to approximately one third of men. Alcohol use was found to be more common among women with no education (4%), in lowest wealth quintile (6%), and belonging to scheduled tribes (14%).Among women who drink alcohol, 40 percent drink alcohol about once a week, and 15 percent

drink alcohol almost every day. Overall, the prevalence of alcohol use among women has been estimated at less than 5 percent by most of the researchers who have included women in their study samples (Isaac, 1998; Mahal, 2000; Mohan et al., 1981; Sundaram et al., 1984). A longitudinal multicentered study among university students (Mohan & Sundaram, 1987) showed drinking in boys to be eight times (21.7%) more common than in girls (2.6%). Mohan et al. (1981), while reporting alcohol use as being at 3.2 percent in women, made an observation that girls had moved from "never use" to "ever use" status. Four large epidemiological studies undertaken in the early 1990s, covering northern, western, southern, and northeastern India, with sample sizes varying from 4,000 to 30,000 (Channabasavanna, Ray, & Kaliaperumal, 1990; Mohan & Desai, 1993; Mohan, Ray, & Sharma, 1993; Singh, Kaul, & Sharma, 1992), indicated that drug and alcohol use was a predominantly male phenomenon, and that 92 to 94 percent of women had never used any psychoactive substance in their lifetimes. The reasons behind this gender gap may be manifold. The physiological differences in acute effects and metabolism (Thomasson, 1995) have been thought to play a role. Certain sociocultural theories have also been put forth to explain the gender differences in the international literature. Drinking is seen as an expression of power dominance and aggression. Alcohol consumption in all-male groups may affirm the privileged status of being a man, rather than a woman (Campbell, 2000), and the ability to consume large amounts of alcohol without apparent impairment may help to demonstrate that the drinker is manly (Neff, Prihoda, & Hoppe, 1991); men have been shown to be more willing or motivated to take risks than women (Weber, Blais, & Betz, 2002); the expectations of enhanced sexual pleasure after alcohol consumption may also be more influential for men than women (Morr & Mongeau, 2004); additionally, greater role responsibilities, particularly at home, may also cause women (more than men) to limit their drinking (Ahlström, Bloomfield, & Knibbe, 2001). Some or all of these social contexts may play a role in India to maintain the gender gaps in drinking behavior.

Recently, there is a suggestion that alcohol use in women has been showing an upward trend. It has been observed in the southern state of Karnataka that 16 percent of women were "ever users," and 5.8 percent of all female respondents reported drinking alcohol in the past year out of a total sample size of 3,000 women (Benegal, Nayak, Murthy, Chandra, & Gururaj, 2005). These findings emerged from a collaborative multinational study called Gender, Alcohol, and Culture: An International Study. It was designed by the International Research Group on Gender and Alcohol using a similar methodology across a number of countries to study gender and cultural differences with regard to alcohol. The results from a state cannot be generalized for the whole of India; neverthe-

less, some useful insights can be gained, as discussed later. A study of drinking habits conducted in Bangalore City (Kumar, 1997) reported that one-fifth of young people who frequented pubs on weekends were girls aged between 13 and 19 years. Changing societal values and liberalization may have formed fertile ground for acceptability of and increased alcohol use in women.

Factors Influencing Initiation of Drug Use by Women

Interviews with female drinkers identified "boredom" and "lack of work at home" as factors for drinking among the high-income group; the low-income group identified "fatigue" and "spousal violence" as triggers for drinking (Kumar, 1997). Women also commonly reported being initiated into alcohol use by family members, and associated alcohol use among key family members was commonly reported. Psychosocial stressors were mentioned as maintaining factors for alcohol use more commonly in women than in their male counterparts (Selvaraj, Prasad, Ashok, & Appaya, 1997). Certain cultural beliefs, such as that alcohol has restorative properties after childbirth, were other common reasons in addition to giving company to husbands (N. V. Murthy, Benegal, & Murthy, 1995). Relief from tension was reported to be a common reason for drug use in less educated, poor, and rural women, while positive expectancies were commonly reported in women from affluent urban and educated backgrounds (Benegal et al., 2005).

Clinical Features of Alcohol Use by Women

The little information that exists about patterns of alcohol use in India indicates that women who drink have patterns of equally heavy alcohol use as men. A study from southern India of a representative sample of 7,445 adult men and 6,919 adult women (Benegal et al., 2003) found that the average consumption on typical drinking occasions, in both men and women, was five standard drinks (12 grams of ethanol per drink). There were no significant differences between male and female current drinkers in terms of age at first drink or the duration of drinking. Eighty percent of male consumers and 65 percent of female consumers drank at hazardous levels (defined as Alcohol Use Disorders Identification Test[6] scores of 8 and above), and a large proportion of drinkers of both genders reported daily or almost daily use. Frequent (weekly or more) heavy drinking (five or more drinks per occasion) was found in 47.6 percent of male drinkers and in 27.9 percent of female drinkers in another large-scale study by Benegal et al. (2005). A significant number of female users (42%) also expressed the opinion that it was really no point having a drink if one did not feel high at the end of it. This supports the fact that the Indian culture of

"drinking to get drunk" among alcohol users is seen among women users as well.

Solitary drinking was commonly seen in female alcohol users seeking treatment (Benegal et al., 2003). Women in urban areas were more likely to drink with meals, at friends' homes, at home, and at parties. The men, on the other hand, were more likely to drink at pubs and bars. The most common reported time of drinking was the weekend after five in the evening, followed by weekday evenings after five (Benegal et al., 2005).

The most commonly used beverages were found to be spirits (Indian-made foreign liquor) for both women (64.3%) and men (70.7%). Wine was drunk by significantly more women than men (Benegal et al., 2005). Data from the alcohol industry also suggest that the sale of hard liquors is much higher in India as compared to beers and wines and that hard liquors constitute more than 95 percent of the market for both men and women. There has been a recent introduction of new products, such as flavored and mild alcoholic products, which is aimed at recruiting nondrinkers and women (Naik, 2002).

Women experience several problems different in nature from men. It was seen that alcohol-using women were less likely to have an intact marriage or stable relationships to provide support, unlike the male alcohol users (N. V. Murthy et al., 1995). Forcible sex after the age of 16 years was reported by significantly more women (10.4%) as compared to men (1.6%), and most abusers were close relatives, spouses, or partners (Benegal et al., 2005). Three hospital-based studies of female alcoholics from southern India (Benegal et al., 2003; N. V. Murthy et al., 1995; Prasad, Murthy, Varma, Mallika, & Gopinath, 1998) reported a high prevalence of medical and psychiatric morbidity along with delayed help seeking until physical health or emotional crises made it inevitable. It was widely believed by both men and women that female users are more likely to experience alcohol-related physical problems compared to men. Overall, half of the total women studied felt that alcohol use in women was acceptable, but these views were expressed by those who had an urban background (Benegal et al., 2005). There was also a strong bias among men against alcohol use in women (even among men who themselves used alcohol).

Emerging Patterns of Alcohol Use by Indian Women

There appear to be two widely divergent patterns of drinking among Indian women based on their socioeconomic status, education, and urban versus rural background (Benegal et al., 2005).

There is a traditional pattern of alcohol use that is confined to less educated, poor, or rural women. Their drinking is marked by binging and drinking to

intoxication, with a high proportion of heavy drinkers (defined as five or more drinks per occasion). They also predominantly use cheaper, high-alcohol beverages: spirits, illicit liquor, and country liquor. Drinking is mostly alone, in undersocialized drinking contexts and at home. Though they drink less frequently, their pattern is close to the male pattern of drinking. Drinking appears to serve the function of tension relief, rather than the enhancement of positive experiences. These women also report more alcohol-related familial, financial, and health-related problems (Benegal et al., 2005).

Benegal et al. (2005) also observed another and recent pattern of alcohol use in educated urban women. They are comparatively younger, educated, with higher spending capacity and a shorter duration of drinking. They drink mostly in socialized circumstances: at restaurants, parties, with spouses, family members, colleagues, and friends. Although spirit drinking is still the most common form, use of low-alcohol beverages like wine and beer is also present. Motivation appears to be equally split between relief of tension and the enhancement of positive experiences. They drink less on a typical drinking occasion and have fewer problems as a result of their drinking. This latter group, with a shorter duration of drinking, reflects the recent change in attitude in a section of society.

Treatment Seeking by Women

Female alcohol users are underrepresented in clinical psychiatric practice in India. Data from treatment centers have shown that female drug users form a minority (3%) of the treatment-seeking population (Ray et al., 2004). A profile of all women treated for alcohol dependence during a 12-year period between 1983 and 1994 revealed a total number of 77, with the majority being from lower socioeconomic backgrounds and having high preceding (33.8%) and concurrent (75.3%) psychiatric and medical (68.7%) morbidity. Only 13 percent of the sample attended follow-up regularly, and the population as a whole showed quite high dropout rates (N. V. Murthy et al., 1995). A retrospective structured chart review of female substance (including alcohol) abusers again indicated low follow-up rates in addition to higher rates of comorbidity and family history (Grover, Irpati, Saluja, Matto, & Basu, 2005). Several factors may be contributing to low treatment seeking: societal disapproval, stigma, presence of a spouse with alcohol use, lack of adequate social support, and lack of separate treatment services for women. There is a need to understand the perspective of female alcohol users and address their barriers to seeking treatment.

Research on Gender Issues Related to Alcohol in India: Future Directions

There is now increasing recognition of alcohol use among women in India, and preliminary information is available regarding clinical and epidemiological aspects of alcohol use. There are several lacunae in the existing research. There is a need to study the (1) course and outcome of alcohol use, (2) measures directed at early identification and prevention of alcohol use, and (3) specific issues, such as reproductive health, in female users. There is also a need to develop effective treatment models catered to female alcohol users. Future studies should be directed to accurately identify the risk and protective factors for alcohol use in both men and women. Changing cultural norms and influences of social factors also should be studied, both quantitatively and qualitatively.

CONCLUSION

India, in several ways, is a unique culture with enormous diversity. Alcohol has been consumed since historical times in India. Traditional religious and cultural beliefs permitted alcohol use in moderation on social occasions and in rituals. Drinking was completely prohibited only for certain sections of society: students, learned men (Brahmins), and those seeking religious purity. Alcohol was never a part of the daily routine in Indian culture. Women were also not permitted to use alcohol, and alcohol was associated with bad character in women. Alcohol use has not been commonly seen in Indian women, and drinking has remained a male phenomenon, with consumption mostly away from home. There has been no normative pattern of alcohol use, and India is closer to an ambivalent drinking culture in many ways. On one hand, abstinence has been held in high regard, and on the other, it has been seen that people who use alcohol "drink to get drunk."

Table 8.5
Changing Patterns of Alcohol Consumption in India

Relatively greater acceptability of drinking as a social norm, especially among the affluent and urban
Wine and beer drinking are gradually emerging
Increase in drinking among women
Heavy drinking is common among male and female drinkers
Alcohol use combined with other high-risk behaviors

Recent times have seen a few changes with increasing liberalization and changing societal values (Table 8.5). Alcohol use has been recognized in women, and generally, the pattern is the same as in men, with most women using in heavy amounts. Women also have higher rates of comorbid physical or psychiatric illnesses. Female alcohol users continue to face stigma from society and even from male alcohol users. There is an emerging segment of urban and affluent women who drink in a socialized context or with family. Drinking appears to be more socially acceptable among them, reflecting the influences of urbanization and globalization. Gender-specific issues by and large remain understudied in India, and there is a need to devote research efforts to them so that effective interventions can be designed aimed at prevention and management.

NOTES

1. The oldest and most revered Indian collection of Vedic Sanskrit hymns dedicated to the gods.
2. A collection of teachings and a guide to proper ritual action.
3. Ancient Indian scriptures divide society into four classes: the Brahmins (teachers, scholars, and priests), the Kshatriyas (kings and warriors), the Vaishyas (traders), and Shudras (agriculturists, service providers, and some artisan groups).
4. The traditional system of Indian medicine attributed to Dhanvantari, the physician to the gods in Hindu mythology.
5. Treatises on the proper righteous way of orderly life for Hindus.
6. Developed by the World Health Organization.

REFERENCES

Achaya, K. T. (1998). *Indian food: A historical companion*. New Delhi: Oxford University Press.

Ahlström, S., Bloomfield, K., & Knibbe, R. (2001). Gender differences in drinking patterns in nine European countries: Descriptive findings. *Substance Abuse, 22*, 69–85.

Assanangkornchai, S., Pinkaew, P., & Apakupakul, P. (2003). Prevalence of hazardous-harmful drinking in a southern Thai community. *Drug and Alcohol Review, 22*, 287–293.

Benegal, V., Gururaj, G., & Murthy, P. (2002). Project report on a WHO multi centre collaborative project on establishing and monitoring alcohol's involvement in casualties, 2000–01. Retrieved April 15, 2008, from http://www.nimhans.kar.nic.in/deaddiction/lit/alcohol%20and%20%20injuries_who%20collab.pdf

Benegal, V., Gururaj, G., & Murthy, P. (2003). Report on a WHO collaborative project on unrecorded consumption of alcohol in Karnataka, India. Retrieved April 15, 2008, from www.nimhans.kar.nic.in/deaddiction/Publications.html

Benegal, V., Nayak, M., Murthy, P., Chandra, P., & Gururaj, G. (2005). Women and alcohol use in India. In I. Obot & R. Room (Eds.), *Gender, alcohol and culture in low income countries* (pp. 89–124). Geneva, Switzerland: World Health Organization.

Benegal, V., Velayudhan, A., & Jain, S. (2000). Social costs of alcoholism: A Karnataka perspective. *NIMHANS Journal, 18,* 6–7.

Bhatt, R. V. (1998). Domestic violence and substance abuse. *International Journal of Gynaecology and Obstetrics, 63,* 25–31.

Blume, S. B. (1991). Women, alcohol and drugs. In N. Miller (Ed.), *A handbook of drug and alcohol addiction* (pp. 147–177). New York: Marcel Dekker.

Brown, J. (1972). *Gandhi's rise to power: Indian politics 1915–1922.* Cambridge: Cambridge University Press.

Campbell, H. (2000). The glass phallus: Pub(lic) masculinity and drinking in rural New Zealand. *Rural Sociology, 65,* 562–581.

Census of India. (2001). *Provisional population totals.* New Delhi, India: Registrar General and Census Commissioner.

Chand, T. (1972). *Liquor menace in India.* New Delhi: Gandhi Peace Foundation.

Chandra, P. S., Krishna, V. A., Benegal, V., & Ramakrishna, J. (2003). High-risk sexual behavior and sensation seeking among heavy alcohol users. *Indian Journal of Medical Research, 117,* 88–92.

Channabasavanna, S. M., Ray, R., & Kaliaperumal, V. G. (1990). *Patterns and problems of non-alcoholic drug dependence in Karnataka,* Bangalore, India: Department of Health and Family Welfare, Government of Karnataka.

Chopra, R. N., & Chopra, I. C. (1965). *Drug addiction: With special reference to India.* New Delhi: Council of Scientific and Industrial Research.

Deb, P. C., & Jindal, B. R. (1974). Drinking in rural areas: A study in selected villages in Punjab in the wake of Green Revolution. *NIMHANS Journal, 16,* 87–91.

Dikshitar, V. R. (1951). *Pre-historic south India.* Madras, India: n.p.

Doniger, W. (1992). *The laws of Manu.* New Delhi: Penguin Classics.

Euromonitor. (2005). *Alcoholic drinks in India.* London: Author.

Government of India. (1996). *The Constitution of India.* New Delhi, India: Ministry of Law and Justice.

Grover, S., Irpati, A. S., Saluja, B. S., Matto, S. K., & Basu, D. (2005). Substance dependent women attending a de-addiction clinic in northern India: Socio-demographic and clinical profile. *Indian Journal of Medical Sciences, 59*(7), 283–291.

Gupta, P. C., Saxena, S., Pednekar, M. S., & Maulik, P. K. (2003). Alcohol consumption among middle-aged and elderly men: A community study from western India. *Alcohol and Alcoholism, 38,* 327–331.

Gururaj, G., & Benegal, V. (2002). *Report on Project on Prevention of Drunken Driving in Bangalore City.* Retrieved April 15, 2008, from http://www.nimhans.kar.nic.in/deaddiction/lit/Female%20Alcoholics.pdf

Gururaj, G., & Isaac, M. (2001). *Epidemiology of suicides in Bangalore City* (Publication No. 43). Bangalore, India: National Institute of Mental Health and Neuro Sciences.

International Institute of Population Sciences and Macro International. (2007). *National Family Health Survey (NFHS-3) 2005-06.* Mumbai, India: International Institute of Population Sciences.

Isaac, M. (1998). India. In M. Grant (Ed.), *Alcohol and emerging markets: Patterns, problems, and responses* (pp. 145–175). Philadelphia: Brunner / Mazel.

Kraus, L., Bloomfield, K., Augustin, R., & Reese, A. (2000). Prevalence of alcohol use and the association between onset of use and alcohol related problems in a general population sample in Germany. *Addiction, 95,* 1389–1401.

Kumar, G.S.U., & Parthasarathy, R. (1994). Alcohol drinking in rural areas: Some observations. *Indian Journal of Behavioral Science and Rehabilitation, 1,* 9–14.

Kumar, V. S. (1997). Behavioral malignancy: Alcoholism, a bleak future? In J. Azariah, H. Azariah, & D.R.J. Macer (Eds.), *Bioethics in India: Proceeding of the International Bioethics Workshop in Madras: Biomanagement of biogeoresources* (pp. 16–19). Madras, India: University of Madras / Eubios Ethics Institute.

Mahal, A. (2000). What works in alcohol policy? Evidence from rural India. *Economic and Political Weekly, 35,* 3959–3968.

Mandelbaum, D. (1965). Alcohol and culture. *Current Anthropology, 6,* 3.

Mohan, D., Chopra, A., Ray, R., & Sethi, H. (2001). Alcohol consumption in India: A cross-sectional study. In A. Demers, R. Room, & C. Bourgault (Eds.), *Surveys of drinking patterns and problems in seven developing countries* (pp. 103–114). Geneva, Switzerland: World Health Organization.

Mohan, D., & Desai, N. G. (1993). *A survey on drug dependence in the community, urban megapolis, Delhi.* New Delhi: Indian Council of Medical Research.

Mohan, D., Ray, R., & Sharma, H. K. (1993). *Collaborative study on narcotic and psychotropic substances.* New Delhi: Indian Council of Medical Research.

Mohan, D., & Sharma, H. K. (1995). Alcohol and culture in India. In D. B. Heath (Ed.), *International handbook on alcohol and culture* (pp. 128–141). Westport, CT: Greenwood Press.

Mohan, D., Sharma, H. K., Sundaram, K. R., & Advani, G. B. (1981). Prevalence and pattern of alcohol abuse in rural community and its correlation with psychosocial sequalae. New Delhi: Ministry of Social Welfare.

Mohan, D., & Sundaram, K. R. (1987). *A multi-centered study of drug abuse among students: A preliminary report.* New Delhi: Ministry of Welfare / All India Institute of Medical Science.

Morr, M. C., & Mongeau, P. A. (2004). First date expectations: The impact of sex of initiator, alcohol consumption, and relationship type. *Communication Research, 31,* 3–35.

Murray, C.J.L., & Lopez, A. D. (1996). Quantifying the burden of disease attributable to ten major risk factors. In C.J.L. Murray & A. D. Lopez (Eds.), *The global burden of disease: A comprehensive assessment of mortality and disability from diseases,*

injuries and risk factors in 1990 and projected to 2020 (pp. 295–324). Cambridge, MA: Harvard University Press.

Murthy, N. V., Benegal, V., & Murthy, P. (1995). *Alcohol dependent females: A clinical profile.* Retrieved April 15, 2008, from http://www.nimhans.kar.nic.in/deaddiction/lit/Female%20Alcoholics.pdf

Murthy, P. (2002). *Women and drug abuse: The problem in India* (R. Ray, Sci. Ed.). New Delhi, India: Ministry of Social Justice and Empowerment / United Nations Office on Drugs and Crime, Regional Office for South Asia.

Musgrave, S., & Stern, N. (1988). Alcohol demand and taxation under monopoly and oligopoly in south India in the 1970s. *Journal of Development Economics, 15,* 15–16.

Naik, S. (2002). *Indian spirits market: Regulatory shackles easing.* Mumbai: Rabo India Finance.

Needham, J. (1980). *Science and civilisation in China* (Vol. 5[4]). Cambridge: Cambridge University Press.

Neff, J. A., Prihoda, T. J., & Hoppe, S. K. (1991). "Machismo," self-esteem, education, and high maximum drinking among Anglo, black, and Mexican-American male drinkers. *Journal of Studies on Alcohol, 52,* 458–463.

Pande, R. (2000). From anti-arrack to total prohibition: The women's movement in Andhra Pradesh, India. *Gender, Technology and Development, 4,* 131–144.

Pittman, D. (1964). Social and cultural factors in drinking patterns, pathological and non-pathological. In *Selected Papers Presented at the 27th International Congress on Alcohol and Alcoholism* (Vol. 1, pp. 1–13). Laussane, Switzerland: International Council on Alcohol and Alcoholism.

Prakash, O. (1961). *Food and drinks in ancient India.* New Delhi: Munshi Ram Manohar Lal.

Prakash, O. (1956). *Grhyasutra.* Bombay, India: Venkateswara.

Prasad, S., Murthy, P., Varma, V., Mallika, R., & Gopinath, P. S. (1998). Alcohol, drugs dependence in women: A preliminary profile. *NIMHANS Journal, 4,* 87–91.

Rahman, A. (1984). Science and technology in medieval India. In A. Rahman (Ed.), *Science and technology in Indian culture: A historical perspective* (p. 123). New Delhi, India: National Institute for Science, Technology and Development.

Rao, S.S.V.V., & Rao, C.R.P. (1976). Drinking in the tribal world: A cross-cultural study in "cultural theme" approach. *Man in India, 57*(2), 97–102.

Ray, R., Mondal, A. B., Gupta, K., Chatterjee, A., & Bajaj, P. (2004). *The extent, pattern and trends of drug abuse in India: National survey.* New Delhi: United Nations Office on Drugs and Crimes / Ministry of Social Justice and Empowerment.

Ray, R., & Sharma, H. K. (1994). Drug addiction—an Indian perspective. In V. P. Bashyam (Ed.), *Souvenir of ANCIPS* (pp. 105–109). Madras: Indian Psychiatric Society.

Reddy, G. P. (1971). When liquor decides everything: Drinking subculture among the tribes of Andhra. *Social Welfare, 17,* 4–5.

Saxena, S. (1999). Country profile on alcohol in India. In L. Riley & M. Marshall (Eds.), *Alcohol and public health in 8 developing countries* (pp. 37–60). Geneva, Switzerland: World Health Organization.

Selvaraj, V., Prasad, S., Ashok, M. V., & Appaya, M. P. (1997). Women alcoholics: Are they different from men alcoholics? *Indian Journal of Psychiatry, 39*, 288–293.

Singh, A. D., Kaul, R. K., Sharma, S. G. et al., (1992). *Survey of drug abuse in Manipur State*. Imphal, India: Department of Science, Technology and Environment, Government of Manipur.

Srinivas, M. N. (1997). *Social change in modern India*.New Delhi, IndiaOrient Longman.

Sundaram, K. R., Mohan, D., Advani, G. B., Sharma, H. K., & Bajaj, J. S. (1984). Alcohol abuse in a rural community in India. Part I: Epidemiological study. *Drug and Alcohol Dependence, 14*, 27–36.

Thimmaiah, G. (1979). *Socio-economic impact of drinking, state lottery and horse-racing in Karnataka.* New Delhi: Sterling.

Thomasson, H. R. (1995). Gender differences in alcohol metabolism: Physiological responses to ethanol. In M. Galanter (Ed.), *Recent developments in alcoholism: Vol. 12. Alcoholism and women*. New York: Plenum.

Weber, E. U., Blais, A.-R., & Betz, N. E. (2002). A domain specific risk attitude scale: Measuring risk perceptions and risk behaviors. *Journal of Behavioral Decision Making, 15*, 263–290.

Wei, H., Derson, Y., Xiao, S., Li, L., & Zhang, Y. (1999). Alcohol consumption and alcohol-related problems: Chinese experience from six area samples, 1994. *Addiction, 94*, 1467–1476.

World Health Organization. (2004). *Global status report on alcohol.* Geneva, Switzerland: World Health Organization.

Chapter **9**

Alcohol and Drug Abuse in Malaysia

See Ching Mey, PhD, MEd, and
Cecilia A. Essau, PhD, MA, HBA

The Federation of Malaysia covers a total area of 329,847 square kilometers distributed between two regions, Peninsular Malaysia and East Malaysia on the island of Borneo, which are separated by approximately 640 miles of the South China Sea. There are 11 states and 2 federal territories in Peninsular Malaysia, and 2 states and 1 federal territory in East Malaysia.

The total population is estimated at 26.6 million (2007) and comprises approximately 60 percent Malays, who are constitutionally defined as Muslims; about 26 percent Chinese; and 14 percent Indians and indigenous peoples. The gross national income per capita is USD 5,620 (World Development Indicators Database, 2007).

The Chinese and principally southern Indians came to Malaysia during the British colonial times to work on the plantations, in the tin mines and to provide mercantile and administrative support services. The ethnic groups coexist in harmony, but there is little cultural integration.

ATTITUDES TO AND PATTERNS OF ALCOHOL CONSUMPTION

Before Islamization, the Malay population could be said to follow a largely Hindu/Buddhist faith. Missionaries brought Islam beginning in the thirteenth century with the establishment of several Muslim states in Peninsular Malaysia. Historically, the coming of Islam is identified with the emergence of the Muslim Sultanate of Malacca under Iskander Shah (formerly the Hindu

ruler Parameswara, who converted to Islam on his marriage to a princess from Pasai) at the beginning of the fifteenth century (Freeman, 2003). The Malay Muslim population can thus be described as, by inclination and religion, abstainers.

The same does not hold true for the rest of the population, who brought with them their traditional alcoholic beverages from their home countries or continued to use their traditional fare. These include, among south Indians, *toddy*, a drink made from the fermentation of the sap from the coconut palm, which has an alcohol content of between 4 and 6 percent and which, until recently, was widely available through government- or estate-regulated *toddy* shops (Arasaratnam, 1970). The estate owners used alcohol as a colonizing and proletarianizing force (Van-Onselen, 1982).

The Chinese brought with them *samsu*, a grain-derived alcoholic drink which, if distilled, can reach between 37 and 70 percent alcohol content. *Samsu* production has become an enterprise worth more than RM 180 million annually (Assunta, 2001). A good portion of this is illegally sold through traditional medicine outlets and sundry shops. Often school dropouts are easy targets of this cheap liquor. The price for a 150-milliliter bottle of *samsu* can be as cheap as RM 3.00 (about USD 1.00), hence making it affordable to these children (Consumer Association of Penang, 2008).

A rice wine, *tapai*, is available among the indigenous population in the East Malaysian states of Sabah and Sarawak and is very much a part of their festivals and hospitality. Refusal to drink may be a breach of etiquette.

A survey on urban lifestyles puts alcohol consumption among Malays at 1.4 percent, among Chinese at 27 percent, and among Indians at 23.4 percent (Inoguchi, Baáñez, Tanaka, & Dadabaev, 2005). Another, the Second National Mental Health Survey, suggests that 23 percent of non-Muslims aged 18 years and above admitted to alcoholic beverage consumption (Rugayah et al., 2000). This survey also reported that the rural rate (27.3%) was higher than the rate for the urban population (21.1%). More men (38.3%) drank compared to women (9.8%; Loe & Maimunah, 1997). The pattern of these findings can be ascribed to the culture and social acceptability of alcohol drinking in the Malaysian community among the non-Muslims.

European forms of alcoholic beverages, such as stout, beer, brandy (cognac), and whiskey, were brought by the British. Guinness Stout went from being imported from Dublin to being brewed locally by a Malaysian company, Guinness Anchor Berhad, in a stronger form (8% alcohol content). To avoid the high import duties on beer, Carlsberg A/S of Denmark, the biggest brewer in the world, built a factory and started brewing Carlsberg Green Label locally in 1972. It has viewed Malaysia as a "very important and attractive market"

(Assunta, 2001, pp. 18–21). Currently, Carlsberg is the undisputed beer market leader (Jernigan & Indran, 1997).

The rest of the alcoholic drink market in Malaysia is expected to continue performing positively. It grew at an annual average rate of 1.2 percent between 2000 and 2005 (Guinness Centre, 2005). Traditionally, the most popular European alcoholic beverages have been cognac (brandy), followed by whisky. Wine has become a favorite and a mark of cultural refinement. Some 3.8 million liters of wine are imported. They come from Australia, United States, Chile, South Africa, Argentina, and France.

Malaysia is the tenth largest consumer of alcohol in the world, with an estimated expenditure of over USD 500 million on alcoholic beverages (Assunta, 2001). Although only about one-fourth of the Chinese and Indians admit to drinking, the statistics suggest that they do so heavily. There is no recent record of the number of alcoholics in Malaysia, but it can be foreseen that alcohol abuse may become a social problem in the near future.

Controlling the Drinking Habit

The government, while remaining liberal toward the consumption of alcohol, has taken action to curb the habit. A set of taxes has increased the retail costs of all beverages. For example, the excise duty on beer rose from RM 2.75 in 1991 to RM 7.40 in 2005. The combined import, excise, and sales taxes on a standard 750-milliliter bottle of wine are approximately RM 15.00 (about USD 5.00).

In Malaysia, there has been concern since the late 1990s about aggressive marketing by international companies, using advertisements and promotional tactics in bars and clubs that would not be permitted in many Western countries (Jernigan, 1997). As a result, Malaysia banned direct alcohol advertising over public radio, television, and on billboards, but it is permitted in cinemas, on video cassettes, and in the print media. Sponsorship activities are allowed.

Assunta (2001) has listed the tactics used by the alcoholic beverage industry to attract and keep drinkers. A few of these follow:

Ethnic Targeting

The 1980s Guinness campaign "Guinness is good for you" has been a successful campaign in capturing the poorer working class, especially among the poorer estate workers and laborers. This stout is promoted as a drink that "will put back what the day takes out," and it contains more alcohol than beer for the same price. Even religious occasions, such as Deepavali, the Hindu festival

of lights, are not spared in the advertising campaign. The targets are generally poorer Indians.

Claiming Health Benefits for Alcoholic Beverages

At least two alcoholic concoctions—Yomeishu, which contains 14 percent alcohol, and DOM Benedictine, which contains 40 percent alcohol—have been advertised as restoratives for work-related fatigue and mothers who have just given birth.

Manufacturers of *samsu* have resorted to many misleading health claims. One brand recommends it for anemia, loss of appetite, indigestion, and healing properties (Consumer Association of Penang, 2008).

The growing popularity of red wines may be attributed to claims that they contain substantial amounts of antioxidants to slow down the aging process.

Sponsoring Activities

Alcohol companies used to sponsor music and sports events before the ban on most forms of alcohol advertising. The industry now undertakes indirect advertising or brand stretching. Events in private clubs may be sponsored, and products are offered at substantial discounts to entice drinkers.

Problems Associated with Alcohol

The victims of alcohol abuse are the poor, particularly the rural Indian laborers who work on rubber and oil palm estates, who drink *samsu* and *toddy*. It is a major cause of poverty. Of an estimated 200,000 drinkers, 75 percent are *samsu* drinkers (Assunta, 2001).

Drinking and driving causes about 0.3 percent of total road accidents nationwide and has become enough of a problem for the government to impose roadside monitoring and increased penalties for driving under the influence.

In Malaysia, it is evident that nonbiological factors, especially unemployment, lower educational level, alcohol use, media influence, the presence of gangs, and access to weapons and firearms, are important precursors of violent crime. Alcohol has a strong association with aggressive behavior and violence. Women who live with heavy drinkers are at greater risk of being a victim of partner violence, and men who are under the influence of alcohol tend to commit more serious violence at the time of assault (World Health Organization, 2006).

The reasons for perpetration and victimization in intimate partner violence in Malaysia are not clearly documented. However, based on cases reported to

the Department of Social Welfare, five common causes for violent acts between husbands and wives in 2004 were financial problems, extramarital affairs, conflicts with in-laws, stress at the workplace, and alcohol abuse (Malaysia Department of Social Welfare, 2004).

Legislation Regulating Alcohol

Malaysia has laws related to sales of alcohol. Under Section 32 (1) and 33 (1) of the Excise Act 1976, no person can sell (either wholesale or retail) intoxicating liquor either for use within a premise or outside without a license. The license issued must specify the precise place where sale is allowed. Any sale without a license is an offense under the act. The legal limit for drinking is 80 milligrams of alcohol in 100 milliliters of blood. If a person is caught driving while over the legal limit, he has to pay a penalty of RM 2,000 (about USD 700) or spend a maximum of six months in jail, or both, for the first offense, along with suffering the loss of his driver's license.

In November 2008 several local government councillors from *Parti Islam Se-Malaysia* (Pan-Malaysian Islamic Party—PAS) raised the issue of banning the sale of alcoholic drinks in Selangor, who voiced their concern that alcoholic drinks were easily available for youths (Bernama, 2008). Selangor is one of the states governed by *Pakatan Rakyat* (People's Pact), a new government formed on April 1, 2008. However, according to Chief Minister Selangor, Tan Sri Abdul Khalid Ibrahim, Selangor will not take the drastic action of totally banning the sale of alcoholic drinks as its residents are multiracial and multireligious. He stated that Selangor state government will intensify enforcement to ensure that alcoholic drinks were not sold to Muslims and youths under the age of 18. The state government will hold discussions with traders, local governments, other government agencies, and non-governmental organizations next year to identify the best methods to enhance enforcement on liquor consumption.

DRUG ABUSE IN MALAYSIA

Like other fast-developing countries, Malaysia also faces a plethora of social problems resulting from economic, technological, and societal growth. One of them is drug abuse. Drug abuse in Malaysia is not a recent phenomenon. It has a history that is closely associated with the early development of the country. Drugs were introduced into the country in the early nineteenth century with the importation of migrant labor from China and south India. The Chinese brought the habit of opium smoking, while Indians introduced cannabis or ganja smoking (How, 1999).

The Bureau for International Narcotics and Law Enforcement Affairs (2008) stated that Malaysia is not a significant source country or transit point for U.S.-bound illegal drugs, but regional and domestic drug trafficking remains a problem, and international drug syndicates are still looking to Malaysia as a regional production hub for crystal methamphetamine and ecstasy, or MDMA. The narcotics imported to Malaysia include heroin and marijuana from the nearby Golden Triangle area, while other drugs such as amphetamine-type stimulants (ATS), including crystal methamphetamine, ecstasy, and ketamine are brought from India. Ketamine and crystal methamphetamine are the fastest-growing illicit drugs used in Malaysia.

Malaysian Drug Scenario

Despite strict laws and penalties for drug use and trafficking, the drug problem has been identified as a security problem since 1983, when specific laws were formulated to curb the epidemic. However, the number of drug users identified each year still increases.

Dato' Dr. Chua Soi Lek, then minister of health, in a speech in 2004, stated that the number of drug addicts in Malaysia detected in 2003 was 36,996—a 16 percent jump from the 31,893 cases detected in 2002, vis-à-vis the small 1 percent increase from 2001 to 2002 (Chua, 2004). Chua also said that a total of 20,194 cases were newly detected cases. Malays made up 71.4 percent of the newly detected cases, Chinese 10.6 percent, and Indians 8.2 percent. Among those who gave reasons for their initiation into drug abuse, 52 percent attributed it to peer pressure, 25 percent to curiosity, and 17 percent to a search for pleasure. Of great concern to the Malaysia government is the fact that 70 percent of the drug addicts detected in 2003 were young adults between 20 and 39 years of age—people in their prime of life who ought to be economically and socially most productive. Heroin (36%), morphine (30%), ganja (23%), and methamphetamine, or *syabu* (7%), constituted the most frequently abused drugs (Chua, 2004).

Interestingly, according to UNICEF Malaysia (2007), in 2006, as many as 22,811 drug users were detected, 12,430 of whom were repeat offenders. There was a 34.47 percent reduction in the number of cases detected in 2006 as compared to the 34,813 cases detected in 2005. Pulau Pinang recorded the highest number of detected cases at 5,127, followed by Kedah (2,634), Perak (2,545), Kelantan (2,243), and Johor (2,329). The profile of the drug user is (UNICEF Malaysia, 2007) 97.97 percent male; 69.48 percent Malay; 71.04 percent youth aged between 25 and 29 years old; 78.30 percent minimum secondary Form 3 education (16 years old); 90.69 percent employed (majority laborers); 60.73

percent heroin/morphine users; and 12.56 percent ATS abusers. About 59.78 percent of users said that their use of drugs was influenced by friends.

In its latest bulletin, the National Anti-Drugs Agency (2007) reported that at the end of June 2007, 14,489 drug users were identified, showing another 36.48 percent decrease compared with 2006 (22,811 drug users). The profile of the drug users are 97.60 percent male; 72.62 percent Malay; 70.00 percent youth aged between 19 and 24 years old; 79.17 percent minimum secondary Form 3 education; 92.03 percent employed (majority laborers); 62.56 percent heroin/morphine users; and 10.28 percent ATS abusers. About 55.65 percent of them said that they were influenced by friends to use drugs. Pulau Pinang still recorded having the highest number of drug abusers (3,433), followed by Johor (2,004), Kuala Lumpur (1,756), and Kedah (1,604).

Drug Treatment and Rehabilitation Programs in Malaysia

Drug abuse and related disorders exert a heavy burden on the country's health care and legal systems. Historically, drug abusers were rehabilitated involuntarily in correctional, rather than health care facilities. This primarily criminal treatment approach had limited effectiveness and led to widespread public dissatisfaction until the introduction of medical treatments for addiction. Naltrexone was introduced in 1999, buprenorphine was introduced in 2001 and methadone in 2003. Agonist maintenance programs were embraced rapidly by the medical community in Malaysia. Currently over 30,000 opiate-dependent patients are treated with agonist maintenance treatments by more than 500 medical practitioners in Malaysia (Mazlan, Schottenfeld, & Chawarski, 2006).

The first phase of the Methadone Maintenance Therapy (MMT) program was officially launched at the national level in October 2005. It involved 1,241 clients spread over eight government hospitals, two community health clinics, and seven private health practitioners (UNGASS Country Progress Report, 2008). The program was able to achieve a retention rate of 75 percent after 12 months of implementation. This result is considered a major achievement when measured against World Health Organization (WHO) retention rate standards of 55 to 60 percent as well as against comparable programs in Poland, Lithuania, and Ukraine.

All the clients of the program were closely monitored using the WHO Quality of Life tests combined with appropriate physical, psychological, social relationship, and environmental evaluations over the treatment period.

The expansion of the MMT program (currently in phase 2) will enable an estimated 25,000 clients to be reached by 2010. However, the target deter-

mined by WHO and the Joint United Nations Programme on HIV/AIDS requires that 60 percent of the intravenous drug using population have access to the intervention for it to have an effective impact. This means that the existing program needs to be further expanded. Though this represents a significant challenge, the results thus far from the program have created an argument for long-term commitment of resources by the government and for the continued expansion of coverage.

Drug treatment and rehabilitation in Malaysia are operated either by the National Anti-Drugs Agency, the Prison Department, the Ministry of Home Affairs, or by nongovernmental organizations (NGOs). The National Anti-Drugs Agency operates 29 treatment and rehabilitation centers, also known as Pusat Serenti. These Serenti drug rehabilitation centers provide rehabilitation services for drug users from various categories of addiction ranging from hard-core to new users (Malaysia Ministry of Health, 2002). Until March 2008, there were 7,328 drug users in 29 Serenti drug rehabilitation centers throughout the country. About 646 of them (8.81%) were HIV-positive (Personal Communication, 2008).

The prison system currently operates 12 programs throughout the country. These programs are based on the therapeutic community (TC) model. The programs include a core program philosophy, daily activity schedule, and specific skills, or "tools of the house," that must be learned by each inmate. The programs consist of three phases: (1) induction (pretreatment), (2) core treatment, and (3) reentry (Gerald & Steven, 2003).

The goal of the programs is not punishment. Many activities are consistent across all of the prison TC programs, including (1) different types of group activities (e.g., encounter, peer confrontation, assertiveness training, morning meeting, and house meeting), (2) different types of individual activities (e.g., individual counseling with staff, pull-ups, haircut, and peer confrontation), (3) vocational training, (4) religious education and counseling, and (5) house chores.

The programs use volunteers to assist with religious components, which provide equal emphasis on Muslim, Hindu, Christian, and Buddhist beliefs. However, follow-up and continuing care programming exceed the authority of the Prison Department. As a result, both are not offered formally at present.

The NGOs offer 52 programs nationwide, and these NGO programs are run primarily by small, faith-based organizations (Gerald & Steven, 2003). Persatuan Pengasih (the word *pengasih* means "compassion") was founded in 1991 as a private NGO treatment and rehabilitation institute. It began offering services in 1993. Participation is voluntary. Services include outreach, primary treatment, and hospice care (Gerald & Steven, 2003).

Persatuan Pengasih drug treatment programs also use the TC model of treatment. Persatuan Pengasih's mission is to facilitate the recovery of drug-dependent individuals, help restore families affected by drug abuse, provide compassionate care for people suffering from HIV/AIDS, and provide preventive education programs in the community. All residents agree that the love and support they receive from peers and the sense of family that exists within the community are the most valued aspects of the program. Persatuan Pengasih offers a full continuum of care, including outreach, primary treatment, and hospice services. The organization serves more than 300 persons per month.

There are several challenges faced by the National Anti-Drugs Agency, the Prison Department, the Ministry of Home Affairs, and NGOs in helping drug users. There are increasing numbers of hardcore addicts who use mixed drugs (*dadah campuran*), leading to cross-addiction. The lack of staff experienced in handling these cases creates further challenges.

Drug Abuse and HIV/AIDS

There is a link between intravenous drug abuse and the spread of HIV. The first AIDS case in Malaysia was reported in December 1986; since then, the number of new cases has risen exponentially.

Intravenous drug use has long been seen as being responsible for the spread of HIV in Asia. In Malaysia, the rapid spread of the virus since the 1990s has been attributed to the use and sharing of contaminated intravenous drug equipment. The profile of intravenous drug users continues to be predominantly male, young, of Malay ethnicity, and heterosexual. Women represent a small percentage of Malaysian drug users (around 2%), and their total numbers remain unknown due to the lack of available data and studies of this population (UNGASS Country Progress Report, 2008).

In 2001, cumulative HIV infections were 44,208 cases, 6,024 of which were AIDS cases. Most of the HIV cases were among men (81.5%) and Malays (72.3%), and a staggering 79 percent of reported cases of infection were from intravenous drug use (Malaysia Ministry of Health, 2002). By the end of 2004, 64,000 Malaysians were reported to be infected with HIV, of whom some 9,400 had developed AIDS. The majority of reported AIDS cases and HIV infections were contracted through intravenous drug use (United Nations Development Program, 2007).

As of December 2006, after more than 20 years into the HIV epidemic in Malaysia, the country has recorded a total of 76,389 persons with HIV since the first reported cases in 1986. Most reported infections occur among young heterosexual males between the ages of 20 and 39. About 75 percent of

the cumulative HIV cases were reported among intravenous drug users. The WHO currently continues to classify Malaysia as having a concentrated HIV epidemic (Malaysia Ministry of Health, 2007).

Drug Legislation in Malaysia

Malaysia has five main statutes that cover drug laws. They are as follows:

Dangerous Drugs Act 1952

This is the major legislation relating to Malaysian drug control. This act gives provision for the regulation of the importation, exportation, manufacture, sale, and use of opium and of certain other dangerous drugs and substances. The act has been revised several times in relation to changes in patterns of drug use. This act includes the death penalty for drug trafficking.

Poisons Act 1952

This act is aimed at regulating the importation, possession, manufacture, compounding, storage, transport, sale, and use of poisons. This includes any mixture, preparation, solution, or natural product. It also includes products used in industry, medicine, and agriculture.

Drug Dependents (Treatment and Rehabilitation) Act 1983

This is a comprehensive legislation covering treatment and rehabilitation. The act provides for compulsory treatment and rehabilitation of any person who has been certified as drug-dependent as well as for the establishment and implementation of voluntary treatment programs. The period of compulsory treatment and rehabilitation (i.e., not voluntary) at a rehabilitation center is two years, and thereafter, one must undergo supervision by an officer for two years.

Dangerous Drugs (Special Preventive Measures) Act 1985

This act allows the government to detain anyone suspected of being a trafficker without having to bring the suspect to a court of law. This act is an amendment to the Dangerous Drugs Act 1952 to provide for matters connected therewith.

Dangerous Drugs (Forfeiture of Property) Act 1988

This act allows the government to trace, seize, and forfeit assets of convicted drug traffickers. This act also makes provisions for offenses in relation to prop-

erty, and for the seizure and forfeiture of property, connected with activity related to offenses under this act, or any foreign law corresponding to these acts or to the provisions for offenses under these acts; for assistance to foreign countries in relation to matters connected with dangerous drugs; and for matters connected with the aforesaid provisions.

Actions against Drug Abuse

It is evident that drug addiction is threatening the future of Malaysians, and actions need to be taken to put a stop to it. The responsibility of combating this ominous threat does not rest solely on any one party. Agencies and centers, such as government agencies, NGOs, law enforcement agencies, rehabilitation centers, social and welfare institutions, and the medical fraternity, need to cooperate and collaborate on efforts to develop more effective approaches to combat and manage drug addiction. Total commitment, involvement, and sustained efforts from everyone are crucial. Only then can we hope to make any significant headway in our quest for a drug addiction–free nation.

Family members and the community have a particularly important role to play. Vision 2020 calls for the establishment of a fully moral and ethical society, whose citizens are strong in religious and spiritual values, and a fully caring society and caring culture, where society will come before self and the welfare of the people will revolve around a strong and resilient family system. As such, parents should work hard to instill the right moral values in their children so that they do not fall prey to drugs. And for drug users who have gone astray in the choices that they have made in life, family members and the community should provide the support, encouragement, acceptance, and understanding necessary for them to wean themselves of their habits. They need to be guided back to the right path and be given a second chance at a productive life.

Medical practitioners and other health care providers play an important role in assisting family members and the community in drug prevention and management. Medical practitioners and other health care providers must be prepared to go beyond the call of duty to provide support, counseling, and guidance to the drug users and to help them work through their problem each step of the way. It is also their duty to help change the mind-set of society and erase the stigma and discrimination attached to drug treatment and rehabilitation so that more families will be willing to bring their drug-addicted members in for treatment.

Government agencies like the National Anti-Drugs Agency, the Association for Drug Prevention Malaysia (*Persatuan Mencegah Dadah Malaysia*), the

Ministry of Education, and the Ministry of Health have already taken steps to prevent drug addiction through education. There are dedicated drug prevention programs targeted at the family and community levels; at primary, secondary, and tertiary institutions; and at workplaces to increase public awareness of drugs and their effects, with the hope that people will be better empowered to make the correct choice not to be involved with drugs and to resist negative peer pressure.

According to the Bureau for International Narcotics and Law Enforcement Affairs (2008), in addition to the actions mentioned previously, Malaysia has also made other efforts such as the following:

Policy Initiatives

Malaysia continues a long-term effort launched in 2003 to reduce domestic drug use to negligible levels by 2015, a goal shared by the whole of Association of Southeast Asian Nations (ASEAN). The National Anti-Drugs Agency is the policy arm of Malaysia counternarcotics strategy, coordinating demand reduction efforts with various ministries. Malaysian law stipulates a mandatory death penalty for major drug traffickers, with harsh mandatory sentences also applied for possession and use in smaller quantities. In practice, however, many minor offenders are placed into treatment programs instead of prison or are referred to the Preventive Measures Act, under which they can be held for two years (renewable to eight years).

Law Enforcement Efforts

Malaysian authorities raided three clandestine drug labs in 2007 and had several successful drug seizures, confiscating large quantities of methamphetamine and ecstasy. Police arrested 36,534 people for drug-related offenses between January and August 2007 and held in detention or rehabilitation centers a total of 111,416 drug addicts during the same period. Enforcement officials continued to show successes in ATS-related seizures and have also recorded a higher level of heroin seizures over the same period than last year. The Royal Malaysian Police recorded a 46 percent increase in confiscated property derived from drug-related cases over the first eight months of 2007 (Bureau for International Narcotics and Law Enforcement Affairs, 2008).

Many suspected traffickers continue to be detained under Malaysian special preventive measures, including the Restricted Residence Act and the Emergency Ordinance 1969, which allow for detention without trial of suspects who pose a threat to public order or national security.

Drug Flow/Transit

Drugs transiting from Malaysia do not appear to make a significant impact on the U.S. market. However, Malaysia's proximity to the heroin production areas and methamphetamine labs of the Golden Triangle leads to smuggling across Malaysian borders of drugs destined for Australia and other markets. Ecstasy from Amsterdam is flown into Kuala Lumpur International Airport for domestic use and distribution to Thailand, Singapore, and Australia. Ketamine comes from Tamil Nadu, (southern) India, and is exported to several countries in the region. There is evidence of increased transit of cocaine, although police have not yet thoroughly developed information on this trend (Bureau for International Narcotics and Law Enforcement Affairs, 2008).

Domestic Programs (Demand Reduction)

The National Anti-Drugs Agency continues to target its demand reduction efforts toward youth, parents, students, teachers, and workers, with extensive efforts to engage schools, student leaders, parent-teacher associations, community leaders, religious institutions, and workplaces through prevention and treatment (Global Demand Reduction Efforts on Drugs, 2000). A demand reduction program in Malaysian public schools and a drug-free workplace prevention program began in 1999.

CONCLUSION

The most important implication of this review is that little is known about the prevalence of alcohol use and abuse in Malaysia. However, given the high expenditure being reported for alcoholic beverages, the rates of alcohol consumption and abuse are expected to be relatively high. As with alcohol, we are unaware of any empirical studies that have examined the prevalence of drug use and abuse. However, according to UNICEF Malaysia (2007), a total of 22,811 drug users were detected in 2006, half of whom were reported to be repeat offenders.

To fully understand alcohol and drug use and abuse in Malaysia, there is an urgent need to conduct a large epidemiologic study. Such a study should provide information on the prevalence, comorbidity, risk and protective factors, and course of substance use and abuse in difference age and ethnic groups. This information will be useful from a public health point of view in terms of preventing the occurrence of substance abuse, particularly among young

adults. Given the negative consequences of substance abuse, several prevention programs have been developed for several age and professional groups. The next important step would be to systematically examine the efficacy of these programs.

REFERENCES

Arasaratnam, S. (1970). *Indians in Malaya and Singapore*. London: Oxford University Press.

Assunta, M. (2001). The alcohol problem in Malaysia. *The Globe*, *4*, 18–21.

Bernama. (2008, November 26). *No total ban on sale of alcoholic drinks in Selangor*. Retrieved December 5, 2008, from http://www.bernama.com/bernama/v3/news_lite.php?id=374617

Bureau for International Narcotics and Law Enforcement Affairs. (2008). *International narcotics control strategy report (2008): Malaysia*. Retrieved May 5, 2008, from http://www.shaps.hawaii.edu/drugs/incsr2008/incsr_ 2008_ Malaysia.html

Chua, S. L. (2004, October 2). Speech presented at the First Malaysian Conference on Addiction Medicine, Kuala Lumpur. Retrieved May 5, 2008, from http://www.moh.gov.my/ MohPortal/speechDetail.jsp?action=view&id=330

Consumer Association of Penang. (2008). *Call for ban on samsu*. Retrieved May 5, 2008, from http://en.cap.org.my/index.php?option=com_content&task=view&id=169&Itemid=3

Freeman, D. B. (2003). *The straits of Malacca: Gateway or gauntlet*. Toronto: McGill-Queens Press-MQUP.

Gerald, R. G., & L. G. Steven. (2003). *Promising practices in drug treatment: Findings from Southeast Asia*. Washington, DC: Bureau for International Narcotics and Law Enforcement Affairs, U.S. Department of State.

Global Demand Reduction Efforts on Drugs. (2000). *International demand reduction initiatives*. Retrieved May 5, 2008, from http://www.fas.org/irp/news/2000/12/irp-001211-drug.htm

Guinness Centre. (2005). *Spirits in Malaysia to 2010*. Dublin: Research and Markets.

How, T. B. (1999). Drug prevention education in schools: The Malaysian experience. *Drug, Education, Prevention, and Policy*, *6*, 3–15.

Inoguchi, T., M. Baáñez, A. Tanaka, & T. Dadabaev (2005). *Values and life styles in urban Asia: A cross-cultural analysis and sourcebook based on the Asia Barometer Survey of 2003*. Mexico City: Siglo XXI.

Jernigan, D. (1997). *Thirsting for markets: The global impact of corporate alcohol*. San Rafael, CA: Marin Institute for the Prevention of Alcohol and Other Drug Problems.

Jernigan, D. H., & S. K. Indran. (1997). *Country profile on alcohol in Malaysia*. New York: Oxford University Press.

Loe, Y. K., & A. H. Maimunah. (1997). Alcohol consumption among non-Muslim adults. In *Report of the Second National Health and Morbidity Survey Conference*,

20–22 November 1997, Hospital Kuala Lumpur. Kuala Lumpur: Public Health Institute, Ministry of Health Malaysia.

Malaysia Department of Social Welfare. (2004). *Perangkaan kebajikan masyarakat Malaysia 2003*. Kuala Lumpur: Ministry of Women, Family, and Community Development of Malaysia.

Malaysia Ministry of Health. (2002). *Estimation of drug users and injecting drug users in Malaysia*. Kuala Lumpur: Author.

Malaysia Ministry of Health. (2007). *Summary of HIV/AIDS cases*. Kuala Lumpur: Author.

Mazlan, M., R. S. Schottenfeld, & M. C. Chawarski (2006). New challenges and opportunities in managing substance abuse in Malaysia. *Drug and Alcohol Review, 25*, 473–478.

National Anti-Drugs Agency. (2007). *Buletin Dadah* (Report No. 4/2007). Kuala Lumpur: Author.

Rugayah, H. B., Rozita Halina, T. H., Sondi, S., Azman, A. B., Tiew, C. T., Lailanoor, H. I., et al. (2000). *Malaysia country report: An essential link to equity in development*. Kuala Lumpur: Institute of Public Health, Ministry of Health Malaysia.

UNGASS Country Progress Report. (2008). *Malaysia*. Retrieved May 5, 2008, from http://data.unaids.org/pub/Report/2008/malaysia_2008_country_progress_report_en.pdf

UNICEF Malaysia. (2007). *Drugs and HIV*. Retrieved May 5, 2008, from http://www.unicef.org/malaysia/hiv_aids_4167.html

United Nations Development Program. (2007). *Malaysia at a glance*. Retrieved May 5, 2008, from http://www.youandaids.org/Asia%20Pacific%20at%20a%20Glance/Malaysia/index.asp#general

Van-Onselen, C. (1982). Studies in the social and economic history of the Witwatersrand: 1886–1914. New York: Longman.

World Development Indicators Database. (2007). *Malaysia data profile*. Retrieved May 5, 2008, from http://devdata.worldbank.org/external/CPProfile.asp?PTYPE=CP&CCODE=MYS

World Health Organization. (2006). *Malaysia national report on violence and health*. Geneva: Author.

This page intentionally left blank

Chapter **10**

Alcohol Use and Binge Drinking in Cyprus

Xenia Anastassiou-Hadjicharalambous, PhD,
Cecilia A. Essau, PhD, MA, HBA, and George Georgiou, PhD

Cyprus is the third largest island in the Mediterranean, with a population of about 800,000. The island of Cyprus is divided by the so-called Green Line, established following the invasion of Turkey in 1974. About 37 percent of the island is under Turkish occupation. Cyprus joined the European Union in 2004.

Its long Mediterranean summers, mild winters, and gentle winds make Cyprus an ideal place for vine growing. In fact, recent archaeological discoveries show that Cyprus has been a vine-growing and wine-producing country for 6,000 years. Historically, wine production can be divided into four periods: ancient, medieval to 1878, between 1878 and 1980, and 1980 onward (Kythreotou, 2003):

ancient. Archaeological evidence seems to indicate that the cultivation of the grape vine started in Cyprus in the second millennium B.C. For example, the discovery of a shipwreck in 1999 showed that wine was being traded as early as 2300 B.C. Other discoveries include wine containers that belonged to the Bronze Age (2500–2000 B.C.). More recently, the discovery of pottery fragments, dating back to the chalcolithic period (i.e., between 3500 and 3000 B.C.) and containing traces of tartaric acid, gave further proof that this pottery was used for wine. Indeed, these and other recent archaeological discoveries place Cyprus as the first Mediterranean country to produce wine.

medieval to 1878. The island's wine history seems to be closely related to its political and administrative history. For example, during the Lusignan occupation, Commandaria wine was the most popular. Commandaria has

been produced on the island since at least 1000 B.C. and remains the oldest wine still in production. During the Ottoman occupation, wine production declined dramatically due to traditions associated with Islam and the heavy burden of taxation.

1878–1980. The handing over of the island from Ottoman rule to the British Empire in 1878 brought a revival in the wine-making industry. This period also saw changes in taxation rules and the expansion of the local wine industry. An important factor that led to the expansion of the Cypriot wines was related to the phylloxera epidemic, which destroyed the majority of wine-producing vines in mainland Europe in the nineteenth century. Cyprus remained relatively unaffected due to the strict quarantine controls that were in place, which subsequently boosted demand for both Cyprus grapes and wines.

1980 onward. From 1980, three initiatives were launched to overhaul the wine sector to improve the quality of wine. These included the introduction of new varieties of grapes (e.g., Cabernet Sauvignon, Cabernet Franc, and Palomino) and financial support for their cultivation; this initiative was believed to improve quality wine production and so make wines more palatable to overseas markets compared to local grapes. The second incentive was to establish small regional wineries near the vineyards: reducing the distance the grapes had to travel vastly improved the quality of the wine. The third incentive was the launching of the new Appellation of Origin in 2007. Specifically, when Cyprus joined the European Union, it had to classify its wine under the new legislature.

In Cyprus, alcohol is easily available in supermarkets, gas stations, grocery stores, and public facilities.

LAW PARADOX

The Children Law of 1956 states that any person who sells alcohol to a person under the age of 14 years, for his or her own use, is guilty of an offense and will be liable, in the case of a first offense, to a fine not exceeding £25 (€43); for a second offense, to a fine not exceeding £25 (€43); and in the case of a third or subsequent offense, to a fine not exceeding £50 (€85). Furthermore, the police would be authorized to seize alcohol in the possession of any person under the age of 14 years found in any street or public place. This providence law applies only to cases where alcohol is purchased by the child for personal consumption. However, a child is entitled to purchase alcohol on behalf of an adult.

The Children Law of 1956 is in conflict with the Selling of Alcohol Law of 1961, article 83, 1998. The Selling of Alcohol Law of 1961, article 83, 1998,

prohibits the trade or distribution of alcoholic beverages to an individual under the age of 17 years, an offense punishable with imprisonment for up to three months or a pecuniary fine up to £1,000 (€1,709), or both. Proof of age documentation is required when purchasing alcohol. The contradiction between the Children's Law of 1956 and the Selling of Alcohol Law of 1961 is obvious. On one hand, the former allows the selling of alcohol to individuals under the age of 14 when the alcohol purchased is not for personal consumption, while the latter prohibits the distribution and selling of alcohol to individuals up until the age of 17.

A further paradox is observed between the Modified Children's Law of 1999 and the Recreational Centres Law (1985–1999). The Recreational Centres Law (1985–1999) does not foresee any prohibition related to underage drinking. In contrast, the Modified Children's Law of 1999 requires that all drinking establishments ensure that no individual under the age of 16 years is allowed to enter their premises. First-time violators of this law will be subjected to a pecuniary fine up to £500 (€854) or up to three months' imprisonment, or both. The person who violates this law for a second time will incur a pecuniary fine up to £1,000 (€1,709) or up to six months' imprisonment, or both. The establishment may also face losing its liquor license. These contradictory laws are compounded by the lack of law enforcement. Indeed, children and adolescents freely obtain alcohol in bars, clubs, and other establishments and outlets without providing proof of age documentation as the law requires. Currently members of parliament are calling for significant legislative progress and harsher penalties in the fight against underage drinking.

The Cyprus Broadcasting Corporation Law, 300A, 2001, 2002, 2004, requires that the advertisement of alcohol follows the following rules: (1) ads must not be addressed to underage individuals or present underage individuals consuming alcohol; (2) ads must not connect the consumption of alcoholic drinks to improved body performance or to driving a vehicle; (3) ads must not cause the impression that the consumption of alcoholic drinks enhances social or sexual success; (4) ads must not allow the impression that alcoholic drinks have therapeutic attributes or that they act as stimulants, tranquillizers, or analgesics; (5) ads must not encourage overconsumption of alcohol and must not allow a negative image of those individuals who abstain from the consumption of alcohol; and (6) ads must not stress as an advantage of the drink the inclusion of a high amount of alcohol.

The Internal Regulation of the Cyprus Broadcasting Corporation on alcohol advertising states that advertisements for alcoholic drinks will be presented as late as possible in the night and not between programs that are addressed to children or young individuals.

PREVALENCE OF ALCOHOL CONSUMPTION IN CYPRUS

Alcohol Use and Binge Drinking in Adolescents

The only information on the patterns of alcohol consumption in adolescents comes from the European Schools Project on Alcohol and Other Drugs (ESPAD). The ESPAD aimed to examine the frequency of consuming different types of alcoholic beverages (including binge drinking) and other substance use in over 50,000 16-year-olds across Europe (Morgan et al., 1999). Three surveys have been conducted so far: the first one in 1995, the second in 1999, and the third in 2003. A total of 26 and 30 European countries participated in the first and second survey, respectively (Hibbel et al., 2001). In the last survey, 35 countries participated, including Austria, Belgium, Bulgaria, Croatia, Cyprus, the Czech Republic, Denmark, Estonia, the Faroe Islands, Finland, France, Germany, Greece, Greenland, Hungary, Iceland, Ireland, Isle of Man, Italy, Latvia, Lithuania, Malta, the Netherlands, Norway, Poland, Portugal, Romania, Russia (Moscow), the Slovak Republic, Slovenia, Sweden, Switzerland, Turkey, Ukraine, and the United Kingdom. During the 2003 ESPAD survey, 2,152 (999 boys and 1,153 girls) Cypriot adolescents participated.

The results of the most recent ESPAD survey can be summarized as follows (Hibbel et al., 2004):

1. About four-fifths of Cypriot 16-year-olds have tried alcohol sometime in their lives. Specifically, 86 percent of the students reported having consumed alcohol at least once in their lives. Of those who had consumed alcohol, 27 percent reported having consumed alcohol on more than 40 occasions. The 12- and 1-month prevalence of alcohol use was 79 percent and 63 percent, respectively.
2. Beer was the most common alcoholic beverage (53%), followed by wine (36%) and spirits (41%).
3. Alcopops (i.e., the assortment of alcoholic beverages) were consumed by quite a high percentage of adolescents (62%), ranking Cyprus among the highest countries where adolescents drink this beverage.
4. Thirty-four percent of the students had engaged in binge drinking (i.e., having five or more drinks in a row); however, most of them only binged once or twice, and only 2 percent had binged more than 10 times.
5. Boys drink more than girls.
6. The majority of the participants had their first experiences with beer, as opposed to other alcoholic beverages; that is, 68 percent of the participants reported having had beer and first drunkenness at the age of 13 years or

younger, compared to those who had consumed wine (44%) or spirits (27%).

7. Thirty-nine percent of the adolescents reported being drunk sometime in their lives; this figure indicates that Cyprus had among the lowest rate of drunkenness among the ESPAD countries.
8. Forty-eight percent expected positive consequences (mostly related to having lots of fun) of alcohol consumption, compared to 31 percent who expected negative consequences (mostly related to getting a hangover).
9. Perceived easy availability of alcohol beverages was as follows: beer (85%), wine (87%), and spirits (83%).

When comparing the percentage of adolescents who consumed alcohol in the last 12 months across the three surveys, Figure 10.1 shows a decrease from 85 percent in 1995 to 79 percent in 1999 and 2003. Across all the ESPAD countries, the corresponding percentages were 80 percent (in 1995), 82 percent (in 1999), and 81 percent (in 2003; European School Survey Project on Alcohol and Other Drugs, n.d.; Figure 10.1). The rate of drunkenness remained stable, ranging from 27 percent in 1995 to 25 percent in 2003 (Figure 10.2), compared to the respective percentages of 47 percent, 51 percent, and 50 percent for all the ESPAD countries

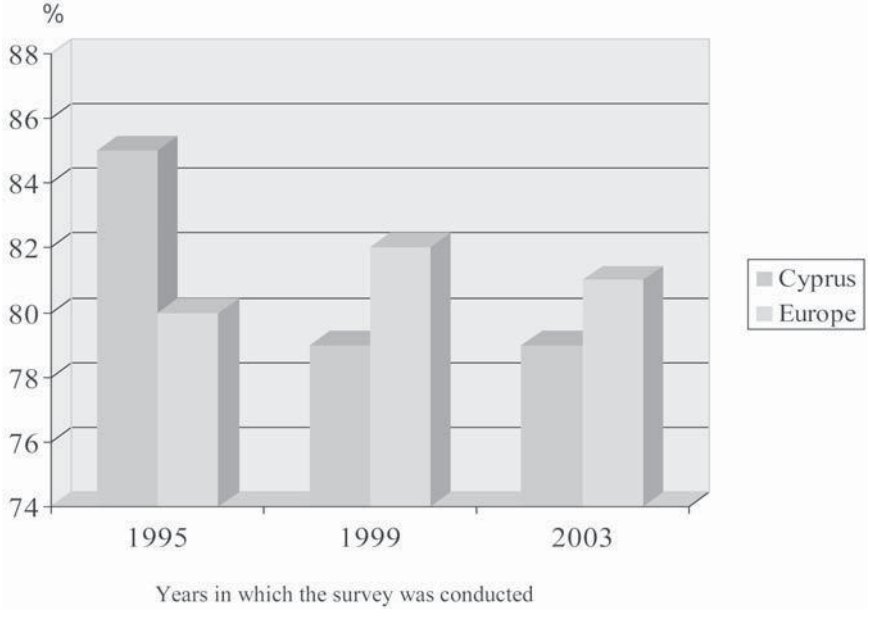

FIGURE 10.1 Percentages of persons drinking any alcoholic drinks in the last 12 months.

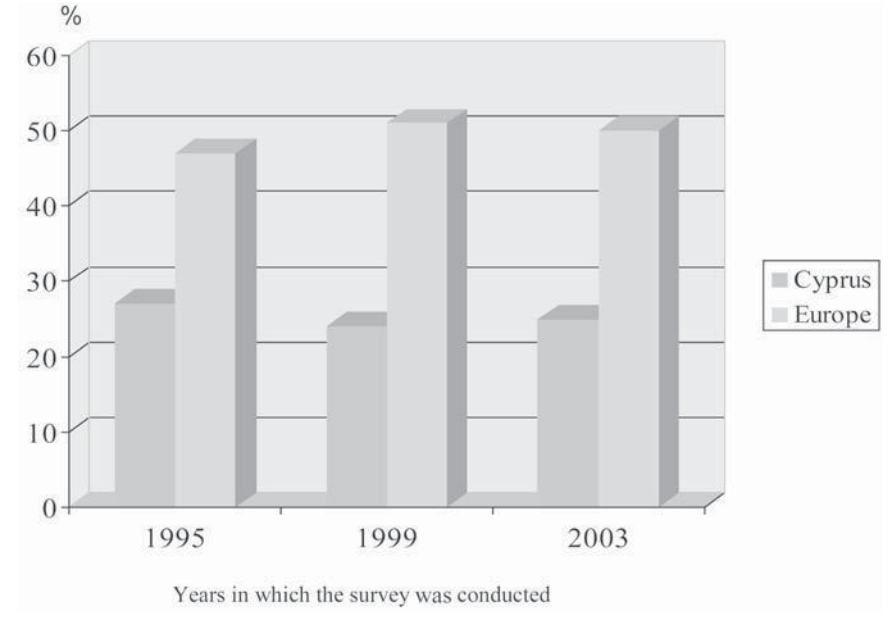

FIGURE 10.2 Percentages of persons experiencing drunkenness in the last 12 months.

Alcohol Use and Binge Drinking in Young Adults

In this section, we describe our recent study (Anastassiou-Hadjicharalambous, Essau, & Georgiou, 2008) on alcohol drinking and binge drinking among young adults in Cyprus.

Participants

A total of 126 students participated in the study (31.5% men and 68.5% women). Their mean age was 23.6 years (*SD* = 5.3). Most of them were still single (84%); 11.9 percent were married and 4 percent were divorced. As for religious affiliation, almost all of the participants reported being a Christian Orthodox (91.9%).

The participants were recruited from various colleges and universities in Nicosia, the capital of Cyprus. They were administered a set of questionnaires that measured frequency, patterns, and motivations for alcohol use.

Instruments

The Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de la Fuente, & Grant, 1993) was used to measure recent alcohol use,

alcohol dependence symptoms, and alcohol-related problems. It consisted of 10 questions. The AUDIT was developed by the World Health Organization (WHO) and has been used for screening for excessive drinking in many settings. A cutoff value of 8 points has been identified as an index of problematic drinking. The psychometric proprieties of the AUDIT have been reported in various studies. In this study, Cronbach's was 0.84.

The Self-Regulation Questionnaire (Carey, Neal, & Collins, 2004) was used to measure self-regulation (i.e., the generalized ability to regulate behavior to achieve desired future outcomes). It contains 31 items that can be answered on a 5-point Likert scale ranging from 1 (strongly disagree) to 5 (strongly agree). In this study, Cronbach's was 0.88.

The College Alcohol Study (CAS; Weitzman, Nelson, & Wechsler, 2003) was used to measure student alcohol use, patterns of use, reasons for not drinking, and problems associated with alcohol use. In this study, the CAS was slightly modified to adjust to the adults in Cyprus. In this study, a drink was defined as the following: one-half pint of ordinary strength beer, lager, or cider (so a pint is two drinks); a 25-milliliter measure of spirits straight or in a mixed drink; a 125-milliliter (small) glass of wine; and two-thirds pint of beer/alcopop (bottles are one and a half drinks per unit).

A gender-specific definition of binge drinking (i.e., heavy drinking behavior) was used: for men, it was five or more drinks in a row in the past two weeks, and for women, it was four or more drinks in a row in the past two weeks.

The Drinking Motives Questionnaire (DMQ; Cooper, Russell, Skinner, & Windle, 1992) was used to measure the participants' motivation to drink alcohol. The DMQ contains 20 items, which can be rated on a 4-point Likert scale ranging from 1 (almost never/never) to 4 (almost always). The items can be grouped into four different subscales: enhancement, coping, conformity, and social motives. Cronbach's was 0.91.

The Spence-Essau Anxiety Scale (Spence & Essau, 2004) is a 33-item measure of anxiety symptoms in children and adolescents. The items reflect symptoms of the main anxiety disorders from the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1994), including social phobia, obsessive-compulsive disorder, panic/agoraphobia, specific fears, and generalized anxiety disorder. Each item is rated on a 4-point scale in terms of its frequency from 0 (never) to 3 (always). The items are summed to yield a total score, with higher scores reflecting higher levels of anxiety symptoms. Cronbach's was 0.91.

The Comprehensive Effects of Alcohol (Fromme, Stroot, & Kaplan, 1993) was used to measure participants' discrete expectancies about alcohol use. It contains 38 items that can be rated on a 4-point scale ranging from 1 (disagree) to 4 (agree). The items could be grouped into positive and negative factors. The

Table 10.1
Frequency of Binge Drinking by Gender and Age Groups

	Gender		Age group		
	Male (*N* = 40), *n* (%)	Female (*N* = 86), *n* (%)	18–19 (*N* = 60), *n* (%)	20–25 (*N* = 43), *n* (%)	26+ (*N* = 18), *n* (%)
No binge drinking	9 (22.5)	52 (60.5)	21 (35.0)	14 (32.6)	3 (16.7)
Binged 1–2 times in the past 2 weeks	16 (40.0)	29 (33.8)	24 (40.0)	23 (53.5)	13 (72.2)
Binged 3+ times in the past 2 weeks	15 (37.5)	5 (5.8)	15 (25.0)	6 (14.0)	2 (11.1)

positive factors can further be divided into four subscales: sociability, tension reduction, liquid courage, and sexuality. Likewise, the negative factors can be divided into cognitive and behavioral impairment, risk and aggression, and self-perception. In this study, Cronbach's was 0.93.

Results

Significantly more men than women reported binge drinking (2 = 20.65, $p < 0.001$; Table 10.1). Specifically, 77.5 percent of the men reported having binged at least once in the past two weeks. The highest rate of binge drinking was reported by participants in the oldest age group (i.e., 26-year-olds and older).

The most common alcoholic beverages that the participants consumed for *four or more* drinks in a row were spirits (37.1%), followed by beer (22.9%) and alcopops (18.6%). The average time to consume *four or more* drinks in a row was two hours, being reported by 32.8 percent of the participants. Almost all (86.4%) of the participants reported that it was "easy" or "very easy" to obtain alcohol.

The most common problem related to binge drinking was having a hangover. Significant group differences were found in 3 out of 12 problem areas (Table 10.2). Participants who binged at least three times in the past two weeks had significantly more problems with missed classes (2 = 27.35, $p < 0.001$), having unplanned sexual activity (2 = 23.58, $p < 0.01$), and not using protection when having sex (2 = 19.05, $p < 0.05$) than participants who did not binge drink or who had binged only once or twice.

Table 10.2
Problems Related to Binge Drinking

	No binge drinking, *n* (%)	Binged 1–2 times in the past 2 weeks, *n* (%)	Binged 3+ times in the past 2 weeks, *n* (%)
Have a hangover	12 (30)	17 (41.5)	11 (57.9)
Miss a class	2 (5)	13 (31)	6 (31.7)
Get behind in school work	3 (7.5)	13 (31)	7 (36.9)
Do something you later regret	8 (20)	12 (30)	8 (42.2)
Forget where you were or what you did	3 (7.5)	7 (16.6)	5 (26.4)
Argue with friends	2 (5)	8 (19)	4 (27.9)
Engage in unplanned sexual activity	–	9 (22)	9 (45.0)
Not use protection when you have sex	–	8 (19.4)	6 (33.4)
Damage property	1 (2.5)	7 (16.7)	2 (10.0)
Get into trouble with the campus security or local police	1 (2.5)	3 (7.2)	1 (5.6)
Get hurt or injured	3 (7.5)	5 (11.9)	2 (10.0)
Require medical treatment for an alcohol overdose	–	–	1 (5.3)

The participants were also asked their reasons for not drinking or for trying to limit their drinking. As shown in Table 10.3, the most important/very important reasons for not drinking were "going to drive," followed by "not wanting to lose control."

Correlational Analyses

The AUDIT correlated significantly positive with all the four main types of drinking motives, with all the correlations at the $p < 0.001$ level. These findings indicate that high levels of drinking were related to enhancement ($r = 0.41$), coping ($r = 0.31$), conformity ($r = 38$), and social motives ($r = 0.45$).

Table 10.3
Reasons for Not Drinking or Trying to Limit Drinking Alcohol

	Very important to important, *n* (%)	Somewhat to not at all important, *n* (%)
Drinking is against my religion	30 (24.2)	94 (75.8)
Drinking is against my values	44 (35.2)	81 (64.8)
People in my family have had alcohol problems	44 (35.5)	80 (64.6)
I'm not old enough to drink legally	25 (20.5)	97 (79.5)
I'm going to drive	102 (80.9)	24 (19.0)
It costs too much money	29 (23.8)	93 (76.3)
I don't like the taste	42 (33.9)	82 (66.1)
My friends don't drink	22 (18.1)	99 (81.8)
I don't want to disappoint someone I care about	50 (40.3)	74 (59.7)
I'm going on a date	41 (3.8)	80 (66.1)
It is bad for my health	84 (67.2)	41 (32.8)
It interferes with my studying	74 (60.2)	49 (39.8)
It interferes with my athletic activity	66 (53.2)	58 (46.8)
I've decided to cut down	50 (41.3)	71 (58.6)
I don't want to lose control	85 (68.5)	39 (31.4)
I recently drank too much	41 (34.4)	78 (65.5)
I've had problems with alcohol	44 (36.7)	76 (63.3)
It's fattening	49 (40.9)	71 (59.2)
I have a fear of getting caught	22 (20.0)	88 (80.0)

The AUDIT also correlated significantly negative with self-regulatory behavior ($r = -0.24, p < 0.05$). Thus a high level of alcoholic consumption was related to less ability to control one's behavior. Surprisingly, none of the anxiety subscales showed any significant correlations with the AUDIT.

Of all the subscales of the Comprehensive Effects of Alcohol, only cognitive and behavioral impairment correlated with the AUDIT ($r = 0–24, p < 0.01$).

Alcohol Use and Binge Drinking in Adults

Information on the prevalence and patterns of alcohol consumption in Cyprus came from the Attitudes towards Alcohol Survey (European Community, 2007). The survey was conducted at the end of 2006, in 25 member states of the European Union, plus in 2 acceding countries (Bulgaria and Romania) and 1 candidate country (Croatia) and in the Turkish Cypriot Community (TCC). The participants were interviewed face-to-face at their own homes. A total of 604 adults in Cyprus and 372 adults in the TCC participated in the survey. The results of this survey can be summarized as follows:

1. As in most European Union (EU) countries, a high percentage of the Cypriots (74%, compared to 75% across the EU) reported having consumed alcohol beverages during the last 12 months. By contrast, about half (53%) of the participants in the TCC claimed not to have drunk any alcohol in the past 12 months; this high rate of abstinence may be due to religious reasons.
2. About 63 percent and 28 percent of the participants in Cyprus and in the TCC, respectively, reported having drunk alcohol during the last 30 days before the survey. Of the Cypriots who consumed alcohol in the last 30 days, 31 percent of them drank alcohol once a week and 8 percent drank daily; in the TCC, it was 24 percent and 8 percent, respectively.
3. About 46 percent (compared to 31% across Europe) of the Cypriots had not drunk five or more drinks on one occasion. In the TCC, it was 29 percent. Interestingly, of those who had drunk more than five drinks on one occasion, 19 percent of the participants in the TCC reported drinking several times a week; the percentage found for participants in Cyprus who drank five or more drinks on one occasion was 6 percent.
4. A high percentage of the Cypriot (77%) and the TCC (68%) participants reported not knowing the legal blood level that is allowed for car drivers.
5. A majority of the Cypriot (72%) and the TCC (56%) participants did not believe that higher prices of alcohol would discourage young people and heavy drinkers from consuming alcohol.
6. Most participants agree to putting warnings on alcohol bottles and advertisements, particularly for pregnant women and car drivers (Cyprus, 92%; TCC, 91%).
7. A high percentage of the Cypriots (72%) and those in the TCC (89%) approved of banning alcohol advertising that targets young people. Equally high percentages were found for those who supported the idea of banning selling and serving alcohol to the underaged (i.e., under 18 years of age).

CONCLUSION

Information on the frequency, patterns, and correlates of alcohol use and binge drinking in Cyprus is rare. According to some recent European-based surveys, the ESPAD and the Attitudes towards Alcohol Survey (European Community, 2007), Cyprus seems to have the lowest percentages in terms of frequency of drinking compared to other European countries. However, our ongoing study seems to indicate that about one-third of the young adults, especially men, binge drink on a regular basis. Those who binge reported a wide range of personal and social problems. Our study also indicated some common correlates of alcohol use and binge drinking such as lack of self-regulatory control and specific drinking motives (i.e., enhancement, coping, conformity, and social motives). It is hoped that such information will be informative for designing methods of prevention of alcohol abuse among Cypriots (Dadds & McAloon, 2002).

The next stage of research in Cyprus should be (1) to conduct methodologically sound treatment studies, using manually guided interventions with established, empirically validated efficacy; (2) to evaluate treatment outcomes that cover abstinence or relapse status and individuals' psychosocial functioning in various life domains; (3) to evaluate the efficacy of different treatment strategies for different groups of individuals (e.g., with comorbid disorders); and (4) to conduct so-called treatment matching. This chapter suggests that much still needs to be done in Cyprus in terms of alcohol research.

REFERENCES

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Anastassiou-Hadjicharalambous, X., Essau, C. A., & Georgiou, G. (2008). *Frequency of alcohol use and binge drinking among young adults in Cyprus*. Unpublished manuscript.

Carey, K. B., Neal, D. J., & Collins, S. E. (2004). A psychometric analysis of the self-regulation questionnaire. *Addictive Behaviors, 29,* 253–260.

Cooper, M. L., Russell, M., Skinner, J. B., & Windle, M. (1992). Development and validation of a three-dimensional measure of drinking motives. *Psychological Assessment, 4,* 123–132.

Dadds, M. R., & McAloon, J. (2002). Prevention. In C. A. Essau (Ed.), *Substance abuse and dependence in adolescence* (pp. 143–182). London: Brunner-Routledge.

European Community. (2007). *Attitudes towards alcohol.* Retrieved May 5, 2008, from http://ec.europa.eu/health/ph_determinants/life_style/alcohol_en.htm

European School Survey Project on Alcohol and Other Drugs. (n.d.). *Key result generator.* Retrieved May 5, 2008, from http://www.espad.org/sa/node.asp?node=730

Fromme, K., Stroot, E., & Kaplan, D. (1993). Comprehensive effects of alcohol: Development and psychometric assessment of a new expectancy questionnaire. *Psychological Assessment, 5(1),* 19-26.

Hibell, B., Andersson, B., Ahlström, S., Balakireva, O., Bjarnasson, T., Kokkevi, A., et al. (2001). *The ESPAD (European School Survey Project on Alcohol and Other Drugs) report: Alcohol and other drug use among students in 30 European countries.* Stockholm: Swedish Council for Information on Alcohol and Other Drugs.

Hibell, B., Andersson, B., Bjarnason, T., Ahlström, S., Balakireva, O., Kokkevi, A., et al. (2004). *The ESPAD report: Alcohol and other drug use among students in 35 European countries.* Stockholm: Swedish Council for Information on Alcohol and Other Drugs.

Kythreotou, F. (2003). *The book of Cyprus wine*. Nicosia: Intercollege Press.

Morgan, M., Hibell, B., Andersson, B., Bjarnason, T., Kokkevi, A., & Narusk, A. (1999). The ESPAD study: Implications for prevention. *Drugs: Education, Prevention, and Policy, 6,* 243–256.

Saunders, J. B., Aasland, O. G., Babor, T. R., de la Fuente, J. R., & Grant, M. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption—II. *Addiction, 88,* 791–804.

Spence, S., & Essau, C. A. (2004). *Spence-Essau Anxiety Scale.* Unpublished instrument.

Weitzman, E. R., Nelson, T. F., & Wechsler, H. (2003). Taking up binge drinking in college: The influences of person, social group, and environment. *Journal of Adolescent Health, 32,* 26–35.

This page intentionally left blank

Chapter **11**

Alcohol Use and Abuse in Poland

Anna Bokszczanin, PhD, Cecilia A. Essau, PhD, MA, HBA,
and Jean O'Callaghan, PhD

DRINKING AND POLISH CULTURE

The cultural model of drinking consists of many elements that describe the manner of alcohol consumption in a given country. Single and Leino (1998) consider the following elements to be of greatest importance: the age and gender of drinking people, the place and time of drinking (during meals, meetings, free time), and the kind of alcoholic beverages and the way of drinking (savoring an alcoholic beverage or drinking with the intention of getting drunk).

The customs of drinking alcohol in Poland are deeply rooted and pervasive. Alcohol accompanies the Polish family virtually at every occasion, starting with the birth of a child and finishing with a funeral reception. Alcohol is also drunk to celebrate name day or birthday parties, during meetings with friends, and to evoke different moods—getting emotional, relieving pain, soothing sadness, or cheering up. A typical beverage is vodka (in Polish, *wódka*), which has been produced in Poland since the early Middle Ages, when it was commonly used as a medicine and believed to serve as an aphrodisiac to increase fertility. Large-scale vodka production began in Poland at the end of the sixteenth century, and the end of the eighteenth century saw the beginning of the vodka industry. The production of vodkas became a Polish government monopoly in 1925. Following the Second World War, all vodka distilleries were under the control of the Communist government. During the 1980s, the sale of vodka was rationed, and following the victory of the Solidarity movement, distilleries in Poland were privatized. This privatization has led to an explosion of various brands of vodka and deregulation of rationing.

In terms of gender differences, alcohol drinking is men's domain in Poland. There are many more nondrinking women than men among the adult population of Poland. Traditional alcoholic beverages are high proof, and their consumption usually ends in excess. Statistical data show that more than 50 percent of adult men usually drink vodka in huge amounts (Dolata, n.d.).

One of the most disturbing phenomena in Poland is the high frequency of premature death that is, to a large extent, related to alcohol consumption. The frequency of such death is about 40 percent higher than the European average, especially among men (Piątek, 2003).

GOVERNMENT REGULATION OF DRINKING

Changes in alcohol consumption in Poland need to be considered in the context of political changes and associated economic climates. For example, the generally low rate of alcohol consumption in the 1980s was understood to be associated with depressed consumption of many goods in Poland. This period was marked by increasing inflation and rationing of goods. The political situation was also critical. Specifically, martial law was declared at the end of 1981 to control democratic movements, especially Solidarity. After martial law was lifted, numerous restrictions remained for several years that reduced the civil liberties of people living in Poland. During this period, the Roman Catholic Church played an active role not only in supporting anti-Communist opposition, but also in the promotion of an alcohol-free lifestyle (Wolniewicz-Grzelak, 1990).

Solidarity was again legalized in 1989, which initiated the democratic process. In the early 1990s, the alcohol industry, which was controlled by the state, began to be privatized, leading to less governmental control over alcohol production, distribution, and importation. Prior to 1989, there were no advertisements for any goods on Polish TV or in any other mass media; however, with the first advertisements in the 1990s, a lot of beer ads were addressed at teenagers (Świątkiewicz, 1997). This led to an increased availability of beer and better-quality beer. At the same time, wine prices increased considerably in comparison to beer and vodka. Between 1992 and 1996, alcohol became a dominant aspect of youth culture, being used by 90 percent of 15-year-olds (Sierosławski, 2002). The increase in alcohol consumption among minors was interpreted as being related to a more permissive attitude toward alcohol (Sierosławski, 2002) and to the availability of a wide variety of alcoholic drinks, which stimulated temptations to experiment. At the same time, a revision of Polish state policy toward alcohol drinking in the 1990s involved the process of liberalization, which resulted in less restrictive supply. Thus, while in the 1980s, alcohol policy was based on an abstinence ideology and limitations of availability, in the 1990s, it focused on

promotion of responsible drinking and risk reduction. In this new climate of easy access to alcohol, while selling alcohol to under 18-year-olds is forbidden, nevertheless, social and legal controls on availability for adolescents are not easily enforced (Sprawozdanie, 2006). Furthermore, during the 1990s, many alcoholic beverages became inexpensive (Sprawozdanie, 2000, 2006).

The transformation of the political system in Poland in 1989 from a socialist to a capitalist system also had far-reaching implications with respect to alcohol consumption, as noted previously with regard to the supply and consumption of alcohol (Szymański, Woynarowska, & Mazur, 2001). In comparison with the period before the system's transformation, the consumption of beer and vodka increased considerably among Polish adolescents, whereas among adults, consumption of vodka decreased considerably.

Another important change was Poland's accession to the European Union (EU) in 2004, which is referred to as the second phase of transformation and which brought about changes in practices and customs associated with drinking (Piątek, 2003). It is predicted that Poland will catch up with Europe with respect to addictive practices that are socially degenerative and reinforce alcohol problems for both genders. Positive aspects connected with Poland's accession to the EU must also be considered, especially the possibility of collaborative research. Poland offers considerable potential for research into the prevention and social regulation of alcohol problems (Piątek, 2003).

The legal drinking age is 18. Advertising of alcoholic drinks is prohibited in Poland. An exception is the advertising of beer when it is not (1) addressed to minors; (2) done in such a way that it creates associations with sexual attractiveness, leisure, body fitness, professional activities, job or life success, or health; (3) shown via television, radio, cinema, or theater between 6:00 A.M. and 8:00 P.M.; (4) on videotapes or other data carriers; (5) featured by youth or children's presses; (6) on the front pages of newspapers or magazines; (7) on posters or billboards, except when at least 20 percent of the advertising space is used for information about the adverse effects of alcohol use and the prohibition against selling alcohol to minors; and (8) used with minors. These restrictions do not include advertising that is conducted inside warehouses or in places that sell alcoholic beverages.

PREVALENCE OF ALCOHOL USE AND ABUSE

Alcohol Use in Adolescents

In 1990, Poland joined 32 other countries involved in the Health Behaviour in School-Aged Children surveys (Woynarowska & Mazur, 2000). This study

showed that almost all Polish 15-year-olds had consumed alcohol sometime in their lives (Currie et al., 2000). Specifically, 92 percent and 91 percent of 15-year-old boys and girls, respectively, had drunk alcohol. About 20 percent of the boys and 8 percent of the girls consumed alcohol on a regular basis (i.e., at least once a week). When comparing the frequencies of drinking alcohol from 1994 to 1998 among 15-year-olds, the data showed a decrease. However, the frequency of drunkenness increased by five times in 15-year-old boys and by three times in girls.

Most frequently, alcoholic beverages are drunk at parties, often at all-night ones (Łosik & Chadaj, 2003). The main reason for adolescent drinking was related to the lack of parental control. This tolerant attitude of parents is supported by a recent study that showed that between 25 and 49 percent of adolescents drank alcohol for the first time in the presence of adults; most often, it was in their parents' company (Hryszkiewicz, 2002). Another reason for juvenile drinking is alcohol's availability, despite the law prohibiting the sale of alcoholic beverages to adolescents. The reason for the lack of law enforcement is related to the strong competition in the alcohol market (Łosik & Chadaj, 2003).

Poland has also participated in the European School Survey Project on Alcohol and Other Drugs (ESPAD) since 1995 (Sierosławski, 2005). ESPAD results from the years 1999 and 2003 showed no significant change in levels of alcohol use by 15-year-olds (Sierosławski, 2005). An additional ESPAD survey was conducted in Poland, and the findings showed a decrease (from 93% in 2003 to 85% in 2005) in the lifetime prevalence of alcohol drinking by 15-year-olds. The results showed a decrease in the number of adolescents who consumed alcohol (from 66% in 2003 to 45% in 2005) and who reported being drunk (from 29% in 2003 to 15% in 2005) during the past 30 days. In the mid-1980s, wine was the most popular alcoholic drink, especially among girls. Beer became the alcoholic beverage of choice in 1988 among boys and in 1996 among girls. In 2004, more girls than boys reported drinking (Okulicz-Kozaryn & Borucka, 2006).

One of the most important studies in Poland was the one conducted by Okulicz-Kozaryn and Borucka (2006). This epidemiological study examined changes in patterns of alcohol use from 1988 to 2004 among 15-year-olds in Warsaw. The study started in 1988 and was conducted at an interval of four years thereafter (i.e., 1988, 1992, 1996, 2000, and 2004). This study is of interest because it covers a 20-year period, including significant political, economic, and social changes, which provide a changing context for adolescent alcohol use.

The results can be summarized as follows:

1. The results showed that in the years 1984–1996, the percentage of adolescents who had never drunk alcohol had fallen from 22 to 10 percent. Between 1996 and 2004, the proportion of nondrinking, frequently drink-

ing, and excessively drinking pupils remained stable. This finding was interpreted as a sign of adaptation to the new political, social, and economic conditions of Polish society.

2. A major change between 2000 and 2004 is the increase of vodka abuse by adolescents.

   The most common alcoholic drinks among adolescents who drink with their peers were beer and vodka: 67 percent of the adolescents drank beer with other adolescents in 1988, compared to 81 percent in 2000, while vodka drinking with peers remained stable (72% in 1988 and 80% to 88% in following years). The prevalence of wine drinking with peers was highest in 1992 (60%) but decreased from the mid-1990s to 41 percent in 2004.
3. A small proportion of adolescents drank alcohol alone. The alcoholic beverage most often used with parents was wine (30% in the 1980s and 1990s; 40% in the twenty-first century).
4. Girls drink more. Some possible explanations for this gender difference include (1) a general trend that girls use alcohol, smoke cigarettes, and try illicit drugs at least as frequently as boys, and (2) biological factors, which show a decrease in the age of onset of menstruation in industrialized countries during the twentieth and twenty-first centuries. Biological maturity has a strong impact on alcohol drinking by girls, and the results of some studies provide support for a positive correlation between the age of menarche and girls' tendency to associate with older boys. (3) In terms of spending free time, girls generally spend more time outside of the home than boys, which exposes them to alcohol use. Boys tend to play computer games more than girls (Ostaszewski et al., 2005), which limits the opportunities for drinking alcohol. (4) There seemed to be gender differences in reasons for drinking: girls tend to initiate and continue consuming drugs to cope with negative emotions; boys drink to increase social bonding (Isralovitz & Rawson, 2006). (5) Girls experience high levels of life stressors, and they tend to drink as a way of coping with these stressors.

In interpreting the high rate of alcohol drinking among adolescents, the authors suggested that the following factors may be responsible: a greater availability of alcohol for juveniles, disobedience of rules prohibiting the sale of alcohol to juveniles below the age of 18, and adults' more liberal attitude to juvenile drinking. It can be assumed that the fast political and economic changes in Poland were stressful factors both for parents and for adolescents. Parents' problems with their new professional and social situations, for example, the growth of unemployment, were directly reflected in the behavior of both grown-ups and children (cf. Bokszczanin & Essau, 2007a, 2007b).

A study by Maciorkowska, Buraczewska, and Sacharewicz (2007) examined the frequency of alcoholic beverage use among children and adolescents in Białystok City and examined the influence of familial and environmental fac-

tors on these young people's alcohol consumption. Their first experiences with alcohol occurred when the young people were 5 to 10 years old, with the greatest alcohol initiation (35% of young people) being reported in the 10- to 15-year-old age group. Adolescents' main reason for alcohol consumption was for company (21.6%); they also drank due to a lack of feeling safe (18.4%) and as part of free-time activities (23.4%). More than half of the children (63.4%) had conflict situations at home, and 19.7 percent were unsatisfied with their lives. Despite the ban on selling alcohol to minors, only a very small proportion (5%) of the adolescents reported being asked by a shop assistant for identification. About 18 percent of the adolescents obtained alcohol at parties, 15 percent bought it themselves, and 7 percent asked their older friends to buy it on their behalf. Alcohol was very easily accessible to young people: 21.7 percent of them buy alcohol frequently in the neighborhood shop, and 12.1 percent bought alcohol in a shop where no one knew them. Furthermore, the findings also showed that 70.8 percent of parents talk about bad habits with their children.

A recent study by Makara-Studzińska and Urbańska (2007) examined frequency of drinking, age of alcohol initiation, and preferences toward alcoholic drinks among people from rural areas in Lublin Province between the years 2002 and 2004. They studied 5,000 persons, aged 14–24 years, who had been recruited from various types of schools (i.e., secondary, technical, vocational, and high schools). Participants who attended vocational schools were reported to have started drinking at the earliest age (64% in primary school and 14% before age 10). Among those who attended technical school, 54 percent drank alcohol in primary school and 10 percent drank before the age of 10; 36 percent started drinking after the age of 16. Similar results were obtained among those who attended secondary school (58% and 36%, respectively). Beer was reported to be the most common alcoholic beverage. About half of the adolescents who consumed alcohol on a regular basis described the atmosphere in their homes as unpleasant, and in 25 percent of cases, it was described as very unpleasant. Most of these young adults reported having fathers who abused alcohol, and they described their fathers as aggressive (53%) and quickly aroused to anger (38%).

Alcohol Use among Adults

In 1937, Poland's consumption of alcohol was not greater than 0.7 liters per capita; in the 1950s, it was three to four liters, and in the 1980s, it reached the level of seven liters. In the 1990s, there was another jump to over 10 liters of pure alcohol per capita (Dolata, n.d.). The results of research conducted by the Social Research Laboratory (Jastrun, 2006) indicate that men drink 3.5 times

more alcohol than women, and the greatest consumption of alcohol is observed between the ages of 18 and 29. The unemployed drink the most, whereas pensioners and senior citizens drink the least. The consumption of alcohol among the poorest people increased, while among the wealthiest, it decreased. The most popular alcoholic beverage among young people is beer. Jastrun (2006) presents the latest statistical data concerning drinking, which show an increase in consumption of spirits by one-third, of vodka by 40 percent, and of beer by 10 percent in the years 2002–2005.

The recent survey on Attitudes towards Alcohol (European Community, 2007) in Europe provided information on the prevalence and patterns of alcohol consumption in Poland. The survey was conducted at the end of 2006, in 25 member states of the EU, 2 acceding states (Bulgaria and Romania), and 1 candidate country (Croatia), and in the Turkish Cypriot Community (TCC). The participants were interviewed face-to-face in their homes. One thousand adults in Poland participated in the survey. The results of this survey can be summarized as follows:

1. A high percentage of the participants in Poland (72%, compared to 75% across the EU) reported to have had alcoholic beverages (i.e., beer, wine, spirit, cider, or other local beverages) during the last 12 months. Seventy-five percent of the Poles reported having drunk alcohol during the last 30 days before the survey. Of those who consumed alcohol in the last 30 days, only 1 percent drank alcohol daily, 4 percent had it four to five times a week, 9 percent had it two to three times a week, and 28 percent drank once a week.
2. The percentages of those who had five or more drinks on one occasion in the last 12 months were lower than reported in many EU countries; that is, only 4 percent of the Poles reported drinking such a considerable amount of alcohol several time a week, compared to about 13 percent of respondents across Europe.
3. A high percentage of the participants (61%) have one to two drinks on the days they drink alcohol.
4. A low percentage of the Poles (27%) reported not knowing the legal blood level that is allowed for car drivers. Most participants (78%) agreed that random alcohol checkpoints conducted by police would decrease alcohol consumption before driving.
5. About one-third (36%) of the Poles did not believe that higher prices of alcohol would discourage young people and heavy drinkers from consuming alcohol.
6. Eighty-nine percent of participants agreed with putting warnings on alcohol bottles and advertisements, particularly for warning pregnant women and car drivers.

7. Almost all (93%) of the participants agreed that selling and serving should be banned to under 18-year-olds, and 79 percent agreed with banning alcohol advertising that targets young people.

RISK FACTORS

Research on risk groups (Sierosławski, 2008) shows that among the consumers of alcoholic beverages, 10.6 percent of Poles abuse alcohol, that is, drink above the safety threshold. The smallest number of those drinking at the level of increased risk are the oldest people, whereas the greatest number are people in their forties. Sierosławski's research also proves that marital status does not differentiate people as far as risky drinking is concerned. The size of the place of living differentiates significantly only men: the largest proportion of those included in the risk group live in villages, while towns and cities do not differ in this respect. Individuals with higher education drink less riskily than individuals with secondary or vocational education. The proportion of those drinking riskily is also significantly higher among unemployed people in comparison with those in steady employment. Practicing believers—both men and women—drink riskily much less frequently than others (Sierosławski, 2008).

A recent study by Bobrowski (2007) provided information on adolescents' risky behavior. A factor that reduced juveniles' tendency for reaching for psychoactive substances, including alcohol, by 50 percent was spending at least one hour daily doing homework, reading books for pleasure, or going to the cinema. On the other hand, staying out more than three hours daily increased the risk of abusing psychoactive substances. Interestingly enough, Bobrowski's research also proved that pupils practicing sports intensively have a stronger tendency for abusing psychoactive substances.

Bokszczanin (1998) examined the extent to which belief about the positive influence of alcohol consumption would increase the tendency to be addicted to alcohol. Her results showed that 22 percent of the adolescents from vocational schools (aged 16–17) believed that "those who have strong heads may drink freely." Twenty-one percent of them thought that "beer and wine are not real alcohol," 11 percent claimed that "a driver may drink a little," 15 percent considered the belief that "only primitive people from bad environments have alcoholic problems" to be true, 83 percent of the surveyed juveniles found the opinion that "strong will is the most important thing in dealing with drinking" to be true, and 27.7 percent believed that "alcohol can be a good remedy for different ailments." It seems that these adolescents' opinions, especially about what is and what is not alcohol and about the role of a strong will in drinking, are a reflection of grown-ups' opinions.

TREATMENT AND PREVENTION OPTIONS

The Polish state undertakes many activities to fight drunkenness and alcoholism such as legislative, medical, preventive, and social initiatives. The legal basis for activities connected with the prevention and solution of alcohol problems is the Sobriety Education and Alcoholism Prevention Act of October 26, 1982. According to this law, provincial local governments are obliged to carry out many activities, among others, activities aimed at limiting alcohol consumption, creating social organizations, and cooperating with churches in the field of sobriety education.

According to statistics, there are about 600,000 to 700,000 people addicted to alcohol in Poland (Dolata, n.d.). Alcoholism is treated in state and private clinics and therapeutic centers. In the last 20 years, the treatment of alcoholism in Poland has changed from medical only to psychological and medical. Within the inpatient care system, there are detoxification centers, wards and subwards of psychiatric hospitals, where such forms of aid as detoxification, group therapy, and occupational therapy are used. In outpatient clinics, education in the field of alcohol problems and individual, group, and family therapy are used. The networks of Alcoholics Anonymous support groups and teetotalers' clubs are also popular (Bełza, 2004).

Moskalewicz, Sierosławski, and Dąbrowska (2006) recently evaluated a system that is involved in psychoactive substance addiction treatment in Poland. The following aspects were included in the analysis: resources (personnel, budget), the scope of services, availability, time of treatment, and remaining in therapy. Their results indicated that the dominant aim of therapy was abstinence from alcohol, and subsequently, the improvement of life's quality. The time spent waiting for treatment is relatively short in outpatient clinics (about a week), but it takes a little longer in detoxification wards (20 days on average). Most financial resources for treatment and rehabilitation come from the National Health Fund (a state fund). Treatment programs are completed by fewer than 50 percent of patients.

About 20 years ago, the age of professional preventive activities began in Poland (Szymańska, 2000), and several of the prevention programs have been evaluated (Koczurowska, 2002). Some examples of evaluated prevention programs that have shown some effectiveness are the programs carried out locally in Opole Province, for example, the prevention program for adolescent leaders called the Activation of Juvenile Preventive Initiatives (Łabudziński, 2007), and Peer Help (Raszka, 2007), which aims to educate university students (volunteers), who will later train adolescent leaders in junior secondary schools and secondary schools in the field of preventing addictions. It is hoped that with

many more young people taking part in such programs, the number of those with alcohol problems will decrease.

REFERENCES

Bełza, M. (2004). Alkoholizm—wybrane sposoby pomagania wychodzenia z nałogu [Alcoholism—selected methods of weaning people of addiction]. *Psychologia i Rzeczywistość, Czasopismo internetowe, 1*. Retrieved April 30, 2008, from http://www.dzieci.bci.pl/strony/nadzieja/psychrzecz.html

Bobrowski, K. (2007). Czas wolny a zachowania ryzykowne młodzieży [Leisure time and risky behavior of young people]. *Alkoholizm i Narkomania, 20*, 267–287.

Bokszczanin, A. (1998). Optymizm i spostrzeganie zagrożeń związanych z piciem przez młodzież pijącą alkohol i palącą papierosy [Optimism and the perception of drinking related threats among young people drinking alcohol and smoking cigarettes]. *Alkoholizm i Narkomania, 2*, 219–228.

Bokszczanin, A., & Essau A. C. (2007a). Impact of unemployment on life satisfaction in Poland and Germany. In A. Bokszczanin (Ed.), *Social change in solidarity: Community psychology perspectives and approaches* (pp. 149–155). Opole, Poland: Wydawnictwo Uniwersytetu Opolskiego.

Bokszczanin, A., & Essau A. C. (2007b). Parental unemployment and its effects on children's coping strategies. In A. Bokszczanin (Ed.), *Social change in solidarity: Community psychology perspectives and approaches* (pp. 197–203). Opole, Poland: Wydawnictwo Uniwersytetu Opolskiego.

Dolata, A. (n.d.). *Alkoholizm w Polsce—problem społeczny*. Retrieved April 30, 2008, from http://209.85.129.132/search?q=cache:EQiOoHgEeoMJ:publikacje.lozywiec.pl/publikacje/234.doc+dolata+alkoholizm+w+Polsce&hl=pl&ct=clnk&cd=3&gl=pl.doc

European Community (2007). Attitudes towards alcohol. Brussels: European Community.

Hryszkiewicz, G. (2002). Alkohol, młodzież, dorośli [Alcohol, youth, adults]. *Świat Problemów, 9*, 18–19.

Jastrun, E. (2006). Wzorce konsumpcji alkoholu w Polsce [Patterns of alcohol consumption in Poland]. *Świat Problemów, 4*, 4–7.

Koczurowska, J. (2002). Standardy jakości programów profilaktycznych (Quality standards of preventive programmes). In G. Świątkiewicz (Ed.). *Profilaktyka w środowisku lokalnym*, 115–124. Warszawa, Poland: Krajowe Biuro ds. Przeciwdziałania Narkomanii.

Łabudziński, M. (2007). Ewaluacja efektów programu dla liderów młodzieżowych, Aktywizacja młodzieżowych inicjatyw profilaktycznych" [The evaluation of the effects of the Programme for Juvenile Leaders: "The activation of juvenile preventive initiatives"]. In *Niepublikowana praca magisterska* (pp. 1–139). Opole, Poland: Uniwersytet Opolski, Instytut Psychologii.

Łosik, L., & Chadaj, J. (2003). Skala zjawiska alkoholizmu u dziewcząt między 14–18 rokiem życia [The range of alcoholism among girls aged 14–18]. In L. Niebrój & M. Kosińska (Eds.), *Rodzina: dziecko i jego potrzeby zdrowotne: Vol. 2. Eukrasia* (pp. 109–114). Katowice, Poland: Wydawnictwo ŚAM.

Maciorkowska, E., Buraczewska, E., & Sacharewicz, A. (2007). Familial and social conditions of alcohol drinking in children and adolescents. *Advances in Medical Sciences, 52*, 115–118.

Makara-Studzińska, M., & Urbańska, A. (2007). Alcohol consumption patterns among young people from rural areas of Lublin province. *Annals of Agricultural and Environmental Medicine, 14*, 45–49.

Moskalewicz, J., Sierosławski, J., & Dąbrowska, K. (2006). Ocena systemu leczenia i rehabilitacji osób uzależnionych od substancji psychoaktywnych w Polsce [The evaluation of the system of treatment and rehabilitation of people addicted to psychoactive substances]. *Alkoholizm i Narkomania, 19*, 327–355..

Okulicz-Kozaryn, K., & Borucka, A. (2006). Zmiany w piciu alkoholu przez warszawskich nastolatków w latach 1984–2004 [Changes in alcohol drinking among Warsaw teenagers in the years 1984–2004]. *Alkoholizm i Narkomania, 19*, 243–258.

Piątek, D. (2003). *Dolnośląski program profilaktyki i rozwiązywania problemów alkoholowych oraz zadań z zakresu zapobiegania narkomanii na lata 2004–2008* [The Lower Silesian Programme of prevention and solution of alcoholic problems and tasks in drug addiction prevention for the years 2004–2008]. Wrocław, Poland: Departament Spraw Społecznych, Biuro Profilaktyki i Rozwiązywania Problemów Uzależnień.

Raszka, M. (2007). Udział w programie profilaktycznym „Rówieśnicze Pomaganie" a postawy wobec środków psychoaktywnych i postawy prospołeczne u studentów wolontariuszy [Participation in the preventive program "Peer Help" and the attitudes toward psychoactive substances and prosocial attitudes among student volunteers]. In *Niepublikowana praca magisterska* (pp. 1–121). Opole, Poland: Uniwersytet Opolski, Instytut Psychologii.

Sierosławski, J. (2005). Badania szkolne na temat używania substancji psychoaktywnych przez młodzież (ESPAD). Polska młodzież na tle Europy. W: Zieliński A. *Ewolucja wzorów używania alkoholu przez młodzież w Polsce w XX wieku*, 75–88. Warszawa, Poland: Wydawnictwo Edukacyjne PARPA.

Sierosławski, J. (2008). *Płeć a picie alkoholu* [Gender and alcohol drinking]. Retrieved April 30, 2008, from http://www.alkoholizm.akcjasos.pl/pliki/Plec%20a%20picie%20alkoholu.doc

Single, E., & Leino, V. E. (1998). Levels, patterns and consequences of drinking. In M. Grant & J. Litvak (Eds.), *Drinking patterns and their consequences* (pp. 7–24). Washington, DC: Taylor and Francis.

Sprawozdanie. (2006). Sprawozdanie, realizacji ustawy o wychowaniu w trzeźwości i przeciwdziałaniu alkoholizmowi w okresie 1 stycznia–31 grudnia 2005 roku, Państwowa Agencja Rozwiązywania Problemów Alkoholowych (National Agency for Solving Alcohol Problems).

Świątkiewicz, G. (1997). Kwestia piwa w okresie transformacji społeczno-ustrojowej w Polsce. *Alkoholizm i Narkomania, 1*, 11–19.

Szymańska, J. (2000). *Programy profilaktyczne. Podstawy profesjonalnej profilaktyki* [Preventive programmes: The rudiments of professional prevention] (p. 107). Warsaw: Centrum Metodyczne Pomocy Psychologiczno Pedagogicznej.

Szymański, J., Woynarowska, B., & Mazur, J. (2001). Picie napojów alkoholowych przez młodzież szkolną w Polsce i w innych krajach. Tendencje zmian w latach 1990–1998 [The drinking of alcoholic beverages among school juveniles in Poland and other countries: Tendencies of changes in the years 1990–1998]. *Alkoholizm i Narkomania, 14*, 245–259.

Warszawa. Report on realization of the act on upbringing in sobriety and counteracting alcoholism (January 1–December 31 2005).

Wolniewicz-Grzelak, B. (1990). Zmiany w spożywaniu przez młodzież napojów alkoholowych. Badania mokotowskie z 1984 i 1988 roku. *Alkoholizm i Narkomania, 4*, 113–132.

Chapter **12**

Alcohol Consumption and Binge Drinking in the United Kingdom

Cecilia A. Essau, PhD, MA, HBA,
Changiz Mohiyeddini, MD, PhD, and Diane Bray, PhD

Like in many European and other Western countries, alcohol use is an accepted part of social interaction in the United Kingdom. The majority of adults in the United Kingdom have been reported to drink alcohol regularly. Among young people, drinking is regarded as integral to a night out, and heavy drinking akin to binge drinking is used by some young people to establish and maintain friendships. In fact, it is the group aspect of the binge drinking session that makes getting drunk so much fun.

Binge drinking is nothing new in British society. It has been endemic in British society for centuries, and binge drinking has been embedded in various social and work practices, for example, the Gin Craze that swept over London in the eighteenth century, to illustrate a parallel with the past (Borsay, 2006). The term *Gin Craze* was used to describe a moral panic that characterized concerns related to the impact of rapid urbanization on law and order; the effect of rising levels of wealth on patterns of consumption; the will to work and the fulfillment of domestic responsibilities; and widespread anxiety about family breakdown and government's failure to take effective action to remedy the problems. An increased disapproval of heavy drinking began in British society after industrialization.

ALCOHOL USE

The drinking of alcoholic beverages is both a popular and a generally accepted activity among young people in the United Kingdom. In fact, first experiences

with alcoholic beverages are made together with family members and friends (Essau, Karpinski, Patermann, & Conradt, 1998). Although alcohol is illegal for adolescents in public places, its use is very widespread. According to the European Schools Project on Alcohol and Other Drugs (ESPAD) (ESPAD; Hibell et al., 2001), 91 percent of the adolescents had been drinking alcohol during the last 12 months; this figure is above the average reported for all ESPAD countries (83%). The percentage of adolescents who reported drunkenness during the same period is also higher than the average among ESPAD countries (68% versus 53%). The percentage of those who use alcohol in combination with pills is 7 percent, which is comparable to percentages found in other countries.

The average age for a first experience with alcohol is 11 years among boys and 11.5 years among girls (Leite & Parrish, 1992). An early experience with alcohol is not uncritical. As shown by Holly, Türk, Nelson, Pfister, and Wittchen (1997), alcohol users who first consumed alcohol at the age of 14 years or younger were at higher risk for meeting a diagnosis of dependence and abuse compared to those who initiated alcohol use at 15 years of age or older.

BINGE DRINKING

There is perhaps no other problem in adolescent and young people that has attracted as much media attention as binge drinking. Headlines such as "Binge-Drinking Now the Norm," "Binge-Drinking Has Become a Fashion," and "Teen Binge Drinking: Common and Risky" are common among major British newspapers. The term began to be used in the late 1990s, and between 1998 and 2002, there were 10 to 20 mentions of the term in the print media a year; in 2003, this figure was about double (39 mentions). In 2004, there was a threefold increase in the number of articles that included the term *binge drinking,* most of which were related to the introduction of the Licensing Act of 2003.

Indeed, studies have indicated that young adults between 18 and 24 years of age are more likely than other adults to binge drink (Moore et al., 1994). There is also concern that binge drinking and alcohol consumption are increasing among young people in the United Kingdom (Harnett et al., 2000).

An accumulative number of studies have examined factors associated with binge drinking among young people in the United Kingdom. These include (Murgraff, Parrott, & Bennett, 1999) (1) binge drinking being typically viewed as socially acceptable and normal youth behavior; (2) personal and social motives, such as curiosity and experimentation, group pressure, the desire to

appear grown-up, and positive expectations of alcohol use; (3) availability and affordability—the introduction of alcopop products and specific promotions (e.g., two for the price of one and all you can drink for £10) are thought to have contributed to binge drinking among young people; and (4) advertising of alcoholic drinks through various media, including television, radio, print, point-of-sale promotions, and the Internet.

As shown by several studies, alcohol advertisements have been found to be related to positive attitudes about alcohol among young people.

IMPACT OF BINGE DRINKING

Binge drinking is responsible for various kinds of health care, societal, and personal costs. According to various government reports (Prime Minister's Strategy Unit, The Academy of Medical Sciences, Health Development Agency),

- About 5.9 million adults (23% men and 9% women) engage in binge drinking
- The number of binge drinkers among young British women has increased more than in any other EU country in the last decade
- Death rates as a result of acute intoxication have doubled in the last 20 years among men and women
- Twenty-nine percent of deaths are related to alcohol (mostly as a result of injuries when in a state of intoxication), especially among 16- to 34-year-olds
- The annual cost of crime/antisocial behavior and the cost of productivity lost related to alcohol misuse is £7.3 billion and £6.4 billion, respectively

In economic terms, binge drinking has been estimated to cost Great Britain £20 billion a year. Billions more are spent clearing up alcohol-related crime and social problems. Alcohol-related problems are recorded to be the cause of 22,000 premature deaths each year. Binge drinking and alcohol consumption are also responsible for a wide range of health care costs. Binge drinking is specifically related to accidents and violence, both of which cost the health service billions of pounds each year. Around 40 percent of admissions to the Accidents and Emergency Units are alcohol related, and at peak times (i.e., between midnight and 5:00 a.m.), this figure rises to 70 percent. The cost of alcohol misuse to the health service is about £1.7 billion a year.

People who binge drink report engaging in a range of risk-taking behaviors, particularly while drunk. Some examples of risk behavior reported by young people while drunk include getting in a car with a stranger or going back to a

stranger's house; pranks that put them in physical danger and involvement in disorderly behavior (e.g., fighting, vandalism, and stealing); and unprotected sex or sex with someone they did not know. In fact, Cooper (2002) showed that drinking was strongly associated with decreased protective behaviors among younger individuals and first intercourse experiences. Furthermore, a recent review by Cook and Clark (2005) indicated that problem drinking is associated with an increased risk of sexually transmitted diseases across populations.

Heavy drinking has been identified as a major risk factor for suicide and suicidal behavior among both young people and adults (Beautrais, 1998). Additionally, the risk of alcohol dependence increases with both the volume of alcohol consumption and binge drinking (Caetano et al., 1997). Indeed, alcohol dependence occurs frequently among young adults (Caetano & Cunradi, 2002), particularly among those with frequent drinking at ages 14–15 years and binge drinking at age 16 years.

To cope with these problems, particularly in reducing violence and antisocial behavior, a new licensing law was introduced in 2005. The government's rationale for this licensing law is to enable premises to stagger closing times and for the drinkers not be tempted to try to beat the clock due to the pub's closing time.

THE NEW LICENSING LAW

The new Licensing Act of 2003 came into effect in November 2005. Its introduction has changed the way alcohol and entertainment licensing is administered in England and Wales (Parliamentary Office of Science and Technology, 2005). The new law allows the extension of opening times to 24 hours (seven days a week), the transfer of licensing powers from magistrates to local authorities, and expanded police powers to deal with troublesome premises. It was argued by the Department for Culture, Media, and Sport (DCMS) that extending opening times may reduce the practice of stocking up with alcohol shortly before closing time. The DCMS also suggests that by staggering the closing times, the number of people disgorging on the streets could be reduced. This new law was not without argument. The Royal College of Physicians, for example, suggests that the extended opening hours might actually increase, rather than decrease, overall alcohol consumption.

ALCOHOL AND CRIME

According to the recent results of the British Crime Survey 2006/07 (BCS; Nicholas, Kershaw, & Walker, 2007), in 39 percent of domestic violence cases,

alcohol somehow was involved. In 46 percent of all violent incidents and 58 percent of cases of stranger violence, victims believed offenders were under the influence of alcohol. Furthermore, BCS 2006/07 showed that in 1,087,000 recorded violent incidents, the victims believed that the offender or offenders were under the influence of alcohol. In 58 percent of incidents involving stranger violence, in 47 percent of incidents of acquaintance violence, and in 39 percent of incidents involving domestic violence, the offender was judged to be under the influence of alcohol.

A growing body of literature and empirical results has contributed to the knowledge and understanding that the link between alcohol and crime is multifaceted (Berry & Brain, 1986; Giancola & Zeichner, 1995; Hayes, 1993; Kelly, Cherek, Steinberg, & Robinson, 1988; Room & Collins, 1983). Dehhan (1999) emphasized that alcohol-related crime covers a continuum of offences that involve alcohol as the cause, as a disinhibitor, or as an excuse or that alcohol can result in crime because the individual has a drinking problem. Therefore alcohol can not only be considered as a causal factor in crime, but also, alcohol can contribute to crime and be associated with it.

According to Hayes (1993), alcohol is related to crime through three different pathways: a causal pathway, a contributory pathway, and a coexistence pathway. Causal relationships comprise three groups of offences:

a*lcohol-defined offences*. This group is defined by combining a lawful behavior (being in public places) with consumption of alcohol (drunk in a public place)

alcohol-induced offences. This group includes offences that emerge because the offender has drunk alcohol (e.g. domestic violence)

alcohol-inspired offences. This group comprises offences committed to obtain alcohol (e.g., shoplifting)

Contributory relationships describe alcohol acting as a trigger to gain courage to facilitate an offence (Dutch courage), whereas coexistence relationships refer to criminal behavior alongside (heavy) alcohol consumption.

The Rule of Physical and Social Drinking Environments

Empirical results indicate that a physical and social drinking environment has a significant impact on alcohol-related violence (Roman, 1981). On the basis of an extensive analysis of the information gathered from the academic, health, and policing sources in the United Kingdom, the British Home Office (Deehan, 1999) recommended controlling certain elements of physical and social drinking environments to reduce alcohol-related crime and violence:

- The licensed premises should be attractive and well maintained; such premises have fewer violent incidents
- The licensed premises should avoid offering discounted alcoholic drink promotions and happy hours.
- Alcoholic beverages should be served with food; serving food is associated with lower rates of aggressive behaviors
- The closing times of licensed premises should be staggered; the availability of fast food and transportation should be increased, reducing the number of people on the streets at the same time looking for transport and food
- The intoxication level of drinkers should be considered, and selling alcohol to already intoxicated drinkers should be avoided
- Staff should actively expect customers to behave in a socially acceptable manner and discourage antisocial behavior
- The door staff should be trained to identify potentially aggressive (groups of) customers, refusing them entrance
- Magistrates should make it a condition of a license to use toughened glass on the premises (each year, almost 5,000 injuries occur in the United Kingdom as a result of glass being used as a weapon)

According to Dehhan (1999), a successful initiative for reducing alcohol-related crime and violence will be based on a partnership approach that includes "written agreements, establishment of ownership, adequate funding, active police involvement and an understanding of the need for businesses to make a profit" (p. 1).

Binge Drinking and Crime: The Rule of Age and Gender

Alcohol-related aggression often results in facial injury. According to Magennis, Shepherd, Hutchison, and Brown (1998), about 125,000 people in Britain suffer facial injuries every year in violent circumstances. In 61 percent of cases, either the victim or the assailant had been drinking alcohol. Eighty percent of injured people are men.

The results of BCS 2006/07 (Nicholas et al., 2007) show that in comparison with all other age groups, young adults (18- to 24-year-olds) have the strongest tendency toward binge drinking. Forty-four percent of this age group were identified as binge drinkers, and in this age group, binge drinking was more common among men (49%) than women (39%). Furthermore, compared with regular drinkers (13%), young binge drinkers committed more offences (27%) in the past 12 months. Sixty-three percent of all young adult binge drinkers admitted to criminal and disorderly behavior during or after drinking, compared with 34 percent of other young regular drinkers.

By the same token, the results of BCS show that the relationship between alcohol consumption and crime at a young age is moderated by gender: 25 percent of young male binge drinkers were involved in fights, while only 12 percent of young women got into a fight.

GOVERNMENT INITIATIVES

A number of initiatives have been introduced by the government to tackle binge drinking, particularly among young people, in the United Kingdom, including the following:

1. The Lewisham's Drug and Alcohol Strategy Team recently introduced the "Don't binge and cringe" campaign, which is an innovative approach to tackling binge drinking and its associated harms. During last year's festive season, volunteers gathered in hot spot areas and handed out goody bags that contained (1) a condom; (2) an alcohol leaflet about drunk driving, what is a unit, safe sex, and how to plan a night to ensure how to get home safely; (3) information about alcohol- and drug-related rape and where to get support; (4) spikeys (i.e., the anti-spiking stopper for bottle drinks) to prevent drug-assisted rape; (5) a lollipop; and (6) an overdose card advising what to do if young people are found passed out in the street.
2. Tackling young people's drinking is a pilot scheme to tackle underage drinking. This binge drinking road show aims to educate parents about the dangers of alcohol and drugs.
3. Greater Easterhouse Alcohol Awareness Project has recently launched a new online campaign highlighting the dangers of binge drinking and to promote awareness of the amount of alcohol units in drinks. This project is for young people between 9 and 17. It involves a series of alcohol-related quizzes with four games, and the players compete against each other; the 10 highest scores achieved for each game will go into a top 10.
4. ALCOHOL CONCERN is the national agency on alcohol misuse, working to reduce the level of alcohol misuse and to develop the range and quality of aid services available to problem drinkers and their families.

In addition to these specific initiatives, the National Strategy to Reduce Alcohol Harm was introduced in 2004. This strategy focuses on (1) better education and communication to change attitudes in relation to irresponsible drinking; (2) the introduction of better health and treatment systems so that alcohol problems can be detected and treated early; (3) fighting alcohol-related crime; and (4) working with the alcohol industry both to establish and develop new initiatives. It remains to be seen whether any of these initiatives will prove effective.

CONCLUSION

Binge drinking, particularly among young people, occurs frequently in the United Kingdom and has been estimated to cost Great Britain £20 billion a year. On an individual level, alcohol consumption is associated with a wide range of physical harms. At a societal level, a high level of alcohol consumption will lead to high level of harm and economic disadvantage. Consequently, numerous alcohol policies have been introduced in the United Kingdom that could have an impact on reducing ill health and premature death due to alcohol.

U.K. alcohol policy needs to balance policies to protect and promote public health, on one hand, and on the other hand, to serve the British economic interests. This balance needs to be made correctly to maintain the well-being of British society and its socioeconomic development.

REFERENCES

Beautrais, A. L. (1998). *Risk factors for serious suicide attempts among young people: A case control study*. In R. J. Kosky, H. S. Eshkevari, R. D. Goldney, & R. Hassan (Eds.), *Suicide Prevention: the Global Context* (pp. 167–181). New York, NY: Plenum Press.

Berry, M. S., & Brain, P. F. (1986). *Neurophysiological and endocrinological consequences of alcohol*. In Brain, P. F. (Ed.), *Alcohol and aggression* (pp. 19–55). Dover, NH, Croom Helm.

Borsay, P. (2006). *A history of leisure: The British experience since 1500*. Basingstoke: Palgrave.

Cook, R. L., & Clark, D. B. (2005). Is there an association between alcohol consumption and sexually transmitted diseases? A systematic review. *Sexually Transmitted Diseases, 32,* 156–164.

Cooper, M. L. (2002). Alcohol use and risky sexual behavior among college students and youth: Evaluating the evidence. *J. Stud. Alcohol,* Supplement, 14, 101–117, 2002.

Dehhan, A. (1999). *Alcohol and crime: Taking stock*. London: Home office.

Essau, C. A., Karpinski, N. A., Petermann, F., & Conradt, J. (1998). Häufigkeit und Komorbidität von Störungen durch Substanzkonsum. *Zeitschrift Kindheit und Entwicklung, 7,* 199–207.

Giancola, P. R., & Zeichner, A. (1995). Alcohol-related aggression in males and females: Effects of blood alcohol concentration, subjective intoxication, personality, and provocation. *Alcoholism: Clinical and Experimental Research, 19,* 130–134.

Harnett, R., Thom, B., Herring, R. & Kelly, M. (2000). Alcohol in transition: Towards a model of young men's drinking styles. *Journal of Youth Studies, 3,* 1–77.

Hayes, P. (1993). A view from the probation service. In J. Russell (Ed.), *Alcohol and crime—Proceedings of a Mental Health Foundation conference* (pp. 26–38). London: Health Education Authority.

Hibell, B., Andersson, B., Ahlstrom, S., Balakireva, O., Bjarnasson, T., Kokkevi, A., et al. (2001). *The ESPAD (European School Survey Project on Alcohol and Other Drugs) report: Alcohol and other drug use among students in 30 European countries*. Stockholm: CAN (Swedish Council for Information on Alcohol and Other Drugs).

Holly, A., Türk, D., Nelson, B., Pfister, H., & Wittchen, H.-U. (1997). Prävalenz von Alkoholkonsum, Alkoholmissbrauch und -abhängigkeit bei Jugendlichen und jungen Erwachsenen. *Zeitschrift für Klinische Psychologie, 26*, 171–178.

Kelly, T. H., Cherek, D. R., Steinberg, J., & Robinson, D. (1988). Effects of provocation and alcohol on human aggressive behaviour. *Drug and Alcohol Dependence, 21*, 105–112.

Leite, J. S., & Parrish, J. K. (1992). Why kids are the way they are. *Adolescence, 7*, 52–54.

Magennis, P., Shepherd, J., Hutchison, H., & Brown, A. (1998). Trends in facial injury. *British Medical Journal, 316*, 325–326.

Moore, L., Smith, C., & Catford, J. (1994). Binge drinking: Prevalence, patterns and policy. *Health Education Research, 9*, 497–505.

Murgraff, V., Parrott, A., & Bennett, P. (1999). Risky single-occasion drinking among young people—Definition, correlates, policy and intervention: A broad overview of the research findings. *Alcohol and Alcoholism, 34*, 3–14.

Nicholas, S., Kershaw, C., & Walker, A. (2007). *Crime in England and Wales 2006/07: Home Office statistical bulletin* (4th ed.). London Home Office.

Parliamentary Office of Science and Technology. (2005). *Binge drinking and public health.* Retrieved December 5, 2008, from http://www.parliament.uk/parliamentary_offices/post/pubs2005.cfm

Room, R., & Collins, G. (Eds.). (1983). Alcohol and disinhibition: Nature and meaning of the link, *NIAAA Research Monograph* No. 12. Washington, DC: United States Government Printing Office.

This page intentionally left blank

Chapter **13**

Alcohol Use and Abuse in Germany

Changiz Mohiyeddini, MD, PhD,
Cecilia A. Essau, PhD, MA, HBA, and Regina Pauli, BSc, PhD

Germany is a federal parliamentary republic that includes 16 states, and the total area of the country is 357,021 square kilometers. Germany comprises over 82 million inhabitants, the largest population in the European Union (EU). With more than 10 million immigrants (almost 12.4% of the state's population), Germany is home to the third highest number of international migrants worldwide. Germany is the third largest economy by nominal gross domestic product worldwide and was classified in 2007 as the largest exporter of goods internationally. According to the Human Development Index (Human Development Report, 2007–2008), Germany has established a very high standard of living and dispenses a comprehensive system of social welfare and security.

As a consequence of World War II, Germany was divided in 1949 into two separate states along the lines of Allied occupation. After 41 years of division, the two states were reunified in 1990. West Germany was a founding member of the EU and, in 1999, adopted the euro (the common European currency). Germany's gross national income per capita is US$36,810 (World Development Indicators Database, 2007).

The chapter is organized as follows. We begin with a brief review of attitudes to and patterns of alcohol consumption in Germany and discuss the problems associated with alcohol consumption. Afterward, we will explore availability, advertising, and legal restrictions on alcohol consumption and will aim to explain the tactics used by the advertisement industry to attract drinkers. Finally, we will explain the restrictions and controls on alcohol advertisement.

ATTITUDES TO AND PATTERNS OF ALCOHOL CONSUMPTION

In Germany, for several decades now, the parameters of the debate as to whether alcohol is a cultural asset or a drug have been determined on the one hand by social policy and on the other by advertising for alcoholic beverages. In Germany, almost 40,000 to 73,000 people die each year directly or indirectly in connection with alcohol (Bühringer et al., 2000). Approximately 2.5 million people in Germany are addicted to alcohol (Bloomfield, Kraus, & Soyka, 2008).

According to the figures of the German federal police for 2006, more than 30 percent of all violent offences, 40 percent of all murders with robbery, 33 percent of all serious bodily harm, 38.5 percent of bodily harm causing death, and 40.5 percent of homicides happened while the offenders were under the influence of alcohol (Bundeskriminalamt, 2008). The economic damage caused by alcohol-related morbidity and mortality is estimated at between €5 billion and €40 billion (Bergmann & Horch, 2002), with the average estimation being €20 billion. Annually, the German national health system spends €150 million to €200 million treating almost 6,000 babies born with serious health conditions caused by alcohol abuse during pregnancy (Fischer, 1987).

Empirical results indicate that many factors influence alcohol consumption and foster the widespread prevalence of alcohol abuse (Martin et al., 2002). Lindenmeyer (2005) has identified five unwritten laws that affect and regulate alcohol consumption in German-speaking countries (Germany, Switzerland, and Austria): (1) "Regular alcohol consumption is normal," (2) "Alcohol is part of the game" (you can drink alcohol in almost any kind of social situation), (3) "Alcohol is good for you" (alcohol makes you humorous, chatty, easygoing, relaxed, self-confident, and sociable), (4) "Drink as much as your drinking partner" (this rule refers to the pure understanding of risk alcohol consumption and binge drinking among alcohol drinkers), and (5) "Drinking alcohol is a private affair" (nobody can interfere with your drinking behavior and habits).

PROBLEMS ASSOCIATED WITH ALCOHOL IN GERMANY

The long-term development of alcohol consumption shows, for the period from 1973 to 2001, an overall decline in the proportion of 12- to 25-year-olds who regularly drink alcohol. However, alcohol and drug consumption by children and adolescents in Germany is a growing problem. The number of young people who die because of alcohol abuse has significantly increased (Haustein,

Pohlmann, & Schreckenberg, 2004). Additionally, almost 30 percent of car accidents occurring under the influence of alcohol are caused by young people aged between 18 and 24 years. Furthermore, the figures of the German federal office for statistics shows a significant increase of 104 percent from the years 2000 (9,500 adolescents) to 2005 (19,400 adolescents) in the number of children and adolescents who were hospitalized because of alcohol poisoning (Statistische Bundesamt Deutschland, 2008).

The German federal office for health education (Bundeszentrale für gesundheitliche Aufklärung, BzgA) has shown that half of children up until the age of 12 have already had experiences with alcohol consumption. Among 16-year-olds, only 3 percent had never drunk alcohol, while the vast majority (78%) had repeatedly drunk alcohol (Bundeszentrale für gesundheitliche Aufklärung [BzgA], 2001). Fifty percent of young people up to the age of 14 years have already been drunk at least one time. This number rose to 83 percent among 16-year-old boys and to 77 percent among girls (Currie, Hurrelmann, Settertobulte, Smith, & Todd, 2000; Kraus, Heppekausen, Barrera, & Orth, 2004). At the ages of 16 and 17, drinking habits of young people become stabile. As in the subsequent years of life, 37 percent indicated alcohol consumption at least once a week, while 21 percent indicated rarely or never drinking alcohol (BzgA, 2001).

In comparison with other European countries, Germany is in a middle position in terms of experience with alcohol and the regular consumption of alcohol among young people. Recent findings on alcohol consumption among German youths in Europe (the European School Survey on Alcohol and Other Drugs, ESPAD) show that almost all students in Germany had an experience with alcohol during the last 12 months (93%), which is substantially higher than the average for all ESPAD countries (83%). Furthermore, the proportion of students who had been drunk during the last 12 months was higher (61%) than the average (53%). Also 77 percent of students in Germany had smoked in their lifetimes (66% average), and significantly more students in Germany had used marijuana or hashish (27%) than the average for all countries (21%). The use of illicit drugs other than cannabis was also higher than the average (10%, compared to 6%), and use of alcohol in combination with pills is more common in Germany (16%) than the average (7%). One study seemed to indicate the influence of German reunification on the drinking pattern in Germany among adolescents (Silbereisen, Robins, & Rutter, 1995): as a consequence of the open market that followed unification, the East German data showed a twofold increase in alcohol consumption from 1989 to 1990.

Among those who drink alcohol, between 4.3 and 9.7 percent meet the diagnostic requirements for alcohol abuse and dependence. According to the results

of the Early Development Stages of Psychopathology study (Holly, Türk, Nelson, Pfister, & Wittchen, 1997; Holly & Wittchen, 1998), 9.7 percent of 14- to 24-years-olds already have all the criteria for a diagnosis of alcohol abuse, according to the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (American Psychiatric Association, 1994), and 6.2 percent for alcohol addiction. Alcohol users who first consumed alcohol at the age of 14 years or younger were at higher risk for meeting a diagnosis of dependence and abuse compared to those who initiated alcohol use at 15 years of age or older. Results of the Bremen Adolescent Study (Essau, Baschta, Koglin, Meyer, & Petermann, 1998; Essau, Karpinski, Petermann, & Conradt, 1998) indicated that 6.7 percent of 12- to 17-year-olds have an abuse diagnosis, and 4.3 percent a dependency diagnosis. A high percentage of those who met the diagnosis for alcohol abuse and dependence also met the diagnosis for other psychiatric disorders. Alcohol use disorders were significantly higher in boys than in girls, and the disorders increased with age.

In addition to being common, alcohol abuse and dependence are frequently associated with a wide variety of problems for the adolescents themselves and their families as well as for society at large. Some examples of problems associated with substance abuse include increased risk for: motor vehicle accidents; consensual sexual behavior resulting in unwanted pregnancy or a high risk of HIV infection; academic underachievement; antisocial behavior; failing to fulfill major role obligations; and, recurrent social or interpersonal problems (Essau, Karpinski, et al., 1998). These results indicate that experiences with alcohol among young people in Germany are very common.

AVAILABILITY, ADVERTISING, AND LEGAL RESTRICTIONS ON ALCOHOL CONSUMPTION IN GERMANY

Converging empirical results show that media and advertisements are crucial predictors of underage drinking behavior, youths' preference for alcohol brands, and current and intended drinking behavior (Gentile, Walsh, Bloomgren, Atti, & Norman, 2001; Martin et al., 2002). By the same token, a considerable body of research during recent years indicates that underage drinking is a main causal factor of many serious problems, for instance, low school performance, early onset of sexual activity, unprotected sex, criminal behavior, sexual victimization, and risk taking (Austin & Knaus, 2000; Cooper, 2002; Fergusson & Lynskey, 1996; Lowry et al., 1994; Mullahy & Sindelar, 1989).

Hence the main targets of the final declaration of the European Ministerial Conference of the World Health Organization on Young People and Alcohol,

2001, were to create a substantial reduction in the number of young people who start consuming alcohol and also delay the onset age of drinking. These targets point to a number of issues and challenges in reducing the pressures on young people to drink that must be considered in national health education and health policy, namely, alcohol promotion, free distribution, advertising, and sponsorship and availability. The charter suggests that each member state should implement strict controls on advertising of alcoholic beverages and ensure that no form of advertising is specifically addressed to young people (e.g., through the linking of alcohol to sports).

According to Kunkel, Wilcox, Cantor, Palmer, and Linn (2004), there is undisputed social agreement that young people need special protection from the unrestrained efforts of the economic marketplace. The issues related to advertising are being increasingly globally recognized. Over the past few years, considerable effort has been made to investigate the impact of advertising and alcohol consumption, which consistently shows a significant correlation between positive evaluations of alcohol advertising and current or intended alcohol consumption (Austin & Nach-Ferguson, 1995; Casswell & Zhang, 1998; Martin et al., 2002; Wyllie, Zhang, & Casswell, 1998). Furthermore, and crucially, the results of the Enforcement of National Laws and Self-Regulation in Advertising and Marketing of Alcohol project indicate that alcohol advertisements are clearly related to positive attitudes and beliefs about alcohol among youths, resulting in an increase in the likelihood that young people will start to drink, the amount they consume, and the amount they drink on any one occasion. By the same token, an enormous body of empirical results indicates that alcohol advertisements increasingly target underage alcohol consumers (Buijzen & Valkenburg, 2002; Grube, 1993; Jones & Donovan, 2001; Waiters, Treno, & Grube, 2001), creating new low-cost products (e.g., designer drinks, alcoholic energy drinks, and premixed cocktails) using packaging designed to be perceived by young people as "in" or "cool."

TACTICS USED BY THE ALCOHOLIC BEVERAGE AND ADVERTISEMENT INDUSTRIES IN GERMANY TO ATTRACT AND KEEP DRINKERS

A substantial body of evidence shows that the drinking behavior of young people is affected by television advertising (Kunkel & Roberts, 1991; Young, 1990). Similar to other countries (Kunkel, 1990), television in Germany is also the predominant medium that the commercial industry has chosen for marketing products to young people. Comparing international advertisement expenditures for alcoholic beverages, Germany occupies third place (behind

the United States and Japan), spending almost €600 million in 2000 for alcohol advertisements (German Advertising Federation, 2006).

Haustein et al. (2004) analyzed the tactics used by the commercial and alcoholic beverage industries to attract and keep consumers. In this study, a sample of TV programs on eight major German TV channels were broadcast over a period of six months and evaluated (174 commercials and product sponsorships in total). The results show that compared to commercials for other alcoholic beverages, advertising spots for alcopops were highly oriented toward young people: they were more strongly characterized by the use of bright colors, youthful music, action, and innovation. On the basis of a content analysis, Haustein et al. concluded that one reason for the increasing popularity of alcopops is the concerted marketing of these products (see also Kraus, Heppekausen, Barrera & Orth, 2004): while in 1998, just 9 percent of 14- to 29-year-olds drank at least once per month (alcopops), the number was already 42 percent in 2002 (BzgA, 2003a), and among 14- to 17-year-olds, it was 48 percent (BzgA, 2003b). The results of Haustein et al. confirm the previous findings in other countries that indicate that young people like the style and image-oriented elements of alcohol television commercials that are delivered with humor and youth-oriented music, colors, and/or characters (Waiters et al., 2001).

Predominant in beer advertising, by far the most frequently advertised alcoholic drink, were images of fun, healthy, pleasure, and socialization with friends and camaraderie, whereas champagne and wine commercials addressed flirtation, love, romance, and a wealthy lifestyle (Aufenanger, Große-Loheide, Hasenbrink, & Lampert, 2002).

A further important result of the study of Haustein et al. pertains to the frequency of alcohol advertisement on Germen television: on average, 1.2 alcohol commercials are shown per hour. Thirty percent of all alcohol commercials are shown on German Sport TV. As previous research indicates, the repetitive character of alcohol advertisements influences the drinking behavior of young consumers (Martin et al., 2002), who believe in information that has been displayed repeatedly without questioning its substance and/or sincerity (Roggeveen & Johar, 2002).

RESTRICTIONS AND CONTROLS ON ALCOHOL ADVERTISING IN GERMANY

As already mentioned, in the EU, alcohol advertising targeting young people is controlled by means of rules suggested in the European Council Recommendation adopted on June 5, 2001, by the Health Ministry. The

members of the EU aimed in this recommendation to take collective action by providing protection for children and adolescents from exposure to alcohol promotion and sponsorship, warranting that alcoholic products do not target children and adolescents, controlling alcohol availability, promoting education through rising awareness of the impact of alcohol, and developing youth-specific health promotion programs. The education programs should support young people to learn and develop a healthy lifestyle, resisting social and the peer group's pressure to consume alcohol (Council of the European Union, 2001; Ugland, 2002). The council recommendation stipulates that national governments, the alcohol and advertisement industries, and retailers are called to ensure, among other things, that advertising does not encourage excessive consumption or disparage abstinence or moderation or link alcohol consumption to driving or create the impression that alcohol enhances physical or mental performance or fosters the sexual attractiveness.

Because of the noncompulsive character of the council recommendation, members of the EU can decide at the national level whether they approach the recommendation through legal, regulatory, or self-regulatory mechanisms (Council of the European Union, 2001). As Ugland (2002) pointed out, this recommendation has no legal character, but rather, it works politically and morally.

In terms of EU-wide legislation, the Television Without Frontiers (TWF) directive has been incorporated into national law. This directive was designed to harmonize regulations so that cross-border transmissions would not contravene national regulations.

In Germany, there is a variety of national restrictions and controls, and mixtures of the statutory and the self-regulatory. The alcohol and advertising industries in Germany argue that Germany's constitution emphasizes the right for everyone to express and disseminate his opinions freely in speech, writing, and pictures—and this includes opinions expressed in the form of advertising (Zentralverband der deutschen Werbewirtschaft, 2006). By voluntary agreement, most spirits are not advertised on TV. On other media, a voluntary code is in operation. The German Advertising Council, which is funded by 41 organizations allied in the German Advertising Federation (ZAW), first issued its Code of Conduct on Commercial Communication for Alcoholic Beverages in 1976. In 1986, the European Commission incorporated the code into the EU TWF directive. The scope of the code was extended by resolution of the ZAW member associations to include teleshopping in 1998 and means of communication in general in 2005. Furthermore, alcohol advertisements and sales are controlled by laws governing fair trade (Gesetz gegen den unlauteren Wettbewerb), food and drink (Lebensmittel- und Bedarfsgegenständegesetz),

and the law for the protection of children and youth (Jugendschutzgesetz and Jugendmedienschutz-Staatsvertrag).

Anyone who feels that advertising violates the German advertising code can complain to the German Advertising Council. The advertising council can also evaluate advertising without a complaint having first been submitted.

Haustein et al. (2004) investigated whether the advertisement industry in Germany considers the guidelines of the European Council Recommendation and the German Advertising Code. According to the results, more than 12 percent of alcohol commercials infringed on the rules of the German Advertising Council by showing excessive alcohol consumption, displaying a dangerous car driving scene, implying that use of alcohol is a coping strategy against social anxiety, and suggesting that alcohol consumption increases social and sexual success.

CONCLUSION

Our review of recent literature on young people in Germany has indicated that a high number of adolescents have had some experience with alcohol. The high rates of alcohol consumption may be related to German law and societal views about the use of alcohol in young people; that is, legal access to alcoholic beverages in Germany begins at age 16 and is accompanied by a widespread acceptance of alcohol use in this age group. This is in contrast to many other countries, where the age for legal access to alcohol is much older. For example, in the United States, the legal age for buying alcoholic beverages is 21 years.

To conclude, numerous studies and government reports have been conducted, which have provided us with information on the trends in alcohol use and abuse in young people. Ideally, such information should be considered in the design of interventions. However, hardly any studies have taken this scientific-based information into consideration in their treatment designs. In this respect, the gap between the practitioner's and the researcher's world is still very wide.

REFERENCES

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: Author.

Aufenanger, S., Große-Loheide, M., Hasenbrink, U., & Lampert, C. (2002). Alkohol Fernsehen Jugendliche. *Programmanalyse und medienpädagogische Praxisprojekte*. Berlin: VISTAS.

Austin, E., & Knaus, C. (2000). Predicting the potential for risky behavior among those "too young" to drink as the result of appealing advertising. *Journal of Health Communication, 5*, 13–27.

Austin, E. W., & Nach-Ferguson, B. (1995). Sources and influences of young school-aged children's general and brand-specific knowledge about alcohol. *Health Communication, 7*, 1–20.

Bergmann, E., & Horch, K. (2002). Kosten alkoholassoziierter Krankheiten. Schätzungen für Deutschland. *Beiträge zur Gesundheitsberichterstattung des Bundes*. Berlin: Robert Koch-Institut.

Bloomfield, K., Kraus, L., & Soyka, M. (2008). Alkoholkonsum und alkoholbezogene Störungen. *Gesundheitsberichterstattung des Bundes Heft 40*. Berlin: Robert Koch-Institut.

Bühringer, G., Augustin, R., Bergmann, E., Bloomfield, K., Funk, W., Junge, B., et al. (2000). *Alkoholkonsum und alkoholbezogene Störungen in Deutschland* (Schriftenreihe des Bundesministeriums für Gesundheit, Band 128). Baden-Baden, Germany: Nomos.

Buijzen, M., & Valkenburg, P. M. (2002). Appeals in television advertising: A content analysis of commercials aimed at children and teenagers. *Communications, 27*, 349–364.

Bundeskriminalamt. (2008). *Polizeiliche Kriminalstatistik*. Retrieved June 17, 2008, from http://www.bka.de/pks/

Bundeszentrale für gesundheitliche Aufklärung. (2001). *Die Drogenaffinität Jugendlicher in der Bundesrepublik Deutschland 2001*. Retrieved August 14, 2001, from http://www.bzgA.de/ studien/daten/stud.htm

Bundeszentrale für gesundheitliche Aufklärung. (2003a). *Bekanntheit, Kauf und Konsum von Alcopops in der Bundesrepublik Deutschland 2003. Ergebnisse einer repräsentativen Befragung*. Retrieved July 12, 2004, from http://www.bzga.de/

Bundeszentrale für gesundheitliche Aufklärung. (2003b). Bekanntheit, Kauf und Konsum von Alcopops bei Jugendlichen. Ergebnisse eine Repräsentativerhebung bei Jugendlichen im Alter von 14 bis 17 Jahren. Retrieved July 12, 2004, from http://www.bzga.de/

Casswell, S., & Zhang, J.-F. (1998). Impact of liking for advertising and brand allegiance on drinking and alcohol-related aggression: a longitudinal study. *Addiction, 93*, 1209–1217.

Cooper, M. L. (2002). Alcohol use and risky sexual behaviour among college students and youth: Evaluating the evidence. *Journal of Studies on Alcohol, 14*, 101–117.

Council of the European Union. (2001, June 16). Council Recommendation of 5 June 2001 on the drinking of alcohol by young people, in particular children and adolescents. *Official Journal of the European Communities, L 161*, 38–41.

Currie, C., Hurrelmann, K., Settertobulte, W., Smith, R., & Todd, J. (2000). *Health and health behaviour among young people*. Retrieved August 14, 2001, from http://www.ruhbc.ed.ac.uk/hbsc/

Essau, C. A., Baschta, M., Koglin, U., Meyer, L., & Petermann, F. (1998). Substanzmißbrauch und -abhängigkeit bei Jugendlichen. *Praxis der Kinderpsychologie und Kinderpsychiatrie, 47,* 754–766.

Essau, C. A., Karpinski, N. A., Petermann, F., & Conradt, J. (1998). Störungen durch Substanzkonsum bei Jugendlichen. *Kindheit und Entwicklung, 7,* 199–200.

Fergusson, D. M., & Lynskey, M. T. (1996). Alcohol misuse and adolescent sexual behaviour and risk taking. *Journal of the American Academy of Paediatrics, 98,* 91–96.

Fischer, A. (1987). Alkoholmissbrauch im Betrieb gefährdet Produktion. Milliardenverluste. *Industrie Anzeiger, 109,* 30–34, 76.

Gentile, D. A., Walsh, D. A., Bloomgren, B. W., Atti, J. A., & Norman, J. A. (2001). *Frogs sell beer: The effects of beer advertisements on adolescent drinking knowledge, attitudes, and behavior.* Paper presented at the biennial conference of the Society for Research in Child Development, Minneapolis, MN.

German Advertising Federation. (2006). *Alcohol and advertising. The facts.* Berlin: Zentralverband der deutschen Werbewirtschaft.

Grube, J. W. (1993). Alcohol portrayals and alcohol advertising on television. *Alcohol Health and Research World, 17,* 61–66.

Haustein, S., Pohlmann, U., & Schreckenberg, D. (2004). *Inhalts- und Zielgruppenanalyse von Alkoholwerbung im Deutschen Fernsehen.* Bonn, Germany: Bundesministerium für Gesundheit und Soziale Sicherung.

Holly, A., Türk, D., Nelson, C. B., Pfister, H., & Wittchen, H.-U. (1997). Prävalenz von Alkoholkonsum, Alkoholmißbrauch und -abhängigkeit bei Jugendlichen und jungen Erwachsenen. *Zeitschrift für klinische Psychologie, 3,* 171–178.

Holly, A., & Wittchen, H.-U. (1998). Patterns of the use and their relationship to DSM-IV abuse and dependence of alcohol among adolescence and young adults. *European Addiction Research, 4,* 50–57.

Human Development Report. (2007–2008). Fighting climate change: Human solidarity in a divided world. New York: United Nations Development Programme.

Jones, S. C., & Donovan, R. J. (2001). Messages in alcohol advertising targeted to youth. *Australian and New Zealand Journal of Public Health, 25,* 126–131.

Kraus, L., Heppekausen, K., Barrera, A., & Orth, B. (2004). *Die Europäische Schülerstudie zu Alkohol und anderen Drogen (ESPAD)* (Vol. 141). Munich: IFT Institut für Therapieforschung.

Kunkel, D. (1990). The role of research in the regulation of children's television advertising. *Knowledge: Creation, Diffusion, Utilization, 12,* 101–119.

Kunkel, D., & Roberts, D. (1991). Young minds and marketplace values: Research and policy issues in children's television advertising. *Journal of Social Issues, 47,* 57–72.

Kunkel, D., Wilcox, B. L., Cantor, J., Palmer, E., & Linn, S. (2004). *Report of the APA Task Force on Advertising and Children—Section: Psychological issues in the increasing commercialization of childhood.* Washington, DC: American Psychological Association.

Lindenmeyer, J. (2005). *Lieber schlau als blau. Informationen zur Entstehung und Behandlung von Alkohol und Medikamentenabhängigkeit.* Weinheim, Germany: Psychologie Verlags Union.

Lowry, R., Holtzman, D., Truman, B. I., Kann, L., Collins, J. L., & Kolbe, L. J. (1994). Substance use and HIV-related sexual behaviors among US high school students: Are they related? *American Journal of Public Health, 84,* 1116–1120.

Martin, S. E., Snyder, L. B., Hamilton, M., Fleming-Milici, F., Slater, M. D., Stacy, A., et al. (2002). Alcohol advertising and youth. *Alcoholism: Clinical and Experimental Research, 26,* 900–906.

Mullahy, J., & Sindelar, J. (1989). Life cycle effects of alcoholism on education, earnings, and occupation. *Inquiry, 26,* 272–282.

Roggeveen, A. L., & Johar, G. V. (2002). Perceived source variability versus familiarity: Testing competing explanations for the truth effect. *Journal of Consumer Psychology, 12,* 81–91.

Silbereisen, R. E., Robins, L., & Rutter, M. (1995). Secular trends in substance use: Concepts and data on the impact of social change on alcohol and drug abuse. In M. Rutter & D. J. Smith (Eds.), *Psychosocial disorders in young people: Time trends and their causes* (pp. 490–543). Chichester, England: John Wiley.

Statistische Bundesamt Deutschland. (2008). Alcohol diagnosis: Strong increase in the number of young hospital patients. Press release No. 240 / 2007-06-13. Statistisches Bundesamt Wiesbaden.

Ugland, T. (2002). *Making policy in the European Union: The Council Recommendation on the drinking of alcohol by young people.* Oslo: Norwegian Institute for Alcohol and Drug Research.

Waiters, E. D., Treno, A. J., & Grube, J. W. (2001). Alcohol advertising and youth: A focus group analysis of what young people find appealing in alcohol advertising. *Contemporary Drug Problems, 28,* 695–718.

Wyllie, A., Zhang, J. F., & Casswell, S. (1998). Responses to televised alcohol advertisements associated with drinking behaviour of 10-17 year olds. *Addiction, 93,* 361–371.

Young, B. (1990). *Television advertising and children.* Oxford: Clarendon Press.

Zentralverband der deutschen Werbewirtschaft. (2006). *Alkohol und Werbung. Fakten als Position der Werbewirtschaft.* Berlin: Zentralverband der deutschen Werbewirtschaft.

This page intentionally left blank

Chapter **14**

From British India to the Taliban: Lessons from the History of the Heroin Market[1]

Kathryn Meyer, PhD

In September 1997, Pino Arlacchi became head of the United Nations International Drug Control Program. He had impressive credentials for an international drug czar: he began his career as a professor of sociology. His excellent books about the Italian Mafia had brought him recognition as a leading expert on organized crime; unlike most scholars, he had put his knowledge into action; he had joined the Italian Parliament and had led an attack against the same criminals whose organization he had researched. By all reports, he was successful in his efforts to bring the Sicilian Mafia under control (see *Financial Times* [London], July 17, 1997; *New York Times*, June 23, 1997).

From his new position Arlacchi announced the beginning of a ten-year plan to eradicate narcotics worldwide. He then opened discussions with the Taliban, the revolutionary Islamic group in control of war-torn Afghanistan, a major poppy-growing area supplying today's heroin market. To his surprise, the Taliban leadership indicated a willingness to work with the United Nations. Thus, in mid-November 1997, Arlacchi went to Afghanistan to discuss concrete plans to provide funds for irrigation projects and a textile factory, all to be built in a key opium-growing area. His trip provided the Taliban with an opportunity to enhance their international image, tarnished by numerous accounts of religious intolerance and poor treatment of women. Because he was criticized in some circles for making compromises with repressive regimes, Arlacchi was careful to point out the positive contributions UN aid would make, including plans for a wool-weaving plant that would employ women (*New York Times*, October 25, 1997, November 14, 1997; Associated Press, November 27, 1997; Lexis/Nexis).

Arlacchi's Afghan trip was the forerunner of a larger global plan to eliminate drug crops through "alternative development." "We would propose an alternative way of life," he told Christopher Wren of the *New York Times*. "They can be rich peasants if they grow opium, but they can die if they don't have roads and hospitals." His plan was not all carrot and no stick, however; it called for enforcement as well. He summarized it as "alternative development, eradication and law enforcement." The program was endorsed by the Clinton administration, and General Barry McCaffrey, the reigning U.S. drug czar, applauded Arlacchi's "focused, high-energy leadership" (*New York Times*, November 25, 1997; December 3, 1997; June 7, 1998).

Yet, in spite of the public professions of compliance by the Taliban, poppy production in Taliban-controlled areas continued. The 1998 harvest was about as large as that of the previous year. In response, Arlacchi told nations contributing aid to Afghanistan that he was prepared to use the earmarked money for border patrols rather than development. Noorullah Zadran, a Taliban spokesman, replied, "God knows we tried. There is a difference in cultures. There is a different perception" (*New York Times*, July 17, 1998). Afghanistan is indeed a place with a distinct culture; however, both Zadran and Arlacchi were wrong if they thought that this made the Afghan situation unique. These two men and their respective organizations fit into a pattern that has been repeated throughout the first hundred years of the illicit heroin market.

Over the course of the twentieth century, the major areas of both poppy production and heroin consumption shifted. Before the Communist revolution of 1949, China was the world's major consumer; since the early 1950s, the United States and Europe have assumed that role. Early in the nineteenth century, heroin made only small inroads into a market dominated by smoking opium; today, it is the most visible illicit opiate available. The opiate market has been defined by the difficulties of delivering a desirable product through an increasing network of international regulations. These laws, rather than ending the traffic, became part of the risk of engaging in a highly profitable trade. Looking at the development of this enterprise may provide some lessons for Mr. Arlacchi.

POPPIES, OPIUM, AND ASIA

The earliest international sanctions against narcotics came in China, only eight years after the invention of heroin. Opium use and cultivation had been legal in that country from 1858 to 1906. Although, at that time, opium smoking was considered to be a Chinese vice, the poppy plant was not native to Asia, nor was the fifty-year-long legality of the drug a policy willingly adopted by

the Chinese government. Legalization was accepted by a weakened Chinese government after China's bold attempts to force British smugglers to withdraw from its shores had enmeshed it in a losing struggle with the British navy. The opium problem was a nagging reminder of a larger attack on the authority of the Chinese state by foreigners.

The best opium came from British India. In 1905, when British diplomats suggested the possibility of opium restriction, the Chinese government responded positively. Statesmen representing the emperor knew they required British participation for any domestic program to prove effective. In 1906 China's opium eradication began with edicts against the cultivation and use of opium in China. This was followed by a British measure restricting exports of opium from India to China. The Qing (Ch'ing or Manchu) dynasty government that began this ambitious project had only five years left in its 270-year reign. On its last legs, it faced general public disorder, economic decline, and increasing foreign encroachment. Yet, as government officials began closing down opium dens and plowing under poppy fields, they were met with public enthusiasm and surprising success (see *North China Herald*, February 8, 1907, April 26, 1907; Hosie 1914, v. 2, 191; and Riens 1991).

In 1911, the Qing dynasty was overthrown, and in 1912, a new republic was declared. Best known in the West for its figurehead leader Sun Yat-sen and his Guomindang, or Nationalist party, the republic was in its early years dominated by the military strongman Yuan Shikai, who immediately began chipping away at the constitution and attacking those who opposed him. Yet, in spite of political conflicts between Sun's democratic ideals and Yuan's authoritarian ambitions, opium reform moved forward after only a short pause. Part of the motivation behind the success was the determination of Chinese patriots to rid their country of a problem that they saw as debilitating to the nation, a plague brought in by foreigners (*North China Herald*, October 12, 1912; Jordan to Grey, March 5, 1912, FO 10168/12644, and Jordan to Grey, January 22, 1913, FO 10481/6308, in Great Britain, Foreign Office 1974).

Narcotics regulation began in this climate of political breakdown, promoted by people with larger political agendas. Suppliers and addicts adapted to the new conditions as best they could. Legitimate opium merchants shifted their business arrangements. Some wealthy addicts bought up the last of the legal supply, while many others tried cures. There were different remedies, however. By 1917, when these first reform campaigns collapsed, "white drugs," including heroin, appeared in China and were advanced as cheaper, more easily transportable alternatives to opium and sometimes as a cure for the opium habit. Thus the introduction of heroin into the Chinese opiate market was dictated by the necessity of circumventing legal barriers. David Courtwright notes that

the same process occurred in America after passage of the Smoking Opium Exclusion Act of 1909 (Courtwright 1982, 83).

THE CHINESE OPIUM BUSINESS TAKES A NEW DIRECTION

The Taliban assured Mr. Arlacchi that the use of intoxicants is contrary to Islam and that, therefore, their own beliefs required them to end Afghan trafficking. At the same time, they obviously relied on opium profits to continue their war efforts, making them appear to be hypocrites of the first order. In fact, the Taliban were responding to a complex political scenario strikingly similar to an episode that occurred in China only ten years after the first opium bans went into effect. The smuggling case associated with the Righteous Yunnan Uprising of 1916 marked the breakdown of the first successful opium reform and demonstrates how illicit delivery systems can become political tools.

One of the most surprising victories during the ten-year opium reform in China came from Yunnan Province. Yunnan shares a border with both Burma and what was then French Indochina, just north of the area known today as the Golden Triangle. Because it had been established as a commercial poppy-growing area during the fifty years in which opium was legal, it came as a surprise to reformers when reports appeared in 1909 that Yunnan was on the road to opium eradication. Local officials were thorough, even giving thought to what we now call crop substitution. Change proceeded despite the obstacle that while opium was moved easily over the rugged Yunnan terrain, rice did not have such an agreeable portability. And when a bumper crop of rice glutted the local market in 1914, officials recommended producing rice wine, to help peasants stave off the temptation to resort to their opium standby (Hosie 1914, v. 2, app. 2; Butler to Grey, February 9, 1910, FO 9392/9773, Great Britain, Foreign Office 1974; *North China Herald,* October 10, 1914).

Then, in 1916, Yunnan became the base of operations for a group of patriotic soldiers who opposed Yuan Shikai. For two years after the 1911 revolution, Chinese politicians had tried to create a constitution and convene a parliament. Yuan, however, dismantled those attempts and became more of a dictator. With each step toward authoritarian rule, Yuan alienated more of his supporters. The last straw came in 1915, when he cast aside all pretense of republicanism and proclaimed himself emperor of China. Yuan's power came from his control of the Chinese army. Many of his officers had supported the revolt against the previous imperial house. The absurdity of Yuan's imperial ambitions made these once-loyal supporters back away from him. Cai E was one of these officers. From his stronghold in Yunnan Province, he formed the Protect the Nation Army and led a revolt against Yuan (Yu 1966 [1917]; Zhang 1986).

Cai E was from a gentry family. He had given up scholarship and trained for a military career because he feared the weakness of China in the modern world. He was ascetic and dedicated to the republican cause. In December 1915, the National Protection Army began an armed revolt, as other officers declared themselves independent of Yuan's erstwhile empire. Yunnan began to attract patriots from all over China, including some members of Sun Yat-sen's Guomindang. In the face of such overwhelming opposition, Yuan Shikai gave up his imperial scheme in May 1916. He died in June 1916 (Ch'en 1972).

This military exercise, though brief, had been expensive. During the struggle with Yuan, the Yunnan patriots remained in contact with sympathetic groups throughout China, including the Guomindang in Shanghai, which sent much needed funds. The Guomindang, in turn, relied on some of the more unsavory characters in Shanghai for fundraising. Shanghai Municipal Police blotters were filled with complaints about the strong-arm tactics used to raise money for the revolt. Also during this period Yunnan opium began flowing once again onto the China market (Shanghai Municipal Police, 1916).

The short revolt and Yuan's subsequent death left a power vacuum in China. Cai E's generals, hailed as heroes, began to plan a greater national role for themselves in the aftermath of victory. In the summer of 1916, the many factions that had united against Yuan came together in Beijing to plan China's political future. Yunnan sent a delegation that left the province on June 23. The generals traveled by train south through French Indochina to Haiphong, where they caught a ship to Shanghai (Figure 14.1).

The Yunnan party hoped to use the visibility they had gained during the war to move into national prominence. To this end, they determined to establish a partisan newspaper and publicity office in Shanghai. They were, however, short on funds, and so they turned to smuggling. Having acquired a supply of opium that had been confiscated by Yunnan police during the earlier poppy eradication campaigns, they loaded it into the trunks that would accompany them on their journey. They felt safe because a prominent member of the old parliament accompanied them, but just to be sure, they packed opium in his trunks as well (Zhang 1986; *Minguo Ribao* [Republican Daily], August 9–23, 1916; *Shi Bao* [Eastern Times], August 9–23, 1916; *North China Herald,* August 9–23, 1916).

Shanghai was the best place from which to move opium onto the Chinese market. As the entrance to the Yangzi (Yangtze) River, it provided access to trade routes running through the country, making it the entrepreneurial center of China. Shanghai also had a significant foreign presence and had been fragmented politically into a French concession; the International Settlement, where British and other foreign nationals lived; and a large, sprawling Chinese city. Each area had its own courts and police jurisdictions, making law enforcement difficult under the best of conditions (Zhang 1985).

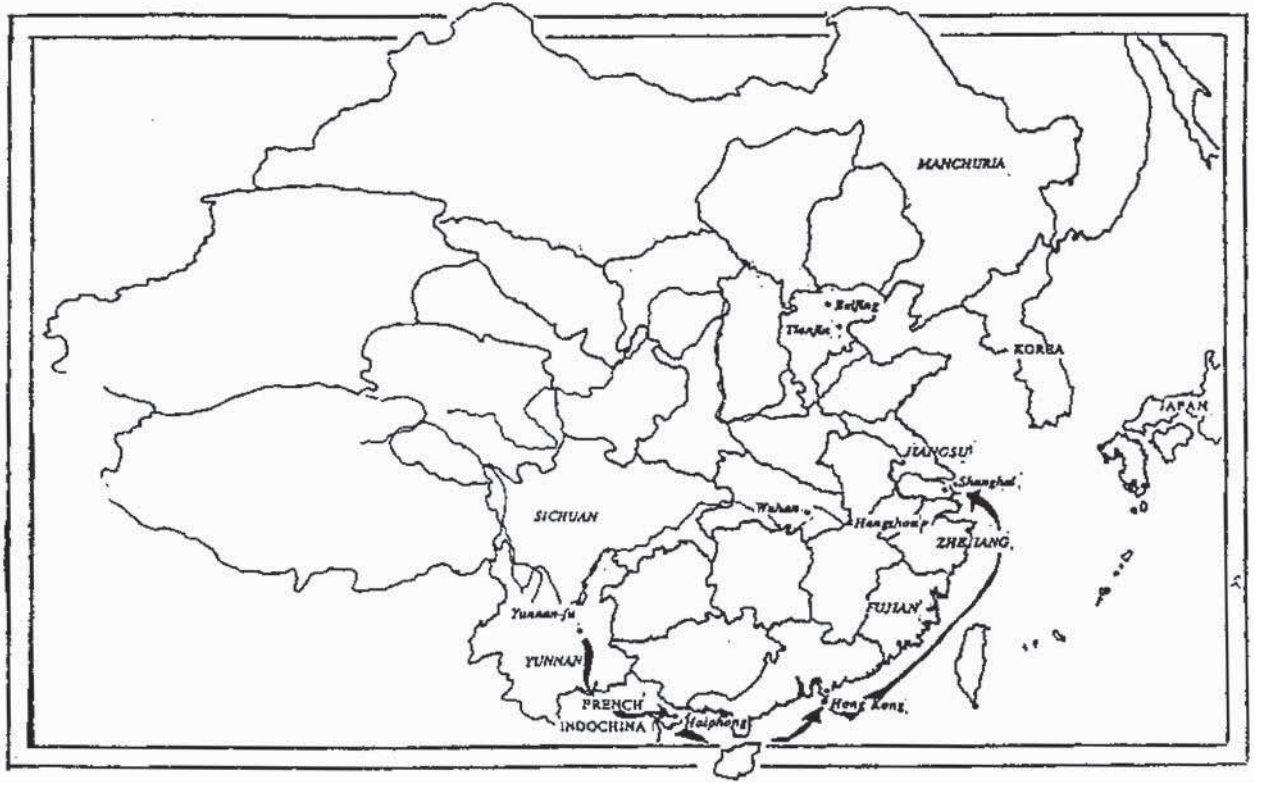

FIGURE 14.1 Route traveled by the Yunnan patriots and their opium-laden baggage.

The opium-laden trunks, sixty in all, easily cleared customs because the Yunnan generals had political connections. The men and their luggage then went to a Chinese inn in Shanghai's International Settlement. But the next evening international police raided the hotel and confiscated four opium-filled trunks. On the following day police traced an additional twenty trunks to the office of a prominent Chinese official, the same man who had signed the customs waiver. When the case reached its conclusion, three members of the Yunnan party were convicted of smuggling, and several Chinese officials were so badly compromised that their careers ended (*Minguo Ribao*, August 22, 1916).

Cai E was never implicated in the scandal, and he probably knew nothing about the opium scheme. During the campaigns he was a busy man, and a sick one. While his subordinates traveled to Shanghai to further their careers, he went there separately, seeking medical attention. Cai's subordinates were more practical and ambitious men than he. Tang Jiyao, the second in command, was very much implicated in the opium scandal. After the fiasco in Shanghai, Tang returned to Yunnan, where he retreated from national politics, making the province into his personal power base. By the 1920s, it was once again a major opium-producing area.

The Yunnan case established a pattern that repeated itself throughout the century, even as heroin replaced opium. The source of opium was in an area with a population poor enough to profit from such labor-intensive endeavors as poppy cultivation and opium manufacture and smuggling. More important, the undertaking was protected by ambitious men willing to make short-term compromises for larger political goals. Putting ideals into practice takes revenue. Many of the Yunnan leaders were against opium because it tarnished the republic, yet the state of civil war made the situation too complex for them to entirely control. Can we call these men simple hypocrites, or was something more complicated going on?

The Yunnan uprising of 1916 solved none of China's problems; Yuan's collapse left a power vacuum that was filled by contending military strongmen, commonly called the warlords, most of whom followed the Yunnan pattern and relied on opium funds. These warlords have such a bad reputation that it is well to recall that the first armed military revolt was filled with promise and is still referred to as the Righteous Yunnan Uprising. In the 1920s and 1930s, this Chinese civil war continued. It changed direction in 1927 when the Guomindang nationalists eclipsed the warlords and fought the Chinese Communists, while facing a serious Japanese threat. Opium, and increasingly, heroin, thrived in this atmosphere.

THE BEGINNINGS OF INTERNATIONAL REGULATION

Pino Arlacchi brought to his career a passionate dedication that transcended the mere filling of a job description. This animated enthusiasm is a requirement for anyone hoping to create successful international reform. Certainly the man who played a pivotal role in the 1920s League of Nations antiopium efforts was equally passionate. Sir Malcolm Delevingne did not invent the international regulatory system, but the force of his personality gave it direction.

International efforts to regulate narcotics traffic began in the first years of the twentieth century. The encouraging signs coming from the Chinese reform effort of 1906 coincided with American legislation to curb narcotics. In the same year that China promulgated its antiopium edicts, the U.S. government passed the Pure Food and Drug Act, the first of its many prohibitionist laws. Americans also were instrumental in calling the first international narcotics conference, convened at Shanghai in 1909, to discuss the issue of opium smoking throughout East Asia. Follow-up conferences at The Hague in 1911–1912, 1913, and 1914 resulted in more substantial agreements that became the basis for League of Nations initiatives (Musto 1973, 24–53; Taylor 1973, 47–122).

The results of these first steps toward international opium control made the Chinese drug market change, rather than disappear. As supplies of both Indian and domestic opium shrank, the skyrocketing price of smoking opium induced many Chinese to satisfy their cravings through heroin. More powerful than opium, it was sometimes injected with a hypodermic syringe but more often was smoked, just as opium had been. Other commodities appeared as well. Red Pills and Gold Pills—capsules containing morphine that had been introduced as cures for opium addiction—soon became popular as cheap opium substitutes. During the ten years of successful opium suppression, a market for refined opiates developed in China. After 1917, when smoking opium became plentiful in China again, heroin use continued, especially among the urban lower classes (Public Record Office, London [hereafter, PRO], "Memorandum Respecting the Opium Problem in the Far East," August 10, 1929, 17–18; "Smuggling Opium into China," *Times* [London], March 27, 1920; Ah Nan 1937).

At first, heroin was not manufactured in China. A few European, American, and Japanese pharmaceutical firms produced the entire world's supply. After World War I, opium control measures expanded to address the additional problem of narcotic substitutes. In 1921 the League of Nations created the Advisory Committee on Traffic in Opium and Other Dangerous Drugs, more commonly known as the Opium Advisory Committee (OAC). Between 1925 and 1936, it produced systems to control narcotics at their source. One such arrangement

was the creation in 1925 of a certificate system to track the international movement of narcotic drugs. The purpose of the agreement was to eliminate the excessive production of narcotic drugs, which resulted in diversion of a substantial quantity into the illicit traffic. In addition to formulating conventions, the OAC did valuable work in less visible ways. It gathered and shared information about the "hot spots" in the drug traffic, and it put political pressure on supply nations, albeit with varying degrees of success (Parssinen 1983, 144, 151; "Foreign Office Minutes 1920," PRO FO 371/ 5307/117–20; "Conference on the Restrictions of Opium," July 19, 1920, PRO FO 371/5307;107–08).

The idea for the OAC came from the British government, and specifically from Malcolm Delevingne, who, as Home Office Undersecretary, had responsibility for the drug traffic. Like Pino Arlacchi, he made elimination of narcotics his life's goal. He became the driving force behind the League's efforts. During the 1920s, information that he received about the sources and movement of narcotics led him to criticize certain European governments and firms for what he considered to be opportunism in finding and using loopholes in international regulations. An instance of this was his censure of the Swiss government's tolerance for the way Hoffmann La Roche slipped around the irregular European laws. This attitude, he said, demonstrated that they "do not take into account the character of the proceedings of the firm from a moral point of view" ("Memorandum Respecting Traffic in Opium," 1920, PRO FO 371/5308/233, 238–41; M. Delevingne to Foreign Office, February 27, 1926, PRO FO 371/11713/156). These efforts had their effect, although not entirely along the lines that Sir Malcolm had envisioned.

A CRISIS IN HEROIN SUPPLY

As a result of the efforts of Delevingne and the OAC, and despite footdragging by certain governments, the situation changed rapidly in Europe by the late 1920s. Enactment of the certificate system, universal enforcement of strictures against trafficking in western Europe by 1930, and the curtailment of production in European factories by 1932 had reduced legitimate opiate production to less than half of what it had been in 1928–1929. This crisis of supply forced a restructuring of the entire industry.

One man whose career demonstrates the changes taking place in Europe is Elie Eliopoulos, a Greek national operating out of Paris. From 1928 to 1931 he sold morphine and heroin to Tianjin, China. He began his career just as it was becoming difficult for illicit traders to make legal purchases of European narcotics. British and American factories were closed to them, and Swiss and German manufacturers were beginning to feel government pressure. However,

a few French factories continued to supply the market. Eliopoulos established connections with two of these—the Comptoir des Alcaloides and the Societe Industrielle de Chimie Organique. In 1930, when the French government passed stronger legislation, restricting narcotics trading licenses to only fifteen or sixteen reputable firms, Eliopoulos adapted. He turned to Istanbul, where his old suppliers opened a factory called ETKIM (Eliopoulos 1932; Moses 1933).

Eliopoulos needed political protection after January 1929, when the French laws took effect. Early in his career, he had struck a bargain with an Inspector Martin of the Paris Prefecture of Police, who supplied protection in return for money. Eliopoulos at first paid Martin 5,000 francs per month; this amount doubled as the business flourished. Eliopoulos could afford even this higher business expense, for morphine and heroin purchased in Europe for £40 or less per kilo could be sold in Tianjin at £70 or more per kilo. Monthly receipts of £21,000 yielded a gross profit of about £9,000 on the Tianjin business alone. From Eliopoulos's perspective, protection was a manageable business expense. Martin protected Eliopoulos until a Surete investigation in 1931 exposed Eliopoulos and made the situation untenable (Eliopoulos 1932).

Eliopoulos's trade with China was not his only narcotics business. Contrary to his profession, "by the memory of my father and all else that is holy," that he never sold drugs to America, he was a regular supplier of Jacob Polakiewitz, also known as Jack Paull, then the largest exporter of narcotics to the United States (Eliopoulos 1932; Moses 1933). Until the 1950s, however, this American market remained secondary to China.

Eliopoulos flourished during a period of crisis and transition in the illicit narcotics industry. At the end of his four-year career, he described important changes taking place in the world market. As restrictive measures in France, Germany, and Turkey made it difficult to divert large quantities of narcotic drugs from European pharmaceutical houses, Asian traffickers had begun to use Chinese opium to manufacture their own narcotic drugs (Eliopoulos 1932, 16–17). It would be Chinese gangsters and Japanese adventurers who would actively develop the heroin market in the 1930s.

BLACK INTO WHITE

After ten years of successful opium reform in China, smoking opium returned to the market, but now it was supplied by smugglers and the price was considerably higher. Poorer addicts, who turned to morphine and heroin, accounted for the demand that Elie Eliopoulos supplied in the 1920s. As Eliopoulos himself observed, however, by the early 1930s, Asian chemists were

learning the secret of turning black (opium) into white (morphine and heroin). And they had developed a better delivery system.

During the years in which Eliopoulos and other Europeans sold heroin to China, the Green Gang of Shanghai rationalized the opium distribution network by perfecting the kind of political connections the Yunnan opium group had tried to create. The Green Gang was well placed to step into the business. It had grown with the port, feeding on protection, gambling, labor racketeering, and prostitution. The gang controlled the wharves of Shanghai, an advantage for smuggling. It was a tip from a Green Gang member to the Shanghai police that had ruined the plans of the Yunnan group, whose biggest mistake was encroaching on someone else's territory (Zhang 1986).

The introduction of opium profits after the breakdown of reform made the 1910s contentious, as Green Gang factions fought over the lucrative trade. Only in the 1920s did three men known as the Three Big Shots—Huang Jinrong, Zhang Xiaolin, and Du Yuesheng—put together what would become in the 1930s a financial empire including both illicit and legitimate enterprises. They would create the kind of syndicate that their American contemporary Al Capone only briefly visualized before his collapse. Huang Jinrong brought with him to this enterprise his Shanghai police connections, and Zhang Xiaolin, his influential friends in the military.

Du Yuesheng, the kingpin of the Green Gang, is as famous to the Chinese as Al Capone is to Americans. Du was able to expand his smuggling empire through building on political connections. His ultimate triumph came in 1927, when the Green Gang helped Chiang Kai-shek purge the Guomindang party of Chinese Communist members in a bloody massacre known as the white terror (Isaacs 1951, 143–85; Marshall 1977, 29–35). After this initial event, Du's opium money contributed to the Nationalist war chest. In return, Du was able to work in Shanghai with relative freedom from police harassment (Xiao 1996).

Du was a diplomat. He ended the violent conflicts that marked the early days of the illicit Chinese opium market through negotiation as much as through force. From 1920 on, he made peace with every military strongman in the Chinese civil war who ventured into or around Shanghai by negotiating agreements that could be mutually profitable. These early deals employed the kind of pure graft exchanged between Eliopoulos and his Inspector Martin. When Du brokered a deal with Chiang Kai-shek, it was made on a pattern already established. Yet Chiang was not an ordinary warlord. He had a vision of national unity that was frustrated by the continuing Communist resistance (Martin 1996, 51–109; Mei and Shao 1987, 50–69).

Scholars have been both kind to and critical of Chiang Kai-shek. He has been praised by some as a nationalist hero and reviled by others as a narrow-minded bully. Those who attack him are quick to point out his alliance with Du; those who honor him respond that Du used his gang to work against the Japanese army during World War II. The truth of the situation is not as simple as either side maintains. Chiang Kai-shek was no friend of the narcotics traffic. He was an abstemious man who thrived under military discipline. But, like the Taliban, he faced a political reality so complex and expensive that his attitudes about drug regulation were easily compromised. In the 1930s, he faced both internal conflict and foreign invasion. He needed money, and he needed information.

One way of appreciating the revenue-generating power of opium at that time is to follow the change in its cost along the Yangzi River from Chongqing, deep in the opium-growing region of Sichuan, to Shanghai, on the coast. In Chongqing the cost of prepared opium was $1.50 per ounce; in Yichang it cost $2.00; in Hankou, $2.80; and in Shanghai, $4.50.[2] These price increases came from taxation. In 1932 the American consul Walter A. Adams conservatively estimated the total revenue produced by the Chinese opium traffic at $300 million per year. Over the decade of the 1930s, the nationalist government initiated opium monopoly schemes, which served to bring the traffic under government control. The profits went into the Agricultural Bank of China, where they funded intelligence operations (Stilwell 1935, 15; Adams 1934; Xiao 1996, v. 20, 609–17).

Du benefited from his alliance with Chiang Kai-shek. Yet there were times when his ambitions clashed with Chiang's sensibilities or with the plans of others also backed by Chiang. One example occurred in 1932, as Du was expanding into morphine and heroin manufacturing. Chiang gave Du a quantity of confiscated opium to make into medical morphine. Du created a processing plant to make the morphine, but he also secretly continued to operate the facility to make illicit heroin from opium that he received from another source. When Chiang learned of the extended operation, he sent police to raid the factory. It took several weeks of negotiation for Du to get himself back in Chiang's good graces.

Why did Chiang decide to raid this factory? There were several explanations at the time. One claimed that Chiang could tolerate opium but would draw the line at morphine and heroin. A second opinion ventured that Dai Li, the head of Guomindang intelligence and a beneficiary of narcotics funds, wanted more direct control of the proceeds of the factory for himself. After this short rupture, Du was able to talk his way back into favor, and he branched into the heroin market as well (Shanghai Municipal Police, November 25, 1933, D 5645; and February 5, 1940, D 9319).

Du clearly was taking advantage of the opportunity presented by the decreasing supply of European opiates. He inherited Eliopoulos's mantle, stepping into the market vacated by European sellers. What Eliopoulos may not have foreseen was that in the 1930s, some Chinese-made morphine and heroin would begin to penetrate into the American market. One exporter who worked this route was Paul Crawley.

Crawley began his career in the California motion picture industry. He first went to Shanghai as a film distributor but later moved into his own variety of the import-export business. Crawley brought pianos and slot machines into China and sent heroin back to his brother in Los Angeles and to a gang of accomplices in San Francisco. Influential friends helped him bring the drug out of China. H. O. Tong, for instance, was one of his Shanghai business associates. A good person for a smuggler to know, Tong worked in the Shanghai Customs Bureau and was a close friend of Chiang Kai-shek's brother-in-law T. V. Soong (Shanghai Municipal Police, November 12, 1931, D 3057).

The relationship between Du and Chiang Kai-shek was mutually beneficial. Chiang continued to make efforts to control opium. In 1935 his government began a six-year plan to eliminate opium use. Du was named to the Shanghai Municipal Opium Suppression Committee. When we consider the pressures Chiang faced in 1935—Communist insurrection, Japanese invasion, opposition from factions within his own party—the appointment seems less a cynical or corrupt calculation than a rational opportunity to increase his control over strong warring forces. If we apply this lesson to the Taliban, we may be able to speculate that they faced a similar situation. Putting questions of revenue aside, the opium producers whom the Taliban were trying to control most likely were factionalized political actors in their own right. This may be part of the meaning of the "cultural differences" to which Noorullah Zadran alluded.

JAPAN CAPTURES THE NARCOTICS MARKET

Although Du Yuesheng ventured into heroin and morphine manufacture, it was his Japanese competitors who became the major producers of opiates for China in the 1930s. From the first, Japanese smugglers had been involved in bringing European pharmaceuticals to China, as can be seen in the statistics. Between 1898 and 1907, Japan imported an annual average of 20,000 ounces of morphine, which represents the standard of morphine consumption for legitimate purposes in the country. Between 1915 and 1920, the average increased to 495,000 ounces of morphine, about twenty-five times the legitimate amount ("Memorandum from the Anti-Opium Association," August 10, 1920, Foreign Office 317/5307/211; "Foreign Office to Board of Trade,

March 23, 1921, Foreign Office 371/6593/162). When European supplies dwindled in the 1930s, Japanese manufacturers picked up the slack.

Until 1895, Japan had little experience with opium. In that year, however, Japan acquired the colony of Taiwan, which had a rich heritage of opium use. Faced with the sudden need for a practical opium policy, Japanese decision makers established a government opium production bureau, issued licenses for existing smokers, and taxed sales, hoping to control and eventually to discourage the use of opium. The architect of this policy was Goto Shimpei, who, in 1895, was the head of the National Board of Health (Ryu 1983, 3–35).

Goto was an unlikely person to become entangled in international narcotics. He was born to a poor samurai family in 1857 and grew up during the excitement of the overthrow of Japan's feudal order. He received a degree in Western medicine, and his articles on public health earned him official recognition and an invitation to help create a modern public health service. Like Sir Malcolm Delevigne and Pino Arlacchi, Goto was a man with a larger social vision. He called his program "biological colonial management" (Tsurumi 1965, v. 1, 872). It included narcotics control.

Through the establishment of the opium monopoly, Taiwan, which initially had been an expensive drain on the Japanese budget, became self-supporting. The policy of gradual withdrawal, along with its monopoly apparatus, became the model used by the Japanese colonial system as it extended through Asia. Goto rose to become a politician of the first rank in early-twentieth-century Japan, serving as minister of foreign affairs, minister of home affairs, and mayor of Tokyo. He was a man of standing and integrity who never thought of himself as encouraging the international opium trade. Yet the system he fashioned leaked at the seams, creating opportunities for traffickers.

As Japan's Asian empire grew, its commercial outposts in and around China also served as staging areas for traffickers. Taiwan became a center of smuggling into south China. Korea, occupied by Japan after 1905 and annexed after 1909, played a similar role in the north. The Japanese presence also penetrated China's sovereign territory, through concession areas and railways. North China became a jurisdictional patchwork in which smugglers could work with ease because, as Japanese subjects, they had special treaty rights that rendered them immune to Chinese law (Minami Manshu Tetsudo Kabushiki Kaisha, Kaisai Chosakai Dai Go Bu 1934).

Periodic scandals exposed this situation. One such drama involved Hoshi Hajime, founder and president of Hoshi Pharmaceuticals. Hoshi came to the narcotics business in 1914, when, through the recommendation of Goto, he received a monopoly license to produce morphine for the Taiwan colonial

administration. By 1921 British authorities suspected that Hoshi's firm was supplying to sources other than the legitimate markets. The British consul in Taiwan estimated that 3,500–4,000 *jin* (4,655–5,320 pounds) of morphine from the Hoshi Pharmaceutical Company reached south China annually (C. Eliot to Lord Curzon, February 26, 1921, PRO, F0371/6594/10-1). These suspicions appeared to have been well founded when, after pressure was put on the Japanese government by the British consular service, a Japanese customs agent discovered Hoshi's opium on a ship headed for Vladivostok without proper papers. Further investigations produced enough evidence that Hoshi had to stand trial for misconduct and was acquitted only on appeal (Hoshi 1971; Hoshi 1926; "Memorandum, Opium Scandal," Japan Chronicle, May 17, 1925, Pro FO 371/12527/108–12; P. Butler to Sir John Tilley, October 16, 1926, PRO FO 371/11714; FO 371/9248/141–47).

White drugs and smoking opium moved between Taiwan and the China coast through the agency of Japanese who had contacts with manufacturers in Japan and dealers in China. One such man was Harry Yamazaki, a sea captain who had learned the smuggler's craft from a Portuguese opium dealer. Yamazaki bought drugs in Taiwan and elsewhere, then sold them in China. In the 1920s and 1930s, his customers were Japanese *ronin*. These adventurers—part samurai, part gangster, part patriot—had gone to China with idealistic intentions of aiding the Chinese in the 1911 revolution. However, as the republic collapsed, many drifted into the service of Chinese warlords, for whom opium and narcotics became a source of income (Nitanosa 1977, 9598; "Note on the Operations of a Syndicate," Shanghai, January 1925, PRO FO 371/10969).

In the early 1920s, narcotics came to Japan from European sources as well as from Hoshi's factory. As the decade came to a close, however, Japanese merchants took advantage of the increasing cost and decreasing availability of European drugs. Japanese alkaloid chemists, trained in Europe, returned to work in factories scattered in concessions on the Chinese mainland. These firms kept costs low, produced close to their market, undercut the prices of their European competitors, and developed a better quality of smoking heroin. The price of morphine plunged from £100–120 per kilo in 1926 to £70 per kilo in 1928 (Yamauchi 1956; Eliopoulos 1932).

One such chemist was Yamauchi Saburo, who worked in Qingdao, Shandong Province, for several years before setting up his own company in Dairen, Manchuria. Producers like Yamauchi sold their product to wholesalers, who in turn distributed it through a system of retail merchants—Korean or Japanese adventurers whose restless lives and political activities in Asia made them familiar with men who could help to distribute the goods. At first there

was much competition among the heroin makers; later, the stronger knocked the weaker out of the market. Those smugglers who survived the competition were the ones who bought protection from the Japanese army.

A 1932 bond issue of 130 million for industrial development was secured by a pledge of anticipated annual revenues of 15 million from the opium monopoly in the Japanese puppet state of Manzhouguo (see Figure 14.2). At this time, Yamauchi tells us, heroin makers donated funds to the military, often receiving decorations in return (Yamauchi 1956).

In 1934 the government of Manzhouguo invited Nitanosa Otozo, a "patriotic agriculturalist of Osaka," to visit and tender some advice. Nitanosa was a peasant with connections to Goto Shimpei. He first became concerned with opium supply in 1895, at the time of the acquisition of Taiwan. He contacted Goto and, with his support, began the experimental planting of opium poppies on his land outside of Osaka. Through the 1910s and 1920s, he used the facilities of the board of health to increase the alkaloid content of his opium plants, clearly a move in preparation for heroin manufacture.

Nitanosa went to Manzhouguo to help make that nation "self-sufficient in opium" and to end its dependence on imports from Persia. Under his guidance, as the Japanese expanded into north China, suitable fields were turned over to poppies. From 1935 to 1936, poppy cultivation increased by 28 percent. In 1937, it increased by another 17 percent. Long after Goto passed from the scene, his legacy remained visible in a colonial structure that aided drug trafficking. Even when there were officials in charge who disapproved of the traffic and wanted the opium monopoly to function as it had originally been intended to do, the potential for abuse was hard to control (Nitanosa 1977, 64, 92).

North China became a haven for heroin supplied by both Japanese and Chinese manufacturers. One reporter paints a vivid description of the "white face houses" where heroin could be purchased in Beijing in the 1930s. Low-class places, located in the eastern and western districts, they were open from sunrise to the middle of the night and supported a continual traffic of pale-faced men and women. Their windows were covered with tattered newspaper. Outside, two or three rickshaws might be parked, each stripped of its cushions and lanterns, which had been pawned to support habits. The better heroin dens could be found in the north of the city. The windows of these houses had glass panes covered with white paper shades. These establishments were run by Koreans, who at that time were Japanese subjects (Ah Nan 1937).

A typical operation involved three people. The manager sat at the counter and acted as both cashier and pawnbroker. At his side sat his wife, who doled out heroin from a bag, using a long-handled brass spoon. She leveled off the powder with a spatula, then emptied the portion onto a square of waxed paper.

FIGURE 14.2 Japanese expansion.

"Pull or stab?" she asked, to find out if the customer intended to smoke the heroin or inject it. If the customer indicated smoking, a pack of matches and a cigarette came with the price. If injection was desired, the heroin was mixed with morphine, because the product from north China was quite pure. Patrons went into a back room to enjoy their purchase. In the darkened room, they lounged on the heated *kong*. Here and there the flair of a match would outline a face in the gloomy room that stank of unwashed bodies and smoke (Ah Nan 1937).

THE FOG OF WAR

In the 1930s, the delivery system for Chinese narcotics became professional, and heroin attracted more of the consumers that system served. Meanwhile, public attention was focused on the dramatic events that would lead the world into war. In north China, reporters followed the Japanese army through a series of low-level conflicts as it expanded its control from Manzhouguo to the outskirts of Beijing (Figure 14.3). Just as today's news junkies are introduced briefly to names and places in the Middle East, only to find them eclipsed by pressing news stories closer to home, average Americans of the Depression years were diverted from following Asian events by their more immediate domestic worries. Once the north China situation escalated into war in 1937, earlier images were recalled, and when Japan became a U.S. enemy, its involvement in narcotics trafficking seemed appropriately sinister.

During World War II, three responses made by the Japanese to their situation made that group of people seem then as cynical as the Taliban seemed half a century later. First, the Japanese armies marching south took over areas previously controlled by the Guomindang opium monopoly and Du Yuesheng's operation. Not only did those commanders need money, but they did not wish to cope with addicts in sudden withdrawal while they were attempting to control a hostile, occupied population. As a solution, they reached for Goto's formula of opium monopoly. In 1939 the Japanese occupation authorities established the Ko-A-In, or Asia Development Board, to facilitate control of the Chinese economy. They attached an opium monopoly bureau to it. Commanders in charge of social control of occupied areas worried about continuing to supply their addicted population. In line with their general war supply problems, the Japanese faced chronic opium shortages for smoking and for opiate production. Manchurian supplies had been adequate to finance local growth during the period of low-level conflict in the north because they had been buttressed

FIGURE 14.3 North China under Japanese control.

by shipments from Persia. The war cut off that supplementary source of supply (Nakamura 1983).

Second, narcotics use in China was a reality that field commanders fighting a total war considered as part of their plans. I have found only one reference to the use of heroin as a way to weaken China, Yamauchi Saburo, a heroin maker who wrote his memoirs in the 1950s. Records from commanders at the front talk instead about undercutting Chinese sources of wealth: they used opium and morphine smuggling schemes to flood the market and drive down the price of Chinese opium. As the war progressed and inflation made currency

all but useless, opium became a form of illegal tender that carried real value. It paid for both strategic materials and information impossible to acquire by using paper money (Yamauchi 1956; Yamamoto 1986; Okada 1980; *Sunday Mainichi*, December 9, 1984).

The Japanese were correct in assessing opium as strategic wealth. Once Shanghai fell to the Japanese, Du Yuesheng left for Hong Kong and, later, Chongqing, where Chiang Kai-shek and the Guomindang had retreated. While Du lived in semiretirement, part of his Green Gang operation worked against the Japanese under the direction of Chiang's chief of espionage, Dai Li, and his Military Statistics Bureau. Du and Dai created at least one company for the purpose of selling opium in Japanese-held territory to pay for much needed supplies. Through this company, exchanges were facilitated by men like the shadowy Xu Caicheng, a double, or perhaps triple, agent whose name appears in police files and memoirs of the time as a Green Gang member, a Japanese espionage asset, a Chinese agent, and an opium merchant with a radio transmitter in his back room (Shen 1961; Zhang 1982).

A third condition the Japanese occupation forces faced was the appearance of antiopium agitation among the Japanese residents of Manzhouguo. Confronted with official propaganda that touted the puppet state as a paradise where five races would live in harmony, many Japanese residents found it difficult to face the corpses of addicts that filled the streets of cities like Harbin and Mukden. In January 1937, a conference of Manchurian governors, who were Japanese, petitioned their government to reduce the number of opium shops and make it harder for addicts to get licenses. "The present conditions might be part of a plan to get rid of the weak," they said, but "it is a disgraceful reflection on the people that they should continue to take poison like candy" (*Shen Ching Shi Bao* 1937, 59–60). This brought a belated effort by the local authorities to curb opium use through the local monopoly.

While the situation improved outwardly, much of the problem retreated to places like the Garden of Grand Contemplation. In spite of its poetic name, this garden was a jerry-built warren of flophouses located in the Chinese slums of Harbin, a city located in the northernmost region of Manchuria. The deserted building had been colonized by a thriving society that included displaced Chinese peasants down on their luck, heroin merchants, prostitutes, and a sprinkling of spies (Hinkosho Chiho Hoankyuko 1941).

The biographies of the residents of the Garden demonstrate the routes people followed into addiction and how heroin use blended in with opium use. Many tenants were peasants who had come north looking for work after floods and war had devastated their fields. Finding no employment, they turned to the slums and the solace available through a Japanese monopoly outlet located

inside the Garden. Most addicts, however, found monopoly opium inferior, so they improved it by adding heroin, a practice called "silver inside gold." The heroin did not come from the monopoly, but it was in plentiful supply through resident smugglers, many of them Korean (Hinkosho Chiho Hoankyuko 1941).

Most of the Garden's residents could afford single rooms in one of the flophouses. Those whose habits brought them to the brink of death rented bunks in dormitories. Skin and bones, these unfortunates often had pawned even their clothes to support their habit. When they became sick and delirious, their fellow tenants tossed them into the bitter winter streets to freeze to death and stole what meager possessions remained. There were programs to help addicts. One resident took a cure, even though he was not an addict, to get the food and new suit of clothes that came with the process. Still, the scope of the problem facing the Japanese in 1941 in places like the Garden was beyond anything they could handle at the same time they fought a war (Hinkosho Chiho Hoankyuko 1941).

Satomi Hajime was a man with an excellent vantage point from which to make sense of the conflicts in Japanese drug policy. Satomi had gone to China in 1913 to study the language. He stayed and became a journalist and a sales agent for the South Manchurian Railway. In 1937 he went to Shanghai, where he ran the city's opium monopoly for the Japanese army. At the end of the war, he was designated a Class A war criminal and served time in Sugamo Prison. When questioned by American prosecutors, he gave them his perspective on the Japanese experience with narcotics. It was not entirely what they wanted to hear.

Satomi felt that the prosecutor wanted him to confirm that the Japanese command had been part of a larger conspiracy to traffic in narcotics. He could not do that, he said. Instead, he told his interrogators that they should stop chasing after the big fish. It was the mid-level officials and officers who were involved in opium. When asked to comment on the Guomindang's supposedly successful campaign against opium in the 1930s, he replied that this assessment was mistaken and cited his own experience in Shanghai. When the Japanese army took over there, he said, they found a healthy opium market in spite of this campaign. He also contradicted the accusation that Japan was flooding China with opium from Manchuria. Satomi said that he had had to scramble for opium supplies because Manchuria could not meet the demand. The frustrated prosecutors asked if he realized that he had been violating international law when he worked in Shanghai. Satomi replied, "Warfare itself is a violation of international law, and the violation of what I call the Opium Treaty was a necessary part of our warfare" (Satomi 1946).

Mr. Arlacchi should consider the words of Satomi Hajime. The good intentions of leaders can indeed be deeply held and sincere. At the same time, if

the leaders also profess an ideology with larger political goals, short-term compromises might be made in order eventually to get those ideals into practice. Today's healthy heroin market continues to thrive on our political passions.

SUPPRESSION OF COUNTERREVOLUTIONARIES

When Pino Arlacchi went to Kandahar in 1997 to meet with the Taliban leadership, he discussed ways to improve their image in the world. He told them that two aspects of their image were in need of change: their opium trafficking and their treatment of women. Should the Taliban leaders indeed decide to undertake poppy eradication and narcotics reform, they could follow the precedent of the programs used by the Chinese Communist Party after its victory in 1949. The Afghan leadership could be influenced to seriously consider such a step by the argument that opium reform programs can be an excellent platform for the advancement of social revolution. Unfortunately, the part of their negative image that involves women is not so amenable to change because the social revolution that the Taliban envision is a regressive one, involving the regulation of ideas and the strict seclusion of women.

On October 1, 1949, the Chinese Communists emerged victorious from the Chinese Revolution, and Chiang Kai-shek's Guomindang troops retreated to Taiwan. Like the other contenders in the struggle to control China, the Communist leadership had trafficked in opium during World War II. Desperate for funds, they had dabbled in the opium trade, though their philosophy decried drug consumption as a weapon of class and imperialist warfare. In February 1950, Zhou Enlai announced stringent opium control laws (Ch'en 1995; Ma 1993).

Satomi Hajime once said, "People who know China know no suppression campaign will work" (Satomi 1946). He had good reason to be cynical. Regulations had been promulgated before the Communist initiative, and no one was in a better position to appreciate how poor the results had been than Satomi. But the new Chinese regime was determined to make a clear distinction between themselves and the past. They looked hard for ways to make the laws work. While they had been holed up in the northern base camp during the war with the Nationalists, they had established a procedure for developing a dedicated Marxist corps of soldiers out of the ragtag volunteers who were drawn to their side. Based on the principle of "unity through criticism," the process involves self-criticism in a public forum. This activity has become familiar to us in the West through reports of the Cultural Revolution of the 1960s. In the early 1950s there was a mass movement called the Three Antis/Five Antis Campaign. Aimed at ridding China of the political corruption that had

plagued the Nationalists, the Chinese Communist leadership included in this larger movement a national campaign against opium use (Ma 1993).

The program included the roundup, trials, and executions of notorious drug dealers. In addition, mass participation in antinarcotics rallies, public confessions by ex-addicts, and neighborhood discussion groups molded popular opinion against drug use. During the time of this movement, China was at war with the United States in Korea. Drug peddlers could be identified with the Guomindang in Taiwan and, by extension, with the United States. This gave the movement a patriotic boost (Ma 1993).

The approach of the Chinese Communists in these campaigns appeared to be harsh but effective. They put forward the message that drug use was not only a degrading habit, but also one that belonged to a decadent capitalist culture. The Taliban could use precisely this kind of propaganda program, substituting their brand of Islam for Chinese Marxism, but the Chinese campaigns, while successful in curbing drug use, also led to suppression of intellectuals and to larger human rights abuses. Considering the repressive social vision of the Taliban, such an antidrug campaign would probably take the same direction. Is this a situation that international regulators would condone?

COLD WAR SOLUTIONS

The Chinese Communist leadership was successful in its antinarcotics campaign because it could link opium and heroin elimination to a larger political program. In the early 1950s, a war-weary Chinese population had been willing to give the new regime the benefit of the doubt, and so it could be inspired by a new vision. In the long run, it became clear that the program of the 1950s had been successful only temporarily. Once Communist China reopened its markets to world trade, it rejoined the world's narcotics users as well. During the thirty years of its isolation, heroin had become the dominant opiate in the United States and Europe. At the end of the twentieth century, China faced a growing heroin epidemic and found that its earlier approach to narcotics control was no longer so effective (Cheng 1993).

It is not surprising that today's heroin problem in China is centered in Yunnan. Not only does Yunnan have a rich legacy as an opium-producing territory, but just across its southern border lies the Golden Triangle, where the heroin trade continued to develop during the years of China's isolation. Because mainland China was perceived as an enemy of the West, American propagandists found it convenient to blame China for the growing heroin traffic flowing into the United States from Burma and Thailand. Harry Anslinger, the longtime head of the Federal Bureau of Narcotics and a dedicated cold

warrior, prominently accused the Communists throughout the 1950s of flooding the United States with heroin (Yates to Narasimhan, June 6, 1962, series 272, box 1, U.N. Archives, New York).

On May 31, 1962, Anslinger repeated this message as he addressed the seventeenth session of the UN Narcotics Commission in Geneva. This created a problem for members of the UN Opium Control Board. Gilbert Yates, the head of that body, privately worried about how to respond to the accusations. According to his information, it was Guomindang irregulars holed up in Burma, on the border with China, who were living off opium. Remnants of Chiang Kai-shek's armies driven out of China at the end of the revolution, in the early 1950s, those soldiers had gone into the Burmese hills, where they staged several attempts to take back the Chinese mainland. After the failure of these efforts, they remained a force in local Burmese politics. It was here that the trafficker Khun Sa later developed his heroin empire (*New York Times*, June 1, 1962; Yates to Narasimhan, June 6, 1962; McCoy 1991, 162– 78).

Yates was observing a shift from the old supply system that had been developed in China during the 1930s. After the Communist revolution in 1949 and the Guomindang retreat, most of the leadership of the defeated forces went to Taiwan, where they nurtured the rhetoric of reconquest of the mainland while building a new society on the island. Some, though, remained around the borders of China in a kind of unholy diaspora. One example is Ke Chaohuang. Ke had been a member of Dai Li's Military Statistics Bureau during the war. As such, he was assigned to infiltrate the secret societies in south China that had already been penetrated by the Japanese secret service. After the success of the revolution, he set up his own organization in Hong Kong, called 14K after the address of its headquarters. Soon 14K began trafficking in heroin from the Golden Triangle (He 1996).

This is the last lesson for Mr. Arlacchi. As the United States lobs missiles into Afghan territory, it is a good time to reflect on the conditions that have nurtured the narcotics trade network during the past century. The raw materials for narcotics production grow in places that not only are poor, but also are strategically located near the conflicts that made that period so violent. Uneven economic development has been the foundation of much of the strife of the last hundred years, but alternative development requires political stability. How should development move forward? Who is to control the resources? Which traditional values and constraints must be sacrificed as the cost of modernization? Each of these questions is difficult to resolve in spite of our modern sophistication. Meanwhile, opium revenues have been too convenient a resource to be forsworn by those caught up in the wars and revolutions that modern tensions have spawned.

NOTES

1. From *One Hundred Years of Heroin*, David F. Musto; Chapter 12, pp 199–226, "From British India to the Taliban," by Kathryn Meyer. Copyright © 2002 by David F. Musto. Reproduced with permission of Greenwood Publishing Group, Inc. Westport, CT.
2. Currency arrangements in China were complicated. In the 1930s China had a managed currency, the Chinese yuan, but foreign currency circulated in China as well. The symbol $ indicates Mexican silver dollars, which were popular and in which currency dollar values are given in this text. In 1930, U.S.$1.00 = Mex. $.3009 = yuan .2992 = £4.8621 = 1.4939. U.S. Department of Commerce, *Statistical* Abstract of the United States, 1932 (Washington: U.S. Government Printing Office, 1932).

REFERENCES

Adams, Walter. 1934. *Opium situation in China*. Report 893.114NARCOTICS/708. Washington, DC: U.S. State Department.

Ah Nan. 1937. White face houses of Beijing. In *Yapian Shi Jin Xi* [Opium today and yesterday], ed. Tao Kangde. Shanghai: Yuzhoufeng She Chuban.

Ch'en, Jerome. 1972. *Yuan Shih-k'ai*. Stanford, CA: Stanford University Press.

Ch'en Young-fa. 1995. The blooming poppy under the red sun: Yet Yenan Way and the opium trade. In *New perspectives on the Chinese Communist Revolution*, ed. Tony Saich and Hans van de Ven. Armonk, NY: M. E. Sharpe.

Cheng Yi, ed. 1993. *Baise Youling: Zhongguo Dupin Newmu* [White ghost: The inside story of Chinese narcotics]. Beijing: Guangming Ribao Chubanshe.

Courtwright, David. 1982. *Dark paradise: Opiate addiction in America before 1940*. Cambridge, MA: Harvard University Press.

Eliopoulos, Elie. 1932.. Report 800.114N16 ELIOPOULOS/27, RG 59, box 4536. National Archives, Washington, DC.

Great Britain. Public Record Office, London, England.

Great Britain, Foreign Office. 1974. *The opium trade, 1910–1941*. Wilmington, Del.: Scholarly Resources.

He Zengxiao. 1996. Guangdong Hengmen Zhongyihui Shimo [History of the Hong League]. In *Zhonghua Wenshi Ziliao Wenku* [A treasury of Chinese source materials], 20:366–83. Beijing: Zhongguo Wenshi Chubanshe.

Hinkosho Chiho Hoankyuko. 1941. *Taikanen no Kaibo* [Autopsy of the Garden of Grand Contemplation]. In *Kamminzoku Shakai Jittai Chosatsu* [Investigation into the social conditions of the Chinese race].

Hoshi Hajime. 1926. *Ahen Jiken* [The opium incident]. Tokyo: Hoshi Pharmaceutical Business School.

Hoshi Shinichi. 1971. *Jinmin wa Yowashi; Kanri wa Tsuyoshi* [The people are weak; the bureaucrats are strong]. Tokyo: Kadokawa Shoten.

Hosie, Alexander. 1914. *On the trail of the opium poppy*. 4 vols. London: George Philip.

Isaacs, Harold. 1951. *The tragedy of the Chinese Revolution.* Stanford, CA: Stanford University Press.

Jordan to Grey. March 5, 1912. FO 10168/12644, and Jordan to Grey, January 22, 1913, FO 10481/6308, in Great Britain, Foreign Office 1974.

Ma Weigang. 1993. *Jinchang, Jindu: Jianguo Chugi de Lishi Wenti* [Prohibit prostitution, prohibit poison: Historical problems from the early period of national construction]. Beijing: Jingjuan Jiaoyu Chubanshe.

Marshall, Jonathan. 1977. Opium and the politics of gangsterism in nationalist China, 1927– 45. *Bulletin of Concerned Asian Scholars* 8: 19–48.

Martin, Brian. 1996. *The Shanghai Green Gang: Politics and organized crime, 1919–1937*. Berkeley: University of California Press.

McCoy, Alfred W. 1991. *The politics of heroin: CIA complicity in the global drug trade.* New York: Lawrence Hill.

Mei Zhen and Shao Pu. 1987. *Haishang Wenren Du Yuesheng* [Shanghai celebrity Du Yuesheng]. Zengzhou, China: Henan Renmin Chubanshe.

Mimeographed police report, labeled "Top Secret." Tokyo: National Diet Library.

Minami Manshu Tetsudo Kabushiki Kaisha, Kaisai Chosakai Dai Go Bu. 1934. *Manshukoku Ahen Seido oan* [Draft proposal for control of opium in Manzhouguo]. South Manchuria Railway Company archives, Library of Congress, Washington, DC.

Moses, Sayas. 1933. Report 800.114N16 ELIOPOULOS/27, RG59, box 4536. National Archives, Washington, DC.

Musto, David. 1973. *The American disease: Origins of narcotic control.* New Haven, CT: Yale University Press.

Nakamura Takahide. 1983. *Senji Nihon no Kahoku Keizei Shihai* [Japan's wartime economic management of North China]. Tokyo: Yamakawa Shupansha.

Nitanosa Nakaba. 1977. *Senso to Nihon Ahen Shi: Aheno Nitanosa Otozo no Shogai* [War and the history of Japanese opium: The life of Nitanosa Otozo, the opium king]. Tokyo: Subaru Shobo.

Okada Yoshimasa. 1980. Chukoku Shihei Gizo Jiken no Zembo [The complete picture of the Chinese currency counterfeiting caper]. *Rekishi to Jimbutsu* 110.

Parassinen, Terry. 1983. *Secret passions, secret remedies: Narcotic drugs in British society, 1820–1930.* Philadelphia: ISHI Press.

Public Record Office. 1929. *Memorandum respecting the opium problem in the Far East.* London: Public Record Office.

Riens, Thomas. 1991. Reform, nationalism, and internationalism: The opium suppression movement in China and the Anglo-American influence, 1900–1908. *Modern Asian Studies* 25: 101–42.

Ryu Meishu. 1983. *Taiwan Tochi to Ahen Mondai* [Control of Taiwan and the opium problem]. Tokyo: Yamakawa Shoten.

Satomi Hajime. 1946. International Prosecutors Office, Interrogation no. 59, case 426 (March 5), International Military Tribunal. National Diet Library, Tokyo.

Shanghai Municipal Police. 1916. Document 10 670. National Archives, Washington, DC.

Shanghai Municipal Police. 1931. Document D 3057. National Archives, Washington, DC.

Shanghai Municipal Police. 1933. Document D 5645. National Archives, Washington, DC.

Shanghai Municipal Police. 1940. Document D 9319. National Archives, Washington, DC.

Shen Zui. 1961. "Wo Suo Zhidaode Dai Li" [The Dai Li I knew]. In *Wenshi Ziliao Xuanzhuan* [Selected historical resources], 22. Beijing: Zhonghua Shuchu.

Sheng Ching Shi Bao [Sheng Ching News]. 1937. Document 9668, exhibit 383. International Military Tribunal, National Diet Library, Tokyo.

Stilwell, Joseph. 1935. *Political issues and problems—The narcotic situation.* Report 893.114NARCOTICS/1547, U.S. State Department, RG 59, box 7202. National Archives, Washington, DC.

Takasugi, Shingo. 1984. Akuma no Boryaku [The devil's strategy]. Series of articles. *Sunday Mainichi*, December 2–30.

Taylor, Arnold. 1973. *American diplomacy and narcotics traffic, 1900–1939: A study in international humanitarian reform.* Durham, NC: Duke University Press.

Times [London]. 1920. Smuggling opium into China. March 27.

Tsurumi, Yusuke. 1965–1967. *Goto Shinpei.* 4 vols. Tokyo: Keisoshobo. United Nations Archives, New York.

U.S. Department of Commerce. 1932. *Statistical abstract of the United States, 1932.* Washington, DC: U.S. Government Printing Office.

U.S. State Department. Record group 59. National Archives, Washington, DC.

World Development Indicators Database. (2007). World Bank. World Bank Publications.

Xiao Juetian. 1996. Jiang Jieshi Jinyan de Neimu [The inside story of Chiang Kaishek's opium suppression]. In *Zhonghua Wenshi Zilaio Wenku* [Treasury of Chinese historical materials], 20:650–58. Beijing: Zhongguo Wenshi Chubanshe.

Yamamoto Tsuneo. 1986. *Ahen to Taiho: Rikugun Showa Tsusho no Nananen* [Opium and cannon: Nine years in the army's Showa Trading Company]. Tokyo: PMC.

Yamauchi Saburo. 1956. Mayaku to Senso: Nitchu Senso no Himitsu Heiki [Narcotics and war: A secret weapon of the China war]. *Jimbutsu Orai*, October.

Yu Enyang. 1917/1966. *Yunnan Shouyi Yunghe Gunghe Shimo Ji* [Record of Yunnan righteous leadership in protecting the republic]. Repr. Taipei: Wenhai Chubanshe.

Zhang Wuihan. 1982. Dai Li yu Juntongpe [Dai Li and the Military Statistics Bureau]. In *Zhijiang Wenshi Ziliao* [Historical sources for Zhijiang Province], 104–7. Zhijiang: Renmin Chubanshe.

Zhang Zaichuan. 1986. Xuanhu Yishi de Tang San [The eminent Tang San (Tang Jiyu)]. In *Panlong Wenshi Ziliao*, 1:13–26. Yunnan: Kunming. Historical Materials Collection, Center for Chinese Studies Library, University of California, Berkeley.

Zhang Zheyong. 1985. *Shanghai Jindai Shi* [The history of modern Shanghai]. Shanghai.

This page intentionally left blank

Part III

ASPECTS

This page intentionally left blank

Chapter **15**

Youth Tobacco Use: The Health Effects, Trends in Smoking Rates, and Reasons Why Kids Use Tobacco[1]

Clete Snell, PhD

Smoking kills an estimated 434,000 Americans every year. The vast majority of these smokers began smoking in their teen and preteen years. Very few people begin tobacco use as adults. In fact, by the age of eighteen, approximately one in three people in America use tobacco products. Moreover, the earlier adolescents initiate tobacco use, the heavier their tobacco use becomes in adulthood and the longer potential time they have to be users. Long-term chronic health problems, such as cancer, emphysema, and heart disease, are related to the duration and amount of tobacco use. The focus of public health officials for the last couple of decades has been to reduce deaths from tobacco by attempting to prevent young people from taking up the habit.

THE HEALTH EFFECTS OF TOBACCO USE AMONG YOUTH

Tobacco use—especially cigarette smoking—is considered the chief preventable cause of premature death in America. Several reports by the Surgeon General's Office has documented a causal link between cigarette smoking and lung cancer and other fatal cancers, arteriosclerosis and coronary heart disease, chronic obstructive pulmonary disease, and a wide array of other serious health problems.[2] More recently, research has found that passive or environmental tobacco smoke can lead to serious health problems—such as lung cancer—in otherwise healthy nonsmokers. In fact, every three weeks in the United States,

secondhand smoke kills about the same number of nonsmokers as were killed in the September 11th terrorist attacks on the World Trade Center.[3]

Many young people have the false impression that the health effects of smoking and other tobacco use are problems they will have to combat much later in life. Many youth who just start smoking also believe that they can quit at any time. The truth is that smoking has immediate and serious effects on the health of children. Cigarette smoking in adolescence appears to retard the rate of lung growth and the maximum lung function that can be achieved. Young smokers are less likely to be physically fit than young nonsmokers, and more likely to experience shortness of breath, coughing spells, wheezing, and other physical health problems.

Heart disease is the leading cause of death in the United States. Atherosclerosis is a precursor to heart disease and may begin in childhood or young adulthood as a consequence of smoking. Smoking by children and adolescents is related to an increased risk of early atherosclerotic lesions that are a risk factor for heart disease.

Smokeless tobacco (frequently termed snuff or chew) is related to health problems that range from halitosis to severe health problems such as several different forms of oral cancer (cancers of the mouth, tongue, and esophagus). Reports by the Surgeon General have documented that smokeless tobacco use is as addictive for young people as it is for adults. Furthermore, smokeless tobacco users are more likely than nonusers to become cigarette smokers.

Drug use among youth has been a public policy concern for decades, but rarely has cigarette smoking been considered to be among serious drug problems. Nicotine dependence from cigarette smoking is the most common form of drug addiction in the nation. Moreover, to emphasize the seriousness of nicotine addiction, it causes more death and disease than all other addictions combined.[4] The two medical disorders associated with nicotine addiction identified by the American Psychological Association are nicotine addiction and nicotine withdrawal. Symptoms of nicotine withdrawal include "craving for nicotine; irritability, frustration, or anger; anxiety, difficulty concentrating; restlessness; decreased heart rate; and increased appetite or weight gain."[5]

Physical dependence on any type of substance refers to the condition in which withdrawal symptoms are present. Physical dependence makes it much more difficult to achieve and maintain abstinence from a drug and makes it more likely that the user will relapse. Surveys conducted by the National Institute on Drug Abuse (NIDA) have found that severe withdrawal symptoms and the inability to maintain abstinence from use of a drug are more commonly associated with heroin and cigarette smoking than any other forms of addictive drugs, including cocaine and marijuana.

Each year about 20 million Americans attempt to quit smoking, but only about 3% have long-term success.[6] In fact, even among addicted cigarette smokers who have lost a lung due to cancer or have had major heart surgery, only about 50% kick the habit for more than a few weeks.[7]

This startling fact is something I witnessed firsthand while visiting my wife who at the time, worked for MD Anderson Cancer Hospital in Houston. Quite frequently cancer patients, who were obviously receiving chemotherapy evidenced by hair loss, weight loss, and pale skin, could be found outside the hospital connected to IV's and smoking cigarettes.

Nicotine's addictive potential is realized very soon after regular use. Data from the 1985 National Household Survey on Drug Abuse (NHSDA) found that 84% of 12- through 17-year-olds who smoked one pack of cigarettes or more per day felt that they needed or were dependent on cigarettes. The NHSDA research found that young people develop tolerance and dependence for nicotine, increase the amount they smoke over time, and are unable to abstain from cigarettes. This suggests that the addictive processes in adolescents are similar or the same as that for adults.[8]

OVERALL TRENDS IN CIGARETTE USE IN THE PAST CENTURY

It is important to develop an understanding of the magnitude of the problem of tobacco use in the United States and how that has changed over recent years. The National Health Interview Survey (NHIS) has been conducted by the Centers for Disease Control (CDC) since 1965. It uses very large probability samples and interviews are conducted within the home. NHIS respondents are considered current smokers if they have ever smoked at least 100 cigarettes and that they continue to smoke. Former smokers are those who have smoked at least 100 cigarettes and are not current smokers. Those classified as having never smoked have either smoked no cigarettes or less than 100 in their lifetimes. The NHIS also asks questions concerning other types of tobacco use such as cigar, pipe, and chewing tobacco.[9]

Total annual consumption of cigarettes in the United States was 2.5 billion cigarettes in 1900, according to records from the U.S. Department of Agriculture. Consumption increased steadily and dramatically over the years, until reaching a peak of 640 billion cigarettes in 1981. The largest increases occurred in the 1910s, 1920s, and 1940s. For instance, total cigarette consumption was 8.6 billion in 1910 and increased to 44.6 billion in 1920. By 1940, consumption had more than tripled to 181.9 billion cigarettes. Since the peak consumption of cigarettes in 1981, cigarette use has steadily declined to approximately 480 billion cigarettes in 2003.[10]

Because overall cigarette consumption does not take into account the overall population changes over the last century, it is more useful to examine cigarette consumption per capita. The trends are similar to that of overall consumption. Per capita use of cigarettes was 54 in 1900 (54 cigarettes consumed per person), 665 in 1920, 1,976 in 1940, and reached a peak of 4,345 in 1963.[11]

The NHIS first asked questions about cigarette smoking in 1965. At that time an estimated 42% of American adults were current smokers, including 52% of men and 34% of women. By 1991, cigarette smoking prevalence had decreased to 26% overall, 28% for men and 24% for women. Throughout this period of time (1965–1991), blacks were more likely to smoke than whites, and Hispanics were less likely to smoke than non-Hispanics. Prevalence by age was highest among persons aged 25–44 years. During the period studied, smoking decreased dramatically for all sociodemographic groups. From 1965 to 1991, the prevalence of cigarette smoking decreased 46% among men and 31% among women. The decrease in cigarette use was about equal between blacks and whites, and decreased more dramatically among Hispanics. Cigarette smoking prevalence also declined most rapidly among younger adults and among people with more education.[12]

There has also been a large increase in the number of smokers who were able to quit smoking. This is generally referred to as *cessation* in the language of epidemiologists. The number of people who were regular smokers but quit nearly doubled between 1965 and 1991. By 1990, more than half of U.S. men and more than half of American white adults who had ever smoked cigarettes had quit. The prevalence of quitting was higher for men than women, for whites than blacks, and for non-Hispanics than Hispanics. The likelihood of quitting the habit increased with both age and education.[13]

TRENDS IN YOUTH TOBACCO USE

There have been a limited number of research projects that have followed adolescent tobacco use over a long period of time. The Monitoring the Future Project is one that has followed teen smoking since 1976. In one publication, daily cigarette smoking among high school seniors was tracked from 1976 to 1993. Daily cigarette smoking was defined as smoking at least one cigarette per day during the past thirty days. The overall prevalence of daily smoking was approximately 35% lower in 1984 than in 1976. However, from 1976 to 1993 there was a 2% increase in daily smoking. In contrast, smoking prevalence among adults in 1991 was about 20% lower than in 1983. Similarly, daily smoking among white high school seniors increased 14% between 1984 and 1993,

while it decreased 20% among white adults. Male high school seniors increased their daily tobacco use by 21% between 1984 and 1993, while it decreased 20% among adult males. Among female high school seniors, on the other hand, the prevalence of daily smoking was 11% lower in 1993 than in 1984. Among adult women between 1983 and 1991, the decrease in current smoking was even more striking (20% reduction).[14]

The greatest reduction in daily smoking among high school seniors was among blacks. Their self-reported prevalence was 51% lower in 1993 than in 1984. But, in keeping with the overall trends among youth during that time period, the reduction in daily smoking among black high school students was less pronounced (19%).[15]

The National Youth Risk Behavior Survey (YRBS) is another source of information regarding cigarette smoking among high school students. The YRBS measures the prevalence of health risk behaviors among high school students through representative biennial national, state, and local surveys. The samples from this study are representative of students in grades 9–12 in all fifty states and the District of Columbia. A recent publication using this data tracked cigarette consumption between 1991 and 2001. Three types of tobacco behavior were assessed: (1) lifetime smoking, defined as having ever smoked cigarettes; (2) current smoking, defined as smoking at least one day in the last 30 days; and (3) current frequent smoking, defined as smoking on at least twenty of the last thirty days. The report concludes that cigarette smoking rates increased steadily from 1991 to 1997, and since that time they have been declining significantly.[16]

The prevalence of lifetime smoking remained stable throughout the 1990s, but declined significantly from 70.4% in 1999 to 63.9% in 2001. The prevalence of current smoking increased from 27.5% in 1991 to 36.4% in 1997, and then declined significantly to 28.5% in 2001. Current frequent smoking increased from 12.7% in 1991 to 16.7% in 1997 and 16.8% in 1999, and then declined significantly to 13.8% in 2001.[17]

Among female students, the prevalence of current smoking increased steadily from 27.3% in 1991, peaked at 34.9% in 1999, and declined significantly to 27.7% by 2001. Similar trends occurred among white female, black male, Hispanic, Hispanic female, Hispanic male, ninth, and eleventh grade students. In all these cases, smoking prevalence peaked by 1999 and then declined significantly by 2001.

The trend was similar for all males; the prevalence of current smoking increased steadily from 27.6% in 1991, peaked at 37.7% in 1997, and declined significantly to 29.2% in 2001. A similar pattern occurred among white, white male, black, tenth, and twelfth grade students.

CURRENT YOUTH TOBACCO USE

According to the National Youth Tobacco Survey (NYTS) in 2002, about 13.3% of middle school students (grades 6–8) reported current use of any tobacco products. Cigarettes (10.1%) were the most popular tobacco choice among middle school youth, followed by cigars (6%), smokeless tobacco (3.7%), pipes (3.5%), bidis (2.4%), and kreteks (2%). Middle school boys were more likely than girls to use all types of tobacco products except for cigarettes. Middle school boys and girls were about equally likely to smoke cigarettes.[18]

High school students used tobacco products at a rate of about 28.4% in 2002. As with middle school youth, high school students were most likely to smoke cigarettes (22.9%), followed by cigars (11.6%), smokeless tobacco (6.1%), pipes (3.2%), kreteks (2.7%), and bidis (2.6%). Males were significantly more likely to use any tobacco products than females students (32.9% for males compared to 23.9% for females). However, male high school students were only slightly more likely to smoke cigarettes than females (24.6% for males compared to 21.2% for females).[19]

To summarize trends in youth tobacco use, smoking among adolescents declined sharply in the 1970s, but the decline slowed significantly in the 1980s, increased throughout most of the 1990s, then decreased in the late 1990s and continues to drop at the present time. Female adolescent smoking actually exceeded that among males by the mid- to late 1970s, however both sexes are about equally likely to smoke at the present time. Boys are much more likely to experiment with other types of tobacco products than girls. Nationally, white adolescents are more likely to use all forms of tobacco products than are blacks and Hispanics. The decline in the prevalence of cigarette smoking among black adolescents has been dramatic and has occurred over a period of about fifty years.

Why have youth smoking rates dropped in recent years? Experts at the Centers for Disease Control believe that the following factors may have been influential: (1) a 70% increase in the retail price of cigarettes from December 1997 to May 2001; (2) an increase in the number and effectiveness of school-based efforts to prevent tobacco use; and (3) an increase in the exposure of youth to national and state mass-media smoking prevention campaigns. Despite the progress, especially among high school students, over one-quarter (28.5%) of high school youth are current smokers and almost 14% are frequent smokers with probable nicotine dependence.[20]

WHY KIDS START SMOKING

The earliest research concerning how to prevent youth from starting to smoke was the result of the landmark 1964 Surgeon General's report on smok-

ing and health.[21] Early research was not theory-based and did not focus on the question of what motivates young people to smoke. This research viewed cigarette smoking as a health behavior, rather than a social behavior with social causes, functions, and reinforcements. By the mid-1970s, support for theory-based research was provided by the National Clearinghouse for Smoking and Health, the National Institutes of Health (NIH), NIDA, and private health organizations such as the American Lung Association, the American Cancer Society, and the American Heart Association.

The application of psychosocial theories to the area of youth smoking was a major breakthrough in attempts to understand why youth begin to experiment with smoking and what sustains their tobacco use. The early works of Leventhal, Bandura, and McAlister, Perry, and Maccoby were pioneering, but limited in the sense that they tended to rely on simple correlations such as testing for a relationship between parental smoking and children's smoking behavior.[22] The problem with research that examines simple correlations is that it is impossible to come to any conclusions about the actual causes of youth smoking. Subsequent research became much more sophisticated, theory driven, and longitudinal in nature. Longitudinal research tracks the same cohort of youth over a period of time (usually several years) and can provide much more useful information as to why kids initiate tobacco use and why they continue to use tobacco products. Conrad, Flay, and Hill reviewed twenty-seven studies concerning why kids start smoking that had been published since 1980.[23] The fairly large number of rather sophisticated studies allows us to begin to draw some conclusions as to why kids smoke.

Developmental Stages of Smoking

Flay developed a useful model of the five primary stages of smoking initiation among children and adolescents.[24] In the first stage or preparatory stage, general attitudes and beliefs about the value of smoking are formed. Even if an adolescent does not begin to smoke, they come to see smoking as having a useful purpose: to appear mature, cope with stress, fit in with a new peer group, or display independence from the influence of authority figures such as parents.[25] Certain psychosocial risk factors discussed in the next section, such as tobacco advertising and adult or sibling role models who smoke, may provide an influence at this stage.

The second stage is termed "the trying stage" where an adolescent actually experiments for the first time with cigarette smoking, usually smoking two or three times. The psychosocial risk factors involved in this stage include peer reinforcement or encouragement to try smoking, the perception that smoking is normative or socially acceptable behavior, and the availability of cigarettes.

The third stage is the experimental stage where adolescents engage in repeated but irregular smoking. Some of the psychosocial risk factors involved in this stage include social situations such as parties where smoking is often prevalent and important peers such as a best friend who supports smoking; low self-efficacy or the inability to refuse offers to smoke; and the availability of cigarettes. These influences may impact a youth enough to smoke in particular social situations or around certain people, but not become a regular smoker.

The fourth stage is termed "regular use" when adolescents smoke on a regular basis, at least weekly, and increasingly across a variety of situations and personal interactions. The psychosocial influences at this stage include socializing with peers who smoke and reinforce smoking, the perception that smoking has personal utility such as reducing stress or weight loss, and the availability of cigarettes.

The final stage is nicotine dependence and addiction. As discussed earlier, this involves a physiological need for nicotine. This need includes a tolerance for nicotine, withdrawal symptoms if the person attempts to quit, and a high likelihood of relapse if the person quits.

While there is much variation among adolescents in the length of time between an initial attempt at smoking and regular use, on average, it takes only two to three years. In fact, McNeill found that of those youths who experimented with cigarettes, about half were smoking on a daily basis within a year.[26] Thus prevention efforts have focused on delaying or preventing the first attempts at smoking as well as preventing the progression to regular use of cigarettes. Because an adolescent may become a regular smoker in a fairly short period of time (two to three years), a high percentage of adolescents at least try cigarettes, and because adolescents who become regular smokers generally progress to addiction as adults, it is critical to target prevention efforts at middle school, junior high school, and high school youths.[27]

Psychological and Social Risk Factors

In the Surgeon General's report *Preventing Tobacco Use among Young People,* various psychological and sociological (psychosocial) risk factors were identified—through a thorough examination of research—as greatly increasing the chances that adolescents will begin using tobacco products. These risk factors were grouped into the following categories: sociodemographic, environmental, behavioral, and personal. These risk factors are believed to be the first link in the causal chain that leads to tobacco-related health problems. Thus prevention efforts should take these factors into account.

Sociodemographic Factors

Sociodemographic factors such as socioeconomic status or gender do not generally directly impact decisions to start smoking, but rather indirectly impact decisions to start smoking by impacting behavioral and personal factors. Tobacco use may vary according to socioeconomic status, family structure, age, gender, and ethnicity.

Low socioeconomic status (SES) has been found to predict smoking initiation in several longitudinal studies.[28] One German study found that seventh- and eighth-grade students from a school in a low-income area had higher rates of tobacco use and were more likely to start smoking over a six-month period than youth from a higher-income area. Low-income students also had greater expectations of certain positive consequences of smoking, more friends who smoked, and lower scores on a self-image scale. A possible explanation is that lower-income students may start cigarette smoking to cope with the stress that comes with a lack of financial and material resources or with living in a one-parent household. They may find that tobacco use is a quick and easy coping strategy for stress or loneliness.[29] Youth from low-income families may also have more peers or other role models who smoke and less supervision to discourage experimentation than youth from higher-income families.[30] While low socioeconomic status is related to initiating cigarette smoking, it is not a strong predictor of experimenting with smokeless tobacco.

The life stage of adolescence has become a consistent and strong predictor of initiating both cigarette and smokeless tobacco use.[31] In particular, the transition between elementary school and high school seems to be a high-risk period for the adolescent's initiation of tobacco use. In fact, the rate of the onset of smoking and the prevalence of smoking may actually level off during the high school years.[32] There are three major types of developmental challenges that youths face during adolescence. First, youth become physically and sexually mature and begin to establish relationships with the opposite sex. Second, adolescence is a time of responding to cultural pressures to begin making the transition to adult social roles and responsibilities. Third, youth during this developmental period begin to establish a coherent sense of self and a set of values to guide future behavior. Many youth confront these challenges through experimentation and risk-taking behavior.[33] Tobacco use is one of many risky behaviors that many youth believe may make them more attractive to certain peers and may contribute to a positive self-image in many social settings.[34]

Currently the rate of smoking among boys and girls is roughly equal in the United States. However, that has not always been the case. Males were much more likely to smoke and use other tobacco products until the mid-1970s

and mid-1980s. During that time, tobacco use among young men dropped from about 45% to 33% but remained constant at about 34% among young women.[35] Two studies suggest that changing gender roles, or more women are in traditionally male positions of power and authority, may explain why young women have been more resistant to the general trend of lower smoking rates. One review of research on the influence of gender on smoking behavior found that peers and parental models equally influenced both girls and boys. The study did find that girls who smoked were more socially skilled (e.g., at ease with their peers, strangers, and adults) than nonsmoking girls, and more socially skilled than boys who smoked.[36] Girls are also more likely to be concerned with body weight and to believe that smoking may help control weight gain.[37]

Environmental Factors

Environmental factors that may influence the decision to initiate smoking include the availability of cigarettes in the community, the social acceptability of smoking, the influences of peer and parental smoking, and the perceptions youth hold of their environment.

Adolescent smoking behavior has been found to be affected by the acceptability and availability of cigarette use within society or the community. The acceptability of smoking and the availability of tobacco products are affected by the pervasive advertising and promotional activities of the tobacco industry. Multiple role models, including family members, friends, and even television and film actors, also affect the social acceptance of tobacco. Finally, community norms, policies, and ordinances impact the acceptability of smoking by either sending clear signals that tobacco use by youth is tolerated through allowing sales or access to underage buyers, or by enforcing strong ordinances that work to keep tobacco out of the hands of kids.

In a study conducted by the National Adolescent Student Health Survey, 79% of eighth graders and 92% of tenth graders considered it to be "very easy" or "fairly easy" to obtain cigarettes.[38] Similarly, the 1991 Monitoring the Future Project found that 73% of eighth graders and 88% of tenth graders reported that it would be "fairly easy" or "very easy" to get cigarettes. Another study conducted by the Centers for Disease Control found that among a national sample, of the 2.6 million underage smokers, 1.5 million purchase their own cigarettes.[39] Among those who buy their own cigarettes, 84% purchase them from small convenience stores, 50% from large stores such as grocery stores, and 14% obtain them from self-serve vending machines. There have been a number of studies observing cigarette buying behaviors of teens that tend to confirm

these reports. The general availability of cigarettes is important because a number of studies have found that the more available cigarettes are in a community, the earlier the onset of smoking among youth.[40]

My own experience in first trying cigarettes was, in most respects, not unlike other adolescents. I was in a car with three of my peers. We were all juniors in high school. We had just left a party and all three of us had been drinking beer. Someone suggested that we drive up to the local ski resort (I lived in Montana at the time). On the way up, another friend passed around cigarettes. I remember smoking two cigarettes and experiencing a terrible burning sensation in my throat. One of my friends asked if I had ever tried Copenhagen, a brand of smokeless tobacco; I lied and said, "of course." I had tried other forms of smokeless tobacco, which were very popular in Montana. He offered his can and I took as small a pinch as I could. When I awkwardly placed the tobacco between my lip and bottom teeth, he laughed, acknowledging that I was not a very experienced "dipper." Within five minutes I felt a distinct light-headedness and a burning sensation in my stomach (I was swallowing the tobacco juices). We eventually made it to the ski resort after climbing to approximately 6,000 feet in elevation. The combination of beer, cigarettes, smokeless tobacco, and high elevation made me as nauseous and dizzy as I have ever been in my life. We pulled up to the parking lot just in time for me to open the car door and vomit in a snowdrift for several minutes. I remember the humiliation of my friends' laughter and the curious look of strangers walking through the parking lot. My first experience with tobacco use had been decidedly negative and I remember thinking, "Why in the world would anyone decide to do this?" Most adolescents' first experiences using tobacco are not positive in terms of physical sensations. It is the social cues in initial tobacco use that reinforce subsequent use: positive reinforcement from peers and the general feeling of social acceptance and fitting in.

The impact of role models such as parents, siblings, and peers on decisions to start smoking have been carefully examined in research. Role models not only provide the necessary attitudes for the social acceptance of tobacco use, they also provide common social settings, such as parties, in which cigarettes are tried for the first time.[41] These role models and social settings provide the context in which youth come to view smoking as either a positive or negative experience.

The evidence concerning the influence of parents' smoking behavior on their children's cigarette use has been mixed. On the one hand, a number of studies have found a strong and consistent relationship between parental and adolescent smoking.[42] By contrast, parental smoking was a weak predictor of children's smoking in other studies.[43] Conrad, Flay, and Hill examined the findings

of twenty-seven prospective studies concerning the onset of smoking among youth.[44] Fifteen of these studies examined parental influences, and fewer than half (seven) found that parental smoking was a strong predictor of their children's smoking behavior. Chassin and his coauthors believe that parental smoking may influence the decision to try cigarettes, but is far less important in the transition to regular smoking behavior.[45]

Parents are not the only family members who may influence decisions to use tobacco products. I started using smokeless or chew tobacco (for only a short time) after seeing my older brothers use it (and before the beer-tobacco-altitude induced vomit incident). Unlike parents, there is considerable support for the influence of the smoking behavior of older siblings on the smoking behavior of younger siblings. One ten-year longitudinal study of over 6,000 adolescents found that sibling smoking was one of only four factors that predicted risk of regular smoking and also predicted whether or not respondents would still be smoking after ten years.[46] A study by Hunter and colleagues found race and gender differences in the effect of sibling smoking.[47] Sibling smoking was a strong influence for white males, a sister's smoking was an influence for white females, and a brother's smoking influenced both black males and females.

The influence of one's peers (persons of about the same age who feel a social identification with one another) on smoking behavior has been a topic of considerable research. The findings suggest that peers may be the single most important factor in determining when and how youth first try cigarettes. As I described in my own experience, smoking is frequently a behavior designed to achieve social acceptance from peers as well as an effort to experiment with an otherwise adult activity within a familiar and friendly social setting.[48]

Studies of varying sophistication from all over the world have found a strong relationship between smoking initiation and the influence of peers' or friends' smoking.[49] In the stage of first experimentation with tobacco, most youth are in the company of their best friends. Hahn and associates found that 60% of the 11- through 17-year-olds in their study admitted to first smoking with close friends, and 72% reported smoking most recently with close friends.[50] Among a group of 12- through 14-year-olds, those whose best friend smoked were four times more likely to smoke than those whose best friend did not smoke. The influence of best friends' smoking influenced both experimentation with smoking and smoking prevalence. In a review of sixteen well-designed longitudinal studies there was a positive association between peer smoking and the decision to start smoking in all but one study, and peers appear to influence other stages of tobacco use as well.[51] In other words, youth with friends who smoke are much more likely to smoke themselves. The fact that this association was found in 88% of well-designed studies suggests a clear link between friends' smoking

behavior and cigarette use. This link appears to be influenced by other personal factors, such as one's self-confidence, and its greatest influence is on the earliest stages of smoking. Finally, the strength of the bond or attachment to a peer that smokes appears to also have a strong impact on smoking behavior.

Perceptions of the Social Environment

The perceptions or beliefs that youth have about their social environments can influence their behavior, regardless of whether those perceptions are true or not. For example, middle school kids may believe that "lots of kids" their age smoke and thus may be more likely to begin smoking to fit in. The reality is that only a small percentage of children that age are regular smokers. Perceptions that have been examined for their influence on youth smoking include smoking-related norms, social support, expectations, reactions by peers and parents, and barriers that youth believe to be in place within their environment.

Norms can be thought of as what a person within a particular group believes he or she ought to do or what is believed to be acceptable behavior for a given age group, gender, or other type of social group. Gerber and Newman asked adolescents how many of their classmates were smokers.[52] Youth who increased their smoking over the course of the year were much more likely to believe that a high number of their classmates were smokers than kids who had decreased their smoking in the same period. Similarly, Leventhal and colleagues found that all kids in their study greatly overestimated the number of adults and kids who smoke.[53] Adolescents in their research believed that 66% of their peers and 90% of adults were smokers. They overestimated the prevalence of smoking in both groups by a factor of three. One large study of approximately 3,300 junior high students in California found that adolescents who made rather high estimates of regular smoking prevalence were more likely to try smoking, to become smokers, or to increase the amount they smoked over the course of the study.[54]

Social support includes the perceived approval or disapproval of adolescent cigarette smoking by parents, siblings, peers, and other important people in a youth's life such as teachers or employers. One important source of social support is peer pressure. Peer pressure is not always negative. Peers may actually discourage smoking and this has been used successfully in many prevention programs.[55] In the community where I grew up, alcohol use was strongly encouraged, cigarette use was accepted but not strongly encouraged, and other types of drug use, such as marijuana smoking were strongly discouraged by my peers.

Despite the lack of strong encouragement to smoke by my own peers, there is tremendous peer pressure to smoke. Hahn and colleagues found that the urging of one or more acquaintances, especially peers or close friends, prompted over half of the adolescents in their study to try a cigarette for the first time.[56] Another study found that girls who believed that their friends were more supportive than critical about their smoking were more likely than those who saw their friends as less supportive to become regular smokers one year later.[57] Similarly, many adolescents in another study claimed "my friends like me because I smoke." In the same study, nonsmokers were more likely to report "my parents don't want me to smoke."[58]

Social support can also include the general support or approval of others who are important in the adolescent's life. This type of support seems to play an important role in the beginning stages of smoking. One study found that adolescents who claimed that their parents were generally supportive of them were less likely to start smoking than youth who believed their parents were not supportive. In contrast, though, the same adolescents who believed that their friends were supportive of them were *more* likely to start smoking. Similarly, male adolescents who believed they had limited involvement in family decisions were more likely to become regular smokers than male adolescents from families where high involvement in family decisions was reported.[59] Finally, adolescents who believed that parents, siblings, friends, and teachers would not care if they smoked were at much greater risk of taking up smoking than those who believed these role models would care.[60]

Behavioral Factors

There are several behavioral factors such as academic achievement, risk taking and other problem behaviors, health enhancing behaviors, and smoking-related skills that have been examined for their influence on the risk of smoking among adolescents. These behavioral patterns may provide opportunities to look at smoking as something that has a purpose; it is useful or appropriate in certain situations.

Poor academic achievement measured in a variety of ways (grades, truancy rates, and future professional or educational aspirations) has repeatedly been found to be related to the onset of smoking. For example, Borland and Rudolph looked at the relationship between scholastic performance, parental smoking, and socioeconomic status among 1,814 high school students in Pennsylvania.[61] They found that among those variables, scholastic performance was the strongest predictor of smoking. In other words, those with the highest grades were much less likely to smoke than adolescents with lower grades. This result has

been found in a study of urban black male adolescents in Harlem, New York, as well as among Hispanic and Asian adolescents in southern California.[62] Similarly, kids who disliked school and feared school failure were more likely to start smoking in early adolescence than those who liked school and expected to be successful in school.[63] Conrad and colleagues found that of the twenty-seven longitudinal studies they examined concerning the onset of smoking, 80% found a positive relationship between low academic achievement and the onset of smoking.[64] Finally, in a study by Newcomb and colleagues, what they termed "academic lifestyle orientation" (measured by grades, educational aspirations, personal and professional plans, and expectations) was the principal influence on teenage smoking behavior, teenage emotional well-being, social relationships with smokers, and adult smoking behavior.[65] Clearly academic achievement plays an important role in adolescent smoking behavior.

Cigarette use among adolescents has been examined in association with other types of problem behaviors. For example, cigarette smoking has been linked to further illegal drug use. This suggests that smoking may be an entry-level or gateway drug in a sequence of increasingly serious drug use. This line of research is not claiming that smoking causes illegal drug use, but rather that those who use illegal drugs have most likely smoked cigarettes prior to becoming involved in illegal drugs. Studies by Fleming and colleagues and Newcomb and Bentler point to the crucial role of cigarette smoking in the progression to marijuana and harder drug use.[66] These researchers have found that the increased use of cigarettes was associated with the increased use of illegal drugs. For example, one study found that among a group of adolescents tracked for eight years, those who were using illegal drugs in adulthood were roughly twice as likely to have smoked than those who had not smoked.[67]

Risk-taking behavior has been defined as youth having an inclination toward excitement and taking chances and is generally considered an indicator of overall deviance and rebelliousness. Risk taking has been found to be related to trying a cigarette for the first time in several studies.[68] However, among the twenty-seven well-designed studies reviewed by Conrad and colleagues, only five found a strong relationship between rebelliousness, risk taking, and proneness to deviance to the onset of smoking.[69]

Health-enhancing behavior, such as participation in sports, has been suggested as one means of decreasing the likelihood of deviant behavior. Swan and colleagues found that girls who were involved in at least one organized sport were less likely to smoke.[70] However, involvement in sports did not appear to impact boys' rate of smoking in this study. Similarly, McCaul and colleagues did not find a relationship between boys' smoking and participation in extracurricular activities such as sports.[71]

Other behavioral factors that have been examined are those related to the more immediate social situations surrounding smoking. For example, one study found that 42% of smoking experimenters had asked for their first cigarette.[72] Another study found that difficulty in refusing an offer to smoke was a strong predictor of smoking.[73] Furthermore, this difficulty in refusing an offered cigarette seems to be strongly influenced by the offering friend's attitudes and behaviors (e.g., being persistent or critical if refused), particularly for high-risk adolescents.[74] In the review of the onset of smoking conducted by Conrad and associates, three studies found that refusal or resistance skills against smoking were associated with lower rates of the onset of smoking.[75]

Personal Factors

Personal factors, those characteristics inherent within a person, include thought processes, personal values, personality, and overall emotional well-being. Personal risk factors are the filters through which sociodemographic and environmental factors pass as they influence behavior. Personal risk factors also help to explain why youth who are exposed to similar social environments may behave differently. Several personal factors have been examined for their relation to cigarette smoking, including knowledge of the health consequences of smoking, the utility of smoking for youth, self-esteem, self-image, self-efficacy, personality, and general emotional well-being.

Many, if not most, youth tobacco prevention programs have a strong emphasis on teaching kids about the negative long-term health consequences of smoking. They talk about death rates among smokers from cancer, heart disease, and emphysema. However, knowledge of the long-term health consequences of smoking has not been shown to be related to adolescent smoking behavior.[76] Virtually all adolescents—smokers and nonsmokers—are aware of the long-term health effects of smoking, but most youth have a very short-term outlook on life and tend to feel invulnerable. Many also believe, falsely, that they can quit smoking at any time. Belief that smoking has short-term consequences appears to have a much stronger influence on kids.[77] One study found that knowing the health consequences of smoking had a minimal impact on adolescent smoking. However, adolescents in the beginning stages of smoking appear to begin actively denying the health consequences of their cigarette use.

In the search for the reasons why youth start smoking, many have begun to examine tobacco use from the perspective of the adolescent. Many adolescents who begin to smoke perceive several functional purposes of smoking. For instance, adolescent smokers are more likely to view smoking as a way to act mature or more adult; have fun; cope with personal problems, stress, and

boredom; or rebel against authority.[78] Castro and colleagues found that adolescents in their study smoked cigarettes to help cope with stressful and disruptive family events.[79] In one important study by Hahn and colleagues, regular smokers were asked why they first tried cigarettes and why they had most recently smoked.[80] The vast majority (60%) reported first trying cigarettes out of curiosity. Much smaller percentages of youth reported trying cigarettes to fit in (13%) or because of feeling pressured (10%). In the case of most recent use, these adolescents were most likely to claim that they smoked for pleasure (27%), as compared to feeling dependent (20%), out of curiosity (17%), and fitting in with a social group (10%). Similarly, Chassin and colleagues suggest that positive attitudes toward smoking, such as the idea that smoking is fun or pleasurable, are a better predictor of regular tobacco use than they are for initial experimentation.[81]

Adolescence is a period of identity formation and a time where a sense of self develops from interactions with parents, school, and peers. Self-esteem, or the qualitative self-evaluation, emerges from this process. Several studies have found a link between self-esteem and the onset of smoking. For instance, Young and Werch found that young nonsmokers and those with no intention to start smoking had higher levels of self-esteem relative to family, school, and peers than frequent users and those who intended to smoke in the future.[82] Conrad, Flay, and Hill concluded in their review that self-esteem received fairly consistent support and that it was much better than what they had expected from their examination of earlier, less rigorous studies.[83]

It has been suggested that many adolescents may compensate for low self-esteem by attempting to improve their external image. They may want to appear to others as if they are mature or look "cool." One large study found that youth in all racial and ethnic groups viewed smoking as a way to improve their self-image.[84] Chassin and colleagues found that among adolescents in their study, smoking role models were viewed as tough, sociable, and sexually attractive.[85] Thus youth who assume that smoking creates these positive attributes may also believe that it is a powerful mechanism for self-enhancement. Smoking becomes a vehicle that they perceive will create a positive social image and improve the way others view them—particularly peers.

An adolescent's efficacy (or confidence) in performing certain behaviors has been found to significantly counter the influences of peers who smoke. De Vries, Kok, and Dijkstra found that self-efficacy in resisting offers to smoke was the strongest factor in predicting adolescent smoking over a one-year period in the Netherlands.[86] Thus self-efficacy, or personal confidence, appears to act as a strong internal buffer in protecting many adolescents from the tremendous social pressure to smoke.[87]

Several studies have found a relationship between cigarette smoking and symptoms of depression among adolescents. For instance, Covey and Tam found a relationship between depressed mood and smoking, and further, that depression scores correlated with the number of cigarettes smoked.[88] Smoking may act as a short-term self-medicating response to depression by increasing alertness and feelings of euphoria and calm, but these effects would dissipate in the long run as tolerance to nicotine increases.[89]

CONCLUSION

Tobacco use is the most common cause of premature death in the United States, killing over 400,000 Americans each year. Despite being linked to chronic health problems, such as cancer, emphysema, and heart disease, many young people suffer under the illusion that they can quit smoking at any time and that any negative health effects will occur much later in life. The truth is that the nicotine within tobacco products is highly addictive; teens who smoke regularly become dependent on cigarettes and find it as difficult to quit as adults. Also, cigarette smoking has immediate health effects ranging from decreased physical fitness to arteriosclerosis, an early precursor to heart disease. Smokeless tobacco products have even more immediate health effects. They may lead to oral or stomach cancers within a few years of use.

Cigarette use increased steadily and dramatically in the 1900s, reaching their peak in the 1960s when 42% of adults were smokers. Cigarette use has steadily declined after the first Surgeon General's Report on the health effects on smoking was released in 1964. While cigarette smoking decreased steadily among adults in the 1970s, 1980s, and 1990s, cigarette use declined less rapidly among youth, and actually increased throughout most of the 1990s. Currently about 13% of middle school and 28% of high school students use some type of tobacco products.

Youth start smoking in progressive stages. In the first stage, general positive attitudes about smoking are formed. Stage 2 is when a youth actually experiments with tobacco for the first time. Stage 3 involves repeated but irregular tobacco use. In stage 4, regular tobacco use leads to addiction and dependence in stage 5. Throughout each of these stages, a number of psychosocial and personal factors are involved in the decision to start smoking and to persist as a smoker including; the availability of cigarettes, the social acceptance of smoking, peer and parenting influences, perceptions of the social environment, self-esteem, and self-image. One of the reasons that many youth come to believe in the social acceptance of smoking has to do with the pervasiveness of pro-tobacco messages. The tobacco industry spends more than $10 billion a year

to convince consumers that smoking will enhance one's personal image, their social acceptance, and to make it look as though tobacco use is normative and valued in American culture. Despite the tobacco industry's claims, these messages continue to impress youth.

NOTES

1. From *Peddling Poison: The Tobacco Industry and Kids*, Clete Snell; Chapter 1, pp 1–18, "Youth Tobacco Use." Copyright © 2005 by Clete Snell. Reproduced with permission of Greenwood Publishing Group, Inc. Westport, CT.
2. U.S. Department of Health and Human Services (USDHHS), *Reducing the Health Consequences of Smoking: 25 Years of Progress*. A Report of the Surgeon General (Atlanta, GA: USDHHS, 1989).
3. W. F. Chinnock, "The Family Courts' Mandatory Duty to Restrain Parents and Other Persons from Smoking around Children," *Arizona Law Review* 45 (2003): 801–20.
4. USDHHS, *Reducing the Health Consequences of Smoking.*
5. American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., text rev. (Washington, DC: American Psychiatric Association, 2000).
6. USDHHS, *The Health Benefits of Cessation: A Report of the Surgeon General* (Atlanta, GA: USDHHS, 1990); Pierce et al., "Trends in Cigarette Smoking in the United States," 61–65.
7. R. R. West and D. A. Evans, "Lifestyle Changes in Long Term Survivors of Acute Myocardial Infarction," *Journal of Epidemiology and Community Health* 40, no. 2 (1986): 103–9.
8. J. E. Henningfield, R. Clayton, and W. Pollin, "Involvement of Tobacco in Alcoholism and Illicit Drug Use," *British Journal of Addiction* 85, no. 2 (1990): 279–92.
9. G. A. Giovino, M. W. Schooley, Z. Bao-Ping, J. H. Chrismon, S. L. Tomar, J. P. Peddicord, R. K. Merritt, C. G. Husten, and M. P. Eriksen, "Surveillance for Selected Tobacco-Use Behaviors—United States, 1900–1994," *Morbidity and Mortality Weekly Report* 43 (1994): 1–43.
10. Ibid., 6–7.
11. Ibid.
12. Ibid., 11, 19.
13. Ibid., 19.
14. Ibid., 34.
15. Ibid., 33.
16. Centers for Disease Control and Prevention (CDC), "Trends in Cigarette Smoking among High School Students—United States, 1991–2001," *Morbidity and Mortality Weekly Report* 51, no. 19 (2002): 409–12.
17. Ibid., 409.

18. J. A. Allen, D. Vallone, M. L. Haviland, C. Healton, K. C. Davis, M. C. Farrelly, C. G. Husten, and T. Pechacek, "Tobacco Use among Middle and High School Students—United States, 2002," *Morbidity and Mortality Weekly Report* 52, no. 45 (2003): 1096–98.
19. Ibid.
20. USDHHS, *Reducing the Health Consequences of Smoking.*
21. Public Health Service (PHS), *Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service,* PHS Publication no. 1103 (Washington, DC: U.S. Department of Health, Education, and Welfare, 1964).
22. H. Leventhal, "Experimental Studies of Anti-smoking Communications," in *Smoking, Health, and Behavior,* ed. E. F. Borgatta and R. R. Evans (Chicago: Aldine, 1968), 95–121; A. Bandura, *Social Learning Theory* (Englewood Cliffs, NJ: Prentice Hall, 1977); McAlister, Perry, and Maccoby, "Adolescent Smoking: Onset and Prevention," 650–658.
23. K. M. Conrad, B. R. Flay, and D. Hill, "Why Children Start Smoking Cigarettes: Predictors of Onset," *British Journal of Addiction* 87, no. 12 (1992): 1711–24.
24. B. R. Flay, "Youth Tobacco Use: Risks, Patterns, and Control," in *Nicotine Addiction: Principles and Management,* ed. J. Slade and C. T. Orleans (New York: Oxford University Press, 1993), 365–84.
25. C. L. Perry, D. M. Murray, and K. I. Klepp, "Predictors of Adolescent Smoking and Implications for Prevention," *Morbidity and Mortality Weekly Report* 3, no. 4 (1987): 41.
26. A. D. McNeill, "The Development of Dependence on Smoking in Children," *British Journal of Addiction* 86, no. 5 (1991): 589–92.
27. R. I. Evans, R. M. Rozelle, M. B. Mittelmark, W. B. Hansen, A. L. Bane, and J. Havis, "Deterring the Onset of Smoking in Children: Knowledge of Immediate Physiological Effects and Coping with Peer Pressure, Media Pressure, and Parent Modeling," *Journal of Applied Social Psychology* 8, no. 2 (1978): 126–35.
28. Conrad et al., "Why Children Start Smoking."
29. N. K. Semmer, P. D. Cleary, J. H. Dwyer, R. Fuchs, and P. Lippert, "Psychosocial Predictors of Adolescent Smoking in Two German Cities: The Berlin-Bremen Study," *Morbidity and Mortality Weekly Report* 36, no. 4 (1987): 35–105.
30. C. L. Perry, S. H. Kelder, and K. A. Komro, "The Social World of Adolescents: Family, Peers, Schools, and the Community," in *Promoting the Health of Adolescents: New Directions for the Twenty-first Century,* ed. S. G. Millstein, A. C. Petersen, and E. O. Nightingale (New York: Oxford University Press, 1993), 73–96.
31. H. M. Alexander, R. Callcott, A. J. Dobson, G. R. Hardes, D. M. Lloyd, D. L. O'Connell, and S. R. Leeder, "Cigarette Smoking and Drug Use in School Children: IV—Factors Associated with Changes in Smoking Behavior," *International Journal of Epidemiology* 12, no. 1 (1983): 59–66.
32. D. B. Kandel and J. A. Logan, "Patterns of Drug Use from Adolescence to Young Adulthood: Periods of Risk for Initiation, Continued Use, and Discontinuation," *American Journal of Public Health* 74, no. 7 (1984): 660–66.

33. G. Konopka, "Adolescence, Concept of, and Requirements for a Healthy Development," in *Encyclopedia of Adolescence,* vol. 1, ed. R. M. Lemer, A. C. Petersen, and J. Brooks-Gunn (New York: Garland, 1991).
34. S. Sussman, C. W. Dent, B. R. Flay, W. B. Hansen, and C. A. Johnson, "Psychosocial Predictors of Cigarette Smoking Onset by White, Black, Hispanic, and Asian Adolescents in Southern California," *Morbidity and Mortality Weekly Report* 36, no. 4 (1987): 11S–16S.
35. M. C. Fiore, T. N. Novotny, J. P. Pierce, E. J. Harziandreu, K. M. Patei, and R. M. Davis, "Trends in Cigarette Smoking in the United States: The Changing Influence of Gender and Race," *Journal of the American Medical Association* 26, no. 1 (1989): 49–55.
36. S. Clayton, "Gender Differences in Psychosocial Determinants of Adolescent Smoking," *Journal of School Health* 61, no. 3 (1991): 115–20.
37. E. R. Gritz and L. A. Crane, "Use of Diet Pills and Amphetamines to Lose Weight among Smoking and Nonsmoking High School Seniors," *Health Psychology* 10, no. 5 (1991): 33–35.
38. American School Health Association, et al., *The National Adolescent Health Survey: A Report on the Health of America's Youth* (Oakland, CA: Third Party, 1989).
39. CDC, "Accessibility of Cigarettes to Youths Aged 12–17 Years—United States, 1989," *Morbidity and Mortality Weekly Report* 41, no. 27 (1992): 485–88.
40. N. K. Semmer, P. Lippert, R. Fuchs, P. D. Cleary, and A. Schindler, "Adolescent Smoking from a Functional Perspective: The Berlin-Bremen Study," *European Journal of Psychology of Education* 2, no. 4 (1987): 387–401.
41. L. Lawrence and L. Robinson, "Self-Efficacy as a Predictor of Smoking Behavior in Young Adolescents," *Addictive Behaviors* 11, no. 4 (1986): 367–82.
42. L. Chassin, C. C. Presson, S. J. Sherman, D. Montello, and J. McGrew, "Changes in Peer and Parent Influence during Adolescence: Longitudinal versus Cross-Sectional Perspectives on Smoking Initiation," *Developmental Psychology* 22, no. 3 (1986): 327–34; see also Fiore et al., "Trends in Cigarette Smoking."
43. E. M. Botvin, "Correlates and Predictors of Smoking among Black Adolescents," *Addictive Behaviors* 17, no. 2 (1992): 97–103; M. B. Mittelmark, D. M. Murray, R. V. Luepker, T. F. Pechacek, P. L. Pirie, and U. E. Pallonen, "Predicting Experimentation with Cigarettes: The Childhood Antecedents of Smoking Study (CASS)," *American Journal of Public Health* 77, no. 2 (1987): 206–8.
44. Conrad et al., "Why Children Start Smoking."
45. L. Chassin, C. C. Presson, S. J. Sherman, D. Montello, E. Corty, and R. W. Olshavsky, "Predicting the Onset of Cigarette Smoking in Adolescents: A Longitudinal Study," *Journal of Applied Social Psychology* 14, no. 3 (1984): 224–43.
46. A. V. Swan, R. Cresser, and M. Murray, "When and Why Children First Start to Smoke," *International Journal of Epidemiology* 19, no. 2 (1990): 323–30.
47. S. M. Hunter, J. B. Croft, I. A. Vizelberg, and G. S. Berenson, "Psychosocial Influences on Cigarette Smoking among Youth in a Southern Community: The

Bogalusa Heart Study," *Morbidity and Mortality Weekly Report* 26, no. 4 (1987): 17S–25S.

48. B. R. Flay, J. R. D'Avernas, J. A. Best, M. W. Kersell, and K. B. Ryan, "Cigarette Smoking: Why Young People Do It and Ways of Preventing It," in *Pediatric and Adolescent Behavioral Medicine*, ed. I. McGrath and P. Firestone (New York: Springer, 1983), 132–83.
49. D. L. O'Connell, H. M. Alexander, A. J. Dobson, D. M. Lloyd, G. R. Hardes, H. J. Springthorpe, and S. J. Leeder, "Cigarette Smoking and Drug Use in Schoolchildren: Factors Associated with Smoking," *International Journal of Epidemiology* 10, no. 3 (1981): 223–31; H. Ogawa, S. Tominaga, G. Gellert, and K. Aoki, "Smoking among Junior High School Students in Nagoya, Japan," *International Journal of Epidemiology* 17, no. 4 (1988): 814–20.
50. G. Hahn, V. L. Charlin, S. Sussman, C. W. Dent, J. Manzi, A. W. Stacy, B. Flay, W. B. Hansen, and D. Burton, "Adolescents' First and Most Recent Use Situations of Smokeless Tobacco and Cigarettes: Similarities and Differences," *Addictive Behaviors* 15, no. 5 (1990): 439–48.
51. M. D. Newcomb, W. J. McCarthy, and P. M. Bentler, "Cigarette Smoking, Academic Lifestyle, and Social Impact Efficacy: An Eight-Year Study from Early Adolescence to Young Adulthood," *Journal of Applied Social Psychology* 19, no. 3 (1989): 251–81.
52. R. W. Gerber and I. M. Newman, "Predicting Future Smoking of Adolescent Experimental Smokers," *Journal of Youth and Adolescence* 18, no. 2 (1989): 191–201.
53. Leventhal, Fleming, and Glynn, "A Cognitive-Developmental Approach to Smoking Intervention," 79–109.
54. L. M. Collins, S. Sussman, C. A. Johnson, W. B. Hansen, and B. R. Flay, "Psychosocial Predictors of Young Adolescent Cigarette Smoking: A Sixteen-Month, Three-Wave Longitudinal Study," *Journal of Applied Social Psychology* 17, no. 6 (1987): 554–73.
55. K. I. Klepp, A. Halper, and C. L. Perry, "The Efficacy of Peer Leaders in Drug Abuse Prevention," *Journal of School Health* 56, no. 9 (1986): 407–11.
56. Hahn et al., "Adolescents' First and Most Recent Use Situations."
57. Chassin et al., "Changes in Peer and Parent Influence."
58. Hunter et al., "Psychosocial Influences on Cigarette Smoking."
59. Mittelmark et al., "Predicting Experimentation."
60. Swan et al., "When and Why Children First Start to Smoke."
61. B. L. Borland and J. P. Rudolph, "Relative Effects of Low Socioeconomic Status, Parental Smoking and Poor Scholastic Performance on Smoking among High School Students," *Social Science and Medicine* 9, no. 1 (1975): 27–30.
62. Brunswick and Messeri, "Origins of Cigarette Smoking," 353–370; Sussman et al., "Psychosocial Predictors."
63. A. Ahlgren, A. A. Norem, M. Hochhauser, and J. Garvin, "Antecedents of Smoking among Preadolescents," *Journal of Drug Education* 12, no. 4 (1982): 325–40.
64. Conrad et al., "Why Children Start Smoking."

65. Newcomb et al., "Cigarette Smoking, Academic Lifestyle."
66. R. Fleming, H. Leventhal, K. Glynn, and J. Ershler, "The Role of Cigarettes in the Initiation and Progression of Early Substance Use," *Addictive Behaviors* 14, no. 3 (1989): 261–72; M. D. Newcomb and P. M. Bentler, "Frequency and Sequence of Drug Use: A Longitudinal Study from Early Adolescence to Young Adulthood," *Journal of Drug Education* 16, no. 2 (1986): 101–18.
67. Brunswick and Messeri, "Causal Factors."
68. Leventhal, Fleming, and Glynn, "A Cognitive-Developmental Approach to Smoking Intervention"; Collins et al., "Psychosocial Predictors"; Sussman et al., "Psychosocial Predictors."
69. Conrad et al., "Why Children Start Smoking."
70. Swan et al., "When and Why Children First Start to Smoke."
71. K. D. McCaul, R. Glasgow, H. K. O'Neill, V. Freeborn, and B. S. Rump, "Predicting Adolescent Smoking," *Journal of School Health* 52, no. 8 (1982): 342–46.
72. Hahn et al., "Adolescents' First and Most Recent Use Situations."
73. Sussman et al., "Psychosocial Predictors."
74. Lawrence and Robinson, "Self-Efficacy as a Predictor."
75. Conrad et al., "Why Children Start Smoking."
76. Conrad et al., "Why Children Start Smoking"; Collins et al., "Psychosocial Predictors."
77. A. D. McNeill, M. J. Jarvis, J. A. Stapleton, M. A. H. Russell, J. R. Elser, P. Gammage, and E. M. Gray, "Prospective Study of Factors Predicting Uptake of Smoking in Adolescents," *Journal of Epidemiology and Community Health* 43, no. 1 (1988): 72–78.
78. Perry et al., "Predictors of Adolescent Smoking."
79. F. G. Castro, E. Maddahian, M. D. Newcomb, and P. M. Bentler, "A Multivariate Model of the Determinants of Cigarette Smoking among Adolescents," *Journal of Health and Social Behavior* 28, no. 3 (1987): 273–89.
80. Hahn et al., "Adolescents' First and Most Recent Use Situations."
81. Chassin et al., "Predicting the Onset of Cigarette Smoking."
82. M. Young and C. E. Werch, "Relationship between Self-Esteem and Substance Use among Students in Fourth through Twelfth Grade," *Wellness Perspectives, Research, Theory and Practice* 7, no. 2 (1990): 31–44.
83. Conrad et al., "Why Children Start Smoking."
84. Sussman et al., "Psychosocial Predictors."
85. L. Chassin, C. C. Presson, and S. J. Sherman, "Social Psychological Contributions to the Understanding and Prevention of Adolescent Cigarette Smoking," *Personality and Social Psychology Bulletin* 16, no. 1 (1990): 133–51.
86. H. De Vries, G. Kok, and M. Dijkstra, "Self-Efficacy as a Determinant of the Onset of Smoking and Interventions to Prevent Smoking in Adolescents," in *European Perspectives in Psychology, Clinical, Health, Stress and Anxiety, Neuropsychology, Psychophysiology*, ed. P. J. Drenth, J. A. Sergeant, and R. J. Takens (New York: John Wiley, 1990), 209–22.
87. Conrad et al., "Why Children Start Smoking."

88. L. S. Covey and D. Tam, "Depressive Mood, the Single-Parent Home, and Adolescent Smoking Behavior," *American Journal of Public Health* 80, no. 11 (1990): 1330–33.
89. Newcomb et al., "Cigarette Smoking, Academic Lifestyle."

BIBLIOGRAPHY

Ahlgren, A., A. A. Norem, M. Hochhauser, and J. Garvin. "Antecedents of Smoking among Preadolescents." *Journal of Drug Education* 12, no. 4 (1982): 325–40.

Alexander, H. M., R. Callcott, A. J. Dobson, G. R. Hardes, D. M. Lloyd, D. L. O'Connell, and S. R. Leeder. "Cigarette Smoking and Drug Use in School Children: IV—Factors Associated with Changes in Smoking Behavior." *International Journal of Epidemiology* 12, no. 1 (1983): 59–66.

Allen, J. A., D. Vallone, M. L. Haviland, C. Healton, K. C. Davis, M. C. Farrelly, C. G. Husten, and T. Pechacek. "Tobacco Use among Middle and High School Students—United States, 2002." *Morbidity and Mortality Weekly Report* 52, no. 45 (2003): 1096–98.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed., text rev. Washington, DC: American Psychiatric Association, 2000.

American School Health Association, et al. *The National Adolescent Health Survey: A Report on the Health of America's Youth*. Oakland, CA: Third Party, 1989.

Bandura, A. *Social Learning Theory*. Englewood Cliffs, NJ: Prentice Hall, 1977.

Borland, B. L., and J. P. Rudolph. "Relative Effects of Low Socioeconomic Status, Parental Smoking and Poor Scholastic Performance on Smoking among High School Students." *Social Science and Medicine* 9, no. 1 (1975): 27–30.

Botvin, E. M. "Correlates and Predictors of Smoking among Black Adolescents." *Addictive Behaviors* 17, no. 2 (1992): 97–103.

Brunswick, A. F., and P. A. Messeri. "Causal Factors in Onset of Adolescents' Cigarette Smoking: A Prospective Study of Urban Black Youth." *Advances in Alcohol and Substance Abuse* 3, nos. 1–2 (1983): 35–52.

Castro, F. G., E. Maddahian, M. D. Newcomb, and P. M. Bentler. "A Multivariate Model of the Determinants of Cigarette Smoking among Adolescents." *Journal of Health and Social Behavior* 28, no. 3 (1987): 273–89.

Centers for Disease Control and Prevention. "Accessibility of Cigarettes to Youths Aged 12–17 Years—United States, 1989." *Morbidity and Mortality Weekly Report* 41, no. 27 (1992): 485–88.

———. "Trends in Cigarette Smoking among High School Students—United States, 1991–2001." *Morbidity and Mortality Weekly Report* 51, no. 19 (2002): 409–12.

Chassin, L., C. C. Presson, and S. J. Sherman. "Social Psychological Contributions to the Understanding and Prevention of Adolescent Cigarette Smoking." *Personality and Social Psychology Bulletin* 16, no. 1 (1990): 133–51.

Chassin, L., C. C. Presson, S. J. Sherman, D. Montello, E. Corty, and R. W. Olshavsky. "Predicting the Onset of Cigarette Smoking in Adolescents: A Longitudinal Study." *Journal of Applied Social Psychology* 14, no. 3 (1984): 224–43.

Chassin, L., C. C. Presson, S. J. Sherman, D. Montello, and J. McGrew. "Changes in Peer and Parent Influence during Adolescence: Longitudinal versus Cross-Sectional Perspectives on Smoking Initiation." *Developmental Psychology* 22, no. 3 (1986): 327–34.

Chinnock, W. F. "The Family Courts' Mandatory Duty to Restrain Parents and Other Persons from Smoking around Children." *Arizona Law Review* 45 (2003): 801–20.

Clayton, S. "Gender Differences in Psychosocial Determinants of Adolescent Smoking." *Journal of School Health* 61, no. 3 (1991): 115–20.

Collins, L. M., S. Sussman, C. A. Johnson, W. B. Hansen, and B. R. Flay. "Psychosocial Predictors of Young Adolescent Cigarette Smoking: A Sixteen-Month, Three-Wave Longitudinal Study." *Journal of Applied Social Psychology* 17, no. 6 (1987): 554–73.

Conrad, K. M., B. R. Flay, and D. Hill. "Why Children Start Smoking Cigarettes: Predictors of Onset." *British Journal of Addiction* 87, no. 12 (1992): 1711–24.

Covey, L. S., and D. Tam. "Depressive Mood, the Single-Parent Home, and Adolescent Smoking Behavior." *American Journal of Public Health* 80, no. 11 (1990): 1330–33.

De Vries, H., G. Kok, and M. Dijkstra. "Self-Efficacy as a Determinant of the Onset of Smoking and Interventions to Prevent Smoking in Adolescents." In *European Perspectives in Psychology, Clinical, Health, Stress and Anxiety, Neuropsychology, Psychophysiology*, edited by P. J. Drenth, J. A. Sergeant, and R. J. Takens, 209–22. New York: John Wiley, 1990.

Evans, R. I., R. M. Rozelle, M. B. Mittelmark, W. B. Hansen, A. L. Bane, and J. Havis. "Deterring the Onset of Smoking in Children: Knowledge of Immediate Physiological Effects and Coping with Peer Pressure, Media Pressure, and Parent Modeling." *Journal of Applied Social Psychology* 8, no. 2 (1978): 126–35.

Fiore, M. C., T. N. Novotny, J. P. Pierce, E. J. Harziandreu, K. M. Patei, and R. M. Davis. "Trends in Cigarette Smoking in the United States: The Changing Influence of Gender and Race." *Journal of the American Medical Association* 26, no. 1 (1989): 49–55.

Flay, B. R. "Youth Tobacco Use: Risks, Patterns, and Control." In *Nicotine Addiction: Principles and Management*, edited by J. Slade and C. T. Orleans, 365–84. New York: Oxford University Press, 1993.

Flay, B. R., J. R. D'Avernas, J. A. Best, M. W. Kersell, and K. B. Ryan. "Cigarette Smoking: Why Young People Do It and Ways of Preventing It." In *Pediatric and Adolescent Behavioral Medicine*, edited by I. McGrath and P. Firestone, 132–83. New York: Springer, 1983.

Fleming, R., H. Leventhal, K. Glynn, and J. Ershler. "The Role of Cigarettes in the Initiation and Progression of Early Substance Use." *Addictive Behaviors* 14, no. 3 (1989): 261–72.

Gerber, R. W., and I. M. Newman. "Predicting Future Smoking of Adolescent Experimental Smokers." *Journal of Youth and Adolescence* 18, no. 2 (1989): 191–201.

Giovino, G. A., M. W. Schooley, Z. Bao-Ping, J. H. Chrismon, S. L. Tomar, J. P. Peddicord, R. K. Merritt, C. G. Husten, and M. P. Eriksen. "Surveillance for Selected

Tobacco-Use Behaviors—United States, 1900–1994." *Morbidity and Mortality Weekly Report* 43 (1994): 1–43.

Gritz, E. R., and L. A. Crane. "Use of Diet Pills and Amphetamines to Lose Weight among Smoking and Nonsmoking High School Seniors." *Health Psychology* 10, no. 5 (1991): 33–35.

Hahn, G., V. L. Charlin, S. Sussman, C. W. Dent, J. Manzi, A. W. Stacy, B. Flay, W. B. Hansen, and D. Burton. "Adolescents' First and Most Recent Use Situations of Smokeless Tobacco and Cigarettes: Similarities and Differences." *Addictive Behaviors* 15, no. 5 (1990): 439–48.

Henningfield, J. E., R. Clayton, and W. Pollin. "Involvement of Tobacco in Alcoholism and Illicit Drug Use." *British Journal of Addiction* 85, no. 2 (1990): 279–92.

Hunter, S. M., J. B. Croft, I. A. Vizelberg, and G. S. Berenson. "Psychosocial Influences on Cigarette Smoking among Youth in a Southern Community: The Bogalusa Heart Study." *Morbidity and Mortality Weekly Report* 26, no. 4 (1987): 17S–25S.

Kandel, D. B., and J. A. Logan. "Patterns of Drug Use from Adolescence to Young Adulthood: Periods of Risk for Initiation, Continued Use, and Discontinuation." *American Journal of Public Health* 74, no. 7 (1984): 660–66.

Klepp, K. I., A. Halper, and C. L. Perry. "The Efficacy of Peer Leaders in Drug Abuse Prevention." *Journal of School Health* 56, no. 9 (1986): 407–11.

Konopka, G. "Adolescence, Concept of, and Requirements for a Healthy Development." In *Encyclopedia of Adolescence.* Vol. 1, edited by R. M. Lemer, A. C. Petersen, and J. Brooks-Gunn. New York: Garland, 1991.

Lawrence, L., and L. Robinson. "Self-Efficacy as a Predictor of Smoking Behavior in Young Adolescents." *Addictive Behaviors* 11, no. 4 (1986): 367–82.

Leventhal, H. "Experimental Studies of Anti-smoking Communications." In *Smoking, Health, and Behavior,* edited by E. F. Borgatta and R. R. Evans, 95–121. Chicago: Aldine, 1968.

McCaul, K. D., R. Glasgow, H. K. O'Neill, V. Freeborn, and B. S. Rump. "Predicting Adolescent Smoking." *Journal of School Health* 52, no. 8 (1982): 342–46.

McNeill, A. D. "The Development of Dependence on Smoking in Children." *British Journal of Addiction* 86, no. 5 (1991): 589–92.

McNeill, A. D., M. J. Jarvis, J. A. Stapleton, M.A.H. Russell, J. R. Elser, P. Gammage, and E. M. Gray. "Prospective Study of Factors Predicting Uptake of Smoking in Adolescents." *Journal of Epidemiology and Community Health* 43, no. 1 (1988): 72–78.

Mittelmark, M. B., D. M. Murray, R. V. Luepker, T. F. Pechacek, P. L. Pirie, and U. E. Pallonen. "Predicting Experimentation with Cigarettes: The Childhood Antecedents of Smoking Study (CASS)." *American Journal of Public Health* 77, no. 2 (1987): 206–8.

Newcomb, M. D., and P. M. Bentler. "Frequency and Sequence of Drug Use: A Longitudinal Study from Early Adolescence to Young Adulthood." *Journal of Drug Education* 16, no. 2 (1986): 101–18.

Newcomb, M. D., W. J. McCarthy, and P. M. Bentler. "Cigarette Smoking, Academic Lifestyle, and Social Impact Efficacy: An Eight-Year Study from Early Adoles-

cence to Young Adulthood." *Journal of Applied Social Psychology* 19, no. 3 (1989): 251–81.

O'Connell, D. L., H. M. Alexander, A. J. Dobson, D. M. Lloyd, G. R. Hardes, H. J. Springthorpe, and S. J. Leeder. "Cigarette Smoking and Drug Use in Schoolchildren: Factors Associated with Smoking." *International Journal of Epidemiology* 10, no. 3 (1981): 223–31.

Ogawa, H., S. Tominaga, G. Gellert, and K. Aoki. "Smoking among Junior High School Students in Nagoya, Japan." *International Journal of Epidemiology* 17, no. 4 (1988): 814–20.

Perry, C. L., S. H. Kelder, and K. A. Komro. "The Social World of Adolescents: Family, Peers, Schools, and the Community." In *Promoting the Health of Adolescents: New Directions for the Twenty-first Century,* edited by S. G. Millstein, A. C. Petersen, and E. O. Nightingale, 73–96. New York: Oxford University Press, 1993.

Perry, C. L., D. M. Murray, and K. I. Klepp. "Predictors of Adolescent Smoking and Implications for Prevention." *Morbidity and Mortality Weekly Report* 3, no. 4 (1987): 41.

Public Health Service. *Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service*. PHS Publication no. 1103. Washington, DC: U.S. Department of Health, Education, and Welfare, 1964.

Semmer, N. K., P. D. Cleary, J. H. Dwyer, R. Fuchs, and P. Lippert. "Psychosocial Predictors of Adolescent Smoking in Two German Cities: The Berlin-Bremen Study." *Morbidity and Mortality Weekly Report* 36, no. 4 (1987): 35–105.

Semmer, N. K., P. Lippert, R. Fuchs, P. D. Cleary, and A. Schindler. "Adolescent Smoking from a Functional Perspective: The Berlin-Bremen Study." *European Journal of Psychology of Education* 2, no. 4 (1987): 387–401.

Sussman, S., C. W. Dent, B. R. Flay, W. B. Hansen, and C. A. Johnson. "Psychosocial Predictors of Cigarette Smoking Onset by White, Black, Hispanic, and Asian Adolescents in Southern California." *Morbidity and Mortality Weekly Report* 36, no. 4 (1987): 11S–16S.

Swan, A. V., R. Cresser, and M. Murray. "When and Why Children First Start to Smoke." *International Journal of Epidemiology* 19, no. 2 (1990): 323–30.

U.S. Department of Health and Human Services. *The Health Benefits of Cessation: A Report of the Surgeon General.* Atlanta, GA: U.S. Department of Health and Human Services, 1990.

———. *Reducing the Health Consequences of Smoking: 25 Years of Progress. A Report of the Surgeon General.* Atlanta, GA: U.S. Department of Health and Human Services, 1989.

West, R. R., and D. A. Evans. "Lifestyle Changes in Long Term Survivors of Acute Myocardial Infarction." *Journal of Epidemiology and Community Health* 40, no. 2 (1986): 103–9.

Young, M., and C. E. Werch. "Relationship between Self-Esteem and Substance Use among Students in Fourth through Twelfth Grade." *Wellness Perspectives, Research, Theory and Practice* 7, no. 2 (1990): 31–44.

This page intentionally left blank

Chapter **16**

Brewing Trouble: Some Employment Implications of Regular Malt Liquor Beer Consumption

Va Nee L. Van Vleck, PhD, and Thomas K. Greenfield, PhD

We examine the association between *regularly* using malt liquor beer products and poorer employment conditions. This study proves that beyond the socioeconomic factors contributing to both choice of alcoholic beverage and employment condition, the choice of high-potency beer products increases the likelihood of nonparticipation and unemployment. Using the 2000 National Alcohol Survey (NAS; including targeted metropolitan oversamples), bivariate probit estimation was used because of the anticipated simultaneity. Regularly drinking malt liquor beer is negatively associated with employment condition even after controlling for endogeneity and sociodemographic characteristics, though the association may not be directly causal. The results suggest that a propensity toward regularly drinking high-potency beer products may exacerbate the likelihood of unemployment and nonparticipation in the labor force.

Alarm over high-potency alcoholic beverages and their disproportionate consumption by racial and ethnic minorities in the United States (National Institute on Alcohol Abuse and Alcoholism, 2000) brings to the fore two related economic problems: first, among those who drink, how much does poor socioeconomic status (SES) increase the likelihood of drinking inexpensive, high-potency alcoholic beverages such as malt liquor? This issue bears on the price sensitivity of specific SES groups. Additionally, how much might high-potency alcohol products adversely affect individual productivity and employability, thereby impairing socioeconomic progress? This second question is the emphasis in the present analysis. There is an urgency to this research question as the demographics of high-potency alcoholic beverage consumers become

more rigorously established (Bluthenthal, Brown Taylor, Guzman-Becerra, & Robinson, 2005; Brown Taylor, 2001). While in the United States beer drinking itself (compared to wine and spirits) is associated with hazardous consumption levels (Rogers & Greenfield, 1999), malt liquor consumption may confer additional risks. For example, in a sample of inner-city, lower-income individuals, predominantly African American, malt liquor use (in contrast to regular beer drinking) was associated with being unemployed, being homeless, or receiving public housing assistance (Bluthenthal et al., 2005). Often costing less than popular beer brands, these products are heavily marketed to and consumed by racial/ethnic younger adults (Allen-Taylor, 1997; Elliott, 2002; Gratz, 1997; Halbfinger, 1997; Herd, 1994, 2005; Powell, 1996)—groups already at increased risk for unemployment, underemployment, and lower wages and earnings in the United States.

Success in the labor market can be elusive for the young and minorities. Obtaining an initial job interview itself can be a significant hurdle. A field experiment by Bertrand and Mullainathan (2004) demonstrated that a comparably qualified résumé submitted under an African American–sounding name (e.g., Lakisha) was significantly less likely to receive a callback compared to a résumé with a white-sounding name (e.g., Emily); this was also true for male names. Even less well understood is *under*employment and work mismatch among the young and racial/ethnic minorities. A greater likelihood of part-time employment, which is less likely to include benefits or offer long-term stability, may put these workers in the lower-income quintiles (Kalleberg, Reskin, & Hudson, 2000; see also Reynolds, 2003).

EMPLOYMENT CIRCUMSTANCES, HEALTH, AND ALCOHOL

The recursive relationship between (long-term) health status and (long-term) economic status is well established and is admittedly complicated. Ill health makes it more difficult to participate (or participate fully) in the labor force or to secure employment, and long and/or repeating spells of unemployment contribute to poorer self-reports of health (Mullahy & Sindelar, 1995; Ross & Mirowsky, 1995; Turner, 1995). Low SES, in addition to race/ethnicity, contributes to health disparities as well as additional vulnerabilities and deficiencies in access to and quality of programs for alcohol problems (Schmidt, Greenfield, & Mulia, 2006; Schmidt, Ye, Greenfield, & Bond, 2007). We have reason to believe that some malt liquor beer drinkers may be unaware of any distinction between malt liquor products and ordinary beer (beyond price); specifically, they may not know that malt liquor beers are higher in ethanol con-

tent (Bluthenthal et al., 2005) than most popular and premium beer brands. (See also Kuo, Wechsler, Greenberg, & Lee, 2003, for a look at the role of prices in the marketing of alcohol to young adults.) Therefore, by choosing the affordable beverage, they may inadvertently consume more alcohol per drinking episode than an otherwise comparable drinker would. Alternatively, some malt liquor beer drinkers for whom the stronger alcohol content is itself a desirable beverage characteristic may be seeking intoxication and feel that the product ensures more bang for the buck. Also, consistent with how the product is advertised, malt liquor drinking may seem associated with celebrity, wealth, and identification with a desirable subculture, as Pampel (2006) has argued for the relationship between SES and smoking. These individuals may have either opted out of the full-time labor market or be willing to risk adverse job and economic consequences in favor of differential sociocultural expression.

Long-term illnesses have been documented as drags on subsequent wages earned, hours worked, and participation in the labor force (Bartel & Taubman, 1979; Bartley & Plewis, 2002; Mullahy & Sindelar, 1995; Pelkowski & Berger, 2004). For example, diabetes has been shown to reduce employment rates, working days, and salaries among men; treatment advances, in turn, have been associated with improved labor force participation (among women) and reduced earnings disparities (Kahn, 1998; see also Brown, Pagan, & Bastida, 2005). Similarly, mental illnesses are associated with reduced employability and lower incomes (Catalano, 1999; Ettner, Frank, & Kessler, 1997; Hamilton, Merrigan, & Dufresne, 1997; see also Marcotte & Wilcox-Gok, 2001). The persistence of these adverse effects may dissipate with time, unless there are recurring episodes of illness (Marcotte, Wilcox-Gok, & Redmon, 2000). Importantly, alcohol dependence (and other chronic substance abuse) shares significant patterns in common with more *values-neutral* chronic diseases (such as diabetes, hypertension, and asthma), including genetic and environmental predictors, physiological impacts, treatment compliance, and relapse rates (McLellan, Lewis, O'Brien, & Kleber, 2000; Weisner, 1997).

ALCOHOL ABUSE AND EMPLOYMENT PROBLEMS

The relationship between alcohol consumption and employment has been addressed most often in the context of problem drinking (variously defined) using cross-sectional data. In a series of articles, Mullahy and Sindelar probed the associations between alcohol abuse and labor market outcomes: schooling, earnings, and occupation (Mullahy & Sindelar, 1989); unemployment (Mullahy & Sindelar, 1991); employment and income levels (Mullahy & Sindelar, 1993); indirect effects via education and marital status (Mullahy & Sindelar, 1994);

employment and unemployment (Mullahy & Sindelar, 1996); and occupational choice (Mullahy & Sindelar, 1998). Mullahy and Sindelar (1989) offered a conjecture of amplified differentials of alcoholism across the life cycle, which now appear to be supported by the findings of Jones and Richmond (2006). Using National Longitudinal Survey of Youth (NLSY) data, Jones and Richmond (2006) quantified significant productivity losses from alcoholism, for both men and women. Recently, *heavy drinking*—episodes characterized by the interaction of quantity, frequency, and temporal pattern, not simply consumption volume—has received some attention in relation to the likelihood of criminal behavior, assessed by arrests (French, Turner, & Greenfield, 2005). However, to date, no economic studies have addressed choice of higher-strength beverages, such as malt liquor, in relation to employment using a suitable national data set.

BEVERAGE CHOICE: REGULAR BEER VERSUS MALT LIQUOR BEER

Beer, wine, and spirits have widely varying alcohol content, conventionally measured by percentage ethanol by volume or alcohol by volume (ABV): a 12-ounce standard drink of beer contains roughly the same volume of alcohol as a 5-ounce glass of wine or 1.5 ounces of hard liquor. But there is a large variance of ethanol content among beer products (Case, Distefano, & Logan, 2000; Kerr & Greenfield, 2003; Kerr, Brown, & Greenfield, 2004; Logan, Case, & Distefano, 1999; Martin & Nirenberg, 1991). The ethanol content of malt liquors in general averages about 6.3 percent ABV, though individual brands contain up to 8 percent ABV (Kerr, Brown, & Greenfield., 2004); in contrast, most mass-market beers have ABV averages of about 5 percent, with the light beer type averaging just over 4 percent. Furthermore, malt liquor is often marketed in large formats, such as 16-ounce cans or 40-ounce bottles with large mouths, in readily accessible coolers found in corner stores in blighted communities, designed to promote immediate, rapid consumption (Greenfield, Brown Taylor, & Bond, 2004 consider also Gruenewald, Millar, & Treno, 1993). Thus the actual consumption of these products may induce higher blood alcohol levels than other beer products (Greenfield, Bond, Korcha, & Kerr, 2003), and greater alcohol-related social and health consequences may be associated with selecting these products in place of regular beer, which holds true even after controlling for overall alcohol intake patterns and other demographic variation (Greenfield et al., 2003. French, Brown Taylor, and Bluthenthal, 2006) show evidence that malt liquor beer consumers are *less* responsive to prices than drinkers of beer or spirits, so that consumption takes on a habitual character.

To the extent that lower-income and underemployed individuals prefer this beverage type, a vicious cycle may become endemic, involving consumption of these less expensive products and experiencing adverse drinking consequences that may affect employment opportunities.

Not surprisingly, malt liquor beer and other fortified alcohol products (wine coolers, fortified wines) have been the target of concern for community groups and public health advocates (Gratz, 1997; Herd, 1994; Mosher, 2005; Powell, 1996) and business ethicists (Brenkert, 1998). A current study from south central Los Angeles already mentioned gives a snapshot of malt liquor beer as the most regular alcoholic beverage choice of inner-city African American men (Bluthenthal et al., 2005). The drinking pattern associated with this subpopulation with multiple economic disadvantages was nearly daily drinking, consuming a higher volume of alcohol per day (than regular beer drinkers), and preferring to drink in outdoor public settings (e.g., a park or parking lot rather than a restaurant or bar). With some differences, U.S. national data have reflected similar profiles. Greenfield et al. (2001) estimated multiple logistic regression models to assess the characteristics associated with use of malt liquor. Self-identified African American non-Hispanic respondents were about 2.8 (CI: 1.9–4.3) times more likely to be regular malt liquor drinkers than whites (2.4 times as likely to report any malt liquor beer use). Younger adults (18–29) and those not completing high school were more likely to drink, and frequently drink, malt liquor, while gender was unimportant. Although lower income was associated with malt liquor use on a bivariate basis, it was not in the multivariate models when controlling for overall drinking pattern; however, in these models, regular malt liquor users compared to beer only drinkers were 1.47 (CI: 1.04–2.07) times more likely to be unemployed, with similarly significant, if weaker, relationships seen for any malt liquor use.

Our principal aim is to probe the influence of regular consumption of malt liquor beer on the probability of *second-best* employment outcomes. We hypothesize that the likelihood of poorer labor outcomes will be positively associated with regularly drinking malt liquor products even after controlling for sociodemographic factors and correcting for possible estimation bias due to endogeneity. Though not the emphasis of the present analysis, we hypothesize that the choice of malt liquor beer may be a response to limited financial resources (reflecting limited income and/or bias against traditional work).[1] Unobservable propensities common to both beverage choice and employment condition (e.g., endogeneity) will result in inconsistent and biased coefficients if either of these outcomes are estimated singly; we use a bivariate probit model to measure the correlation of the error terms and produce unbiased parameter estimates (Ashford & Snowden, 1970; Greene, 1993) of the impact of beverage

choice on employment. Estimates of the error term correlations and their statistical significance supports our hypothesis that endogeneity is present, and so univariate analyses would be biased. (If the error term correlations obtained had been insignificant, then there would have been "no statistical advantages to employing a more sophisticated estimator," Feng, Zhou, Butler, Booth, & French, 2001, p. 515.)

DATA AND METHODS

Data Source

These analyses are based on data from the year 2000 U.S. NAS (N10), which was the first in this series of five-yearly surveys conducted by the Alcohol Research Group to include a sequence of items directly addressing malt liquor beer consumption. Extensively utilized in alcohol epidemiology, previous fieldings of the NAS have been used for labor economic-specific analysis only once (Heien, 1996). Designed for health investigations, it does not have as detailed a set of economic variables as, for example, the NLSY, but on the other hand, it includes a more robust set of alcohol measures and a number of relevant employment questions. The survey's epidemiological design included oversamples of African American and Hispanic individuals and an oversample of 13 low-population states (details of the survey design can be found in Greenfield et al., 2001); Greenfield, Nayak, Bond, and Midanick, 2006; and Kerr, Greenfield, Bond, Ye, and Rehm, 2004).

The national sample for N10, including the designed oversamples, yielded an overall sample totaling 9,044 individuals. The main Random Digit Dial (RDD) sample involved 6,021 individuals including all ethnic groups; oversamples of black and Hispanic respondents added 1,250 cases, and the low-population state oversample added 341 cases (to ensure at least 50 cases in each sate); and finally, oversamples were drawn from five targeted metropolitan areas (Denver, Houston, Kansas City, Philadelphia, and San Francisco), adding 1,432 metropolitan cases. Together, this sample resulted in 1,341 black non-Hispanics (17.9%) and 869 Hispanics (12.8%). There were 5,715 current drinkers overall, but incomplete response reduces our useable sample for analysis to n = 5,509 current (past year) drinkers.

Key Measures

The N10 survey includes item series that allow alcohol use to be measured two ways: (1) a Knupfer Series beverage-specific (beer, wine, or spirits) quantity frequency (QF) measure and (2) the Graduated Frequencies (Greenfield,

2000; Room, 1991). The N10 survey augmented the beer QF questions with the question, "When you drink *beer*, how much of the time do you drink malt liquor?" Here we identify beer drinkers as those respondents who chose to drink beer at some time in the last 12 months. We distinguish beer drinkers (relative to *only* wine and/or spirits), and then we further narrow the focus to examine consumers of malt liquor (among beer drinkers).[2] Respondents who said they had consumed malt liquor beer more than "once in a while" are categorized for this analysis as using malt liquor beer regularly.

The strata of the employment conditions we address are as follows: (1) nonparticipation in the labor force (e.g., due to retirement, school enrollment, or discouragement), (2) unemployment (i.e., available and seeking work), and (3) part-time-only work. Because we are interested in investigating whether or not malt liquor beer is associated with second-best (less remunerative or less secure) outcomes, two of our employment state variables are defined so that an affirmative response is the *poorer* alternative; these are labor force nonparticipation and unemployment. However, we also want to probe whether malt liquor beer decreases the likelihood of even part-time-only employment. For part-time-only employment to serve this litmus test purpose, we have constructed the alternative state as *unemployment* for the estimation analyses. (An affirmative response to part-time only means a person is employed fewer than 35 hours per week; a negative response means being unemployed.) We are assuming part-time only is less desirable than full-time work but more desirable than unemployment. Strictly speaking, part-time work is not always a second-best condition (Barrett & Doiron, 2001; cf. Kalleberg et al., 2000; Reynolds, 2003); yet while recognizing this fact, the N10 data offer us no way to distinguish whether the part-time employment is individually *voluntary* (i.e., first-best) or *involuntary* (i.e., second-best).

Other key measures included in this analysis are variables for religiosity, the age when the respondent began drinking alcohol, regional wetness/dryness, and four count variables of various problems the respondent attributes to his or her drinking. These variables (or variations thereof) are common in this literature. Religiosity can be a protective factor in the development of problem drinking behaviors among adolescents (Brown Taylor, 2001; Mason & Windle, 2002; Wells, Graham, Speechley, & Koval, 2005); this variable is derived from the item, "How important would you say religion is in your life—very important, somewhat important, not really important, or not at all important?" The response "very important" was taken as the positive indicator of religiosity generally found to be related to lower alcohol consumption (Greenfield, 2000). An earlier age of onset is a strong predictor of many later manifesting alcohol problems (e.g., Grant & Dawson, 1998). Regional attitudes toward alcohol were

also considered—broadly speaking, *wet* indicates a relative tolerance of alcohol, whereas *dry* identifies less approval (Hilton, 1988, 1991).[3] Four "alcohol-related problems" variables are components of a larger tangible consequences scale used to assess consequences of alcohol abuse (Hilton, 1991; Midanik & Greenfield, 2000).[4] Current drinker respondents were asked, "Did this happen in the last 12 months?," followed by a series of negative-consequence instances. Problems with the law were assessed by four items, for example, "I had trouble with the law about drinking when driving was not involved," "I have been arrested for driving after drinking," and "a police officer questioned or warned me because of my drinking"; the legal problems variable is the count of affirmative responses. In similar fashion, problems associated with work or employment are positive response counts to the three statements, such as "I have lost a job, or nearly lost one, because of drinking." Consequences affecting health included intoxication lasting several days, concern for physical health, and so on, and were counted from three questions. And the interpersonal consequences of drinking measure covered two questions in which a spouse or coworker expressed concern for the respondent's drinking behavior. In several studies, internal reliabilities of these measures (Cronbach's) ranged from 0.74 to 0.97, excepting health, which was somewhat lower (0.58–0.67), but deemed adequate for brief research measures (Midanik & Greenfield, 2000). Sociodemographic and geographic indicator variables complete the set of independent variables used for these estimations.

Sample means are given in Table 16.1 (including statistics for drinkers who only consumed wine and/or spirits [but not beer] in the interest of comparison). Beer drinkers are unlike drinkers limited to wine and spirits: they are more often male; they are younger; they started drinking alcohol at a younger age; they identify religion as "important" less often; and they report more alcohol-related problems in all four domains (legal, health, work, and interpersonal relationships). Self-identified blacks are *less likely* and Hispanics are *more likely* to drink beer than only wine or spirits. Finally, regular malt liquor beer drinkers have different group characteristics than general beer drinkers. Regular malt liquor beer drinkers tend to be male; have only a high school education or less; are more often unmarried and unemployed; and self-identify more often as black, Hispanic, or other. With the exception of problems related to work, the subgroup who regularly drink malt liquor also report more instances of alcohol-related legal and health problems as well as more interpersonal problems due to drinking than general beer drinkers.

Analytical Framework

Economic modeling of the effects of substance abuse (alcohol, cigarettes, and drugs) on labor market outcomes traces its lineage to human capital (Becker,

Table 16.1
Sample and Beverage Group Means

	All current drinkers		Wine and spirit drinkers		Difference	All beer drinkers		Beer, but not regular malt liquor beer		Difference	Regularly malt liquor beer	
Variable	Mean	SD	Mean	SD		Mean	SD	Mean	SD		Mean	SD
Beverage	0.052	0.223				0.081	0.272					
Labor force nonparticipation	0.207	0.405	0.259	0.438	***	0.183	0.387	0.172	0.377		0.171	0.377
Unemployed	0.027	0.163	0.019	0.136	***	0.031	0.173	0.026	0.159	***	0.078	0.268
Works part-time (<35 hours/week)	0.115	0.319	0.133	0.340	**	0.108	0.311	0.100	0.300	***	0.166	0.372
High school or less	0.652	0.476	0.657	0.475		0.652	0.476	0.675	0.468	***	0.524	0.500
Respondent is male	0.495	0.500	0.247	0.431	***	0.592	0.492	0.616	0.486	**	0.654	0.476
Age at interview	40.2	15.146	43.6	16.400	***	38.6	14.161	38.9	13.847	***	31.4	13.651
Married	0.461	0.499	0.458	0.498	*	0.462	0.499	0.479	0.500	***	0.236	0.426
African American	0.131	0.337	0.181	0.385	***	0.110	0.312	0.094	0.292	***	0.216	0.412
African American and female	0.075	0.263	0.136	0.343	***	0.050	0.217	0.042	0.200	*	0.065	0.247
Hispanic	0.104	0.305	0.079	0.269	***	0.113	0.317	0.108	0.311		0.161	0.368
Hispanic and female	0.048	0.214	0.058	0.234		0.043	0.204	0.040	0.195		0.034	0.182
Other	0.076	0.266	0.069	0.254		0.076	0.266	0.072	0.259		0.123	0.329
Other and female	0.034	0.182	0.049	0.216		0.027	0.162	0.023	0.151		0.038	0.191

Table 16.1
continued

	All current drinkers		Wine and spirit drinkers		Difference	All beer drinkers		Beer, but not regular malt liquor beer		Difference	Regularly malt liquor beer	
Religion is "important"	0.476	0.499	0.581	0.494	***	0.431	0.495	0.414	0.493		0.435	0.497
Age started drinking	18.2	4.799	19.6	6.376	***	17.6	3.940	17.5	3.773	***	16.7	3.335
Legal problems	0.039	0.266	0.006	0.099	***	0.053	0.310	0.051	0.306	*	0.140	0.481
Health problems	0.048	0.269	0.019	0.166	***	0.060	0.299	0.058	0.292		0.116	0.431
Work problems	0.019	0.172	0.007	0.099	**	0.025	0.195	0.023	0.186		0.079	0.348
Interpersonal problems	0.051	0.246	0.022	0.165	**	0.063	0.271	0.059	0.266	**	0.178	0.426
Residence												
Midwest	0.188	0.391	0.173	0.379	**	0.193	0.395	0.193	0.395		0.185	0.389
Pacific	0.264	0.441	0.241	0.428		0.276	0.447	0.275	0.446		0.339	0.474
South	0.290	0.454	0.309	0.462	**	0.278	0.448	0.277	0.448		0.205	0.405
Intermountain West	0.078	0.269	0.069	0.253	*	0.084	0.278	0.085	0.279		0.086	0.280
Regional alcohol attitude	1.447	0.497	1.451	0.498		1.445	0.497	1.444	0.497		1.380	0.486
Obs	5,715		1,512			3,997		3,320			292	

*Significant at 10%. $p \leq 0.10$
**Significant at 5%. $p \leq 0.05$
***Significant at 1%. $p \leq 0.01$

1962; Mincer, 1958) and health capital (Grossman, 1972; Grossman, 1989) theories. However, there are few investigations of beverage-specific associations with labor market outcomes and none related to individuals' choices of high-strength alternative beverages like malt liquor; the present analysis begins to fill this gap.

Single-equation probit models of current employment status as a function of beverage choice and other explanatory variables are summarized in the following:

$$\Pr(\text{Employment State}_i = 1) = \Phi(\alpha A\text{Beverage\_Type}_i + \beta \text{AX}_i). \quad (1)$$

In our analysis, there two versions of the binary variable Beverage_Type$_i$: first, current drinkers who chose beer in the last 12 months (yes or no), and second, beer drinkers who chose malt liquor beer products more than infrequently (yes or no). The placeholder variable Employment_State$_i$ in the generic equation (1) describes three distinct employment situations in our analysis: (1) out of the labor force (yes or no); (2) unemployed (yes or no); and (3) underemployed, our construction of part-time-only work versus unemployed. The coefficients α and β are parameters to be estimated; Φ represents the cumulative normal function when estimating a probit model. However, if the Employment_State condition is itself a contributing factor influencing beverage choice (as we expect), estimation of an equation such as equation (1) will produce biased coefficients and inaccurately reflect any association between beverage choice and employment situation.[5]

Our estimation applies a *bivariate* probit model specification in which the variables Beverage_Type$_i$* and Employment_State$_i$* are latent variables that reflect beverage choice(s) and current employment state(s), respectively (following the approaches of Brown et al., 2005; Feng et al., 2001; Hamilton et al., 1997). The multivariate, simultaneous estimation is summarized in the system

$$\begin{bmatrix} \text{Beverage\_Type}_i \\ \text{Employment\_State}_i \end{bmatrix} = \begin{bmatrix} X_i\theta + Z_i\gamma \\ X_i\beta + \text{Beverage\_Type}_i\alpha \end{bmatrix} + \begin{bmatrix} \varepsilon_i \\ \nu_i \end{bmatrix}$$
$$\begin{bmatrix} \varepsilon_i \\ \nu_i \end{bmatrix} \sim NID\left(0, \begin{bmatrix} \sigma^2 & \rho\sigma \\ \rho\sigma & 1 \end{bmatrix}\right) \quad (2)$$

Two versions of Beverage_Type and three versions of Employment_State obtain six bivariate probit systems to be estimated. The independent variables included in $\mathbf{X_i}$ are indicator variables—high school education or below, sex, age, marital status, self-reported race and ethnicity, three Race × Gender (e.g., black women, Hispanic women, and other (non-White) women) interaction terms, and four variables for geographic region—believed relevant both to drinkers' beverage choices (Kerr, Greenfield, Bond, Ye & Rehm, 2004) and

the respondents' employment condition. The matrix $\mathbf{Z}_i$ includes the variables for age started drinking; religiosity; the alcohol-related legal, health, work, and social problems; and wet/dry—these are the instruments for beverage choice.

We assume that ε_i and υ_i are distributed bivariate normal with zero mean and variance-covariance structure described by the parameters σ and ρ. These error terms may be the result of omitted variables that are related to the probabilities of an individual's particular beverage choice and/or employment circumstance but are unspecified either because they are not measurable (e.g., effort or attitude) or because they are not available from the existing N10 questionnaire (e.g., job tenure, length of current unemployment spell, etc.). If ρ is significantly different from zero, there is endogeneity between beverage choice and employment circumstance. Estimates obtained from simply including Beverage_Type directly in a single-equation probit of Employment_State equation(s) will be biased. The sign of ρ reveals the direction of bias: if ρ is positive, a single-equation probit estimate will exaggerate the effect of beverage choice on employment state, and the coefficient will be *overstated;* if ρ is negative, the single-equation estimate will *understate* any impact since the effects of the unobserved factors affect affirmative choice of alcoholic beverage and employment situation in opposite directions, in a diluting effect.

RESULTS

Odds ratios (OR) obtained by a logistic regression (for brevity of exposition results not reported) for the three respective employment states that simply included beer as an independent variable would appear to (very weakly) obtain that choosing beer (rather than wine or spirits) is unrelated to the odds of being out of the labor force (OR = 1.013, *SE* = 0.084, *p<=0.15*) and significantly increases the odds of being unemployed (OR = 1.582, *SE* = 0.358, $p < .05$); furthermore, beer significantly reduced the odds of working part-time-only relative to unemployment (OR = 0.622, *SE* = 0.140, $p < .01$). The same approach, including instead malt liquor beer as the chosen alcoholic beverage, indicates that regularly choosing malt liquor (rather than ordinary beer) looks to be associated with 50 percent greater odds of being out of the labor force (OR = 1.498, *SE* = 0.278, $p < .01$) and almost double the odds of being unemployed (OR = 1.878, *SE* = 0.488, $p < .05$) but would seem to have no significant association with part-time-only work (OR = 0.706, *SE* = 0.200, *NS*).

Bivariate Probit Analysis

The results from estimating the six bivariate probit systems are shown in Table 16.2.[6] The estimates of ρ are statistically significantly nonzero in five of

the six systems (in all three of the malt liquor beer systems but in only two of the beer systems). The estimated ρs are negative in the cases of labor force nonparticipation and unemployment (both second-best outcomes), which means that simple single-equation estimates, such as the logit OR reported earlier, are understated. And in the part-time work systems, endogeneity is also not trivial; the rs are positive and statistically significant.

First, consider that the initial expectation that choosing beer (over wine or spirits) would have no association with labor force participation is now demonstrably inaccurate. Beer is positively associated with an increased likelihood of being out of the labor force and of being unemployed rather than working, *including part-time-only work*, after controlling for human capital and beverage choice covariates. The ubiquity of beer drinking (among those who drink) is visible in the lack of statistical significance for the education and marital status variables, both factors known to be significant to employment condition. Men and younger drinkers prefer beer—contributing to the fact that drinking beer, in contrast to wine or spirits, other things equal, elevates the risks of drinking and driving (Greenfield & Rogers, 1999). Thus it is not surprising to observe the positive association of legal problems in the beer choice equations. Religiosity and an older age of onset appear to have negative effects on choosing beer. Conversely, there is a positive association of health problems related to drinking and the choice of beer.

Beer choice and employment conditions are known to differ by race. This is also reflected in our results. Blacks are significantly less likely to choose beer (versus wine or spirits), which, all else equal, is a plus for avoiding second-best employment outcomes. And still blacks face an increased likelihood of being unemployed. Hispanics and others are as likely to choose beer as are whites, but Hispanic men have a higher probability of being unemployed relative to any employment, and others have higher probabilities of all three second-best employment outcomes.

Given that our main interest was to examine the effects of regular malt liquor beer consumption (not simply alcohol), we now turn to the results for the malt liquor beer and employment systems (the last six columns of Table 16.2). The role of endogeneity is heightened in these three systems: regularly malt liquor beer is significantly positively associated with the second-best outcomes (nonparticipation and unemployment relative to any work). As ubiquitous as the choice of beer was, the choice of regularly drinking malt liquor beer is a relatively enclaved phenomenon associated largely with markers of race and SES. Blacks, Hispanics, and others (though not as strongly for black or Hispanic women) are more likely to choose malt liquor beer. More than occasional choice of malt liquor beer is negatively associated with a later age of drinking onset, education beyond high school, older age, and marriage. Religiosity has

Table 16.2
Bivariate Probit Estimation of Beverage-Type Choice and Employment Status.

	System 1: beer and out of the labor force		System 2: beer and unemployment		System 3: beer and part-time only	
	Out of labor force	Beer	Unemployed	Beer	Part-time	Beer
Beer	0.569**		0.875**		–1.382***	
	–0.238		–0.390		–0.275	
HS&below	–0.144***	–0.013	–0.311***	–0.005	0.285***	0.096
	–0.043	–0.041	–0.074	–0.048	–0.102	–0.108
Sex	–0.492***	0.814***	–0.392**	0.818***	0.136	0.808***
	–0.083	–0.048	–0.155	–0.055	–0.198	–0.162
Age	0.034***	–0.010***	–0.008	–0.010***	–0.003	–0.013***
	–0.002	–0.001	–0.005	–0.002	–0.004	–0.004
Married	–0.067	–0.008	–0.166**	–0.043	0.120	0.027
	–0.042	–0.040	–0.084	–0.047	–0.118	–0.114
Black	0.131	–0.337***	0.461***	–0.260**	–0.329	0.011
	–0.104	–0.093	–0.145	–0.105	–0.232	–0.285
Black&fem	–0.265**	0.057	–0.056	–0.138	–0.324	–0.430
	–0.128	–0.114	–0.196	–0.128	–0.292	–0.328
Hispn	–0.034	0.056	0.407***	0.080	–0.343*	–0.048
	–0.109	–0.107	–0.137	–0.118	–0.204	–0.279
Hispn&fem	0.178	0.038	–0.403*	0.033	0.324	0.230
	–0.144	–0.136	–0.214	–0.153	–0.305	–0.346
Other	0.292***	–0.164	0.481***	–0.208*	–0.502**	–0.182
	–0.113	–0.113	–0.148	–0.122	–0.225	–0.314
Other&fem	–0.113	0.056	–0.155	0.016	0.195	0.126
	–0.156	–0.150	–0.230	–0.167	–0.314	–0.371
Midwest	–0.103	0.129*	–0.063	0.207**	0.140	0.466**
	–0.066	–0.073	–0.142	–0.085	–0.186	–0.205
Pacific	0.016	0.056	0.238*	0.075	–0.256	–0.031
	–0.063	–0.059	–0.123	–0.067	–0.156	–0.146
South	–0.026	0.025	0.148	0.153	–0.313**	0.141
	–0.060	–0.107	–0.121	–0.126	–0.158	–0.311
Mountn	–0.054	0.140	0.261	0.253*	–0.242	0.630*

System 4: Malt Liquor Beer & Out-of-the-Labor Force		System 5: Malt Liquor Beer & Unemployment		System 6: Malt Liquor Beer & Part-time Only	
ofl	regmlb	unemploy	regmlb	part-time	regmlb
0.834***		1.625***		−1.967***	
−0.309		−0.441		−0.160	
−0.117**	−0.216***	−0.239***	−0.215***	0.177	−0.174
−0.056	−0.068	−0.087	−0.074	−0.129	−0.140
−0.299***	−0.044	−0.283**	−0.085	0.012	−0.054
−0.063	−0.087	−0.118	−0.095	−0.175	−0.190
0.034***	−0.016***	−0.006	−0.021***	−0.005	−0.013*
−0.002	−0.004	−0.005	−0.004	−0.005	−0.007
−0.100*	−0.358***	−0.108	−0.364***	−0.004	−0.481***
−0.054	−0.071	−0.094	−0.078	−0.146	−0.164
−0.125	0.709***	0.114	0.783***	0.151	0.865***
−0.126	−0.120	−0.166	−0.126	−0.231	−0.242
0.045	−0.389**	−0.012	−0.304	−0.144	0.012
−0.179	−0.185	−0.241	−0.200	−0.345	−0.367
−0.068	0.356***	0.373**	0.374***	−0.478**	0.175
−0.122	−0.117	−0.152	−0.125	−0.225	−0.250
0.140	−0.437**	−0.536*	−0.376*	0.658*	−0.131
−0.184	−0.207	−0.276	−0.223	−0.354	−0.376
0.288**	0.303**	0.539***	0.357**	−0.760***	0.114
−0.125	−0.135	−0.155	−0.149	−0.235	−0.284
−0.143	−0.047	−0.280	−0.086	0.378	−0.173
−0.205	−0.232	−0.286	−0.264	−0.418	−0.512
−0.218**	−0.136	0.017	−0.117	−0.032	0.154
−0.085	−0.127	−0.166	−0.146	−0.241	−0.269
−0.069	−0.014	0.189	0.030	−0.294	0.111
−0.080	−0.100	−0.147	−0.108	−0.211	−0.212
−0.106	−0.512***	0.273*	−0.562***	−0.537**	−0.398
−0.079	−0.181	−0.149	−0.204	−0.213	−0.352
−0.027	−0.197	0.354*	−0.211	−0.521*	−0.074

Table 16.2
continued

	System 1: beer and out of the labor force		System 2: beer and unemployment		System 3: beer and part-time only	
	Out of labor force	Beer	Unemployed	Beer	Part-time	Beer
	−0.084	−0.121	−0.166	−0.143	−0.239	−0.354
Religi		−0.144***		−0.117**		−0.084
		−0.042		−0.048		−0.101
Startage		−0.031***		−0.040***		−0.030**
		−0.004		−0.006		−0.012
Legalpr		0.337**		0.308**		6.239***
		−0.141		−0.149		−0.251
Healthpr		0.266**		0.214*		0.704**
		−0.103		−0.125		−0.321
Workpr		0.104		0.244		0.123
		−0.150		−0.182		−0.344
Reactpr		0.075		0.027		−0.290
		−0.105		−0.110		−0.203
Wetdry		0.037		−0.055		−0.277
		−0.090		−0.107		−0.271
Constant	−2.300***	1.209***	−1.958***	1.468***	1.936***	1.555***
	−0.164	−0.143	−0.298	−0.177	−0.249	−0.384
ρ	−0.357**		−0.419		0.695**	
Wald test	4.892		2.242		5.957	
p value	0.027		0.134		0.015	
$\chi 2$	1,279.07		719.09		4,178.39	
Log-likelihood	−5244.33		−2774.0031		−761.62346	
Observations	5,509		4,385		792	

Robust standard errors in parentheses
* significant at 10%; ** significant at 5%; *** significant at 1%

System 4: Malt Liquor Beer & Out-of-the-Labor Force		System 5: Malt Liquor Beer & Unemployment		System 6: Malt Liquor Beer & Part-time Only	
ofl	regmlb	unemploy	regmlb	part-time	regmlb
−0.104	−0.202	−0.189	−0.222	−0.268	−0.379
	0.099		0.151**		0.125
	−0.070		−0.074		−0.117
	−0.020**		−0.019*		−0.020
	−0.010		−0.010		−0.018
	0.053		0.021		−0.034
	−0.083		−0.085		−0.122
	−0.004		−0.238*		−0.228
	−0.116		−0.139		−0.175
	0.128		0.265*		0.532*
	−0.132		−0.158		−0.312
	0.225**		0.225**		0.107
	−0.099		−0.100		−0.166
	0.183		0.213		0.145
	−0.150		−0.163		−0.285
−1.995***	−0.508*	−1.589***	−0.424	1.382***	−0.448
−0.126	−0.290	−0.248	−0.305	−0.296	−0.508
	−0.307**		−0.647***		0.975***
	4.517		7.059		12.213
	0.034		0.008		0.001
	500.02		354.60		479.94
	−2361.1927		−1168.5438		−411.2358
	3612		2991		492

(at best) no inducement and is (at worst) positively associated with the choice of higher alcohol beer among those individuals in the labor force. Among labor force participants, health problems are negatively associated with choosing this higher-strength product. And drinkers of malt liquor beer, more likely to be nonparticipating or to be unemployed, exhibit positive associations with "problems involving negative reactions from others" and "work problems" not observed among drinkers of ordinary beer.

Once controlling for the beverage choice and covariates, self-identifying as black *fails to increase* the probability of any of the second-best employment conditions and also *fails to decrease* the probability of part-time-only work over unemployment. The influence of Hispanic identification is somewhat more mixed: Hispanic men in the labor force are more likely to be unemployed, but Hispanic women are more likely to work part-time-only than to be unemployed. Other things equal, regularly consuming malt liquor beer increases the likelihood of nonparticipation and unemployment, independent of these covariates.

CONCLUDING COMMENTS

The amplified association between regularly drinking malt liquor beer and unemployment highlighted by our analysis gives substance to the niggling unease of community activists and leaders that there is something amiss. Admittedly, it may not be malt liquor beer itself that does the damage; rather, perversely, the individual's choice of malt liquor beer may be an expression of some underlying characteristic that is severely discounted by the labor market. For instance, in the interview callback experiment comparing "Emily" (or "Greg") to "Lakisha" (or "Jamal"), the name itself is not a deterministic signal of ability, yet the outcome illustrates biased assessment of the applicant—*on the basis of the applicant's name*—by the prospective employer. In our analysis, malt liquor beer may be the observable choice that proxies some latent propensity that also increases the likelihood of second-best labor outcomes. The focus for further concern about high-potency beer products is if these contribute to behaviors deleterious to contemporaneous productivity and employability and if they might even be harmful to health (e.g., via an ethanol dose-response mechanism), thereby further limiting future employability and financial security, which themselves feed back to the individual's health status.

There are some limitations of the data and of the analyses that qualify our findings. The NAS analysis assumes the verity of self-report for alcohol consumption, employment, and personal circumstances. As the data are cross-sectional, personal history is only available through respondent recall. A more

robust analysis of the evolution and interaction of specific beverage choices, the several employment-related statuses, and personal income would require a longitudinal panel survey. The results reported here are categorized only in terms of alcoholic beverage chosen (in the last 12 months) and do not incorporate measures of the total volume of alcohol consumed or the frequency of heavy drinking, which is known to have effects on risks of a range of problems (Midanik, Tam, Greenfield, & Caetano, 1996), including nonfatal injuries (Cherpitel, Tam, Midanik, Caetano, & Greenfield, 1995) and even all-cause mortality in men (Rehm, Greenfield, & Rogers, 2001).

Second-best employment outcomes are generally individually and socially costly, affording lower lifetime earnings capacity and/or less employment security and requiring greater social finance, both currently and into the future. We suggest that the ramifications of the popularity during the 1990s of malt liquor beer (and related products), especially among ethnic minority groups, are likely to emerge only gradually. Young racial/ethnic consumers are already at nontrivial risk in terms of health and economic outcomes, without the additional risk identified here.

NOTES

1. For example, young adults across racial/ethnic groups are the marketing audience for rap music; as documented by Herd (2005), the prevalence of references to alcohol use and its association with positive consequences in this genre have increased sharply in the last decade. It is possible, then, that increased consumption of malt liquor beer products by the very populations that are also the audience for rap music is a substitution of relatively inexpensive but high-ABV beverages for an unaffordable (higher ABV) product that has been successfully associated with positive social conditions by admired musical artists.
2. Gill and Michaels (1992) made a distinction between any "drug use" and "hard-drug use" (which was limited to narcotics regarded as more potent or addictive); also, the contrast is implicit in the comparison of marijuana and cocaine in Register and Williams (1992).
3. A more contemporary and potentially more robust/rigorous characterization of regional drinking behaviors has been proposed by William Kerr (2005) of the Alcohol Research Group; ideally, this richer typology will be more informative regarding the drinking attitudes and environments of survey respondents.
4. Our model is a bivariate probit; each of the dependent variables is a binary (0,1). To our knowledge, no a priori endogeneity tests have been developed for this estimation specification.
5. The analysis presented is not an estimation of population parameters; it is an estimation of the relationship between beverage type and employment condi-

tion within individuals. The parameter estimates reported here are obtained from unweighted data (Groves, 1989).

6. We repeated these analyses to consider whether heavy drinking (two characterizations were used) more so than beverage choice was the risk factor. We were surprised to obtain that heavy drinking was only statistically significant in the out of labor force systems, and even so, was small in magnitude.

REFERENCES

Allen-Taylor, J. D. (1997, October 2–8). Malt assault: The malt-liquor industry, drunk on high-octane sales to the black hip-hop nation, has set its sights on the Latino youth market. *Sonoma County Independent*, pp. 10–12.

Ashford, J. R., & Snowden, R. R. (1970). Multivariate probit analysis. *Biometrics, 26*, 535–642.

Barrett, G. F., & Doiron, D. J. (2001). Working part time: By choice or by constraint. *Canadian Journal of Economics, 34*, 1042–1065.

Bartel, A., & Taubman, P. (1979). Health and labor market success: The role of various diseases. *Review of Economics and Statistics, 61*, 1–8.

Bartley, M., & Plewis, I. (2002). Accumulated labour market disadvantage and limiting long-term illness: Data from the 1971–1991 Office for National Statistics Longitudinal Study. *International Journal of Epidemiology, 31*, 336–341.

Becker, G. S. (1962). Investment in human capital: A theoretical analysis. *Journal of Political Economy, 70*, 9–49.

Bertrand, M., & Mullainathan, S. (2004). Are Emily and Greg more employable than Lakisha and Jamal? A field experiment on labor market discrimination. *American Economic Review, 94*, 991–1013.

Bluthenthal, R. N., Brown Taylor, D., Guzman-Becerra, N., & Robinson, P. L. (2005). Characteristics of malt liquor beer drinkers in a low-income, racial minority community sample. *Alcoholism: Clinical and Experimental Research, 29*, 402–409.

Brenkert, G. G. (1998). Marketing to inner-city blacks: PowerMaster and moral responsibility. *Business Ethics Quarterly, 8*, 1–18.

Brown, H. S., Pagan, J. A., & Bastida, E. (2005). The impact of diabetes on employment: Genetic IVs in a bivariate probit. *Health Economics, 14*, 537–44.

Brown Taylor, D. (2001). *Dispelling the myth regarding who drinks malt liquor beer in south central Los Angeles*. Los Angeles: Charles R. Drew University of Medicine and Science / Alcohol Research Group, Berkeley.

Case, G. A., Distefano, S., & Logan, B. K. (2000). Tabulation of alcohol content of beer and malt beverages. *Journal of Analytical Toxicology, 24*, 202–210.

Catalano, R. (1999). Labor market conditions and employment of the mentally ill. *Journal of Mental Health Policy and Economics, 2*, 51–54.

Cherpitel, C. J., Tam, T. W., Midanik, L. T., Caetano, R., & Greenfield, T. K. (1995). Alcohol and non-fatal injury in the U.S. general population: A risk function analysis. *Accident Analysis and Prevention, 27*, 651–661.

Elliott, S. (2002, March 6). Slightly sweet malt beverages get some heavy marketing, with young drinkers the targets. *New York Times*, p. C10.

Ettner, S. L., Frank, R. G., & Kessler, R. C. (1997). The impact of psychiatric disorders on labor market outcomes. *Industrial and Labor Relations Review, 51*, 64–81.

Feng, W., Zhou, W., Butler, J. S., Booth, B. M., & French, M. T. (2001). The impact of problem drinking on employment. *Health Economics, 10*, 509–521.

French, M. T., Brown Taylor, D., & Bluthenthal, R. N. (2006). Price elasticity of demand for malt liquor beer: Findings from a US pilot study. *Social Science & Medicine*, 62, 2101–2111.

French, M. T., Turner, H. L., & Greenfield, T. K. (2005). *The role of alcohol consumption in the probability of arrest* (Working Paper No. F605). Emeryville, CA: Alcohol Research Group.

Gill, A. M., & Michaels, R. J. (1992). Does drug use lower wages? *Industrial and Labor Relations Review, 45*, 419–434.

Grant, B. F., & Dawson, D. A. (1998). Age of onset of drug use and its association with DSM-IV drug abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey. *Journal of Substance Abuse, 10*, 163–173.

Gratz, P. (1997, June 15). Blunt talk about a 40-ounce problem. *Santa Cruz County Sentinel*.

Greene, W. H. (1993). *Econometric analysis* (2nd ed.). Upper Saddle River, NJ: Prentice Hall.

Greenfield, T. K. (2000). Ways of measuring drinking patterns and the difference they make: Experience with graduated frequencies. *Journal of Substance Abuse, 12*, 33–49.

Greenfield, T. K., Bond, J., Korcha, R., & Kerr, W. C. (2003, June 21–25). *Alcohol problems among regular malt drinkers: The role of maximum rate of drinking in an occasion*. Fort Lauderdale, FL: Research Society on Alcoholism (also Working Paper No. E719, Emeryville CA: Alcohol Research Group).

Greenfield, T. K., Brown Taylor, D., & Bond, J. (2001). *Ethnic subgroups' consumption of malt liquor beer based on the year 2000 U.S. National Alcohol Survey* (Working Paper No. E605). Emeryville, CA: Alcohol Research Group.

Greenfield, T. K., Nayak, M., Bond, J., & Midanick, L. T. (2006). Maximum quantity consumed and alcohol-related problems: Assessing the most alcohol drunk with two measures. *Alcoholism: Clinical and Experimental Research, 30*, 1576–1582.

Greenfield, T. K., & Rogers, J. D. (1999). Alcoholic beverage choice, risk perception, and self-reported drunk driving: Effects of measurement on risk analysis. *Addiction, 94*, 1735–1743.

Grossman, M. (1972). On the concept of health capital and the demand for health. *Journal of Political Economy, 80*, 223–255.

Groves, R. M. (1989). *Survey errors and survey costs*. New York: John Wiley.

Gruenewald, P. J., Millar, A. B., & Treno, A. J. (1993). Alcohol availability and the ecology of drinking behavior. *Alcohol Health and Research World, 17*, 39–45.

Halbfinger, D. M. (1997, July 24). Icy, fruity malt liquor draws fire. *New York Times*, p. A16, B1.

Hamilton, V. H., Merrigan, P., & Dufresne, E. (1997). Down and out: Estimating the relationship between mental health and unemployment. *Health Economics, 6*, 397–406.

Heien, D. (1996). Do drinkers earn less? *Southern Economic Journal, 63*, 60–68.

Herd, D. A. (1994). Contesting culture: Alcohol-related identity movements in contemporary African-American communities. *Contemporary Drug Problems, 20*, 739–758.

Herd, D. A. (2005). Changes in the prevalence of alcohol use in rap song lyrics, 1979–97. *Addiction, 100*, 1258–1269.

Hilton, M. E. (1988). Regional diversity in United States drinking practices. *British Journal of Addiction, 58*, 155–161.

Hilton, M. E. (1991). A note on measuring drinking problems in the 1984 national alcohol survey. In W. B. Clark & M. E. Hilton (Eds.), *Alcohol in America: Drinking practices and problems* (pp. 51–70). Albany: State University of New York Press.

Jones, A. S., & Richmond, D. W. (2006). Causal effects of alcoholism on earnings: Estimates from the NLSY. *Health Economics, 15*, 849–871.

Kahn, M. E. (1998). Health and labor market performance: The case of diabetes. *Journal of Labor Economics, 16*, 878–899.

Kalleberg, A. L., Reskin, B. F., & Hudson, K. (2000). Bad jobs in America: Standard and nonstandard employment relations and job quality in the United States. *American Sociological Review, 65*, 256–278.

Kerr, W. C. (2005, December 10–14). *Categorizing US state drinking practices and recent consumption trends*. Paper presented at the Annual Meeting of the American Public Health Association (Presentation No. E885, Emeryville, CA: Alcohol Research Group).

Kerr, W. C., Brown, S., & Greenfield, T. K. (2004). National and state estimates of the mean ethanol content of beer sold in the US and their impact on per capita consumption estimates: 1988 to 2001. *Alcoholism: Clinical and Experimental Research, 28*, 1524–1532.

Kerr, W. C., & Greenfield, T. K. (2003). The average ethanol content of beer in the U.S. and individual states: Estimates for use in aggregate consumption statistics. *Journal of Studies on Alcohol, 64*, 70–74, 439–440.

Kerr, W. C., Greenfield, T. K., Bond, J., Ye, Y., & Rehm, J. (2004). Age, period and cohort influences on beer, wine and spirits consumption trends in the US National Surveys. *Addiction, 99*, 1111–1120.

Kuo, M., Wechsler, H., Greenberg, P., & Lee, H. (2003). The marketing of alcohol to college students: The role of low prices and special promotions. *American Journal of Preventive Medicine, 25*, 204–211.

Logan, B. K., Case, G. A., & Distefano, S. (1999). Alcohol content of beer and malt beverages: Forensic consideration. *Journal of Forensic Science, 44*, 1292–1295.

Marcotte, D. E., & Wilcox-Gok, V. (2001). Estimating the employment and earnings costs of mental illness: Recent developments in the United States. *Social Science & Medicine, 53,* 21–27.

Marcotte, D. E., Wilcox-Gok, V., & Redmon, D. P. (2000). The labor market effects of mental illness: The case of affective disorders. In D. Salkever & A. L. Sorkin (Eds.), *The economics of disability* (pp. 181–210). Greenwich, CT: JAI Press.

Martin, C. S., & Nirenberg, T. D. (1991). Alcohol content variation in the assessment of alcohol consumption. *Addictive Behavior, 16,* 555–560.

Mason, W. A., & Windle, M. (2002). A longitudinal study of the effects of religiosity on adolescent alcohol use and alcohol-related problems. *Journal of Adolescent Research, 17,* 346–363.

McLellan, A. T., Lewis, D. C., O'Brien, C. P., & Kleber, H. D. (2000). Drug dependence, a chronic medical illness: Implications for treatment, insurance, and outcomes evaluation. *Journal of the American Medical Association, 284,* 1689–1695.

Midanik, L., & Greenfield, T. K. (2000). Trends in social consequences and dependence symptoms in the United States: The National Alcohol Surveys, 1984–1995. *American Journal of Public Health, 90,* 53–56.

Midanik, L. T., Tam, T. W., Greenfield, T. K., & Caetano, R. (1996). Risk functions for alcohol-related problems in a 1988 U.S. national sample. *Addiction, 91,* 1427–1437.

Mincer, J. (1958). Investment in human capital and personal income distribution. *Journal of Political Economy, 66,* 281–302.

Mosher, J. (2005). Transcendental alcohol marketing: Rap music and the youth market. *Addiction, 100,* 1203–1204.

Mullahy, J., & Sindelar, J. L. (1989). Lifecycle effects of alcoholism on education, earnings, and occupation. *Inquiry, 26,* 272–282.

Mullahy, J., & Sindelar, J. L. (1991). Gender differences in labor market effects of alcoholism. *American Economic Review, 81,* 161–165.

Mullahy, J., & Sindelar, J. L. (1993). Alcoholism, work, and income. *Journal of Labor Economics, 11,* 494–520.

Mullahy, J., & Sindelar, J. L. (1994). Alcoholism and income: The role of indirect effects. *Milbank Quarterly, 72,* 359–375.

Mullahy, J., & Sindelar, J. L. (1995). Health, income, and risk aversion. *Journal of Human Resources, 30,* 439–459.

Mullahy, J., & Sindelar, J. L. (1996). Employment, unemployment, and problem drinking. *Journal of Health Economics, 15,* 409–434.

Mullahy, J., & Sindelar, J. L. (1998). Drinking, problem drinking, and productivity. In M. Galanert (Ed.), *Recent developments in alcoholism: Vol. 14. The consequences of alcoholism* (pp. 347–434). New York: Plenum Press.

National Institute on Alcohol Abuse and Alcoholism. (2000, May 31). *Report on high alcohol content malt beverages and related products.* Paper presented at the conference of the National Institute on Alcohol Abuse and Alcoholism, Berkeley, CA.

Pampel, F. C. (2006). Socioeconomic distinction, cultural tastes, and cigarette smoking. *Social Science Quarterly, 87,* 19–35.

Pelkowski, J. M., & Berger, M. C. (2004). The impact of health on employment, wages, and hours worked over the life cycle. *Quarterly Journal of Economics and Finance, 44,* 102–121.

Powell, A. (1996). *Message'n a Bottle: The 40-oz. scandal.* Chicago: Renaissance Press.

Register, C. A., & Williams, D. R. (1992). Labor market effects of marijuana and cocaine use among young men. *Industrial and Labor Relations Review, 45,* 435–448.

Rehm, J., Greenfield, T. K., & Rogers, J. D. (2001). Average volume of alcohol consumption, patterns of drinking and all-cause mortality: Results from the U.S. National Alcohol Survey. *American Journal of Epidemiology, 153,* 64–71.

Reynolds, J. (2003). You can't always get the hours you want. *Social Forces, 81,* 1171–1199.

Room, R. (1991). Measuring alcohol consumption in the U.S.: Methods and rationales. In W. B. Clark & M. E. Hilton (Eds.), *Alcohol in America: Drinking practices and problems* (pp. 26–50). Albany: State University of New York Press.

Ross, C. E., & Mirowsky, J. (1995). Does employment affect health? *Journal of Health and Social Behavior, 36,* 230–243.

Schmidt, L., Greenfield, T. K., & Mulia, N. (2006). Unequal treatment: Racial and ethnic disparities in alcoholism services. *Alcohol Research & Health, 29*(1), 49–54.

Schmidt, L. A., Ye, Y., Greenfield, T. K., & Bond, J. (2007). Ethnic disparities in clinical severity and services for alcohol problems: Results from the National Alcohol Survey. *Alcoholism: Clinical & Experimental Research, 31,* 48–56.

Turner, J. B. (1995). Economic context and the health effects of unemployment. *Journal of Health and Social Behavior, 36,* 213–229.

Weisner, C. (1997). Chronic alcohol and drug abuse. In R. J. Newcomer & A. E. Benjamin (Eds.), *Indicators of chronic health conditions: Monitoring community-level delivery systems* (pp. 260–301). Baltimore: Johns Hopkins University Press.

Wells, S., Graham, K., Speechley, M., & Koval, J. J. (2005). Drinking patterns, drinking contexts and alcohol-related aggression among late adolescent and young adult drinkers. *Addiction, 100,* 933–980.

Chapter **17**

The Roads to H: The Emergence of the American Heroin Complex, 1898–1956[1]

David T. Courtwright, PhD

For some years I have been working on a history of the reception and impact of novel psychoactive drugs. The other day it occurred to me that, at bottom, the story is really a very simple one. It is that of the sorcerer's apprentice. Promising new drugs—aqua vitae, tobacco, morphine, cocaine, barbiturates, amphetamines—are introduced. Therapeutic claims are made and evaluated. Doctors argue among themselves about indications, dosage, and toxicity. The intramural debates seldom attract public notice. But, sooner or later, the new drug slips the bonds of medical discourse and control. It escapes into a larger world of popular pleasure and mischief, prompting official intervention.

The history of heroin in the United States fits neatly into this pattern. In 1898 it was just another promising new product from the Bayer Pharmaceutical Company. In the 1910s it developed a growing underworld following and by the 1920s had become the mainstay of the black market. The federal government outlawed the manufacture of heroin in 1924; as existing stocks dwindled to a few hundred ounces, the drug virtually disappeared from medical practice. The last remaining supplies were swept up by the Narcotic Control Act of 1956, which required the surrender of all remaining pharmaceutical heroin to the federal government. Heroin became, so to speak, America's first Schedule I drug. Its use was totally prohibited, except for restricted research purposes, as cannabis, LSD-25, DMT, and mescaline would be in later years. However, the fact that heroin shared the fate of other potent psychoactive drugs is not an adequate explanation of its prohibition. We need to look more closely at the process of its transformation. We need to understand precisely when, how, and

why heroin became H, the top enforcement priority of the Bureau of Narcotics and the basis of an underworld subculture—predominantly white before World War II, and minority thereafter.

MEDICAL USE AND ADDICTION

Heroin use and addiction were originally medical phenomena. What is not generally understood, however, is that the percentage of medical addicts who used heroin was never large. A retrospective study of fifty mostly medical cases published in 1918 showed only two heroin users, or 4 percent of the total (Scheffel 1918, 853–54). Lawrence Kolb, the government's leading medical authority on addiction, carried out a similar study of 150 medical addicts whose use began between 1898 and 1924, that is, after heroin was introduced and before it was effectively outlawed in the United States. The result: two heroin cases, or just 1.3 percent of the total. "My idea has been that the use of heroin in medical practice seldom resulted in addiction," Kolb wrote, "although when used in the underworld for dissipation only it doubtless has produced numerous addicts."[2]

The infrequency of iatrogenic heroin addiction was due to several factors. Heroin, in contrast to morphine and cocaine, was not touted for virtually every physical and medical affliction. Heroin was discussed in the medical literature primarily as a cough suppressant and means of alleviating respiratory distress. It was "recommended chiefly for the treatment of the air passages attended with cough, difficult breathing and spasm, such as different forms of bronchitis, pneumonia, consumption, asthma, whooping cough, laryngitis, and certain forms of hay fever," summed up a 1906 JAMA literature review ("Heroin Hydrochloride" 1906, 1303). While some authorities also recommended the drug as an analgesic, this idea was controversial and, early on, was challenged by several German and American authorities (for example, Floret 1898, 512; Manges 1898, 770; Wood 1899, 89–90).[3]

Advertising stressed heroin as a specific for respiratory symptoms. This was true even when heroin was combined with other analgesic products such as Antikamnia, a popular medication whose name means "against pain." The promotional literature for Antikamnia and heroin mentioned several possible indications, but concentrated on glowing clinical reports of cases involving cough and respiratory ailments. The medication came in the form of a tablet consisting of 5 grains of Antikamnia (47 parts acetanilide, 50 parts sodium bicarbonate, and 3 parts tartaric acid) and just 1/12 grain (5 mg) of heroin.[4]

This was typical. When heroin was prescribed for cough and respiratory ailments, it was given in small doses in tablets, pills, pastilles, elixirs, or glyc-

erin solutions. Some preparations contained only 1 or 2 mg per dose (see, e.g., "Herotopine" and "Hermonal," n.d., W. H. Schieffelin and Company Collection). The ingestion of small amounts of an opiate was a good deal safer, from the standpoint of addiction, than hypodermic injection, which was how morphine was often administered.

Death was also a good protection against addiction. Pneumonia and tuberculosis had particularly high mortality rates in the period before antibiotics. Patients with these conditions who were treated with heroin presumably would not have lasted long as addicts, assuming they reached the point of physical dependence.

While some early reports gave assurances that heroin was not habit forming, and even recommended it as a treatment for morphine addiction, physicians were quickly disabused of these notions. By early 1900 there were several cautionary statements about heroin's toxic and habit-forming potential; by 1903, if not sooner, there were firm and unambiguous declarations with such titles as "The Heroin Habit: Another Curse" (Pettey 1903; see also Wood 1899, 90; Manges 1900, 82; "Caution Regarding Heroin" 1900, 44).

These warnings about iatrogenic heroin addiction came sooner than comparable warnings about morphine. They were received by physicians who were better educated, more therapeutically conservative, and more mindful of specific treatments for specific diseases than their counterparts of the 1870s and 1880s, when morphine reigned as panacea. In fact, most American (and, ironically, German) doctors in private practice eventually became so wary of heroin that they gave up prescribing it before they were legally required to do so. Government and military physicians had less choice in the matter. The U.S. Public Health Service ceased dispensing heroin in 1916; the army, in 1923; the navy, in 1924 (see "Symposium on The Doctor and the Drug Addict" 1920, 1591; New York State Narcotic Drug Control Commission 1920, 41; Wolff 1932, 2180; Anslinger 1936).

Now contrast the heroin situation with that of aspirin. Introduced commercially in 1899, the year after heroin, aspirin was Bayer's best-selling drug by 1906 and one of the most widely prescribed drugs in the world by 1914 (McTavish 1987, 104).[5] Not only was aspirin useful in treating a wide variety of conditions, but it was relatively safe and not habit forming (in the sense of physical dependence). Indeed, I have long believed that one of the reasons for the decline of all forms of iatrogenic opiate addiction in the early twentieth century was the availability of aspirin and kindred preparations to treat rheumatism, colds and flu, toothaches and headaches, and other common aches and pains.

Going further, I believe that degree of exposure is the single most crucial variable in accounting for the prevalence of heroin or any other type of addic-

tion. Yes, set and setting matter; and yes, personality and genetic makeup play a role; and yes, social integration and cultural norms can militate against abuse; and yes, only a minority of those exposed to a given drug typically end up as full-blown addicts. The simple fact remains, however, that those who are never exposed to a drug will never become addicted, and those who are seldom exposed are at much lower risk than those frequently exposed. Aspirin reduced the odds that millions of people from all walks of life who were suffering from common afflictions would come into contact, through either prescription or self-medication, with powerful opiates like morphine or heroin. The net effect was less opiate addiction.

NONMEDICAL USE AND ADDICTION

How, then, were nonmedical users exposed? No simple answer is possible here. In the underworld, many roads led to H. One story has it that prisoners in a state penitentiary were given heroin for cough. News spread among the inmates that the cough pills were "good dope." Word spread outside the prison, and eventually to tenderloins all over the country (Kane et al. 1917, 503).[6] The story is plausible except in one detail. It seems more likely that word of heroin's psychoactive potency spread from multiple sites, rather than one particular prison.

We know that nonmedical heroin addiction was definitely in play by 1910, the year when Leroy Street, pseudonymous author of *I Was a Drug Addict*, and his teenage friends first started sniffing the drug (Street and Loth 1953, 11).[7] We also know that many of the early nonmedical heroin users—mostly sniffers—had previously used other drugs, notably opium, cocaine, and tobacco cigarettes.

Police pressure and a national import ban (enacted 1909) had made opium smoking riskier and more expensive. Heroin sniffing was cheaper, quicker, and much harder to detect. By 1916 several reports described opium smokers who had expediently switched to heroin (see, e.g., Bailey 1916, 314; McIver and Price 1916, 477, 478). Something similar happened with cocaine. The illicit market for cocaine existed well before passage of the 1914 Harrison Narcotic Act. It was created by a combination of informal professional controls—druggists wouldn't sell cocaine to just anybody—and proliferating legal controls of varying stringency (Spillane 2000). The result was an illicit market and higher prices. Decks of cocaine retailed on the streets of New York City for 25 cents, but contained only 1.3 grains (<85 mg), making the actual price eleven times that of the legitimate wholesale price (Musto 1990, 322–23).

Heroin, by contrast, was cheaper and more readily available from druggists. "Dope users have turned to [heroin]," Boston reformers complained in 1912,

"and as the drug is not so well known we find apothecaries who would not sell cocaine who are selling heroin apparently quite freely" (Chase et al. 1912, 9; see also Towns 1916, 221). Heroin was taken in the accustomed form, sniffing; didn't require injection; and had the added advantage of alleviating unpleasant symptoms, such as depression, that might be experienced when quitting cocaine. In 1923 Lawrence Kolb began a systematic study of 230 cases of narcotic addiction, including 40 heroin addicts. His records show that twenty-six of the forty heroin cases used cocaine prior to or concurrently with their first use of heroin (Kolb Papers, Box 6; Courtwright 1982, 161–62 n. 9; see also Stokes 1918, 756–57; Farr 1915, 893–94).

Not all of the heroin sniffers were veteran opium or cocaine users. Another type was the teenage working-class boy, often of immigrant parents, who lived in a "dirty, noisy, cheap, and tough" neighborhood (Stokes 1918, 757). He began using heroin out of curiosity or peer pressure after he had been introduced by someone in his gang—more like a younger, scruffier version of the Bowery Boys than the Crips or the Bloods. If he took any other drug on a regular basis, it was tobacco. No observation about the first generation of nonmedical heroin addicts is more commonplace than that they were inveterate cigarette smokers (e.g., Blanchard 1913, 142; Stokes 1918, 756).

Is there a connection? The chronology, geography, and sociology are all suggestive. Cigarette use was exploding at the same time nonmedical heroin use was taking off. New York City, the center of heroin use, was also the center of the cigarette revolution. In 1910 the city accounted for 25 percent of all U.S. cigarette sales, despite having only about 5 percent of the U.S. population. Cigarettes were particularly popular with New York's immigrants—and many heroin addicts, though born in the United States, came from immigrant families.[8]

Theories developed. R. M. Blanchard, an army doctor, speculated that inhaling smoke increased the absorption of heroin (Blanchard 1913, 142). Harvey Wiley argued that the boy who acquired the cigarette habit would be "brought into sympathetic association with boys who are going to the bad" and that, among other things, he would "more readily become a victim of alcohol, cocain [*sic*], opium, and other narcotic drugs"—an early version of the gateway theory.[9] The addiction specialist Charles Towns likewise argued that cigarettes to alcohol to opiates was a natural progression, both because smokers developed a need for stimulation and because they ran with bad companions (Towns 1916, 152–53, 167, 172).[10] It is hard to know how much weight to assign cigarettes in the etiology equation, but it is at least plausible that their growing use—even more a symbol of defiance and deviance in 1910 than today—by groups of teenage boys paved the way for heroin experimentation.

HEROIN OUTLAWED

The emerging stereotype of the heroin addict was far more frightening than that of the old-fashioned morphine addict. Heroin addicts were boys and young men who ruined themselves in the prime of life. Morphine addicts were older and sicker and more often female. Heroin addiction was a vice. Morphine addiction had often originated in response to pain or chronic disease. Heroin addicts were dumb, greedy for drugs, visible, and rude, "members of gangs who congregate on street corners particularly at night, and make insulting remarks to people who pass." Morphine addicts were "more intelligent, secretive as to their habit, and usually temperate in the dosage of their drug" (Leahy 1915, 256; Stokes 1918, 757). Heroin addicts came from the lower and "criminal" classes of the big cities. Morphine addicts came from more diverse and, on the whole, better social backgrounds. The data in Figure 17.1, based on Treasury Department questionnaires, show where nearly ten thousand addicts were institutionalized during the period 1916–1918 (U.S. Treasury Department

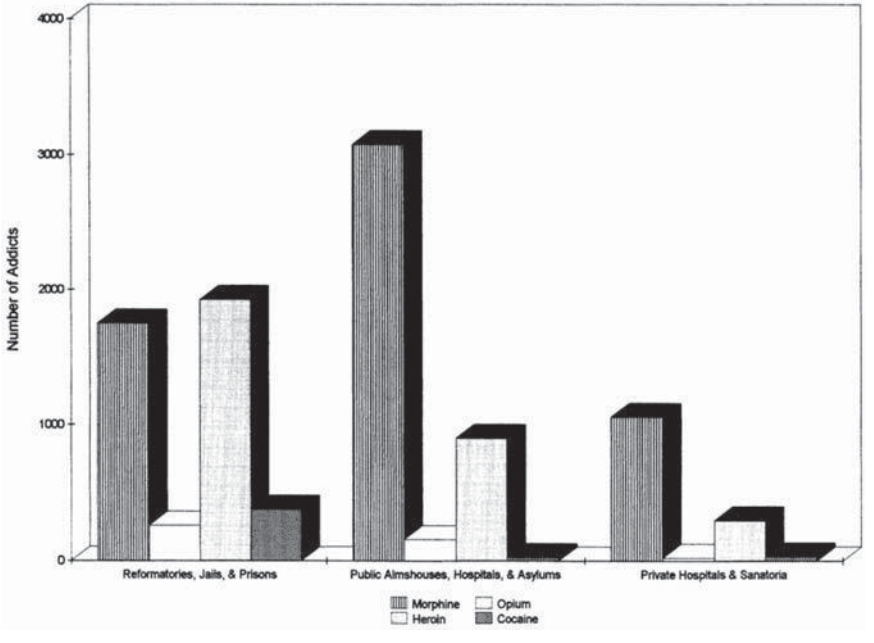

FIGURE 17.1 Institutionalized addicts, 1916–1918 (9,822 cases available to Treasury Department).

1919, 15–19). Heroin addicts were more numerous than morphine addicts in jails and prisons, while morphine addicts were far more numerous than heroin addicts in private hospitals and sanatoria. Unsurprisingly, opium smokers and cocaine addicts were also concentrated in penal or public institutions.

Subsequent studies showed a similar pattern. Of 632 convicted male addicts judged suitable for custodial treatment at New York City's Correction Hospital from March 1927 to June 1928, 588 (93%) used heroin alone or in combination with other drugs. Of 200 female addicts, 168 (84%) did likewise. Though straight morphine addicts were still common in the South in the late 1920s, they were increasingly rare in the North, particularly in correctional settings (Tuttle n.d.).

Why were so many heroin addicts behind bars? In objective terms, the answers are plain enough: because they stole or dealt drugs to support their habits; because they were reared in the slums; because they were single males in their teens and twenties, prime time for crime with or without drugs. But inventive propagandists like Richmond P. Hobson gave another answer: because of the action of heroin itself. It "exalted the ego," he testified, making the addict "suited for daring crimes, holdups, robberies, and such crimes as bandits of old never dared" (U.S. Senate, Committee on Printing 1924, 17).[11] Even if heroin addicts did not commit crimes, they were dangerous because of their tendency to proselytize (Bailey 1916, 315). "He has a mania to see others become addicts," said Hobson, who quoted with approval a comparison between heroin addiction and leprosy (U.S. Senate, Committee on Printing 1924, 16–17). The metaphor stuck.

Those who have lived through the middle to late 1980s do not need to be reminded what happens when the public associates addictive drugs with crime sprees by slum dwellers. In 1920 the American Medical Association's House of Delegates passed a resolution that heroin be eliminated from all medicines and that its importation, manufacture, and sale be banned in the United States. The measure was endorsed by police and penal authorities, and ultimately by Congress, which forbade the importation of opium to manufacture heroin in 1924. Stephen G. Porter, the bill's sponsor, hoped that other nations would emulate America's heroin ban, making it more difficult to divert the drug into the illicit traffic ("History of Heroin" 1953; "Heroin and the International Conferences" 1953, 55–58; Musto 1974, 200–2).[12]

THE HEROIN COMPLEX

Yet within ten years of its prohibition, heroin was the mainstay of the illicit traffic practically everywhere in the United States. In the mid-to-late 1910s most

heroin addicts lived (and died) in the New York–Philadelphia area. That was where most of the large heroin manufacturing and distribution companies were located and where diversion was therefore easiest, as when the American Drug Syndicate Company's Long Island plant was robbed of 150 pounds of heroin tablets in 1920. In other parts of the country, morphine or morphine-and-cocaine addicts were the norm (Courtwright 1982, 101–2; Jonnes 1996, 77).

By the end of 1920 the government had forbidden maintenance and had closed almost all of the municipal narcotic clinics. Some of the displaced patients were able to find doctors willing to write prescriptions. Those who suffered from chronic diseases like advanced tuberculosis, sympathetic figures in the eyes of both physicians and narcotic agents, had the most success in securing a continuous legal supply. But those who were "merely" addicted to narcotics—including virtually all heroin users—generally had to turn to the black market. Most black-market drugs were diverted from legitimate pharmaceutical manufacturers, who were making far more drugs than medically necessary during the 1920s. Enforcement of the Jones-Miller Act (1922) and a series of diplomatic agreements, culminating in the 1931 Limitation Convention to regulate international manufacturing, made diversion more difficult and brought licit production more into line with medical and scientific needs. Supplying the U.S. black market increasingly became a matter of smuggling and then distributing narcotics illicitly manufactured in such distant places as Turkey, the Balkans, and Shanghai.[13]

Heroin was the smuggler's drug par excellence. It was compact and potent, and did not spoil. It could be, and was, adulterated after arrival to increase profits. Even discounting for the adulteration, it was "cheaper for the amount of kick in it" (Helbrant 1941, 30).[14] Neophytes, wary of the needle, could sniff the drug. They could also afford it. Decks of adulterated heroin sold for as little as 50 cents apiece in Harlem during the Depression (Courtwright et al. 1989, 105).[15] These advantages made heroin the primary black-market narcotic throughout the United States, in fact throughout most of the world, by the mid-twentieth century ("History of Heroin" 1953; D'Erlanger 1936, 94).

Not all addicts approved of the trend. American opium smokers, who regarded heroin as dangerous and déclassé, were especially upset. They would have continued as before, but their accustomed drug was becoming progressively scarcer and more expensive. They were victims of what the physician and anthropologist Joseph Westermeyer calls the pro-heroin effects of anti-opium laws. The rigorous enforcement of drug prohibition inevitably drives traffickers and users away from bulkier and more perishable narcotics like opium and toward heroin, with generally evil consequences (Westermeyer 1976, 1135–39).

Let me invoke the work of two other distinguished anthropologists, Vera Rubin and Lambros Comitas. They coined the phrase "ganja complex" to describe a particular pattern of chronic working-class cannabis use in Jamaica and other cultures (Rubin and Comitas 1975, ch. 4; Rubin 1975, 5–6). It seems to me that what had evolved in America by the 1930s was a distinctive "heroin complex." Its attributes can be summarized briefly. Most addicted users were male, poorly educated, irreligious, and engaged in part- or full-time hustling; most lived in cities—about half in New York. Many female addicts were prostitutes. Initiated by sniffing, addicts switched to skin popping and mainlining to maximize the effect of adulterated heroin. They suffered high rates of morbidity, mortality, incarceration, and violence tied to disputes over drugs and money. They were part of a deviant, stratified subculture that revolved around the acquisition and use of heroin, had its own specialized language, and was at once mutually supportive and exploitative. "Never trust a junkie" was more than a prejudice of the straight world.

What happened to the heroin complex? Before World War II, it was mostly white and, as social problems go, of modest significance. Harry Anslinger, longtime head of the Bureau of Narcotics, estimated that there were thirty-five thousand to fifty thousand nonmedical addicts in the country in 1938 (Anslinger 1938). By contrast, Joseph Greenwood, a Drug Enforcement Administration epidemiologist, estimated that there were 504,000 to 578,000 addicts (that is, a 95 percent confidence interval around an estimate of 546,000) in 1975 (Greenwood n.d.). Bearing in mind the critical things I and others have said about official prevalence estimates, there is still no question that the heroin complex of the 1930s was small-time compared with that of the 1970s or, for that matter, of the 1990s.

But before the heroin complex got larger, it got smaller. Addiction hit a record low during World War II. The country was prosperous. There was a sense of national purpose. Jobs were abundant and wages were high. Millions of susceptible youths joined the armed forces. There they might acquire the vices of smoking, drinking, and swearing, but not heroin using, at least not in the 1940s (Burnham 1993, 70–71, 101, 220–21). Back on the streets, prices were high and so was adulteration; black-market heroin was often only 1 percent pure by 1944, when it could be found at all.[16] Addicts had to boil down paregoric, eat *yen-shee* (the residue from smoking opium), bribe physicians, forge prescriptions, rob drugstores, switch to other drugs (such as barbiturates), or just plain quit (see Courtwright et al. 1989, 89, 107–8, 131, 168, 193–94, 268; "What's Cooking?" 1945, 99–100; U.S. Treasury Dept., Bureau of Narcotics 1945, 17; "History of Heroin" 1953; Maisel 1945). Some evidently stayed clean for the duration. Voluntary commitments of drug addicts in New York City,

the nation's erstwhile heroin capital, dipped to almost zero by 1943 (Rosenthal 1951).

Heroin receded from the national consciousness during and immediately after the war. "In 1947 I was as innocent about drugs as I was about sex," William Styron wrote in his quasi-autobiographical *Sophie's Choice* (Styron 1979, 311):

> Our present-day drug culture had not seen, that year, even the glimmerings of dawn, and my notion of addiction (if I had ever really thought of such a thing) was connected with the idea of "dope fiends"—goggle-eyed madmen in straight jackets immured in backwater asylums, slavering molesters of children, zombies stalking the back streets of Chicago, comatose Chinese in their smoky dens, and so on. There was the taint about drugs of the irredeemably depraved, almost as evil as certain images of sexual intercourse—which until I was almost thirteen I visualized as a brutish act committed in secrecy upon dyed blondes by huge drunken unshaven ex-convicts with their shoes on. As for drugs, certainly I knew nothing about the types and subtle gradations of these substances.

But then, in the late 1940s and early 1950s, the narcotic problem became more visible as the flow of smuggled heroin resumed (Jonnes 1996, chs. 8 and 10). The rituals of postwar addicts, the heroin complex, remained much the same. But their background was changing. They were now younger and darker skinned. Just how many teenage addicts there were in the 1950s was much disputed. One of the more comprehensive studies, of newly reported addicts in New York State from 1952 to 1958, showed that about 19 percent were under twenty-one and just 5 percent under eighteen years of age (Schlesinger et al. 1959, 4389). Still, any teenage addiction was alarming, particularly in the apprehensive Cold War climate of the 1950s (see Campbell 2000).

Latino and, especially, black narcotic use was up sharply in the late 1940s and 1950s. From 1935 to 1947 only about 10 percent, on average, of those admitted to the Lexington and Fort Worth hospitals were black. By the mid-1950s the figure had risen to over 40 percent. "I saw a shift in the population," recalled one veteran of Lexington, who was readmitted in 1956. "They came mostly from Chicago and New York, the big cities. They used to say, 'Well, here comes a bunch on the chain from D.C.'—there were a lot of blacks from D.C. If you were arrested and brought there you were hooked up and chained, handcuffed to one another" (Courtwright et al. 1989, 15, quotation at 307).

Explanations for this dramatic development are various and politically sensitive. Some still cling to the "Godfather" conspiracy theory: the Mob decided to dump drugs in black neighborhoods. More plausible is Jill Jonnes's hepster role model theory. When word got out that the coolest of the cool—black

jazzmen like Charlie Parker—used heroin, scores of imitators, "conscientious objectors to the American Dream that never included them," quickly followed (Jonnes 1996, ch. 7, quotation at 121). This began a chain reaction. Older users initiated younger ones, who looked up to them. Dealers became admired figures. Bumpy Johnson, who dealt kilos and drove a Cadillac, was an "idol" to the author Claude Brown's generation, as were "other less well-known, neighborhood racketeers" (Brean 1951, 119; see also Brown 1984, 54). Brown, of course, grew up in Harlem. Underlying all of this was the shift of black population from the rural South, where heroin was practically nonexistent, to the crowded urban slums, where the traffic was well established. Exposure matters.

The postwar revival of the heroin traffic, the involvement of organized crime (and, according to Anslinger, Communist China), the perceived spread of addiction among teenagers, and the very real spread of addiction in the barrios and ghettos contributed to a further hardening of narcotic policy. The outstanding feature of the 1951 Boggs Act, the 1956 Narcotic Control Act, and analogous state legislation ("Little Boggs Laws") was increasingly stiff mandatory minimum sentences. The 1956 statute even permitted juries to recommend the death penalty for those convicted of sales to minors, an indication of the symbolic importance of the endangered-youth issue.[17]

HEROIN AS THE CENTERPIECE OF AMERICA'S DRUG WARS

The events that followed 1956 properly belong to other contributors to this volume. I would, however, like to make one general observation about heroin's first century. Like any psychoactive drug that escapes the realm of healing for that of self-indulgence, heroin provoked a legislative response. What set heroin apart was the strength and persistence of that response.

By my count, there were five, or perhaps four and a half, federal drug wars in the twentieth century. Heroin figured prominently in three of them. The first, longest, and most significant lasted from 1909 to about 1924. Driven by concerns about heroin and other narcotics, it resulted in the de facto criminalization of nonmedical addiction. The postwar expansion of the inner-city heroin complex triggered another major campaign in the 1950s, engineered by the Bureau of Narcotics and its congressional allies. Yet another heroin epidemic in the late 1960s gave rise to Nixon's drug war, a more enlightened and flexible undertaking than either its predecessors or its successor. Heroin was missing in action only in the marijuana skirmish of the mid-1930s and the Reagan-era drug war. The latter grew out of the increased use of marijuana and cocaine, especially in the form of crack. Though crack eclipsed heroin as drug

enemy number one in the late 1980s, heroin made a slow but steady comeback during the 1990s, when increasing supplies and unprecedented levels of street purity provoked new concern. Once again, cocaine users, burned out and jittery, began turning to heroin, just as they had earlier in the century. While it seems unlikely that heroin will again completely supplant cocaine, as it did during the 1920s and 1930s, or will recover the black-market prominence that it enjoyed during the 1950s and 1960s, its suppression remains a critical object of American drug policy.

NOTES

1. From *One Hundred Years of Heroin*/David F. Musto; Chapter 1, pp 3–22, "The Roads to H: The Emergence of the American Heroin Complex, 1898–1956," by David T. Courtwright. Copyright © 2002 by David F. Musto. Reproduced with permission of Greenwood Publishing Group, Inc. Westport, CT.
2. "Questionaire [*sic*] re Drug Habit," Box 6, and Kolb to John Remig, November 14, 1927, Box 4 (Kolb Papers n.d.). This was an almost universal judgment by the 1920s. William White (pers. comm.), an authority on treatment history, adds that early-twentieth-century proprietary drug cures of the mail-order variety almost never mentioned heroin in their advertising.
3. The authoritative *Merck's 1907 Index* refers only to heroin's use as a "cough-sedative" and antispasmodic, recommended in cases of phthisis (tuberculosis), bronchitis, asthma, and so forth. For further discussion of the controversy surrounding heroin's use as an analgesic, see Courtwright (1982, 93). For more on early warnings, see Musto (1974).
4. See the "Heroin" and "Antikamnia" brochures in the Antikamnia Chemical Company Collection (n.d.). Similar items can be found in the Charles L. Mitchell Company Collection (n.d.) and the Lehn and Fink Collection (n.d.). I am grateful to Charles Greifenstein for calling this material to my attention. See also Fiedler (1979) and Haussmann (1891).
5. Another interesting case involves Bromo-Seltzer, an over-the-counter preparation consisting of acetanilide, bromide, caffeine, and citric salts. In the 1930s several deaths were attributed to the product, much to the discomfiture of its manufacturers and advertisers. It turned out, however, that the victims were using Bromo-Seltzer as an analgesic to cope with headaches and other painful symptoms arising from chronic diseases that were the real causes of their demise. Fifty years earlier, they probably would have been using opiates (see J. Walter Thompson Company Archives 1937).
6. Leroy Street tells a version of the story in which the first users were Chinese "hop-heads" arrested sometime during or after 1909 for attempting to secure smoking opium in defiance of the national import ban. "A lot of them had a bad cough; when they were in jail they gave them heroin, which is a *marvelous* cure for a cough.

But aside from that, it's a hell of an addictive drug" (Courtwright, Joseph, and Des Jarlais 1989, 289).

7. The date of the first heroin addiction case admitted to Bellevue also was 1910 (Bloedorn 1917). In 1911 Harlow Brooks and H. R. Mixwell noted that "the habit is by no means infrequent especially on the extreme east and west sides of [New York City]" (Brooks and Mixwell 1911, 386). In short, all available historical evidence suggests that nonmedical heroin use took hold in New York City during or just before 1910 and expanded rapidly thereafter. The number of Bellevue heroin cases went from 1 in 1910 to 649 in 1916 (Bloedorn 1917).
8. For national cigarette production figures, see Brecher et al. (1972, 230). For New York consumption, see Kluger (1996, 62). A study conducted by Sylvester Leahy showed a preponderance of children of immigrants (n = 58) over children of native-born parents (n = 53) among heroin addicts (Leahy 1915).
9. Wiley (1917, 150). I am grateful to H. Wayne Morgan for calling this source to my attention.
10. The idea that tobacco leads to drunkenness and other vices has a long history (see, e.g., Short 1750, 250; Rush 1798, 270; Grimshaw 1853, 27–28).
11. Lawrence Kolb, the man fated to deal with America's narcotic obsessives, thought that these views could not be crazier. He diplomatically said so in his testimony at the same hearings (U.S. Senate, Committee on Printing 1924, 26–27). Nevertheless, the direct heroin-crime link pushed by Hobson and others affected public perception.
12. For a critical account of Porter's subsequent diplomatic maneuverings, see McAllister (2000, chap. 3).
13. Meyer and Parssinen (1998, chaps. 1, 9) offer a comprehensive overview of international developments and how they impacted on the U.S. black market. For more on the 1931 convention as a watershed in international control efforts, see McAllister (2000, chap. 3). Global figures on the declining licit manufacture are found in "History of Heroin" (1953).
14. William S. Burroughs, the "master addict" of his generation, was of the opinion that heroin was eight times as powerful as morphine (from letter to Allen Ginsberg in Burroughs 1993, 215).
15. In the 1920s decks reportedly sold for $1.00, $1.50, $2.00, $3.00, and $5.00 (New York State Commission on Prisons 1924).
16. Well before Pearl Harbor (December 1941), Anslinger had been stockpiling heroin as a strategic material, which drove up the price. The war itself brought tighter border controls, shipping disruptions, and travel restrictions. Seats on the remaining commercial airplanes, for example, were assigned on a priority basis to key government and military personnel. The term *VIP* was born of wartime flight rationing.
17. For more on postwar minority use and the federal laws of the 1950s, see Courtwright Joseph, and Des Jarlais (1989, 14–20) and Musto (1974). The subject is also treated at length in Courtwright (2001).

REFERENCES

The American disease: Origins of narcotic control. 1987. Enl. ed. New York: Oxford University Press.

Anslinger, Harry J. 1936. Memorandum to the Secretary of the Treasury. September 3. Vertical file "Heroin-History." Drug Enforcement Administration Library, Arlington, VA.

Antikamnia Chemical Company Collection. n.d. Medical Trade Ephemera. College of Physicians of Philadelphia Library.

Bailey, Pearce. 1916. The heroin habit. *New Republic* 6 (April 22): 314–16.

Blanchard, R. M. 1913. Heroin and soldiers. *Military Surgeon* 33.

Bloedorn, W. A. 1917. Studies of drug addiction. *U. S. Naval Medical Bulletin* 11.

Brean, Herbert. 1951. Children in peril. *Life*. June 11.

Brecher, Edward M., et al. 1972. *Licit and illicit drugs*. Boston: Little, Brown.

Brooks, Harlow, and H. R. Mixwell. 1911. Two cases of heroin habituation. *New York State Journal of Medicine* 11.

Brown, Claude. 1984. Manchild in Harlem. *New York Times Magazine*, September 16.

Burnham, John. 1993. *Bad habits: Drinking, smoking, taking drugs, gambling, sexual misbehavior, and swearing in American history*. New York: New York University Press.

Burroughs, William S. 1993. *The letters of William S. Burroughs, 1945–1959*. Edited by Oliver Harris. New York: Viking Press.

Campbell, Nancy D. 2000. *Using women: Gender, drug policy, and social justice*. New York: Routledge.

Caution regarding heroin. 1900. *Druggists Circular and Chemical Gazette* 44 (March): 44.

Charles L. Mitchell Company Collection. n.d. Medical Trade Ephemera. College of Physicians of Philadelphia Library.

Chase, J. Frank, et al. 1912. *The dope evil.* Boston: New England Watch and Ward Society.

Courtwright, David T. 1982. *Dark paradise: Opiate addiction in America before 1940*. Cambridge, MA: Harvard University Press.

———. 2001. *Dark paradise: Opiate addiction in America before 1940*. Enl. ed. Cambridge, MA: Harvard University Press.

Courtwright, David, Herman Joseph, and Don Des Jarlais. 1989. *Addicts who survived: An oral history of narcotic use in America, 1923–1965*. Knoxville: University of Tennessee Press.

D'Erlanger, Baron Harry. 1936. *The last plague of Egypt.* London: Lovat Dickson and Thompson.

Farr, Clifford B. 1915. The relative frequency of the morphine and heroin habits: Based upon some observations at the Philadelphia General Hospital. *New York Medical Journal* 101.

Fiedler, William C. 1979. Antikamnia: The story of a pseudo-ethical pharmaceutical. *Pharmacy in History* 21.

Floret, [Theobald]. 1898. Klinische Versuche abet die Wirkung and Anwendung des Heroin. *Therapeutische Monatshefte* 12.

Greenwood, Joseph A. n.d. Estimated drug abusers, estimated addicts, 1969–1975. TS memorandum, vertical file "Addiction-Incidence, 1973–1975." Drug Enforcement Administration Library, Arlington, VA.

Grimshaw, A. H. 1853. *An essay on the physical and moral effects of the use of tobacco as a luxury*. New York: William Howard.

Haussmann, F. W. 1891. Antikamnia. *American Journal of Pharmacy* 63.

Helbrant, Maurice. 1941. *Narcotic agent*. New York: Vanguard.

Heroin and the International Conferences on Narcotics of 1925 and 1931. 1953. *Bulletin on Narcotics* 5.

Heroin hydrochloride. 1906. *Journal of the American Medical Association* 47.

History of heroin. 1953. *Bulletin on Narcotics* 5.

Illicit price of cocaine in two eras: 1908–14 and 1982–89. 1990. *Connecticut Medicine* 54.

Jonnes, Jill. 1996. *Hep-cats, narcs, and pipe dreams: A history of America's romance with illegal drugs*. New York: Scribner.

J. Walter Thompson Company Archives. 1937. Forum Series. Emerson Drug Company. January 12. Special Collections Library, Duke University.

Kane, Francis Fisher, et al. 1917. Drugs and crime: Report of Committee '0' of the Institute. *Journal of the American Institute of Criminal Law and Criminology* 8.

Kluger, Richard. 1996. *Ashes to ashes: America's hundred-year cigarette war, the public health, and the unabashed triumph of Philip Morris*. New York: Knopf.

Kolb Papers. n.d. National Library of Medicine. Bethesda, MD.

Leahy, Sylvester. 1915. Some observations on heroin habitués. *Psychiatric Bulletin of the New York State Hospitals* 8.

Lehn and Fink Collection. n.d. Medical Trade Ephemera. College of Physicians of Philadelphia Library.

Maisel, Albert Q. 1945. Getting the drop on dope. *Liberty*, November 24.

Manges, Morris. 1898. The treatment of coughs with heroin. *New York Medical Journal* 68.

———. 1900. A second report on the therapeutics of heroin. *New York Medical Journal* 71.

McAllister, William B. 2000. *Drug diplomacy in the twentieth century: An international history*. London: Routledge.

McIver, Joseph, and George E. Price. 1916. Drug addiction. *Journal of the American Medical Association* 66.

McTavish, Jan R. 1987. Aspirin in Germany: The pharmaceutical industry and the pharmaceutical profession. *Pharmacy in History* 29.

McWilliams, John C. 1990. *The protectors: Harry J. Anslinger and the Federal Bureau of Narcotics, 1930–1962*. Newark: University of Delaware Press.

Memorandum and data sheet. 1938. March 16. Treasury Department file 0120-9, record group 170. National Archives II, College Park, MD.

Meyer, Kathryn, and Terry Parssinen. 1998. *Webs of smoke: Smugglers, warlords, spies, and the history of the international drug trade*. Lanham, MD: Rowman and Littlefield.

Musto, David E. 1974. Early history of heroin in the United States. In *Addiction*, ed. Peter G. Bourne. New York: Academic Press.

New York State Commission on Prisons. 1924. Special Report on Drug Addiction. Vertical file "Addiction—Incidence [to] 1959." Drug Enforcement Administration Library, Arlington, VA.

New York State Narcotic Drug Control Commission. 1920. *Second annual report*. Edited by Walter R. Herrick. Albany: J. B. Lyon.

Pettey, George E. 1903. The heroin habit: Another curse. *Alabama Medical Journal* 15: 174–80.

Rosenthal, Herbert C. 1951. How much of a menace is the drug menace? *Pageant*, October.

Rubin, Vera, ed. 1975. *Cannabis and culture*. The Hague: Mouton.

Rubin, Vera, and Lambros Comitas. 1975. *Ganja in Jamaica: A medical anthropological study of chronic marijuana use*. The Hague: Mouton.

Rush, Benjamin. 1798. *Essays: Literary, moral and philosophical*. Philadelphia: Thomas and Samuel E. Branford.

Scheffel, Carl. 1918. The etiology of fifty cases of drug addictions. *Medical Record* 94.

Schlesinger, Edward R., et al. 1959. Experience with reporting of habitual users of narcotic drugs in New York State. *New York State Journal of Medicine* 59.

Short, Thomas. 1750. *Discourses on tea, sugar, milk-made wines, spirits, punch, tobacco, etc*. London: T. Longman and A. Millar.

Spillane, Joseph E. 2000. *Cocaine: From medical marvel to modern menace in the United States, 1884–1920*. Baltimore: Johns Hopkins University Press.

Stokes, Charles E. 1918. The problem of narcotic addiction of today. *Medical Record* 93.

Street, Leroy, with David Loth. 1953. *I was a drug addict*. New York: Random House.

Styron, William. 1979. *Sophie's choice*. New York: Random House.

Symposium "The Doctor and the Drug Addict." 1920. *Journal of the American Medical Association* 75.

Towns, Charles B. 1916. *Habits that handicap: The menace of opium, alcohol, and tobacco, and the remedy*. New York: Century.

Tuttle, Charles H. n.d. Memorandum to the National Commission on Law Observance and Enforcement. Box 56. Richmond C. Hobson Papers. Library of Congress.

U.S. Senate, Committee on Printing. 1924. *Hearing . . . on a resolution to print fifty million additional copies of an article entitled "The peril of narcotic drugs" . . . 3 June 1924*. Washington, DC: U.S. Government Printing Office.

U.S. Treasury Department. 1919. *Traffic in narcotic drugs: Report of Special Committee of Investigation appointed March 25, 1918, by the secretary of the treasury*. Washington, DC: U.S. Government Printing Office.

U.S. Treasury Department, Bureau of Narcotics. 1945. *Traffic in opium and other dangerous drugs for the year ended December 31, 1944.* Washington, DC: U.S. Government Printing Office.

Westermeyer, Joseph. 1976. The pro-heroin effects of anti-opium laws in Asia. *Archives of General Psychiatry* 33.

What's cooking? 1945. *Newsweek,* April 9.

W. H. Schieffelin and Company Collection. n.d. Medical Trade Ephemera. College of Physicians of Philadelphia Library.

Wiley, Harvey W. 1917. The alcohol and drug habit and its prophylaxis. In vol. 9 of *Proceedings of the Second Pan American Scientific Congress*. Washington, DC: U.S. Government Printing Office.

Wolff, Paul. 1932. Drug addiction—a world-wide problem. *Journal of the American Medical Association* 98.

Wood, Horatio C., Jr. 1899. The newer substitutes for morphine. *Merck's Archives* 1.

This page intentionally left blank

Chapter **18**

Methadone: The Drug, the Treatment, the Controversy[1]

Herbert D. Kleber, MD

The current methadone controversy is the most recent round in a policy struggle that has been going on since 1964, when the first program began. An appropriate quotation with which to begin a discussion of this rocky history might be the old line "No good deed goes unpunished." Many consider methadone treatment one of the really good deeds, if not great deeds, in the history of the treatment of addiction, yet it clearly is also one of the most assailed.

Methadone was discovered by the Germans during World War II, when they had opiate shortages because of problems with sea routes. After the war it was taken over by Lilly Pharmaceutical and named Dolophine. Some of the anti-methadone groups have claimed the name came from Adolph Hitler and that it is used to weaken the African American community. Actually, Dolophine was named from the Latin *dolor* (pain). Supporters believe that its use can relieve addicts' pain and free them from enslavement to heroin.

After World War II, research on methadone was carried out at the Addiction Research Center in Lexington, Kentucky. The papers from those early days are fascinating. Researchers had little to go on in terms of appropriate dosage. In some studies they put addicts on doses as high as 350 to 400 mg, and then abruptly stopped. The patients would withdraw cold turkey, and the staff would carefully chart what the patients went through. As one of them said, "Man, this stuff never lets you go." Three weeks later, four weeks later, they would still be suffering withdrawal symptoms.

As more became known about the drug, however, unwanted effects were reduced and the benefits became clear. It began to replace morphine or codeine,

the drugs in main use in the late 1940s and early 1950s, as the preferred agent to detoxify individuals from heroin. In 1964 Vincent Dole, an internist, and Marie Nyswander, a psychiatrist, developed the use of methadone as a maintenance drug. Dole had started from the point of view that heroin addiction was really a metabolic disease. Using an insulin model, he had looked for a maintenance drug. Nyswander had written a book based on her experience in treating addicted jazz musicians and the frustrations and difficulties that ensued. She was searching for a medication that would improve outcome. The two, working at Rockefeller University, were trying different maintenance agents; when they got to methadone, they found that their patients behaved very differently. They began to make their beds; they began to talk about wanting to go to work. From that humble beginning, the idea of methadone as a maintenance drug took shape.

There are many key players in this history. Jerry Jaffe, Mary Jean Kreek, Frances Gearing, and Joyce Lowinson have all played major roles in the spread of methadone since the mid-1960s. The controversy about methadone then and now is captured in an anecdote from 1966. At that time I was at the U.S. Public Health Service hospital in Lexington, Kentucky, running one of the addiction treatment units. A program called Daytop Village had just started in New York, and I invited the director to give a talk to the patients at Lexington—although the word "patients" may not be quite accurate: Lexington was a prison. Two-thirds of the thousand residents were serving sentences of from one to ten years. The remaining one-third were voluntary admittees, who signed in because at that time it was the only place to get treatment for narcotic addiction in the United States east of the Mississippi, as its counterpart prison hospital in Fort Worth, Texas, was for west of the Mississippi. The Daytop director talked about therapeutic communities, and, during a question-and-answer period, one of the patients asked about methadone, which they had just heard about. The response was a classic remark: "I think methadone maintenance is a great idea: we should give money to bank robbers, women to rapists, and methadone to addicts."

That was 1966. Five years later the speaker went on to head a methadone program. So life treats us in funny ways. But it is one of my favorite anecdotes because it captures the feeling that a lot of people still have about methadone maintenance—that it constitutes immoral pandering to criminals rather than a scientific treatment of a disease.

In 1968 methadone began to spread out of New York. Jerry Jaffe began his program in Chicago, Bill Weiland started in Philadelphia, and I began in New Haven. All of these programs were supported by NIMH grants. In New Haven we set up a model multimodality treatment program with central intake.

Methadone was just one of the modalities we were using. We also had a therapeutic community, an adolescent program, and narcotic antagonists. In those days, cyclazocine was the antagonist of choice. Also during that era, we learned you didn't need to admit everyone as an inpatient to start, as they did at Rockefeller. Bill Weiland began outpatient induction. We started patients on a day program, which became a fixture in New Haven for many years, to deal with the fact that many of our addicts basically needed resocialization. Anyone who was not working was required to come from 9:00 A.M. to 4:30 P.M. five days a week. During that time they had intensive group therapy, individual therapy, and help with vocational and educational issues. I credit the success of our methadone program in large part to those initial six weeks, which helped to break the code of the streets and to get people involved in wanting to lead not simply a life on methadone, but a whole different life.

As methadone was spreading, so was the controversy. There was continued opposition from therapeutic communities. I was at a number of meetings in those days where, if it was predominantly a TC audience and someone got up to speak about methadone, he would get booed; if it was primarily a methadone audience and someone got up to speak about TCs, *he* would get booed.

There was also a lot of opposition from the black community, some of whom described methadone maintenance as genocidal—an attempt by the white establishment to control young black males by keeping them permanently addicted. I remember vividly one episode during the Black Panther trial in New Haven. Some of the members of that group came to me and said, "If you don't close your methadone program, we're going to burn it down." Their rationale was not that it was not successful, but that it was *too* successful: it helped addicts quit heroin but did not deal with the conditions in the ghetto that they believed *produced* addiction—such as racism, unemployment, and poverty. They viewed it as putting a sort of Band-Aid on the ghetto, an attempt to avoid the total revolution they believed would soon happen.

I believe the thinking was somewhat similar to what the Communists did in the 1930s, trying to kill off the labor unions. The feeling was that labor unions would give the workers half a loaf, and if you did that, they would not rise up and overthrow the whole economic system. These feelings about methadone from some members of minority communities have never totally gone away, although at times they are more prominent than at others. In any event, our clinics neither closed down nor burned down, but instead grew and prospered, because of the help patients were getting.

In retrospect, the 1970s was the golden era of methadone. Bob DuPont in Washington, D.C., showed that you could rapidly develop a large system and bring a great number of the addicts in the city into quality treatment. The fed-

eral government encouraged the spread of methadone maintenance via Bud Krogh, Jerry Jaffe, and SAODAP. By 1973 there were approximately four hundred methadone programs throughout the United States. The National Institute on Drug Abuse (NIDA) was founded, and it was used initially not simply to do research, but also to spread methadone programs.

During that same era, there were a number of studies on methadone safety. There were a lot of myths on the street then about methadone: it got into your bones, it ate away your teeth, it destroyed your vision. In systematically looking at those systems where the street rumors had it that methadone was dangerous—including the kidneys, the eyes, the musculoskeletal system, and the heart—our group at Yale in five-year studies, and Mary Jean Kreek in New York in eight-year studies, found no evidence of any damage due to protracted methadone use. We concluded that addicts could be on methadone for at least five to eight years without adverse physical effects.

Another question began to emerge: Should people be on methadone for life, or, if you rehabilitate them, can they be detoxed and remain abstinent? Dole and Nyswander advocated very strongly, if not the universal lifetime model, then at least keeping patients on methadone for many years—some, perhaps, for their whole life. They believed that being on heroin for many years changed the addict's brain and changed his metabolism, probably permanently.

Our program in New Haven took a different direction. We believed that if you could rehabilitate addicts, you would be able to get them off methadone, especially if you could solve the withdrawal problem. We spent a lot of energy looking at a variety of ways of doing detoxification. From that interest, we demonstrated that clonidine, on the market for hypertension, could markedly decrease withdrawal symptoms, and that combining clonidine and naltrexone could yield a rapid opioid detox with even better results. These methods increased the likelihood that people who had been successfully rehabilitated could get off methadone. While these techniques improved on existing approaches, we still do not have good enough methods by which to end reliance on methadone, and I believe that is one of the major problems. We're not going to know how long people need to be on methadone until we have a much better way of detoxifying them. Buprenorphine is one of the drugs that may help in the future to resolve the controversy as to what percentage of recovering addicts need to remain on methadone versus what percentage can successfully detox from it.

I have a favorite anecdote about the detox controversy. Dole and Nyswander came up to visit our program and give a seminar. One of our counselors, a former methadone patient who had been clean for about six months, asked a lot of pointed questions about the theory that methadone had to be taken for life.

When the seminar was over and we went out for dinner, Marie and Vince said, "Tell us about that guy who was asking all the questions. Has he been on methadone and is now withdrawing?" I replied, "Yes, he's been clean for six months." They then said, "Ah, we thought so. It's that irritability due to the protracted methadone withdrawal that really got him to ask all those questions." Perhaps that was the case, perhaps not, but it reveals the strong feelings of even such wonderful researchers as these two. Parenthetically, the counselor was able to stay off heroin, and methadone, for decades.

While there were other problems in the 1970s, including concerns about methadone overdose among children and nonaddicts who might become exposed to the drug and about methadone diversion, in many ways it was the golden era, a time when, for the most part, methadone was respected and seen as an important part of the treatment system for addicts.

In the 1980s there was a backlash. Early in that decade, the federal block grant replaced direct NIDA grants as a way of funding drug programs. Each state received a certain amount of money that could be used for methadone or for other kinds of treatment, and you began to see a discontinuation of methadone in a number of places. The best study of this natural experiment was the one in Bakersfield County, headed by Bill McLaughlin. What they found when they looked at the patients who were detoxed of necessity, because the clinics were closed, was that, while some stayed clean and some found treatment in other counties, many did not fall into either of those categories. Among this latter group, there were an increased number of deaths and of return to heroin addiction, and an increased amount of criminal activity. Unfortunately, this study did not carry much weight. One of the things I learned in my days in government is that, much as we are reluctant to admit it, science does not drive policy. It may push it a bit—policy makers say they would like to know the science—but, at the end of the day, that is not how they make their decisions.

During the Reagan years, some of the White House drug policy advisers were quite antimethadone. I think of Carlton Turner, for example. That viewpoint peaked in 1988 with the White House Conference for a Drug Free America, which was vehemently antimethadone. They demonized it, as well as NIDA, which they viewed as being "soft on drugs," and they called for a congressional investigation of that agency. This time was probably the lowest point methadone reached, in terms of national policy.

Also during the 1980s, however, evidence was accumulating about the relationship of methadone programs and HIV status. Studies showed that individuals on methadone had a much lower incidence of conversion to HIV-positive status. In England the harm reduction movement was taking hold, driven by the concern over AIDS. The English made a conscious decision in the mid-to-late

1980s that preventing AIDS was more important than dealing optimally with drug abuse, and that, therefore, the main focus of their drug program should be reducing the spread of the HIV virus rather than rehabilitating addicts. The philosophy that flowed from this decision was that no matter how badly a patient is doing on methadone—no matter how much cocaine he is doing, how much alcohol he is drinking, whether he is involved in criminal behavior, and so forth—you keep him on methadone, because if you throw him off, instead of using drugs once a day or once every other day, he will go back to using three or four times a day, and increase the likelihood of HIV conversion.

This attitude took hold in the United States as well, although some of us continued to take the position that the best way to prevent HIV was to offer good-quality treatment with high rather than low expectations. Also during the 1980s, we had the rise of private, for-profit methadone programs. One of the things I am proud of is that during my years in Connecticut I fought to keep for-profit methadone out of the state. Unfortunately, after I left Connecticut in 1989, that Horatio-at-the-bridge position was lost. In other states it was lost a lot earlier. In California, for example, by 1990, 90 percent of their two hundred methadone clinics were privately owned. The major source of funds was patient fees. Between 1980 and 1990, there was an 80 percent increase nationally in reliance on patient fees as the primary source of funding for methadone programs. Along with the rise of the for-profit programs came a curtailment in services.

In the mid-1970s the "slot cost" for a methadone slot was roughly $1,700 a year. In 1990 the Institute of Medicine Committee on Treatment Effectiveness, on which David Courtwright and I, among others, served, showed that from 1976 to about 1986, funding for methadone had increased by only about 9 percent, from $1,700 a slot to $1,850 a slot, while costs went up by at least 35 to 40 percent during those years of high inflation. This disparity eroded the ability of clinics to provide services, and the spread of for-profit programs had a similar effect: if you provided fewer services, you could make more money.

Clinics developed where you would go in, and a drawer like the one on an ATM would come out. You'd put in your credit card, your cash, or your Medicaid card, the drawer would be pulled back, and then it would be pushed forward again, and you'd get your card back, with your dose of methadone. On the wall there typically would be a sign that said, "If you want counseling, please ask one of the staff." Unfortunately, from being the exception in the 1970s, that kind of program has proliferated, and it has had, I believe, a negative impact on public perception. If the methadone patients are basically being maintained but not treated, if the public sees that these patients continue to use cocaine and continue to engage in criminal behavior, they are going to ask, Why should we be paying for this? Why should we permit it?

In the 1990s, when I was Deputy Director for Demand Reduction under Bill Bennett at the Office of National Drug Control Policy, we tried to reverse this movement and to increase respect again for methadone treatment. We put out a White Paper that said very clearly that methadone maintenance was both a legitimate and an important part of the spectrum of drug abuse treatment. We doubled the federal funding for treatment, so that services would be expanded and improved. Unfortunately, the resulting increase in funding accomplished less than we had hoped, because, as we found out later, while we were putting money in the front door via the block grant, the states were pulling out their contributions to program funding through the back door. I tried to get Congress to pass a law that would have made that more difficult, but they were not interested. They said that the Department of Health and Human Services (HHS) already had the power to do that if they wanted to. Unfortunately, HHS was not interested in such a law, nor even in using the power they already had to prevent this supplanting of funds.

The issue of "interim methadone," or "low-threshold methadone," was raised during this period in relation both to the AIDS issue and to the fact that it was very hard to expand methadone treatment. During much of the 1990s, we had about 120,000 methadone slots for somewhere between 750,000 and 850,000 heroin addicts. That's less than one slot for every six potential patients, so there was a push by many people concerned with drug treatment to adopt the so-called Hong Kong model with ten thousand people on methadone and eleven social workers doing therapy. Similarly, in the Netherlands, the dose of methadone was kept low so that patients could continue to use heroin if they wished, and addicts could drop in and out of the program.

I don't like these models. The definitive study, I believe, was Tom McLellan's beautifully designed study, published in *JAMA* in 1984, which randomly assigned addicts to one of three groups: methadone only, methadone as usual (methadone with one counseling session a week), or enhanced methadone (not just drug counselors but also psychologists, psychiatrists, and medical help). At the end of three months, they found that the first group was doing so badly that, for ethical reasons, they felt they had to take two-thirds of them out of the methadone-only protocol and move them into one of the other two types of programs. The enhanced methadone group had markedly lower levels of heroin and cocaine use, needle sharing, and unemployment. Within a few months, the new patients in the groups with counseling were doing just as well as the people who had been there from the beginning. So it clearly was not the patients who accounted for the different results; it was the program. If you have low expectations and minimal services for your patients, they respond appropriately. If you have higher expectations and better services, they respond to that, too.

At the same time these studies were going on, the Dutch were beginning to find out that their methadone policy was flawed. The Amsterdam Health Department published a paper showing that its low-threshold, low-dose methadone approach did not reduce HIV risk. That led to some rethinking of their position. They retained the low-threshold requirement but adopted higher dose levels.

Increasingly, however, states curtailed the length of time patients were allowed to be on methadone maintenance. Oklahoma limited addicts to one year on methadone over a lifetime. Office-based dispensing also aroused disagreements. The central question was, Should people continue indefinitely to have to come to a methadone clinic to obtain the drug, or should they be able to get methadone in a doctor's office? Some people argue for allowing physicians to dispense the drug at the beginning of treatment; others want it only after stabilization and improvement, as one way of increasing the number of methadone slots. I have trouble with the former. I don't have trouble with the latter.

At the same time as these controversies were taking place, the scientific community was confirming the efficacy of methadone treatment. An NIH consensus statement in 1997 asserted the value of methadone. In 1998 General McCaffrey pronounced probably the strongest support I have heard from any director of the Office of National Drug Control Policy: "Methadone is very important to our strategy."

Support of the harm reduction philosophy in the 1990s, however, contributed to decreasing services in methadone programs. At the second national methadone consumers meeting in October 1998, for instance, the argument was the following: "Since methadone doesn't treat cocaine, and that's not why we're in methadone treatment, use of cocaine should never be considered an indication for termination from methadone. Patients should get lower or higher doses as they choose, and they should have the right to have low doses so they can continue to use heroin without the harder override caused by a high dose of methadone."

As is probably evident, I do not agree with much of that harm reduction position. One of the things I've learned in running a methadone program is that drug use is contagious. If you have a program with vigorous support and high expectations, your patients live up to that: you create a program with a relatively low use of other drugs compared to the average clinic. Since many methadone patients want to use other drugs—they are, after all, individuals who enjoy the effects of drugs, whether they be heroin, cocaine, alcohol, or marijuana—once a program permits some patients to use without consequences, the amount of illicit drug use that goes on in the program markedly increases. My experience has been that the more relaxed programs are, and the more tolerant of drug use,

the more they have both drug use and other illicit activities. No one should be surprised by this outcome.

In the summer of 1998, New York City launched its welfare-to-work program. Only about 20 percent of methadone patients were legally employed, and Mayor Giuliani concluded that methadone programs needed to do a better job of getting their patients to work. Unexpectedly, he announced a plan to eliminate methadone maintenance in the city. Of the approximately thirty-six thousand methadone slots in New York City at the time, only about two thousand were controlled by the city. The rest were controlled by New York State, which indicated no interest in cutting methadone. Nonetheless, the plan was highly controversial and generated heated debate.

In the rush to defend methadone and methadone patients that ensued, advocates lost sight of some unpleasant realities. According to the city Health and Hospitals Corporation, for instance, the employment rate in methadone programs in the city ranged, at that time, from a low of 15 percent in the Bronx to a high of 25 to 30 percent in Staten Island and Queens. The reasons are not mysterious. Addicts in many of these programs are on Medicaid, which pays the program over $100 per week. If the patient goes to work and earns perhaps $15,000–$20,000 a year, he may be able to pay the program only $10–$20 per week. If the program accepts that, they are losing up to a hundred dollars. So it is neither in their interest to encourage the patient to go to work, nor in the patient's interest to do so.

I hear from colleagues that the employment rate is a lot higher than 15 to 30 percent, but often it is off the books. Neither the programs nor the patients have an interest in making employment public knowledge when it might result in patients losing welfare and Medicaid—and, perhaps, access to methadone. While this may not be as much an issue in programs less dependent on reimbursement, it certainly is in other methadone programs. A reasonable solution might be to permit people to continue in treatment for a period after they leave welfare—through interim funding, for example.

How was the mayor's statement received? There was no outcry from the majority of people in New York, including the leaders of the black community, many of whom had complained about the mayor in other circumstances. The black community was ambivalent about methadone at best; many of its leaders were hostile toward methadone treatment. Thus they were not unhappy when the mayor attacked methadone clinics. The policy also played very well in conservative upstate New York and other places in the country. While various prestigious scientific organizations, such as the American Psychiatric Association, defended methadone, they were joined by neither key political leaders nor a substantial number of voters.

This should worry treatment specialists more than what a single city official does. Giuliani's order to eliminate methadone and substitute abstinence programs was rescinded after six months. In fact, the mayor decided to add $5 million in new funding to the city-run methadone programs, to improve staffing levels and overall quality of service. I was asked to work with the New York City programs to help achieve this, and spent a year doing so. The programs are now called Narcotic Addiction Treatment Programs. Eventually, they will offer both maintenance and abstinence programs. Some patients will be on methadone maintenance, others on buprenorphine or naltrexone. I see this as a future direction for existing methadone programs in general.

Over the long run, programs that lack political support have trouble competing for resources. Methadone supporters should acknowledge reasonable concerns, such as getting patients to work and preventing Medicaid abuses. High levels of cocaine abuse should be a signal for needed change, not defended as inevitable. On the wall of the therapeutic community that is part of the New Haven program I directed, there is a plaque that says, "You are neither the giant of your dreams nor the pygmy of your fears." Methadone is not the panacea for our heroin problem; it is not the perfect treatment for heroin addiction, but neither is it the demon that some people would like to make it. The truth lies somewhere in the middle.

NOTE

1. This overview of medical practitioners' experience with methadone is adapted from an after-dinner talk, thus the informal tone and absence of scientific citations. From *One Hundred Years of Heroin*/David F. Musto; Chapter 9, pp 149–158, "Methadone: The Drug, the Treatment, and the Controversy," by Herbert D. Kleber.

Chapter **19**

Prevalence of Use and Manufacture of Methamphetamine in the United States—Is the Sky Falling or Is It Not Really a Problem?[1]

Herbert C. Covey, PhD

Methamphetamine has been available for years; why all of the recent alarm? The answer is easy—many of the indicators of illegal substance use indicate that meth use is spreading throughout much of the United States, and authorities report that meth use has increased to "epidemic" proportions (Rawson, Anglin, & Ling, 2002). This spread has been likened to an epidemic moving from the West and Southwest to the rest of the country.

Is this spread unique to the United States? No. The World Health Organization recently reported that amphetamine and methamphetamine are the second most abused drugs in the world. The World Health Organization estimates that over 35 million people use or abuse amphetamine or meth (Rawson et al., 2002). This compares with 15 million for cocaine and 10 million for heroin. Meth abuse has taken over in some countries as the illegal drug of choice. For example, in Thailand methamphetamine abuse now accounts for almost 70 percent of addictions, and other Asian countries report a similar pattern of increasing abuse (Zickler, 2005c).

THE SPECIAL CHALLENGES OF METH

Does meth pose special challenges for agencies and staff trying to curb its use or address its affects on children and families? Some authorities suggest that meth use and addiction are similar to other stimulants, such as cocaine, and should be addressed using the same approaches. Others conclude that meth use and addiction are unique and require special strategies. Its use does, in fact, pose special challenges.

The drug is relatively abundant compared to illegal substances that have to be imported across national boundaries. It is often locally produced in home laboratories using materials such as household batteries and cold medicines (Bonné, 2001a). Although authorities have made efforts to control the precursor chemicals used to produce meth, it is impossible to eliminate them altogether (Rawson et al., 2002) because they are found in multiple household products.

Meth manufacture has been targeted in rural areas, which are attractive for dealers and manufacturers because the odors and by-products of meth production are more easily concealed from authorities and thus they are less likely to be discovered. Some of the methods of production involve chemicals and substances that are associated with farming and ranching, and their possession is not viewed as extraordinary. For example, anhydrous ammonia and iodine, both chemical precursors for meth manufacture, are commonly used on farms and ranches.

Meth can be produced on a small scale for personal use. Authorities have found labs in car trunks, mobile homes, microwave ovens, and small rooms. For example, one child welfare caseworker found a lab setup in a suitcase she planned to use to pack a child's clothing during the child's removal from a home.

More than a decade ago illicit meth production was restricted to a few "cooks" who kept their recipes relatively secret. Today, instructions for making meth are readily available in publications and over the Internet. Any Internet search engine will generate numerous recipes for producing meth. Internet chat rooms are full of self-proclaimed chemists (cooks) who are willing to provide free advice to aspiring cooks. For example, www.totse.com has a Web site that provides amateur recipes for cooking meth and making other illegal substances. If the Internet is not used, there are books that provide easily accessible recipes on how to produce the drug. The most widely known is Uncle Fester's (2002) *Secrets of Methamphetamine Manufacture: Including Recipes for MDA, Ecstasy, and Other Psychedelic Amphetamines*(6th ed.). Other similar books are available.

Another challenge of meth is that it is intense and powerfully addictive. It affords a strong sense of euphoria from its first use onward and does so for a relatively low cost when compared to other stimulants such as crack cocaine. Because of its effect on the brain and nervous system, heavily involved addicts have difficulty experiencing pleasure that is comparable to being high on the drug.

Users perceive other attractive benefits beyond being high on meth. Initial users and addicts view the drug as effective in reducing fatigue and allowing them to work for prolonged periods of time. For example, over-the-road truck

drivers use meth and amphetamines to stay awake on long road trips. Some students view it as a study aid that allows them to study and stay focused for hours. Other individuals, especially females, view it as an effective way to lose weight.

Treatment approaches must be modified to the special needs of the meth addict. For example, treatment information must be easy to understand (simple) and repeated. This is due partially to the effect meth has on brain functioning. The reasoning portion of the brain is affected by prolonged use. Meth addicts struggle with sequential thinking and instructions. In addition, the depression associated with long-term withdrawal must be addressed.

The distribution of meth is different from other illegal substances. It is not generally a street drug but is sold or provided by family, friends, and associates. This makes law enforcement activities challenging as the drug is less on the street than marijuana, heroin, cocaine, and other street drugs.

TRENDS IN THE UNITED STATES

Many substance abuse authorities believe that meth use and distribution has been a significant problem that is expanding in the United States (Freese, Obert, Dickow, Cohen, & Lord, 2000; Gibson, Leamon, & Flynn, 2002; Herrell, Taylor, Gallagher, & Dawud-Noursi, 2000; Hser, Evans, & Huang, 2005; Huber et al., 1997). Some basic data indicate this growth:

- Meth is showing up in the workplace. Between 1999 and 2003, the percentage of positive workplace drug tests containing amphetamines doubled, from 4.5 percent to 9.3 percent (Center for Substance Abuse Research, 2004).
- There are indications that it is spreading among white, low-income, rural Americans (Bonné, 2001a; Kraman, 2004).
- The National Drug Intelligence Center (2002) reported that crystal meth's availability is expanding in several states. The center reported that 31 percent of all state and local law enforcement agencies considered meth their primary drug threat, and 58 percent considered the availability of the drug in their communities to range from medium to high (National Drug Intelligence Center, 2003).
- The Office of National Drug Control Policy (2003a) reported that federal meth lab seizures increased from 327 in 1995 to 13,092 in 2001. This represents an increase of over 4,000 percent, or a 40-fold increase in the number of labs seized. This number does not reflect all of the undiscovered labs or small and undetected operations.
- The methamphetamine problem is becoming so large nationally that at the 2005 National Association of Counties (NACo) annual meeting, members viewed meth as an "epidemic" and voted to request the president to provide more funding for meth research, treatment, enforcement, education, and

cleanup (Leinwand, 2005; National Association of Counties, 2005). In the same vein, NACo released two surveys on the impact of meth on hospital emergency rooms and the challenges of treating its abuse (National Association of Counties, 2006a, 2006b).

- *National Drug Threat Assessment 2003* data reveal that, nationally, 36.2 percent of state and local law enforcement agencies identified meth as their greatest drug threat, ranking second only to cocaine (37%). State and local law enforcement agencies in the Pacific (90.9%), West Central (80.2%), and Southwest (51.6%) regions were more likely to identify meth as their greatest drug threat than were agencies in the Great Lakes (29.4%), Southeast (28%), and Northeast/Mid-Atlantic regions (2.7%; National Drug Intelligence Center, 2004, p. 1).

MEDIA ATTENTION

In addition to some general indicators, the topic of meth has recently been the subject of television, the press, music, and film. The mass media and pop culture have embraced the topic of meth use and manufacture, which had always been present but were less visible to the general public. As a result of all this attention, increased public awareness of meth, and increased media attention, it seems that the drug is reaching epidemic proportions in many regions of the country.

The Drug Enforcement Administration (DEA; Web site at http://www.usdoj.gov/dea/pubs/pressrel/meth) and the Partnership for a Drug-Free America (http://www.drugfreeamerica.org) contain examples of recent press releases regarding meth, as do other Web sites. There are a number of other examples of recent media titles and press clips regarding meth use:

- "Meth's Deadly Buzz—Sioux City, Iowa" In many ways, this article observes that meth is the crack cocaine of the new millennium. Much like crack, which swept across the nation in the 1980s and 1990s, meth use has hit epidemic proportions in the past several years. Crack plagued inner cities and the black community; methamphetamine is thriving in cities like San Francisco and is sweeping across the Midwest and headed east. It has quietly become America's first major homegrown drug epidemic (Bonné 2001, 2001a).
- "Methamphetamine's Young Victims: Homes Doubling as Drug Labs Pose Serious Dangers to Kids," *Denver Post*, October 20, 2002 (Herdy, 2002).
- "The Meth Epidemic: Inside America's Drug Crisis" ("Meth Epidemic," 2005).
- "Potent Mexican Meth Floods in as States Curb Domestic Variety," *New York Times*, January 23, 2006 (Zernike, 2006).

- "Methamphetamine Use Increasing: Public Health Officials Voice Concerns about Infants Born to Addicted Mothers," *American Medical News,* July 26, 2004 (Elliot, 2004).
- "Children of Ice." A series of articles about methamphetamine addiction in Hawaii noted, "The ice epidemic has touched tens of thousands of lives in Hawaii. It has an impact on our families, our children, our schools, our crime rate, our prisons, our businesses. Our community has never faced a problem quite like this, and we are still searching for the right responses," *Honolulu Advertiser,* September 14, 2003 ("Indicators," 2003).
- "Counties Say Meth Is Top Drug Threat." This article noted that meth is a bigger problem than cocaine, marijuana, or heroin for most communities, according to a survey of law enforcement agencies in 500 counties in 45 states by the National Association of Counties (*USA Today*, July 4, 2005).

Documentaries and films have been produced on meth use and production. For example, a journalistic documentary titled *Crank—Made in America* (2003) shows meth use and the interpersonal relationships among users. The PBS documentary *Frontline* (2006) produced a special titled "The Meth Epidemic."

The music industry has also paid attention to meth. The industry has produced songs about the risks and experiences of meth use. The following songs all refer to meth's use: Eminem, "These Drugs"; Third-Eye Blind, "Semi-Charmed Kind of Life"; Bush, "The People That We Love: Speed Kills"; Eminem, "Purple Pills"; and Green Day, "Brain Stew." The song "Brain Stew" provides typical lyrics that describe meth use.

With all of the attention meth has received in the media in recent years, in an open letter, Dr. David Lewis and Dr. Donald Millar (July 27, 2005) warned that caution should be exercised in drawing unproven conclusions about what meth use means to the unborn and newborns. They and others criticize recent media references to "meth babies" or "ice babies" as being unfounded and damaging. Such labels are detrimental because as they grow up, such children face lower expectations and harmful stereotypes about their abilities. They cite recent media coverage, such as *CBS News,* "Generation of Meth Babies" (April 28, 2005) at CBSNews.com as an example of the misuse of the label "meth baby" in the mass media. They also contend that there is no such thing as a "meth-addicted baby," because babies do not act compulsively in spite of adverse consequences as addicts do.

Lewis and Millar (2005) and other substance abuse experts are concerned that stating that meth use by pregnant women results in severe health consequences for infants has not been established by medical research and may

actually do more harm than good. As was the case for crack babies in the 1980s and 1990s, they argue that there has not been enough scientific research to warrant any medical or addictive conclusions about meth use and infants. With at least two decades of research on the effects of the use of the stimulant cocaine on fetuses and newborns, they observe that the feared epidemic of crack babies never materialized, and the same may be true for meth. Although they do not specifically refer to there being a panic, they do stress that the media has consistently overexaggerated meth at a social cost to infants and new mothers.

WHO USES METH? IS THERE A PROFILE OF METH USERS?

What populations are more likely to use meth? Summarizing the literature, Wermuth (2000, p. 423) wrote, "Low-income and unemployed young white men continue to be the group most likely to use methamphetamine, but by the mid-1990s the drug had increased in popularity in more diverse populations and regions." Numerous studies have found that meth use is predominantly a white phenomenon (Anglin, Burke, Perrochet, Stamper, & Dawud-Noursi, 2000; Yacoubian & Peters, 2004). According to the Substance Abuse and Mental Health Services Administration's (SAMHSA; 2005b) *DASIS Report*, January 7, 2005, treatment admissions for methamphetamine or amphetamine abuse in 1992 were 55 percent male, and that percentage remained stable through 2002. The mean age for treatment admissions increased to 29 years in 2002. In 1992, 62 percent of treatment admissions were white. Whites increased to 66 percent of the admissions in 2002. Of those being admitted for treatment, 25 percent indicated that they had part-time or full-time jobs. A SAMHSA (2005a) report estimated a prevalence of about 0.7 percent for whites.

However, meth users come from a wide variety of socioeconomic profiles. For example, one Sacramento, California, study found that users came from all walks of life, and some users started in their teens (Gibson et al., 2002). The penetration of meth into other demographic circles is occurring. Anglin, Kalechstein, Maglione, Annon, and Fiorentine (1997) reported that other groups such as Latinos, gay-bisexual males, older adult arrestees, and adolescents are increasingly involved. Joe (1996) found and reported on meth abuse among Asian-Pacific women in Honolulu, San Francisco, and San Diego. There are numerous anecdotal reports on the broad spectrum of users, such as those describing high-achieving students who use the drug to perform better in school and college, and others, such as athletes, cheerleaders, beauty pageant participants, and models, who use it to improve performance (Rawson et al., 2002).

While many are at risk of meth addiction, some populations identified in the literature are of special concern:

- Women who have emotional relationships with males who are addicted to meth. The males encourage and often demand that the females in their lives also use meth to "bond" with them. If the male is using, it is likely that the female is also using. Anecdotally, a common pattern is for an older male to introduce the drug and encourage its use of meth by a younger female. Another group to use it is stay-at-home adults, usually females, who begin using meth for its short-term benefits such as its energy boost, to treat depression, or for its related weight loss (Rawson, 2005).
- Workaholics or low-income adults who use it to stay awake and perform in multiple jobs. Working low-income individuals find meth attractive because they must work several jobs or long hours to support themselves or their families. They find that higher energy and alertness (ability to stay awake for prolonged periods) help them cope with the demands of multiple jobs. Probably the best example of this are over-the-road truckers, some of whom use meth (or amphetamine), allowing them to keep driving long distances.
- Gay men who use meth to enhance their sexual experiences and stimulate sexual behavior. According to Freese et al. (2000), urban gay males living along the West Coast of California represent a large segment of the meth-using population. The user's sense of euphoria from using the drug reduces normal inhibitions about sex and multiple partners. Meth use can increase the libido, but long-term use may result in sexual dysfunction. There are reports of gay men using meth to prolong and enhance sexual encounters with several partners without appropriate protection, thus increasing the potential for transmission of HIV among this population. Some suggest that associating the use of meth among this population is important to addressing the spread of HIV (Rawson et al., 2002; Specter, 2005). Aware of the association between meth use and the spread of HIV, some activists have made posters that proclaim, "Buy Crystal. Get HIV for Free," or "Crystal Free and Sexy" (Join Together, 2005). Because of this tie to sexual activity, the gay community poses unique challenges for meth treatment. The problem has grown to such a level in New York City that the gay community has developed Meth Anonymous support groups (New York State Office of Alcoholism and Substance Abuse Services, 2004). Some authorities are also concerned about the spreading of HIV through the sharing of needles, which is common among injectors (Gibson et al., 2002).
- Rural residents who are interested in manufacturing meth for profit and personal use. Meth is particularly attractive in rural areas where farming and the economy are in decline (Wermuth, 2000). North Carolina profiled the typical user as, "young, white, small-town residents with limited education and a blue-collar career" (North Carolina Division of Social Services and

the Family and Children's Resource Program, 2005). One study comparing rural with urban illegal substance use in Nebraska found that while meth use was present in urban Omaha, in rural areas meth use was more common (Herz, 2000). One report found that rural and small-town youth were more likely than their urban counterparts to become substance abusers (National Center on Addiction and Substance Abuse at Columbia University, 2000). The same report found that rural eighth graders were 104 percent more likely than urban eighth graders to use amphetamines. This same group was 59 percent more likely to use methamphetamine than urban eighth graders.

- Heterosexuals drawn to sexual experimentation and sexual experiences who use the drug to alter their sexual pleasure (Wermuth, 2000). Meth is known to lower sexual inhibitions, making it attractive to those interested in sexual activity and seduction.
- Youths and young adults involved with the rave subculture who mix meth with other substances for entertainment and excitement.
- Native Americans, including those living on reservations. Native American meth use is spreading across the reservations. In a special *Newsweek* report, journalist Andrew Murr (2004) found that it was becoming a "scourge" affecting Native Americans living on reservations. SAMHSA (2005a) estimates the prevalence of meth use among American Indians and Alaska Natives at about 1.7 percent. Freese et al. (2000) summarize the literature by noting that new approaches need to be designed to address the meth problem in Native American communities. They noted that Native American communities in Wyoming and Montana are moving forward in addressing the problem at the community level.

Evidence indicates that meth use has the potential to spread to other populations. Rawson et al. (2002) wrote,

> Meth use is expanding from a purely Caucasian, English-speaking clientele to Hispanic and Asian populations. Although the use of methamphetamine appears to be minimal among African Americans, increases among Hispanics and Asians suggest expansion of the methamphetamine problem to new markets. (p. 8)

Whether meth spreads to new ethnic and cultural groups remains to be seen. Some research has found that strong ethnic identification and segregation may offset some drug risks (Zickler, 2005a). For example, meth use has not made serious inroads into the African American population. The Drug Abuse Warning Network (DAWN) is a public health surveillance system that monitors drug-related visits to hospital emergency departments (EDs) and drug-related deaths investigated by medical examiners and coroners. DAWN data reflect less popularity of meth use among African Americans than among

whites and Hispanics (Wermuth, 2000). This pattern may or may not hold steady over time.

What is apparent is that users come from a wide variety of backgrounds. As they continue to use and move on to heavy use, their outward appearances, lifestyle disruptions, thought patterns, and behaviors become more similar. They begin the race at different points and end at the same place, becoming more similar with each dose.

GENDER DIFFERENCES AND METH USE

Research has been conducted on gender and patterns of meth acquisition, initiation, use, motivation, problems, treatment, and manufacture. Before the 1980s, female use of any substance was viewed as dependent on relationships with men. Females were either viewed as "good" citizens with addiction problems, or "bad" ones whose drug use mirrored their roles as deviants (Morgan & Joe, 1996). Since the 1980s, increasing amounts of research have found that women have reasons for drug use outside of their relationships with men.

Today, researchers are exploring gender differences among male and female meth users. For example, Brecht, O'Brien, Mayrhauser, and Anglin (2004) studied 350 people engaged in substance abuse treatment. They found gender differences in selected aspects of meth involvement. Some of the differences found were:

- Females were more likely than males to be introduced to meth and gain access through spouses or boyfriends than were males from girlfriends or spouses. Males were more likely to be introduced and gain access through friends and co-workers.
- Males were more likely to be injectors than were females.
- Males more likely to sell it than were females.
- Females were more likely to report skin and high-blood-pressure problems than were males.
- Females had longer first-time treatment episodes than males did.
- Male polydrug use included a wider variety of drugs than females reported.

Hser et al. (2005) also studied gender differences in meth users in a sample of treatment programs in California. In summary, Hser et al. (2005, p. 84) concluded, "Women in our sample, most of whom were of childbearing age or had children, demonstrated more severe problems than did men." They added, "Many were unemployed, relied on public assistance, and suffered from severe psychiatric problems." Hser et al. (2005) specifically found:

- Women reported meth use at an average earlier age (19.2 years) than males (20.6 years).
- Both genders used meth regularly for about the same number of years (8.7–8.8 years).
- Women reported significantly more numbers of prior treatments for drug use.
- The majority of women in the study had children under 18, but most did not live with their children within the last 30 days. Women were significantly more likely to be living with their children than were men. Though the findings were not statistically significant, more women than men had someone else taking care of their children through court order and had their parental rights terminated.

Research findings support gender differences regarding treatment needs. Hser et al. (2005) summarized the literature and concluded that females in treatment tend to have more psychological symptoms, lower self-esteem, heightened anxiety and depression, and higher rates of childhood sexual abuse than males. Women also had greater issues regarding employment, children, job skills, and incomes than males. Women have additional concerns if they are pregnant because of preliminary research suggesting negative effects of meth use on the fetus. Females are more concerned with child-rearing responsibilities, work, and the other seemingly overwhelming demands on their lives than males. This can affect their willingness to stop using a drug that they believe helps them get it all done. In contrast, males were more likely to be involved in criminal activities and be under criminal system supervision.

MEASURES OF THE EXTENT OF METH USE IN THE UNITED STATES

It is possible to get a sense of the extent of meth use and manufacture in the United States by looking at a number of national statistical indicators. While no measure captures everything, when a number of measures are viewed together, a sense for its use in the United States is possible. The following are some national indicators that give a sense of the extent of meth use, manufacture, and treatment.

A number of national efforts are made to track the extent of drug use, treatment, production, arrests, and other data. These data are useful in identifying trends. One of the most widely known is the Monitoring the Future Survey (MTF). This study is an annual national survey that assesses the beliefs, attitudes, and behaviors of high school students in grades 8, 10, and 12 as well as young adults. The study does not include school dropouts, who could represent a high-risk group for meth use. The MTF study surveys about 50,000 subjects.

Since 1999, meth has been included in the survey. Table 19.1 shows that meth use is not common among high school–aged youths. The numbers are low, which is encouraging. However, before becoming too optimistic, one must realize that the usage reported is at least three years old and does not capture recent regional expansion of the drug. In addition, note the percentage of 12th graders indicating a decline in lifetime use from 1999 to 2005 from 8.2 percent to 4.5 percent, respectively.

Emergency Department Drug Mentions—Drug Abuse Warning Network (DAWN)

Drug-related emergency department (ED) mentions are provided by SAMHSA through its Drug Abuse Warning Network (DAWN), which provides national information on morbidity and mortality related to substance abuse, including meth, collected from short-stay medical and medical examiner offices across the United States. The DAWN study is an annual survey of ED episodes from metropolitan areas. The DAWN data are useful in understanding meth and other substance abuse patterns. The DAWN data show

Table 19.1
Percentage of Methamphetamine Use by Secondary School Students by Grade, 1999–2005

Grade	1999	2000	2001	2002	2003	2004	2005
				Lifetime			
8	4.5	4.2	4.4	3.5	3.9	2.5	3.1
10	7.3	6.9	6.4	6.1	5.2	5.3	4.1
12	8.2	7.9	6.9	6.7	6.2	6.2	4.5
				Annual			
8	3.2	2.5	2.8	2.2	2.5	1.5	1.8
10	4.6	4.0	3.7	6.1	3.3	3.0	2.9
12	4.7	4.3	3.9	6.7	3.2	3.4	2.5
				Past 30 days			
8	1.1	0.8	1.3	1.1	1.2	0.6	0.7
10	1.8	2.0	1.5	1.8	1.4	1.3	1.1
12	1.7	1.9	1.5	1.7	1.7	1.4	0.9

Source: National Institute on Drug Abuse (2005).

Table 19.2
Number of Emergency Department Visits for Methamphetamine Mentions (Drug Abuse Warning Network), 1995–2003

1995	1996	1997	1998	1999	2000	2001	2002	2003
15,933	11,002	17,154	11,486	10,447	13,505	14,923	17,696	25,039

Source: Substance Abuse and Mental Health Services Administration (2004).

that meth-related deaths and ED episodes have increased in many urban areas over recent years. For example, in 2003, according to the DAWN data, meth was involved in 25,039 emergency room visits (SAMHSA, 2004). Table 19.2 shows DAWN data from 1995 to 2003. The data show that ED meth mentions have increased from 1995 to 2003 by 57.1 percent.

Treatment Episode Data Set (TEDS)

The Treatment Episode Data Set (TEDS), once labeled the Client Data System, collects data on clients admitted to substance abuse treatment programs. TEDS is an annual compilation of data on the demographic characteristics and substance abuse problems of those admitted for substance abuse treatment. In 1992–1993, with 42 states and the District of Columbia reporting, admissions due to meth abuse increased in 23 out of 29 reporting states for a net increase of 43 percent (Wermuth, 2000). Meth was the primary substance of abuse in over 116,595 substance abuse treatment admissions in 2003 reported to TEDS (SAMHSA, 2005c). In comparison, the same TEDS data show that the number of meth admissions for 1993 was 20,776, which indicates that admissions increased by 461 percent over this 10-year span. SAMHSA (2005c) reported that from 1992 to 2002, the amphetamine and meth treatment admission rate in the United States increased from 10 to 52 per 100,000. In California, a state at the forefront of meth use and manufacture, publicly funded meth treatment increased 226 percent from 1992 to 1998 (Brecht, 2001). The state of Washington treatment admissions increased more than 1,000 percent from 1992 to the first half of 1998 (Mills, 1999). Nationally, this trend continued, as reported admissions to publicly funded treatment programs for meth abuse grew from 12,122 in 1992 to 55,582 in 2002, representing more than a fourfold increase (Hser et al., 2005). This may be reflective of increased interest by meth addicts in getting treatment and/or of more court-ordered treatment.

SAMHSA's March 15, 2006, *DASIS Report* summarized that meth and amphetamine were identified in more than 136,000 cases as the primary sub-

stance of abuse in treatment admissions. This represented about 7 percent of all treatment admissions. The same report noted that between 1993 and 2003, the meth and/or amphetamine admission rate increased from 13 to 56 admissions per 100,000 population aged 12 or older. This represents a 330 percent increase over the period. The report also noted that 18 states had meth and/or amphetamine treatment rates that were higher than the national average of 56 per 100,000 population 12 or older. In addition, the report found that "the proportion of primary methamphetamine/amphetamine admissions referred to treatment by the criminal justice system increased from 36 percent in 1993 to 51 percent in 2003." This later conclusion indicates that methamphetamine and amphetamine users are being ordered into treatment by the courts more frequently.

Reported Lifetime Use

Another way to measure substance use is to survey a sample of households. The National Household Survey on Drug Abuse (NHSDA) is an annual national survey of drug use by household. During the year 2000, the DEA found that 4 percent of the U.S. population reported trying meth at some time over their lifetime (SAMHSA, 2001). According to its 2004 survey, about 12 million people aged 12 and older reported they had used meth at least once in their lifetime. These people represent about 5.3 percent of the total population. The percentages of reported use from the 2004 household survey broken down by age are shown in Table 19.3.

Quest Diagnostics Data

Quest Diagnostics, a private substance use testing firm, develops an index of use every six months as part of its substance testing operations. Quest

Table 19.3
Percentage of Lifetime Methamphetamine Use among U.S. Population by Age Group, 2004

Age group	Lifetime	Past year	Past month
12 and over	4.9	0.6	0.2
12–17	1.2	0.6	0.2
18–25	5.2	1.6	0.6
26 and older	5.3	0.4	0.2

Source: Substance Abuse and Mental Health Services Administration (2005a)

conducted 7.1 million drug tests in 2003 and has been indexing the results of drug tests since 1988. Quest Diagnostics makes its index of positive results available on its Web site (http://www.questdiagnostics.com). The company reported major increases in positive test results for amphetamines and meth. It recently reported (Quest Diagnostics, 2004),

> Drug tests suggest that greater use of Meth among a large group of general U.S. workforce employees during 2003 may have caused the increase in amphetamines use overall. For this group of workers, the incidence of positive drug tests attributed to methamphetamine increased by more than 68% in 2003 from 2002, reaching 0.32% of all drug tests. During 2002, methamphetamine was 0.19%.)

For the period between January and June 2005, Quest Diagnostics data indicate that of the over 3 million general workforce members who took drug tests between January and June 2005, 0.50 percent tested positive for amphetamine use, which includes meth (Quest Diagnostics, 2005). This percentage was slightly down from the 2004 tests.

Meth Arrests and Seizures

The Drug Enforcement Administration (DEA) tracks the number of drug seizures made each year by reporting law enforcement agencies. The number of seizures only reflects the general amount of drug trafficking known to law enforcement authorities. Law enforcement agencies vary in their effectiveness and efficiency in seizing illegal drugs. Thus these data tend to undercount the amount of true activity. Table 19.4 shows DEA drug seizures for the United States from 1990 to September 2005. For this time span, the number of meth kilograms seized increased by 500 percent. In comparison, seizures for the illegal stimulant cocaine increased by 48.9 percent.

Arrestee Drug Abuse Monitoring Program (ADAM)

The Arrestee Drug Abuse Monitoring Program (ADAM), established in 1987 but no longer in existence, collected urine samples from arrestees in 42 jurisdictions across the United States. It was one of the few national efforts to collect verifiable data on actual drug use by urine specimen. ADAM data for adults are available from 1987 to 2003.

ADAM data from 1991 to 2001 indicate that western states had the highest concentration of positive tests for meth among adult arrestees (Yacoubian & Peters, 2004). ADAM 2002 data reveal that the median percentage of adult male arrestees who tested positive for meth use in 2002 was 5.3 percent. The highest proportions of arrestees testing positive for meth were in the West

Table 19.4
Drug Enforcement Administration Drug Seizures, 1990–September 2005

Calendar year	Cocaine, kg	Heroin, kg	Marijuana, kg	Methamphetamine, kg	Hallucinogens, dosage units
Sept. 2005	84,959	479	222,943	1,817.7	8,425,499
2004	117,622	672	264,714	1,647.5	2,483,663
2003	73,720	789	254,188	1,676.9	2,879,528
2002	61,594	705	195,644	1352.8	11,661,811
2001	59,426	752	271,785	1634.1	13,756,939
2000	58,627	546	331,964	1771.4	29,306,453
1999	36,167	351	337,832	1,488.8	1,716,954
1998	34,448	371	262,176	1,202.7	1,075,257
1997	28,630	399	215,348	1,143.2	1,100,912
1996	44,765	320	190,453	751.0	1,719,096
1995	45,326	876	219,830	875.1	2,768,165
1994	75,051	491	157,182	767.6	1,366,817
1993	55,158	616	143,030	559.8	2,710,063
1992	69,323	722	201,507	352.7	1,305,177
1991	67,016	1,170	98,601	289.6	1,295,874
1990	57,031	532	127,694	274.5	2,826,966

Source: Drug Enforcement Administration (2006).

Coast, Southwest, and West Central regions. Honolulu led all ADAM reporting cities for the percentage of male arrestees (44.8%) in 2002 who tested positive for methamphetamine. Hawaii has a long history of crystal meth abuse.

Regionalization of Meth

Meth use and manufacture is not evenly distributed across the United States. For years, meth use and production was concentrated in Hawaii, California, and the western United States. Across the border, Mexico is a country noted for meth production, with targeted sales in the United States. Motorcycle gangs often operated clandestine labs in California and western states and for years held the advantage in production. Meth use in other regions, while occurring, remained relatively rare. This regionalization of meth is rapidly changing as the drug is creeping across the United States. For example, midwestern states, such as Missouri, are now major areas of meth production.

In recent years, production has been spreading to other regions of the country. Some states have passed laws that have slowed this progression down but not halted it entirely. There are several reasons why this is occurring. The popularity of the drug, tightening of the borders due to the war on drugs, and the Internet are among the possibilities. The Internet has probably played a critical role in the spread of the manufacture of the drug because of the easily accessible Web site recipes for its manufacture. Virtually anyone who has access to common household chemicals can find a recipe for cooking meth within a few minutes of searching the Internet. The ability to produce small batches of the drug at home has made users throughout the country less dependent on western or Mexican sources of the drug. The trend continues that most users buy the drug rather than manufacture it for personal use.

Figure 19.1 displays three maps that show the percentages of agencies in each state for the years 2003–2005 reporting meth as their greatest threat, broken down by year. States with 0–24.9 percent of agencies reporting meth as the greatest threat are shown in black; 25–49.9 percent in middle gray; 50–74.9 percent in light gray; and 75–100 percent in dark gray. According to the DEA (2006), in 2003, black states were Connecticut, Delaware, Florida, Illinois, Louisiana, Maine, Maryland, Massachusetts, Michigan, New Hampshire, New Jersey, New York, North and South Carolina, Ohio, Pennsylvania, Rhode Island, Vermont, Virginia, West Virginia, and Wisconsin. Middle gray states were Alabama, Alaska, Georgia, Indiana, Kentucky, Mississippi, and Texas. Light gray states were Arkansas, Colorado, Missouri, New Mexico, and Tennessee. The dark gray states of Arizona, California, Hawaii, Idaho, Iowa, Kansas, Minnesota,

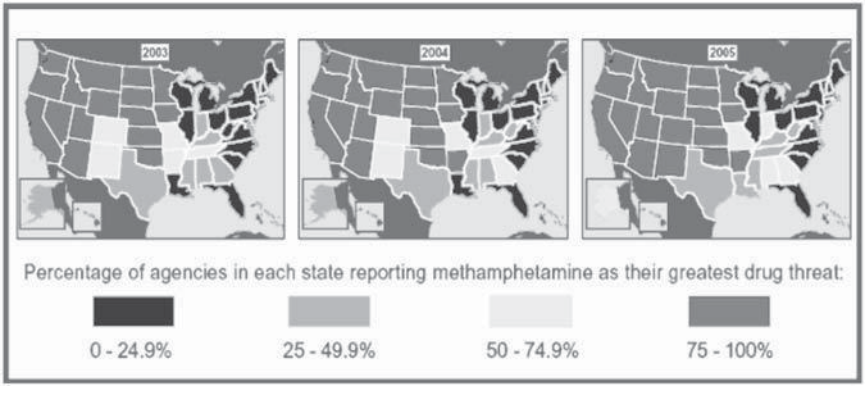

FIGURE 19.1 Methamphetamine threat progression, 2003–2005 (Drug Enforcement Administration, 2006).

Montana, Nebraska, Nevada, North and South Dakota, Oklahoma, Oregon, Utah, Washington, and Wyoming reported meth as their greatest threat. In 2004, West Virginia changed from black to middle gray; Georgia changed from middle gray to light gray; and Arkansas changed from light gray to dark gray. In 2005, Tennessee changed from light gray to middle gray; Louisiana changed from black to middle gray; Alabama, Alaska, and Indiana changed from middle gray to light gray; and New Mexico and Colorado changed from light gray to dark gray. The maps show that meth is marching from the West to the East and South. It has not made serious inroads in the Northeast.

Clandestine Lab Identification

Karen P. Tandy (2004), administrator for the DEA, testified before a congressional hearing that meth was "most frequently produced clandestinely in the United States." These labs can be anywhere. Missouri often shows up as a state with numerous meth labs. It is ideal because of its central location and rural landscape, which helps cooks hide their labs from officials. Leinwand (2003) reported, "In fiscal 2002, local police and U.S. agents shut down 1,039 labs in Missouri, 321 in Illinois, 89 in Florida, and 85 in Georgia." She added for comparison, that "seven years earlier, officials had reported finding 29 labs in Missouri and two each in Illinois, Florida, and Georgia." Other states have reported similar experiences:

- California reported 1,262 meth lab busts from September 30, 2002, to September 30, 2003, more than double the number from seven years earlier (Leinwand, 2003).
- The number of DEA meth lab seizures rose from 8,000 in 1999 to 10,000 in 2003.
- The DEA reported breaking up more than 16,203 clandestine labs in 2002 and 2003 (Ray, 2004).
- The Hazardous Substances Emergency Events Surveillance (HSEES) system reported that meth-related health events increased from 184 in June 2000 to 320 in June 2004. The system reported a total of 1,791 events in 16 states that use the reporting system in 2004. These events resulted in 960 injuries (Edwards, 2005).

The number of lab seizures may undercount the true extent of the lab problem. As states have passed restrictions on the purchase of precursors, illegal meth labs have become fractured. *Fractured* means the production process and acquisition of precursors are spread out over larger geographical areas to avoid detection by authorities. For example, with fracturing, cooks buy precursors from different stores in different towns to avoid detection by law enforcement.

PREVENTION EFFORTS TO CONTROL THE SPREAD OF METH

Prevention is an important component of any community response to meth use and manufacture. Many of the Web sites such as http://www.methresources.gov, http://www.mappsd.org, http://www.stopmeth.org, http://www.preventionpathways.samhsa.gov, and http://www.modelprograms.samhsa.gov provide prevention brochures, ads, video clips, fact sheets, literature, strategies, links, and materials. Some Web sites such as http://www.justthinktwice.com and http://www.montanameth.org provide prevention materials geared toward youth. The Montana Meth Project (http://www.montanameth.org) has films, posters, radio clips, and other excellent prevention resources. SAMHSA can provide prevention documents and materials to any community wanting to prevent meth use. States such as California, Hawaii, Illinois, Indiana, Iowa, Kansas, Michigan, Missouri, Montana, New York, Oregon, South Dakota, Tennessee, and others have launched meth prevention efforts. These are summarized in chapter VII of the SAMHSA-funded *Methamphetamine: A Resource Kit* (Center for Substance Abuse Prevention/National Prevention Network, 2006).

In addition, the Methamphetamine Interagency Task Force (2000a, 2000b) made specific prevention recommendations that are worth noting:

- Address meth issues through broad-based drug prevention and education efforts that target all forms of drug use and that are based on research and established prevention principles.
- Develop science-based prevention program planning and intervention guidelines in communities where meth is already a problem.
- Involve the entire community in prevention efforts, including educators, youths, parents, vendors of materials used in meth manufacture, law enforcement officials, business leaders, members of the faith community, social service providers, and representatives of government agencies and organizations.
- Identify the changing population characteristics of users, their motivations, risk factors, and demographics.
- Involve parents and other adults in prevention and education programs for youths, particularly in the areas of monitoring "latchkey" children, enhancing parent-child communication skills, and providing consistent family/home rules for youth behavior and leisure time activities.
- Ensure that media campaigns proceed with caution, focusing on raising awareness of meth and using messages designed to minimize unintended effects such as arousing curiosity about meth.
- Develop or augment programs aimed at educating those communities in which meth is an emerging or chronic problem.

In an effort to prevent manufacturing of meth, lawmakers in several states and Congress have been active in taking precursors and drugs such as pseudoephedrine off store shelves and restricting access to them. Law enforcement agencies in some jurisdictions have worked with local merchants to monitor suspicious or high-quantity sales of over-the-counter medicines (those containing pseudoephedrine) and chemicals (including fertilizer) that might be used for the purpose of manufacturing meth.

The Comprehensive Methamphetamine Control Act of 1996 identified selected chemicals, such as iodine, as List II chemicals. Using iodine as an example, the DEA requires that detailed records be maintained for sales of iodine crystals that exceed the threshold of 0.4 kilograms (about 14 ounces) to anyone or any entity. The act also requires that distributors and retailers report any suspicious activities to the DEA. Section 21 U.S.C. 841 § (c) (1)–(3) prohibits any person from knowingly possessing or distributing a listed chemical with the intent, knowledge, or belief that the chemical will be used to manufacture a controlled substance such as meth. Section 21 U.S.C. 843 § (a) (7) prohibits the individual from knowingly or intentionally manufacturing, distributing, exporting, or importing any equipment, chemical, or material that may be used to manufacture a controlled substance or listed chemical. Penalties for conviction for each violation are 10 years of imprisonment, a $250,000 fine, or both, and they double with a second or subsequent convictions. States have passed laws to stiffen the penalties. For example, in North Carolina, individuals possessing ingredients in quantities sufficient to make the drug can be punished with up to five years in prison if prosecutors can prove intent to manufacture meth (Eisley, 2004).

Some states have passed laws that hold store owners accountable for large-quantity sales of precursors (ingredients) used to make meth. States that have passed legislation that restricts the sales of precursors are Arkansas, Georgia, Iowa, Illinois, Kentucky, Mississippi, Oklahoma, Oregon, South Dakota, Tennessee, West Virginia, and Wyoming. There are a number of states that have passed laws that restrict the sale of precursors used to manufacture meth:

- Iowa passed legislation that restricts access to over-the-counter cold medicines that can be used to cook meth.
- California, a state with some of the earliest laws restricting the sale of precursors needed to produce meth, believes that it has had positive effects in controlling the spread of meth. After it enacted laws limiting the sale of key chemicals, such as ephedrine and pseudoephedrine, lab busts were cut in half, from 2,090 in 1999 to 1,130 in 2002 (Lacour & Gregory, 2004). California also has strict regulations regarding the sale of iodine crystals. California Code (11107.1) requires all sales to be recorded, limits the quantity, and has other limits.

- Oklahoma passed legislation that requires that pseudoephedrine be kept behind the counter.
- In April 2004, the U.S. Food and Drug Administration (FDA) banned drugs containing the stimulant ephedrine. In 2005, a Utah federal court limited the scope of a FDA rule banning the sale of all ephedrine-alkaloid dietary supplements. The judge ordered the FDA to develop rules for the legal distribution of the drug.

Short of passing restrictions, other efforts have been made to control the distribution of precursors. For example, Illinois issues bulletins to farmers and fertilizer sellers encouraging them to protect their supplies of anhydrous ammonia. In addition, as part of this antimeth campaign, some farmers and suppliers have secured their anhydrous ammonia storage tanks (known in some meth circles as "white buffalos") with fences, motion detectors, and locks.

CLOSING OBSERVATIONS

Meth accounts for a small percentage of the total number of people affected by drug and alcohol problems. However, almost all of the data in this chapter reveal that meth use, manufacturing, and distribution are increasing throughout much of the nation. Some have likened the spread of meth to an epidemic in some regions of the country (Anglin et al., 1997). It is clear that meth has impacted the West, Southwest, and Midwest and is beginning to expand into the Northeast. How extensive this expansion will be is unknown. The other question is whether meth use will grow in prevalence in minority populations. To date Latino, Hispanic, and African American populations have not embraced meth to the extent that Anglos have. If this changes, the negative effects could be substantial. Whether the upward spiral of meth use and manufacture continues remains to be seen. How communities, service agencies, law enforcement, families, elected officials, treatment providers, the courts, and businesses respond to the challenge will influence how, and even whether, this trend can be reversed.

NOTE

1. From *The Methamphetamine Crisis*, Herbert C. Covey; Chapter 2, pp 23–40, "Prevalence of Use and Manufacture of Methamphetamine in the United States," by Herbert C. Covey. Copyright © 2006 by Herbert C. Covey. Reproduced with permission of Greenwood Publishing Group, Inc. Westport, CT.

REFERENCES

Anglin, M. D., Burke, C., Perrochet, B., Stamper, E., & Dawud-Noursi, S. (2000). History of the methamphetamine problem. *Journal of Psychoactive Drugs, 32,* 137–141.

Anglin, M. D., Kalechstein, A., Maglione, M., Annon, J., & Fiorentine, R. (1997). *Epidemiology and treatment of methamphetamine abuse in California: A regional report.* Unpublished manuscript, University of California, Los Angeles, Drug Abuse Research Center.

Bonné, J. (2001a). *Meth's deadly buzz.* Retrieved from http://www.msnbc.msn.com/id/3071772/

Bonné, J. (2001b). *Meth's deadly buzz: Lab-busting in the Northwest.* Retrieved from http://msnbc.msn.com/id/3071775

Brecht, M.-L. (2001). *Update on methamphetamine use trends in California.* Unpublished manuscript, University of California, Los Angeles, Integrated Substance Abuse Program.

Brecht, M.-L., O'Brien, A., Mayrhauser, C., & Anglin, M. D. (2004). Methamphetamine use behaviors and gender differences. *Addictive Behaviors, 29,* 89–106.

Center for Substance Abuse Prevention/National Prevention Network. (2006). *Methamphetamine: A resource kit.* Rockville, MD: Substance Abuse and Mental Health Services Administration.

Center for Substance Abuse Research. (2004). *Workplace drug tests patterns changing.* Retrieved from University of Maryland Web site: http://www.cesar.umd.edu/cesar/cesarfax/vol13/13–41.pdf

Counties say meth is top drug threat. (2005, July 4). *USA Today*.

Drug Enforcement Administration. (2006). *National Drug Assessment 2006.* Washington, DC: Author.

Edwards, E. M. (2005, April 18). *CDC: Rising danger of meth labs.* Retrieved from http://www.jointogether.org/sa/news/summaries

Eisley, M. (2004, December 1). New meth laws worsen penalties. *Raleigh News and Observer*.

Elliot, V. S. (2004, July 26). Methamphetamine use increasing: Public health officials voice concerns about infants born to addicted mothers. *American Medical News*.

Freese, T. E., Obert, J., Dickow, A., Cohen, J., & Lord, R. H. (2000). Methamphetamine abuse: Issues for special populations. *Journal of Psychoactive Drugs, 32,* 177–182.

Gibson, D. R., Leamon, M. H., & Flynn, N. (2002). Epidemiology and public health consequences of methamphetamine use in California's central valley. *Journal of Psychoactive Drugs, 34,* 313–319.

Herdy, A. (2002, October 20). Methamphetamine's young victims: Homes doubling as drug labs pose serious dangers to kids. *Denver Post*.

Herrell, J. M., Taylor, J. A., Gallagher, C., & Dawud-Noursi, S. (2000). A multisite study of the effectiveness of methamphetamine treatment: An initiative of the

Center for Substance Abuse Treatment. *Journal of Psychoactive Drugs, 32,* 143–147.

Herz, D. C. (2000). *Drugs in the heartland: Methamphetamine use in rural Nebraska.* Washington, DC: National Institute of Justice.

Hser, Y.-I., Evans, E., & Huang, Y.-C. (2005). Treatment outcomes among women and men methamphetamine abusers in California. *Journal of Substance Abuse Treatment, 28,* 77–85.

Huber, A., Ling, W., Shoptaw, S., Gulati, V., Brethen, P., & Rawson, R. A. (1997). Integrating treatments for methamphetamine abuse: A psychosocial perspective. *Journal of Addictive Diseases, 16,* 41–50.

Indicators of a worsening problem in Hawai'i. (2003, September 14). *Honolulu Advertiser.* Retrieved from http://www.the.honoluluadvertiser.com/dailypix/2003/sep/14/icebox2.gif

Joe, K. (1996). The lives and times of Asian-Pacific American women drug users: An ethnographic study of their methamphetamine use. *Journal of Drug Issues, 26*(1), 199–218.

Join Together. (2005, April 5). *Gay community fights growing meth problem.* Retrieved from http://www.jointogether.org

Kraman, P. (2004). *Drug abuse in America—Rural meth.* Lexington, KY: Council of State Governments.

Lacour, G., & Gregory, A. (2004, March 21). Meth is invading Carolinas: Frightening, devastating, spreading. *Charlotte Observer.* Retrieved from http://www.charlotte.com

Leinwand, D. (2003, July 29). "Meth" moves east. *USA Today.*

Leinwand, D. (2005, July 4). Counties say meth is top drug threat. *USA Today.*

Lewis, D. C., & Millar, D. G. (2005, July 27). *Open letter: To whom it may concern.*

The meth epidemic: Inside America's drug crisis. (2005, August 8). *Newsweek.*

Mills, K. (1999, December 13). Meth of old has morphed into epidemic proportions. *Seattle Post-Intelligencer.*

Morgan, P., & Joe, K. A. (1996). Citizens and outlaws: The private lives and public lifestyles of women in the illicit drug economy. *Journal of Drug Issues, 26,* 125–142.

Murr, A. (2004, September 27). A new menace on the rez. *Newsweek,* p. 30.

National Association of Counties. (2005, February). *Funding of methamphetamine research, treatment, enforcement, cleanup and education* (NACO Fact Sheet). Washington, DC: Author.

National Association of Counties. (2006a). *The challenge of treating meth abuse.* Retrieved from http://www.naco.org

National Association of Counties. (2006b). *The effect of meth abuse on hospital emergency rooms.* Retrieved from http://www.naco.org

National Center on Addiction and Substance Abuse at Columbia University. (2000). *No place to hide: Substance abuse in mid-size cities and rural America.* New York: Author.

National Drug Intelligence Center. (2002, August). *Crystal methamphetamine* (Information Bulletin No. 2002-L0424-005). Johnstown, PA: U.S. Department of Justice.

National Drug Intelligence Center. (2003). *National drug threat assessment 2003*. Johnstown, PA: U.S. Department of Justice.

National Drug Intelligence Center. (2004). *National drug threat assessment 2004*. Johnstown, PA: U.S. Department of Justice.

National Institute on Drug Abuse. (2005). *Monitoring the future: National results of adolescent drug use*. Retrieved from http://monitoringthefuture.org/data/

New York State Office of Alcoholism and Substance Abuse Services. (2004, March 29). *Amphetamines and methamphetamines*. Retrieved from http://www.oasas.state.ny.us/AdMed/drugs/fyimeth.htm

North Carolina Division of Social Services and the Family and Children's Resource Program. (2005). Methamphetamine: What child welfare workers should know. *Children's Services Practice Notes, 10*(2). Retrieved from http://info.dhhs.state.nc.us/olm/manuals/dss

Quest Diagnostics. (2004). *News from Quest Diagnostics*. Teterboro, NJ: Author.

Quest Diagnostics. (2005). *Amphetamines drug-test positives are up among safety-sensitive workers in the first half of 2005, according to Quest Diagnostics' drug testing index*. Teterboro, NJ: Author.

Rawson, R. A. (2005). *Methamphetamine addiction: Cause for concern—hope for the future*. Retrieved from University of California, Los Angeles Web site: http://www.apa.org/ppo/rawson62805.ppt#257

Rawson, R. A., Anglin, M. D., & Ling, W. (2002). Will the methamphetamine problem go away? *Journal of Addictive Diseases, 21*, 5–19.

Ray, S. (2004). Methamphetamine: The "walk-away" drug. *Social Work Today, 4*, 30–33.

Specter, M. (2005, May 23). Higher risk: Crystal meth, the Internet, and dangerous choices about AIDS. *New Yorker*. Retrieved from http://www.newyorker.com/fact/content/articles/0505223fa_fact

Substance Abuse and Mental Health Services Administration. (2001). *Summary of findings, 2000 National Household Survey on Drug Abuse*. Rockville, MD: Author.

Substance Abuse and Mental Health Services Administration. (2004). *Drug Abuse Warning Network, 2003: Interim national estimates of drug-related emergency department visits*. Rockville, MD: Author.

Substance Abuse and Mental Health Services Administration. (2005a, October 2). *National Survey on Drug Use and Health 2004*. Rockville, MD: Author.

Substance Abuse and Mental Health Services Administration. (2005b, September 16). *The NSDUH Report: Methamphetamine use, abuse, and dependence: 2002, 2003, and 2004, in brief*. Rockville, MD: Author.

Substance Abuse and Mental Health Services Administration. (2005c). *Treatment Episode Data Set (TEDS) 1993–2003*. Rockville, MD: Office of Applied

Tandy, K. P. (2004, March 24). Statement before the U.S. House of Representatives, Committee on Appropriations, Subcommittee for the Departments of Commerce, Justice, State, the Judiciary and Related Agencies.

Uncle Fester. (2002). *Secrets of methamphetamine manufacture: Including recipes for MDA, ecstasy, and other psychedelic amphetamines* (6th ed.). Port Townsend, WA: Loom-panics Unlimited.

Wermuth, L. (2000). Methamphetamine use: Hazards and social influences. *Journal of Drug Education, 30,* 423–433.

Yacoubian, G. S., & Peters, R. J. (2004). Exploring the prevalence and correlates of methamphetamine use: Findings from Sacramento's ADAM program. *Journal of Drug Education, 34,* 281–294.

Zernike, K. (2006, January 23). Potent Mexican meth floods in as states curb domestic variety. *New York Times*.

Zickler, P. (2005a). Ethnic identification and cultural ties may help prevent drug use. *NIDA Notes, 14,* 1–3.

Zickler, P. (2005b). Methamphetamine, cocaine abusers have different patterns of drug use, suffer cognitive impairments. *NIDA Notes, 16,* 1–2.

Zickler, P. (2005c). Thailand conference focuses on methamphetamine research. *NIDA Notes, 16,* 1–2.

Chapter **20**

Interaction between Methamphetamine Use and HIV Infection

Mary F. Holley, MD

BACKGROUND

Methamphetamine use is associated with higher rates of all types of sexually transmitted diseases. This association is both behavioral and physiological in nature. Methamphetamine use increases sexual behavior in both animals and humans. Methamphetamine stimulates increased sexual desire and promotes prolonged sexual encounters by delaying ejaculation. It greatly increases the dopamine response to sexual stimuli in the nucleus accumbens of rats (Fiorino & Phillips, 1999). Methamphetamine is also disinhibiting, which encourages promiscuous behavior and discourages the use of condoms. Genital ulcers caused by other sexually transmitted diseases also increase the risk of HIV transmission (Dickerson, Johnston, Delea, White, & Andrews, 1996). In addition, methamphetamine is a vasoconstrictor that leads to drying of mucous membranes, both vaginal and rectal, increasing the likelihood of abrasions and the transmission of disease.

The most deadly of the sexually transmitted diseases promoted by meth use is AIDS. Both hetero and homo sexuals are at risk. The incidence of new HIV infections has risen in recent years, particularly in America's coastal cities. Homosexual men from all over the nation relocate to the coastal cities in an effort to find a community. Once there, they may find that the harmonious communities and meaningful relationships they had hoped for do not materialize. They may find themselves even lonelier than they were in the small Midwestern towns they came from. While hoping to connect and make friends, they may be reduced to chance encounters, often at a price. The price is more than just monetary. They often pay with their lives.

The interaction between the methamphetamine epidemic and the recent rise in sexually transmitted diseases, including HIV, is not an accident, and is not confined to the homosexual community. Methamphetamine use increases the transmission of HIV and also promotes its invasion into the immune system and central nervous system. Drug resistance is increased in situations where medication compliance is poor, and co-occurring infections like hepatitis C also accelerate the progression of HIV disease. The mental illnesses associated with methamphetamine are exacerbated in patients who are also HIV-positive, particularly depression and dementia.

The implications for public health in the United States alone are staggering. Our tenuous grip on the HIV epidemic is seriously threatened by the accelerating methamphetamine epidemic. As use of methamphetamine spreads across the nation, more people of all ages are exposed to so-called big-city diseases with potentially devastating consequences for our collective public health.

INCREASED TRANSMISSION OF HIV

Methamphetamine is a powerful aphrodisiac, and many people are using it primarily for its sexual effects. In high doses, especially smoked or injected, it causes a rapid increase in dopamine levels in the midbrain pleasure center, the nucleus accumbens (Fiorino & Phillips, 1999). Methamphetamine stimulates sexual desire, enhances the sexual response, and reduces perception of pain, permitting a wide variety of sexual activities. Methamphetamine users report having more sex partners, more unprotected anal intercourse with casual partners, engaging in sex in more types of venues, and more esoteric sex (Rawstorne, Digiusto, Worth, & Zablotska, 2007). The association between meth and sex is cross cultural, with whites, blacks, and Latinos citing enhanced sexual performance as a primary reason for their methamphetamine use (Diaz, Heckert, & Sanchez, 2005). This is not just a coastal city phenomenon. My personal experience as a practicing gynecologist has shown that methamphetamine use among heterosexual women in rural Alabama skyrocketed between 1991 and 2008, and with it a significant increase in anonymous sex with multiple partners, often as payment to a drug dealer.

Methamphetamine users were also more likely to be taking Viagra than nonusers due to the loss of erectile function seen in more advanced methamphetamine addicts. These men still experience intense sexual desire but are unable to obtain a lasting erection and so take Viagra to maintain an erection or choose to engage in receptive sex predominantly (Mansergh et al., 2006). Both methamphetamine and Viagra use are strongly associated with HIV infection in homosexual men (Drumright et al., 2006).

We are seeing a resurgence of all types of STDs, including syphilis, gonorrhea, and AIDS, particularly in drug-abusing populations. Methamphetamine users are often apathetic about their HIV status and careless about their sexual practices (Taylor, M. M., et al., 2007). Users often engage in unprotected sex, which is further associated with transmission of HIV and other sexually transmitted diseases (STDs). A longitudinal study of men who have sex with men revealed that seroconversions were strongly related to the use of drugs before sex, high numbers of partners, and unprotected anal intercourse (Koblin et al., 2006).

These factors are most obvious and measurable in homosexual communities, which have seen a resurgence of HIV infection since the mid-1990s (Catania et al., 2001). Methamphetamine use is 10–20 times more common in homosexual men than in the population at large (Mimiaga et al., 2008; Shoptaw et al., 2006). Even the occasional use of methamphetamine is associated with increased unprotected and serodiscordant sexual activity—activity with persons of opposite or unknown HIV status across the nation, as documented by Colfax et al. (2005) in San Francisco and Plankey et al., (2007) in Washington, D.C.

In a New York City study, methamphetamine seemed to attract a subset of hypersexual risk-taking gay men who do not take precautions, regardless of HIV serostatus. Equivalent rates of extreme sex acts were reported while high and while sober in this meth-abusing population (Halkitis, Shrem, & Martin, 2005). Failure to disclose HIV positivity is strongly associated with methamphetamine use, and condoms are seen as obstacles to pleasure and reminders of the burden of being HIV-positive (McCready & Halkitis, 2008).

The magnitude of the association between meth and HIV is illustrated by recent data from San Francisco. In a study of people seeking HIV testing at a San Francisco STD clinic, those who admitted to recent methamphetamine use were three times more likely to test HIV-positive The incidence of HIV seroconversion is 6.3 percent per year among homosexual methamphetamine users, three times the 2.1 percent conversion rate among non-meth users Those who used methamphetamine during sexual encounters were four times more likely to test positive with a seroconversion rate of 7.7 percent (Buchacz et al., 2005). These individuals were not predominantly IV users of the drug, but rather recreational users. Among homosexuals who use intravenous (IV) methamphetamine, 68 percent are trading sex for money or drugs, and these people are six times more likely to test HIV-positive than non–meth users (Bacon et al., 2006). In the IV drug-using homosexual population, 70 percent of men reported unprotected anal intercourse (Kral et al., 2005) and 42 percent of the HIV-infected men were not aware of their infection (Bacon et al., 2006).

Methamphetamine abuse contributing to HIV transmission is commonly associated with homosexual communities but is also seen in heterosexuals and adolescents who are also using methamphetamine primarily for its sexual effects (Springer et al., 2007). More than 63 percent of adolescents who are involved with drug abuse have engaged in five or more unsafe sexual practices (Tepline et al., 2005). Juveniles in the justice system are much more likely to engage in unsafe sexual practices, and more than 66 percent of them persist in doing so despite counseling and incarceration (Romero et al., 2007). Heterosexual men are also engaging in unsafe sex under the influence of methamphetamine, even when they know they are HIV-positive (Purcell et al., 2006)

Heterosexual meth-using women have similarly high rates of unsafe sexual activity, including multiple partners, anonymous partners, commercial sex work, and unprotected sex (Semple, Grant, & Patterson, 2004). The prevalence of sexually transmitted disease among homosexual women is comparable to that among heterosexual women, and risk factors are similar, with number of partners and unprotected sex being primary risk factors (McNair, 2005). While exclusively homosexual women have lower rates of STDs, many meth-using lesbians have additional risk factors such as prostitution—both homosexual and heterosexual—and IV drug use. IV drug use is eight times more prevalent in homosexual women, with corresponding increases in hepatitis C and HIV risk factors (Fethers, Marks, Mindle, & Estacourt, 2000).

GENERAL HEALTH EFFECTS OF METHAMPHETAMINE USE

Methamphetamine use is associated with poor hygiene, crowded living conditions, and increased general infection rates. Personal hygiene is often compromised as addicts become homeless or live in poverty. Methamphetamine use frequently causes loss of olfactory function as the nasal mucous membranes are compromised by near-constant exposure to a potent vasoconstrictor. Nasal perforations are seen in addicts who snort the drug frequently. As the sense of smell is compromised, the addict does not realize the severity of his hygiene problem.

Methamphetamine is a powerful anorectic and causes malnutrition in general, and vitamin deficiencies, anemia, and poor wound healing in particular. The immune system is especially vulnerable to poor nutrition, accounting for much morbidity, and even mortality. Generalized weakening of immune function related to vitamin and protein deficiencies predisposes addicts to infection from a variety of sources, in addition to STDs. Increased infection rates with community-acquired methacillin resistant staphylococcus aureus (MRSA) are

noted in methamphetamine addicts, with skin picking behavior so common that it is referred to as meth mites. Addicts also have increased rates of sinusitis and pneumonia among those who smoke the drug, and bacterial endocarditis among those who inject it.

Methamphetamine use is also associated with significant psychiatric comorbidity, as dopamine and serotonin neurotransmission is impaired. Anxiety disorders, major depressive symptoms, and bipolar spectrum are all strongly associated with methamphetamine use. Psychotic symptoms are very common among methamphetamine addicts, including persecutory delusions and frank paranoia (McKetin, 2007). Severe mood and thought disorders increase the likelihood of exposure to infectious diseases, especially STDs, by impairing rational decision making and judgment. Among methamphetamine-dependent gay and bisexual men, the incidence of STDs was increased in those with anxiety disorders, social phobias, bipolar disorder, and major depression (Shoptaw, Pack, Reback, & Rotheram-Fuller, 2003).

METHAMPHETAMINE ACCELERATES HIV PROGRESSION

A single case of a rapidly progressive, multiply resistant form of HIV in a New York man who used methamphetamine lead to speculation of a superinfective form of the disease spread by meth addicts (Centers for Disease Control and Prevention, 2006). And indeed, methamphetamine use is associated with one form of drug resistance in HIV: primary nonnucleoside reverse transcriptase inhibitor resistance (Colfax et al., 2007). Gorbach et al. (2008) reported that methamphetamine users were four times more likely to acquire a drug-resistant form of HIV.[1] Reinfection with a different strain of HIV is a common scenario in situations where HIV-positive individuals are engaging in unprotected intercourse with other HIV-positive partners (seroconcordant sex). But viral loads have not been noted to be higher among methamphetamine users in most studies.[2] The finding of rapid progression to clinical AIDS is also related to immune modulation directly or indirectly caused by methamphetamine. Methamphetamine is also a potent direct immunosuppressant, particularly in relation to HIV and HCV (Hepatitis C virus) infections. Tallóczy et al. (2007) published a series of experiments demonstrating the nature of immunosuppression associated with methamphetamine use, even at low concentrations. Methamphetamine acts biochemically as an adjuvant for HIV infection by reducing the immune response to infection both at the splenic level and in peripheral blood.

Dendritic cells in the spleen serve as the first line of defense against HIV. They recognize and internalize pathogens and subsequently activate T cells.

They are also the initial targets of the HIV virus, adhering to and invading these immune cells as a first step in infection. Methamphetamine interferes with dendritic cell function on several levels. First, it decreases dendritic cell expression of genes associated with chemokine regulation, cytokinesis, apoptosis, and cell cycle regulation (Mahajan et al., 2006). The clarion call for reinforcements is impaired. Methamphetamine also influences expression of two dendritic cell genes coding for adhesion-related proteins (Nair, Mahajan, Sykes, Bapardekar, & Reynolds, 2006), which are important for defense against HIV infection. Methamphetamine thus acts as a cofactor in the invasion of HIV into the dendritic cells of the spleen.

Methamphetamine is also active at the intracellular level in the spleen, inhibiting dendritic cell function by collapsing the Ph gradient these cells use to process antigens in the lysosome. It inhibits autophagosome processing, leading to an accumulation of autophagosomes and halting antigen delivery to immune cells (Tallóczy et al., 2007). Meth decreases T cell profilerative responses to intact antigen, impairing the cells' ability to recognize and repel an invasion. Saito et al. confirmed in 2008 that methamphetamine reduced natural killer cell function in splenic lymphocytes, which did not recover, even with interferon treatment.

Peripherally, methamphetamine also interferes with immune function in peripheral blood. Single and repeated methamphetamine injections acutely reduced peripheral blood leukocyte counts (Saito et al., 2008). Methamphetamine also collapses Ph gradients in peripheral blood macrophages, blocking phagocytosis in these primary immune cells concerned with clearance of bacteria. This is thought to underlie the severity of bacterial infections such as MRSA, periodontal disease, and various forms of pneumonia in methamphetamine addicts. Methamphetamine also inhibits phagocytosis of *Candida* and *Cryptococcus* by macrophages by 40 percent and, in fact, increased proliferation of these pathogenic fungi within macrophages (Tallóczy et al., 2007), even in the absence of HIV infection.

Methamphetamine thus acts synergistically with HIV infection to cause more rapid and severe disease progression to AIDS. Animal models involving retroviruses have elucidated the effects of methamphetamine on HIV infection in humans. Methamphetamine exposure further impaired immune function in mice infected with a murine retrovirus, causing reductions in interleukin 2, interferon, and cytokines in retrovirus-infected mice (Yu et al., 2002) Methamphetamine impaired immune function in the thymus by reducing CD8 and CD4 cells, inhibiting IgM formation, impairing the proliferation of mitogen-stimulated B and T cells, and inhibiting granulocyte-macrophage interactions (In, Son, Rhee, & Pyo, 2005). Similar changes have been noted in

the immune systems of HIV-infected humans who abuse alcohol and cocaine (Chiappelli et al., 2006).

METHAMPHETAMINE ACCELERATES CENTRAL NERVOUS SYSTEM INVOLVEMENT

Methamphetamine produces a massive release of neurotransmitters, including dopamine, serotonin, and nor-epinephrine. As receptors are overloaded and ultimately destroyed, mood regulation is impaired, resulting in symptoms of depression and anxiety, and in manifestations of bipolar disorder. Methamphetamine is a well-known neurotoxin, producing oxygen free radials in many tissues, including the central nervous system (Riddle, Fleckenstein, & Hanson, 2006). These toxic compounds cause direct cellular damage to central areas in the brain that regulate emotion, memory, and perception. The development of psychosis and dementia is especially problematic as both can persist long into recovery and complicate treatment efforts (McCann et al., 2007; Yui, Ikemoto, Ishiguro, & Goto, 2000).

HIV infection also affects the central nervous system with the development of AIDS dementia in up to 60 percent of patients with advanced disease. Cognitive impairment is seen, with loss of memory and concentration, and motor symptoms of clumsiness and abnormal gait. Development of dementia is strongly related to increased viral load in the cerebrospinal fluid (Ellis et al., 2002), decreased CD4+ counts, and poor survival, and the incidence of AIDS dementia has decreased in the HAART (Highly Active Anti Retroviral Therapy) era (Bhaskaran et al., 2008). Progression to AIDS dementia is significantly accelerated in patients who also use methamphetamine.

In Los Angeles, 50 percent of white male homosexuals testing positive for HIV are methamphetamine users (Wohl, Frye, & Johnson, 2007). The more rapid progression of HIV in the presence of methamphetamine abuse has been noted, with more severe deterioration in immune and neurologic function. HIV-infected macrophages invade the central nervous system early in the course of disease, causing inflammatory changes and neurodegeneration. Increased neurotoxicity in these patients is related primarily to increased inflammation independent of viral load (Vitiello et al., 2007).

HIV infection with concurrent methamphetamine abuse results in severe and rapidly progressive HIV-associated dementia, with 58 percent of HIV-positive/meth-positive men showing deterioration versus 38 percent of HIV-positive/meth-negative men showing impairment on neuropsychiatric testing (Rippeth et al., 2004). HIV infection in the brain causes encephalopathy and dementia by releasing a protein called Tat (transactivator of transcription) in

the striatum (motivation and motor control) and frontal cortex (judgment and reasoning, executive function). HIV-infected individuals who also abuse methamphetamine have more severe encephalitis and more neural damage than those who do not abuse meth (Taylor, M. J. et al., 2007). Both Tat and methamphetamine activate glia (neural scar tissue) and induce cytokine production, and cytokine levels are further increased in cell preparations exposed to both. The interaction between meth and HIV resulted in increased destruction of dopaminergic terminals in the striatum of rats (Theodore, Cass, & Maragos, 2006).

Hippocampal (memory storage) neurons exposed to both Tat and meth showed early evidence of neural damage at 6 hours and extensive cell death at 24 hours. These changes were associated with a dysregulated mitochondrial calcium potential (Langford et al., 2004); increased levels of oxidative stress; inflammation; and mitochondrial damage in the frontal cortex, hippocampus, and striatum (Flora et al., 2003). Injury to these areas is strongly associated with memory loss and cognitive impairment in HIV-positive methamphetamine users (Chana et al., 2006).

In human studies, higher viral load was strongly associated with increased cellular damage on Magnetic Resonance Spectroscopy scanning (a measure of cellular metabolic function) in patients who also used methamphetamine, but significantly less in those who did not use meth (Taylor, M. J. et al., 2007). Brain metabolite MRS scanning revealed an additive effect of methamphetamine on the HIV-related brain injury in the frontal lobe and basal ganglia (Chang, Ernst, Speck, & Grob, 2005). On magnetic resonance imaging (a measure of cellular volume and structural integrity), atrophy was seen in the HIV-positive patients, while significant swelling was noted in the cortical areas and striatum of HIV-positive methamphetamine users, and atrophy in the hippocampus (Jernigan et al., 2005). Lower CD4 counts were also associated with more neuropsychiatric impairment in meth-using patients (Carey et al., 2006). When HIV is poorly controlled, methamphetamine exposure results in worsening brain injury.

HIV viral control is markedly impaired in methamphetamine users, related both to the intoxicated state and to the mental health consequences of meth use, including depression and anxiety problems. In a study of new-onset HIV patients, 63 percent had a mental illness (including depression, anxiety, psychosis, or a personality disorder), 45 percent had a substance abuse disorder, and 38 percent had both. Those with untreated mental illness or a substance abuse disorder were in a more advanced disease state—higher viral loads and lower CD4+ counts—than those who did not have these problems (Tegger et al., 2008). Methamphetamine-addicted HIV patients are sicker at presentation, and are also less likely to comply with treatment. A longitudinal study

showed markedly reduced HAART medication compliance in drug-using patients, with a fourfold greater risk of medication errors and noncompliance. Stimulant users were at greatest risk of noncompliance, particularly when they were actively using drugs and thus frequently intoxicated (Hinkin et al., 2007). Active cocaine users had only 27 percent adherence to antiviral medications, versus 68 percent for nonusers. Consequently, only 13 percent of cocaine users maintained viral suppression, versus 46 percent of nonusers (Arnsten et al., 2002). As a result of poor compliance, drug users and patients with mental health problems such as anxiety or depression showed slower rates of virologic suppression and faster rates of virologic failure (Pence, Miller, Gaynes, & Eron, 2007). The risk of developing resistance to HAART is greatly increased in situations of late diagnosis and poor compliance with medical regimens, and these resistant strains are then transmitted to others.

The prevalence of hepatitis C coinfection in HIV-positive patients is around 30 percent over all (Rockstroh et al., 2005) and between 70 and 90 percent among injection drug users (Lauer & Walker, 2001; Sherman et al., 2002). Not surprisingly, persons coinfected with HIV and hepatitis C had poorer response to HAART, higher mortality rates, and were more likely to die from HIV-related causes, even with similar viral loads (Weis et al., 2006) and CD4 counts (Anderson, Guest, & Rimland, 2004). Hepatitis C coinfection makes HIV disease worse. Also not surprisingly, persons coinfected with HIV and hepatitis C had increased rates of HCV persistence (95%) with a lack of CD4 T cell response during acute disease contributing to failure of early disease control (Danta et al., 2008). Coinfected individuals who have cleared HCV spontaneously remain at risk for recurrence of hepatitis C viremia if their CD4 counts fall (Kim et al., 2006). HIV coinfection makes hepatitis C disease worse. Methamphetamine use further impairs the immune response to hepatitis C, resulting in more severe liver disease and poorer response to interferon treatment (Ye et al., 2008). Even co-infected individuals who have cleared their HCV spontaneously remain at risk for recurrence of hepatitis C viremia if their CD4 counts fall (Kim et al., 2006).

HCV is itself associated with cognitive impairment, even in the absence of cirrhosis, in one-third of chronically infected persons (Perry, Hilsabeck, & Hassanein, 2008). Neuropsychological function is further impaired by concurrent hepatitis C infection in HIV-positive patients (Richardson et al., 2005; Aronow, Weston, Pezeshki, & Lazarus, 2008). Hepatitis C infection is associated with higher levels of HIV RNA in the cerebrospinal fluid of HIV-positive patients, reflecting more central nervous system invasion by HIV (Letendre et al., 2005). Both cognitive function and affective symptoms were adversely affected by concurrent hepatitis C infection (Clifford, Evans, Yang, & Gulick, 2005; Ryan, 2004). Patients with concurrent HCV infection, HIV infection,

and methamphetamine use were even more severely impaired, with deficiencies in learning, abstraction, motor skills, information processing, and delayed recall (Cherner et al., 2005). The impact appears to be directly attributable to the viral infections since differences in biochemical measures of liver function did not explain the neuropsychological differences in the hepatitis-infected persons (Morgello et al., 2005).

The cognitive effects of HIV and HCV are not trivial. They can in fact result in frank dementia. Clearly HIV and HCV status must be considered in academic studies of cognitive function in methamphetamine users and in treatment planning for methamphetamine addicts. Treatment involving cognitive behavioral therapy will likely require much more time and attention in cases involving concurrent HIV and/or HCV infection.

INTERVENTIONS

The opportunity for intervention in the progression of HIV-related disease is significant. With the advent of HAART treatments for HIV infection, more infected people are living almost full life spans, and more of them are developing HIV-related dementia. The impact methamphetamine has on HIV-related dementia is substantial, and much morbidity could be prevented by reducing the meth use of HIV-positive patients. Also, many HIV infections could be prevented by reducing the high-risk sexual behaviors of meth users.

Reaching the populations at risk for HIV infection has proven difficult since so many people whose sexual behavior places them at risk do not consider themselves members of the at-risk groups. Youths are increasing identifying with a homosexual lifestyle and yet do not consider themselves at risk for the diseases of the older generation of gays. Adult men who have sex with men often have a wife and kids at home, and though they participate in same-sex adventures frequently, they do not consider themselves gay. Closeted homosexuals or bisexuals also are not likely to participate in HIV risk prevention programs and are unlikely to change their behavior in response to such a program.

Concurrently IV methamphetamine and cocaine addicts often do not consider themselves IV drug abusers because they do not use heroin. IV methamphetamine addicts often do not consider themselves at risk for HIV even though 45 to 50 percent of them are HIV-positive. Public perception of what constitutes a risk factor for HIV is significantly outdated (Bull, Piper, & Reitmeijer, 2002). Safe sex programs aiming only at self-identified homosexuals miss more than half of the at-risk population, including virtually all of the IV drug abusers at risk via needle sharing.

Even among self-identified homosexuals, compliance with safe sex measures is sporadic, especially among meth users. Use of party drugs in relation to sex,

including methamphetamine, increases risk of unprotected sexual encounters with HIV-positive individuals, with reliance on assumptions of seroconcordence (Purcell, Moss, Remien, Woods, & Parsons, 2005). Unprotected serodiscordant sex is considered an act of violence among homosexuals, yet many methamphetamine users regularly engage in it. Risky behavior has escalated in the years since highly active anti-retroviral therapy became available. HIV seroconversion is strongly associated with use of methamphetamine, poppers, and/or Viagra. Methamphetamine use is also associated with unprotected anal insertive or receptive sex, number of partners, and complacency regarding the effectiveness of medical treatments for HIV (Plankey et al., 2007; Schwarcz et al., 2007).

Efforts to reduce unsafe sexual practices among homosexual meth-using men have been disappointing. Cognitive behavioral therapy aimed at reducing unsafe sexual practices in the face of ongoing methamphetamine use improved actual behavior only minimally. Participants engaged in safe sex 25 percent of the time, as opposed to 18 percent of the time for control groups 12 months posttreatment in HIV-positive men (Mausbach, Semple, Stradthdee, Zians, & Patterson, 2007). A similar study with peer-led behavioral intervention reduced unsafe sex among HIV-positive men from 26 percent of encounters to 21 percent at three months' postintervention (Wolitski, Gómez, & Parsons, 2005). The low rates of safe sex among these HIV-positive men suggest that we face a serious challenge in our attempts to control transmission of HIV.

Studies show more success in behavior modification if the drug addiction is addressed directly rather than focusing on the consequent unsafe sexual behaviors. Successful addiction treatment with contingency management, cognitive behavioral therapy, or both was more effective in reducing sexual risk behaviors than counseling directed toward the sexual risk factors themselves (Shoptaw, 2006). Reduced methamphetamine use itself resulted in declining depression scores and reduced sexual risk behavior. This suggests that lowering methamphetamine use has a synergistic effect on reducing unsafe sex among homosexual men (Jaffe, Shoptaw, Stein, Reback, & Rotheram-Fuller, 2007). This beneficial effect on sexual risk behavior was persistent in sustained recovery. Successful drug treatment resulted in reduced sexual risk behaviors at one year follow-up, with fewer anonymous partners, reduced anal intercourse, and an increased sense of responsibility to disclose HIV status (Reback, Larkins, & Shoptaw, 2004).

Unavailability of drug treatment facilities has been a continuing problem in areas hard hit by methamphetamine, particularly in the homosexual communities. A majority of homosexual men who use methamphetamine report concern over the effects meth is having in their lives. Of 174 men surveyed at a San Diego STD clinic, 70 percent had tried to quit their methamphetamine use, and 52 percent were interested in obtaining treatment, but only 12 percent had ever been in drug treatment (Menza et al., 2006). Some men feel that if

they quit using meth, their sex life will be over, and they need a role model of successful recovery to take the first step. The vast majority deal with persistent depression and anxiety and are in need of mental health care (Mimiaga et al., 2008). Greater availability of effective drug treatments offers the best hope for reducing HIV transmission in these populations.

Education programs that reach people where they are, physically and emotionally, are much more effective than programs that require the participant to come to a physical location, or admit something that they do not wish to acknowledge. There is some promise in an Internet-based outreach program that takes advantage of the partner meeting Web sites that abound online. This is the starting point for many crystal methamphetamine–related hookups and offers a potential starting point for interventions and educational efforts (Mimiaga et al., 2008). Specifically, posters and pictures depicting the physical degeneration common in addicts may be effective in communicating with young men who highly value their physical appearance.

Our nation's public health is at significant risk if we fail to address the needs and challenges of our IV drug abusing and our homosexual communities. Diseases that are permitted to flourish in one community will most certainly compromise the health and vitality of the entire nation. Taken solely from an economic point of view, we cannot afford the loss of so many talented young people to a preventable disease. We also cannot afford the large number of liver transplants and expensive medications that will be required in coming years if we refuse to take action.

Drug education and prevention work must be done more effectively, with attention to the needs of our youth, our homosexual communities, recent immigrants, and others who have been marginalized. Tuberculosis control was made possible by raising the standards for health and hygiene over all of the United States. Methamphetamine addiction and HIV control can only be accomplished by raising the standards for drug education all over the nation, including gay America.

NOTES

1. Bushkin et al. (2008) described a cluster of 9 methamphetamine using men with phylogenetically related multiple drug resistant HIV strains with resistance to most anti-retroviral drugs. Only 2 of those men had prior experience with anti-retrovirals. All had had unprotected sex with multiple anonymous partners.
2. A recent study by Carrico et al. (2008) found markedly higher viral loads, higher neopterin levels and lower tryptophan levels (both indicators of immune activiation) in stimulant users, regardless of compliance with antir-retroviral therapy.

REFERENCES

Anderson, K. B., Guest, J. L., & Rimland, D. (2004). Hepatitis C virus co-infection increases mortality in HIV infected patients in the highly active anti-retroviral therapy era: Data from the HIV Atlanta VA cohort study. *Clinical Infectious Diseases, 39,* 1507–1513.

Arnsten, J. H., Demas, P. A., Grant, R. W., Gourevitch, M. N., Farzadegan, H., Howard, A. A., et al. (2002). Impact of active drug use on antiretroviral therapy adherence and viral suppression in HIV-infected drug users. *Journal of General Internal Medicine, 17,* 377–381.

Aronow, H. A., Weston, A. J., Pezeshki, B. B., & Lazarus, T. S. (2008). Effects of co-infection with HIV and hepatitis C virus on the nervous system. *The AIDS Reader, 18,* 43–48.

Bacon, O., Lum, P., Hahn, J., Evans, J., Davidson, P., Moss, A,, et al. (2006). Commercial sex work and risk of HIV infection among young drug injecting men who have sex with men in San Francisco. *Sexually Transmitted Diseases, 33,* 228–234.

Bhaskaran, K., Mussini, C., Antinori, A., Walker, A. S., Dorrucci, M., Sabin, C., et al. (2008). Changes in the incidence and predictors of human immunodeficiency virus associated dementia in the era of highly active antiretroviral therapy. *Annals of Neurology, 63,* 312–321.

Buchacz, K., McFarland, W., Kellogg, T. A., Loeb, L., Holmberg, S. D., Dilley, J., et al. (2005). Amphetamine use is associated with increased HIV incidence among men who have sex with men in San Francisco. *AIDS 19,* 1423–1424.

Bull, S. S., Piper, P., & Reitmeijer, C. (2002). Men who have sex with men and also inject drugs—profiles of risk related to the synergy of sex and drug injection behaviors. *Journal of Homosexuality, 42,* 31–51.

Buskin, S. E., Ellis, G. M., Pepper, G. G., Frenkel, L. M., Pergram, S. A., Gottlieb, G. S., et al. (2008). Transmission cluster of multiclass highly drug-resistant HIV-1 among 9 men who have sex with men in Seatle/King Count, WA, 2005–2007. *Journal of Acquired Immune Deficiency Syndrome, 49,* 205–211.

Carey, C. L., Woods, S. P., Rippeth, J. D., Gonzalez, R., Heaton, R. K., & Grant, I. (2006). Additive deleterious effects of methamphetamine dependence and immunosuppression on neuropsychological functioning in HIV infection. *AIDS and Behavior, 10,* 185–190.

Carrico, A. W., Johnson, M. O., Morin, S. F., Remien, R. H., Riley, E. D., Hecht, F. M., & Fuchs, D. (2008). Stimulant use is associated with immune activation and depleted tryptophan among HIV-positive persons on anti-retroviral therapy. *Brain Behavior and Immunology, 22,* 1257–1262.

Catania, J. A., Osmond, D., Stall, R. D., Pollack, L., Paul, J. P., Blower, S., et al. (2001). The continuing HIV epidemic among men who have sex with men. *American Journal of Public Health, 91,* 907–914.

Centers for Disease Control and Prevention. (2006). Investigation of a new diagnosis of multidrug resistant, dual-tropic HIV-1 infection—New York City in 2005. *Morbidity and Mortality Weekly Report, 55,* 793–796.

Chana, G., Everall, I. P., Crews, L., Langford, D., Adame, A., Grant, I., et al. (2006). Cognitive deficits and degeneration of interneurons in HIV+ methamphetamine users. *Neurology, 67,* 1486–1489.

Chang, L., Ernst, T., Speck, O., & Grob, C. S. (2005). Additive effects of HIV and chronic methamphetamine use on brain metabolite abnormalities. *American Journal of Psychology, 162,* 361–369.

Cherner, M., Letendre, S., Heaton, R. K., Durelle, J., Marque-Beck, J., Gragg, B., et al. (2005). Hepatitis C augments cognitive deficits associated with HIV infection and methamphetamine. *Neurology, 64,* 1343–1347.

Chiappelli, F., Shapshak, P., Younai, F., McCoy, C., Page, B., & Prolo. P., (2006). Cellular immunology in HIV 1 positive African American women using alcohol and cocaine. *Frontiers in Bioscience, 11,* 2434–2441.

Clifford, D. B., Evans, S. R., Yang, Y., & Gulick, R. M. (2005). The neuropsychological and neurological impact of hepatitis C virus co-infection in HIV infected subjects. *AIDS, 19*(Suppl. 3), S64–S71.

Colfax, G. N., Coates, T. J., Husnik, M. J., Huang, Y., Buchbinder, S., Koblin B., et al. (2005). Longitudinal patterns of methamphetamine, popper (amyl nitrite), and cocaine use and high risk sexual behavior among a cohort of San Francisco men who have sex with men. *Journal of Urban Health, 82*(Suppl. 1), i62–i70.

Colfax, G. N., Vittinghoff, E., Grant, R., Lum, P., Spotts, G., & Hecht, F. M. (2007). Frequent methamphetamine use is associated with primary non-nucleoside reverse transcriptase inhibitor resistance. *AIDS, 21,* 239–241.

Danta, M., Semmo, N., Fabris, P., Brown, D., Pybus, O. G., Sabin, C. A., et al. (2008). Impact of HIV on host-virus interactions during early hepatitis C infection. *Journal of Infectious Diseases,* doi:10.1086/587843.

Diaz, R. M., Heckert, A. L., & Sanchez, J. (2005). Reasons for stimulant use among Latino gay men in San Francisco: a comparison between methamphetamine and cocaine users. *Journal of Urban Health, 82* (Suppl 1), i71–i78.

Dickerson, M. C., Johnston, J., Delea, T. E., White, A., & Andrews, E. (1996). The causal role for genital ulcer disease as a risk factor for transmission of human immunodeficiency virus: An application of the Bradford Hill criteria. *Sexually Transmitted Diseases, 23,* 429–440.

Drumright, L. N., Little, S. J., Strathdee, S. A., Slymen, D. J., Araneta, M. R., Malcarne, V. L., et al. (2006). Unprotected anal intercourse and substance use among men who have sex with men with recent HIV infection. *Journal of Acquired Immune Deficiency Syndrome, 43,* 344–350.

Ellis, R. J., Moore, D. J., Childers, M. E., Letendre, S., McCutchan, J. A., Wolfson, T., et al. (2002). Progression to neuropsychological impairment in human immunodeficiency virus infection predicted by elevated cerebrospinal fluid levels of human immunodeficiency virus RNA. *Archives of Neurology, 59,* 923–928.

Fethers, K., Marks, C., Mindle, A., & Estacourt, C. S. (2000). Sexually transmitted infections and risk behaviors in women who have sex with women. *Sexually Transmitted Infections, 76,* 345–349.

Fiorino, D. F. & Phillips, A. G. (1999). Facilitation of sexual behavior in male rats following d-amphetamine-induced behavioral sensitization. *Psychopharmacology (Berlin), 142,* 200–208.

Flora, G., Lee, Y. W., Nath, A., Henning, B., Maragos, W., & Toborek, M. (2003). Methamphetamine potentiates HIV-1 Tat protein mediated activation of redox sensitive pathways in discrete regions of the brain. *Experimental Neurology, 179,* 60–70.

Gorbach, P. M., Drumright, L. N., Javanbakht, M., Pond, S. L., Woelk, C. H., Daar, E. S., et al. (2008). Antiretroviral drug resistance and risk behavior among recently HIV infected men. *Journal of Acquired Immune Deficiency Syndrome, 47,* 639–643.

Halkitis, P. N., Shrem, M. T., & Martin, F. W. (2005). Sexual behavior patterns of methamphetamine using gay and bisexual men. *Substance Use and Misuse, 40,* 703–719.

Hinkin, C. H., Barclay, T. R., Castellon, S. A., Levine, A. J., Durvasula, R. S., Marion, S. D., et al. (2007). Drug use and medication adherence among HIV-1 infected individuals. *AIDS and Behavior, 11,* 185–194.

In, S. W., Son, E. W., Rhee, D. K., & Pyo, S. (2005). Methamphetamine administration produces immunomodulation in mice. *Journal of Toxicology and Environmental Health, Series A, 68,* 2133–2145.

Jaffe, A., Shoptaw, S., Stein, J., Reback, C. J., & Rotheram-Fuller, E. (2007). Depression ratings, reported sexual risk behaviors, and methamphetamine use: Latent growth curve models of positive change among gay and bisexual men in an outpatient treatment program. *Experimental and Clinical Psychopharmacology, 15,* 301–307.

Jernigan, T. L., Gamst, A. C., Archibald, S. L., Fennema-Notestine, C., Mindt, M. R., Marcotte, T. D., et al. (2005). Effects of methamphetamine dependence and HIV infection on cerebral morphology. *American Journal of Psychiatry, 162,* 1461–1472.

Kim, A. Y., Schulze zur Wiesch, J., Kuntzen, T., Timm, J., Kaufmann, D. E., Duncan, J. E., et al. (2006). Impaired hepatitis C virus specific T cell responses and recurrent hepatitis C virus in HIV co-infection. *PLoS Medicine, 3,* e492.

Koblin, B. A., Husnik, M. J., Colfax, G., Huang, Y., Madison, M., Mayer, K., et al. (2006). Risk factors for HIV infection among men who have sex with men. *AIDS, 20,* 731–739.

Kral, A. H., Lorvick, J., Ciccarone, D., Wegner, L., Gee, L., Martinez, A., et al. (2005). HIV prevalence and risk factors among men who have sex with men and inject drugs in San Francisco. *Journal of Urban Health, 82*(Suppl. 1), 143–150.

Langford, D., Grigorian, A., Hurford, R., Adame, A., Crews, L., & Masliah, E. (2004). The role of mitochondrial alterations in the combined toxic effects of human immunodeficiency virus Tat protein and methamphetamine in calbindin positive neurons. *Journal of Neurovirology, 10,* 327–337.

Lauer, G., & Walker, B. (2001). Hepatitis C infection. *New England Journal of Medicine, 345,* 41–52.

Letendre, S. L., Cherner, M., Ellis, R. J., Marque-Beck, J., Gragg, B., Marcotte, T., et al. (2005). The effects of hepatitis C, HIV, and methamphetamine dependence on neurophysiological performance: Biological correlates of disease. *AIDS, 19*(Suppl. 3), 572–578.

Mahajan, S. D., Hu, Z., Reynolds, J. L., Aalinkeel, R., Schwartz, S. A., & Nair, M.P. (2006). Methamphetamine modulates gene expression patterns in monocyte derived mature dendritic cells: Implications for HIV-1 pathogenesis. *Molecular Diagnosis and Therapy, 10,* 257–269.

Mansergh, G., Shouse, R. L., Marks, G., Guzman, R., Rader, M., Buchbinder, S., et al. (2006). Methamphetamine and sildenafil (Viagra) use are linked to unprotected receptive and insertive anal sex, respectively, in a sample of men who have sex with men. *Sexually Transmitted Infections, 82,* 131–134.

Mausbach, B. T., Semple, S. J., Stradthdee, S. A., Zians, J., & Patterson, T. L. (2007). Efficacy of a behavioral intervention for increasing safer sex behaviors in HIV positive MSM methamphetamine users: Results from the EDGE study. *Drug and Alcohol Dependence, 87,* 249–257.

McCann, U. D., Kuwabara, H., Kumar, A., Palermo, M., Abbey, R., Brasic, J., et al. (2007). Persistent cognitive and dopamine transporter deficits in abstinent methamphetamine users. *Synapse, 62,* 91–100.

McCready, K. C., & Halkitis, P. N. (2008). HIV serostatus disclosure to sexual partners among HIV positive methamphetamine using gay, bisexual, and other men who have sex with men. *AIDS Education and Prevention, 20,* 15–29.

McKetin, R., McLaren, J., Lubman, D. I., & Hides, L. (2007). The prevalence of psychotic symptoms among methamphetamine users. *Addiction, 101,* 1473–1478.

McNair, R. (2005). Risks and prevention of sexually transmissible infections among women who have sex with women. *Sexual Health, 2,* 209–217.

Menza, T. W., Colfax, G., Shoptaw, S., Fleming, M., Guzman, R., Klausner, J. D., et al. (2006). Interest in a methamphetamine intervention among men who have sex with men. *Sexually Transmitted Diseases, 34,* 209–214.

Mimiaga, M. J., Fair, A. D., Mayer, K. H., Koenen, K., Gortmaker, S., Tetu, A. M., et al. (2008). Experiences and sexual behaviors of HIV- infected MSM who acquired HIV in the context of crystal methamphetamine use. *AIDS Education and Prevention, 20,* 30–41.

Morgello, S., Estanisloa, L., Ryan, E., Gertis, P., Simpson, D., Verma, S., et al. (2005). Effects of hepatic function and hepatitis C virus in the nervous system of advanced stage HIV infected individuals. *AIDS, 19*(Suppl. 3), S116–S122.

Nair, M. P., Mahajan, S., Sykes, D., Bapardekar, M. V., & Reynolds, J. L. (2006). Methamphetamine modulates DC SIGN expression by mature dendritic cells. *Journal of Neuroimmune Pharmacology, 1,* 296–304.

Pence, B. W., Miller, W. C., Gaynes, B. N., & Eron, J. J., Jr. (2007). Psychiatric illness and virologic response in patients initiating highly active antiretroviral therapy. *Journal of Acquired Immune Deficiency Syndrome, 44,* 159–166.

Perry, W., Hilsabeck, R. C., & Hassanein, T. I. (2008). Cognitive dysfunction in chronic hepatitis C: A review. *Digestive Diseases and Sciences, 53,* 307–321.

Plankey, M. W., Ostrow, D. G., Stall, R., Cox, C., Li, X., Peck, J. A., et al. (2007). The relationship between methamphetamine and popper use and risk of HIV seroconversion in the multicenter AIDS cohort study. *Journal of Acquired Immune Deficiency Syndrome, 45,* 85–92.

Purcell, D. W., Mizuno, Y., Metch, L. R., Garfein, R., Tobin, K., Knight, K., et al. (2006). Unprotected sexual behavior among heterosexual HIV positive injection drug using men: Associations by partner type and partner serostatus. *Journal of Urban Health, 83,* 656–668.

Purcell, D. W., Moss, S., Remien, R. H., Woods, W. J., & Parsons, J. T. (2005). Illicit substance use, sexual risk, and HIV positive gay and bisexual men: Differences by sero-status of casual partners. *AIDS, 19,* 537–547.

Rawstorne, P., Digiusto, E., Worth, H., & Zablotska, I. (2007). Associations between crystal methamphetamine use and potentially unsafe sexual activity among gay men in Australia. *Archives of Sexual Behavior, 36,* 646–654.

Reback, C. J., Larkins, S., & Shoptaw, S. (2004). Changes in the meaning of sexual risk behaviors among gay and bisexual male methamphetamine abusers before and after drug treatment. *AIDS and Behavior, 8,* 87–98.

Richardson, J. L., Nowicki, M., Danley, K., Martin, E. M., Cohen, M. H., Gonzalez, R., et al. (2005). Neuropsychological functioning in a cohort of HIV and hepatitis C virus infected women. *AIDS, 19,* 1659–1667.

Riddle, E. L., Fleckenstein, A. E., & Hanson, G. R. (2006). Mechanisms of methamphetamine induced dopaminergic neurotoxicity. *American Association of Pharmaceutical Scientists Journal, 8,* E413–E418.

Rippeth, J. D., Heaton, R. K., Carey, C. L., Marcotte, T. D., Moore, D. J., Gonzalez, R., et al. (2004). Methamphetamine dependence increases risk of neuropsychological impairment in HIV infected persons. *Journal of International Neuropsychology, 10,* 1–14.

Rockstroh, J. K., Mocroft, A., Soriano, V., Tural, C., Losso, M. H., Horban, A., et al. (2005). Influence of hepatitis C virus infection in HIV-1 disease progression and response to highly active antiretroviral therapy. *Journal of Infectious Diseases, 192,* 992–1002.

Romero, E. G., Teplin, L. A., McClelland, G. M., Abram, K. M., Welty, L. J., & Washburn J. J. (2007). A longitudinal study of the prevalence, development and persistence of HIV/sexually transmitted infection risk behaviors in delinquent youth: Implications for health care in the community. *Pediatrics, 199,* e1126–e1141.

Ryan, E. L., Morgello, S., Isaacs, K., Phil, M., Naseer, M., & the Manhattan Brain Bank. (2004). Neuropsychiatric impact of hepatitis C on advanced HIV. *Neurology, 62,* 957–962.

Saito, M., Terada, M., Kawata, T., Ito, H., Shigematsu, N., Kromkhun, P., et al. (2008). Effects of single or repeated administrations of methamphetamine on immune response in mice. *Experimental Animals, 57,* 35–43.

Schwarcz, S., Scheer, S., McFarland, W., Katz, M., Valleroy, L., Chen, S., et al. (2007). Prevalence of HIV infection and predictors of high transmission sexual risk behaviors among men who have sex with men. *American Journal of Public Health, 97*, 1067–1075.

Semple, S. J., Grant, I., & Patterson, T. L. (2004). Female methamphetamine users: Social characteristics and sexual risk behaviors. *Women's Health, 40*, 35–50.

Sherman, K. E., Rouster, S. D., Chung, R. T., & Rajicic, N. (2002). Hepatitis C virus prevalence among patients infected with Human Immunodeficiency Virus: A cross-sectional analysis of the US adult AIDS Clinical Trials Group. *Clinics in Infectious Disease, 34*, 831–837.

Shoptaw, S. (2006). Methamphetamine use in urban gay and bisexual populations. *Topics in HIV Medicine, 14*, 84–87.

Shoptaw, S., Klausner, J. D., Reback, C. J., Tierney, S., Stansell, J., Hare, C. B., et al. (2006). A public health response to the methamphetamine epidemic: The implementation of contingency management to treat methamphetamine dependence. *Bio Med Central Public Health, 6*, 214.

Shoptaw, S., Pack, J., Reback, C. J., & Rotheram-Fuller, E. (2003). Psychiatric and substance dependence co-morbidities, sexually transmitted diseases, and risk behaviors among methamphetamine dependent gay and bisexual men seeking outpatient drug abuse treatment. *Journal of Psychoactive Drugs, 35*(Suppl. 1), 161–168.

Springer, A. E., Peters, R. J., Shegog, R., White, D. L., & Kelder, S. H. (2007). Methamphetamine use and sexual risk behaviors in U S high school students: Findings from a national risk behavior survey. *Prevention Science, 8*, 103–113.

Tallóczy, Z., Martinez, J., Joset, D., Ray, Y., Gacser, A., Toussi, S., et al. (2008). Methamphetamine inhibits antigen processing, presentation, and phagocytosis. *PLoS Pathogens, 4*, e28.

Taylor, M. J., Schweinsburg, B. C., Alhassoon, O. M., Gongvatana, A., Brown, G. G., Young-Casey, C., et al. (2007). Effects of human immunodeficiency virus and methamphetamine on cerebral metabolite measured with magnetic resonance spectroscopy. *Journal of Neurovirology, 13*, 150–159.

Taylor, M. M., Aynalem, G., Smith, L. V., Montoya, J., & Kerndt, P. (2007). Methamphetamine use and sexual risk behavior among men who have sex with men diagnosed with early syphilis in Los Angeles County. *International Journal of Sexually Transmitted Diseases and AIDS, 18*, 92–97.

Tegger, M. K., Crane, H. M., Tapia, K. A., Uldall, K. K., Holte, S. E., & Kitahata, M. M. (2008). The effect of mental illness, substance abuse, and treatment for depression on the initiation of highly active antiretroviral therapy among HIV infected individuals. *AIDS Patient Care and STDs, 22*, 233–243.

Teplin, L. A., Elkington, K. S., McClelland, G. M., Abram, K. M., Mericle, A. A., & Washburn, J. J. (2005). Major mental disorders, substance use disorders, co-morbidity, and HIV—AIDS risk behaviors in juvenile detainees. *Psychiatric Services, 56*, 823–828.

Theodore, S., Cass, W. A., & Maragos, W. F. (2006). Involvement of cytokines in human immunodeficiency virus—1 protein Tat and methamphetamine interactions in the striatum. *Experimental Neurology, 199,* 490–498.

Vitiello, B., Goodkin, K., Ashtana, D., Shapshak, P., Atkinson, J. H., Heseltine, P. N., et al. (2007). HIV-1 RNA concentration and cognitive performance in a cohort of HIV positive people. *AIDS, 21,* 1415–1422.

Weis, N., Lindhardt, B. O., Kronborg, G., Hansen, A. B., Larursen, A. L., Christensen, P. B., et al. (2006). Impact of hepatitis C virus co-infection on response to highly active anti-retroviral therapy and outcome in HIV infected individuals: A nationwide cohort study. *Clinical Infectious Diseases, 42,* 1481–1487.

Wohl, A. R., Frye, D. M., & Johnson, D. F. (2007). Demographic characteristics and sexual behaviors associated with methamphetamine use among MSM and non-MSM diagnosed with AIDS in Los Angeles County. *AIDS and Behavior,* doi:10.1007/s10461-007-9315-7.

Wolitski, R. J., Gamez, C. A., & Parsons, J. T. (2005). Effects of a peer led behavioral intervention to reduce HIV transmission and promote serostatus disclosure among HIV seropositive gay and bisexual men. *AIDS, 19*(Suppl. 1), S99–S109.

Ye, L., Peng, J. S., Wang, X., Wang, Y. J., Luo, G. X., & Ho, W. Z. (2008). Methamphetamine enhances hepatitis C virus replication in human hepatocytes. *Journal of Viral Hepatitis, 15,* 261–270.

Yu, Q., Zhang, D., Walston, M., Zhang, J., Liu, Y., & Watson, R. R. (2002). Chronic methamphetamine exposure alters immune function in normal and retrovirus infected mice. *International Immunopharmacology, 2,* 951–962.

Yui, K., Ikemoto, S., Ishiguro, T., & Goto, K. (2000). Studies of amphetamine or methamphetamine psychosis in Japan: Relation of methamphetamine psychosis to schizophrenia. *Annals of the New York Academy of Science, 914,* 1–12.

This page intentionally left blank

Index

This page intentionally left blank

About the Editor and Contributors

EDITOR

Angela Browne-Miller, PhD, DSW, MPH, is the Set Editor of the *Praeger International Collection on Addictions* (2009), Director of Metaxis Institute for Personal, Social, and Systems Change, based in northern California, United States; Director of Browne and Associates Violence, Substance Abuse, and Trauma Treatment and Prevention Program, also in northern California; has been a keynote speaker at conferences around the world on addiction, violence, and behavior change; and is the author of numerous books, including *To Have and to Hurt: Seeing, Changing or Escaping Patterns of Abuse in Relationships* (2007) and *Rewiring Your Brain to Change Your Behavior* (2009). Dr. Browne-Miller earned two doctorates and two master's degrees at the University of California, Berkeley, where she lectured in three departments for 14 years, and has served as a National Institute of Mental Health Postdoctoral Fellow, a U.S. Department of Public Health Fellow, the public relations director for Californians for Drug Free Youth, the Research Education and Treatment Director for the Cokenders Alcohol and Drug Program, a member of the board of directors of the Employee Assistance Society of North America, an advisor to addiction treatment programs in the United States and several other countries, and project director on three California Department of Health violence prevention projects.

CONTRIBUTORS

Jemel P. Aguilar, PhD, MSW, is an assistant professor at the University of Texas at Austin School of Social Work and the Center for Women and Gender Studies in Austin, Texas, United States. Dr. Aguilar conducts research on offender populations and educational structures' influences on juvenile offending. His primary research interests include gender studies, criminal and juvenile justice, and theory and model development. Dr. Aguilar has published articles on adolescent experiences in juvenile corrections settings, accountability systems in education and social services, and theoretical articles about the psychosocial risks facing low-income youth in urban areas.

Atul Ambekar, MD, is assistant professor at the National Drug Dependence Treatment Centre and Department of Psychiatry, All India Institute of Medical Sciences in New Delhi, Delhi, India. He earned his MD degree from the All India Institute of Medical Sciences, a prestigious and internationally recognized institute in India. He worked as senior resident and later joined the United Nations Office on Drugs and Crime (UNODC), Regional Office for South Asia, New Delhi, as a research officer. Subsequently, he joined All India Institute of Medical Sciences as an assistant professor. He is a resource person for training and research for various national and international organizations such as UNODC, the Joint United Nations Programme on HIV/AIDS, and the government of India. He has several publications to his credit.

Xenia Anastassiou-Hadjicharalambous, PhD, was born and raised on the Mediterranean island of Cyprus. She obtained her undergraduate degree from Aristotelian University (Thessaloniki, Greece), her MSc in psychology from Essex University (Colchester, United Kingdom), and her PhD from Strathclyde University (Glasgow, United Kingdom). She held a research postdoctoral position at the University of Strathclyde before joining the University of Nicosia in Nicosia, Cyprus in September 2004 as a lecturer of developmental psychopathology. Dr Anastassiou-Hadjicharalambous has received several research grants from various national and international funding bodies. She has four ongoing research projects and has published six articles in peer-reviewed journals.

Anna Bokszczanin, PhD, is an assistant professor at the Institute of Psychology, Opole University, in Opole, Poland. She has published intensively in the areas of the impact of stress on children's and adolescents' well-being and mental health in the context of stressful life events at both individual and community levels (e.g., natural disaster, unemployment). She is the author of the books *Spoleczne i psychiczne reakcje dzieci i mlodziezy na powódz 1997 roku* (Social and

Psychological Response of Children and Adolescents to the 1997 Flood) published by *Warszawa: Wydawnictwo Instytutu Psychologii PAN* (2003); and *Social Change in Solidarity: Community Psychology Perspectives and Approaches* (2007). She is the editor of *Opole: Wydawnictwo Uniwersytetu Opolskiego*.

Diane Bray, PhD, is a principal lecturer and currently head of psychology at Roehampton University in London, England. She obtained her undergraduate degree from Bradford University and doctorate from the North East London Polytechnic (now University of East London). Before joining Roehampton, she held a number of teaching posts and was a research officer at the Centre for Criminological Research (Oxford). Her research interests include risk perception, individual differences, and the relationship between cognition and emotion.

David T. Courtwright, PhD, received his undergraduate degree at the University of Kansas in Kansas City, Kansas, United States and his graduate training in history at Rice University in Houston, Texas, United States. He is the author of *Addicts Who Survived* (1989), *Dark Paradise: A History of Opiate Addiction in America* (rev. ed., 2001), and *Forces of Habit: Drugs and the Making of the Modern World* (2001). He is currently presidential professor at the University of North Florida.

Herbert C. Covey, PhD, is deputy director of Adams County Social Services Department and instructor at the College of Continuing Education, University of Colorado at Boulder in Boulder, Colorado, United States, and has taught juvenile delinquency for over two decades. Dr. Covey received his PhD in sociology from the University of Colorado at Boulder. He has published several articles on a wide array of subjects, including community corrections, disabilities, religion, street gangs, crime measurement, boomtown crime, research methods, determinant sentencing, violent mentally ill offenders, and social gerontology, among other topics. His current research involves substance abuse treatment and the dietary practices of enslaved African Americans. He has also served as an academic journal reviewer for *Criminal Justice Review, Journal of Contemporary Criminal Justice, The Gerontologist, Journal of Applied Sociology,* and *Journal of Crime and Justice.* He has encountered many families and youth who have been involved with meth use and manufacture. He has authored or coauthored nine books and numerous academic articles. He is the editor of *The Meth Crisis* (Praeger, 2006), to which he contributed three chapters. He is a coauthor of the text *Youth Gangs* (2006) and sole author of *African American Slave Medicine: Herbal and Non-Herbal Treatments* (2007). He coordinated four major symposia on methamphetamine for judges, caseworkers, public health officers, law enforcement officers, child protection caseworkers, public administrators, ex-users, and other stakeholders.

Ann N. Dapice, PhD, is director of education and research of T. K. Wolf Inc., a nonprofit American Indian organization in Tulsa, Oklahoma, United States. With PhDs in psychology, sociology, and philosophy from the University of Pennsylvania, she has served as professor and administrator at a number of universities, including the University of Pennsylvania, Widener University, Penn State University, and Goddard College, teaching social sciences and Native American studies. Her cross-cultural research has been reported in professional journals, books, and academic presentations regionally, nationally, and internationally. Speaking engagements include topics related to addiction, stalking, and American Indian issues. She has served on the Elders Council for the American Indian Chamber of Commerce and is a member of the boards of directors of the Mental Health Association in Tulsa and the Tulsa Metropolitan Ministry/Domestic Violence Intervention Services Committee. She consults with the University of Pennsylvania on Native American issues, where she is founder and chair of the Association of Native Alumni.

Cecilia A. Essau, PhD, MA, HBA, obtained her undergraduate and master's degrees in psychology from Lakehead University (Canada), her PhD from the University of Konstanz (Germany), and her postdoctoral degree (professorial qualification in Germany) from the University of Bremen (Germany). She held numerous academic positions in Canadian, Austrian, and German universities before joining Roehampton University in London, England, in 2004 as a professor of developmental psychopathology. With research grants from numerous national and international institutions, her research has focused on understanding the interacting factors that can lead children and adolescents to have serious emotional and behavioral problems, and she has used this research to (1) enhance the assessment of childhood and adolescent psychopathology and (2) design more effective interventions to prevent and treat such problems. She has published more than 150 articles in peer-reviewed journals or edited volumes, and 12 books.

Elaine R. Feeney, PhD, RN, observed that the use of drugs of abuse, including alcohol, caused or exacerbated the health problems for which many of her clients (patients) received medical and nursing care. This awareness spurred her interest in the relationship between drugs of abuse and health status and services. Her doctoral dissertation, "Health Services Utilization by Addicted Women before and after the Initiation of Substance Dependence Treatment," clarified her thinking and guided her view of addictive disease as a public health problem. During her career, she has received numerous awards for scholarship, leadership, advocacy, and teaching. Dr. Feeney has practiced her profession in the role of clinician, manager, researcher, teacher, and consultant to nursing specialty organizations. She is currently serving as the president of the Foundation for Addictions Nursing in Baltimore, Maryland, United States.

George Georgiou, PhD, obtained his BS (honors) and PhD from Goldsmiths College, University of London, England. His PhD focused on the role of clinical and nonclinical anxiety in modulating attention, with particular reference to conscious versus nonconscious processing. After completing his PhD, he worked at a medical health center, where he was actively involved in the treatment of patients diagnosed with various anxiety disorders. He then took up a postdoctoral position at the University of Essex before spending four years as an assistant professor at the University of Nicosia, in Cyprus. George is currently a lecturer at Roehampton University. His current main research interest lies in the information processing biases in affective disorders and implications for therapy, and the role of attentional control in anxiety. He has published several articles in peer-reviewed journals in this area and has recently received two European Union grants primarily aimed at tackling binge drinking and alcohol-related road traffic accidents in Cyprus and the United Kingdom.

Thomas K. Greenfield, PhD, is a senior scientist and the scientific director of the Alcohol Research Group (ARG) in Emeryville, California, United States. He also serves as an adjunct clinical faculty member in the University of California (UC) San Francisco's Department of Psychiatry in San Francisco, California, United States. His research interests include the epidemiology of alcohol use and problems, alcohol policy studies, consumer satisfaction, national alcohol surveys and consumption measurement, drinking patterns and mortality, and services research. His responsibilities include overseeing ARG's national alcohol surveys, which are sponsored by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) and conducted every five years. He has served as vice president and secretary of the Kettil Bruun Society for Social and Epidemiological Study of Alcohol and currently serves on the Extramural Advisory Board of the NIAAA. He grew up in Zimbabwe and lives in Oakland with his wife, a professor at UC, Berkeley's Haas School of Business; they have an adult son.

Julia F. Hastings, PhD, MSW, is assistant professor in the School of Social Welfare at the University of California Berkeley in Berkeley, California. Globally, her research interests cover welfare policy, depression, and health disparities among African Americans. Her research projects focus on the interrelationships between race, mental health outcomes, health conditions, risk and protective factors, and poverty. Furthermore, she conducts research studies on methodological issues associated with understanding how researchers can better diagnose depression among African Americans. She has published on culturally competent research methods within African American communities, welfare participation, depression, and body weight. She received her MSW from the University of Washington and her PhD from the University of California, Los Angeles. Her

postdoctoral training occurred at the University of Michigan in the Program for Research on Black Americans at the Institute for Social Research; the School of Public Policy; and the School of Social Work. She also completed a certification in epidemiology from the Department of Epidemiology at the University of Michigan's School of Public Health.

Sean R. Hogan, PhD, MSW, is assistant professor in the Department of Social Work at California State University Fullerton in Fullerton, California, United States. In 2005, he received his doctorate from the School of Social Welfare at the University of California, Berkeley. Dr. Hogan's teaching interests are in the areas of substance abuse and social welfare policy. He also has an interest in the effectiveness of service learning as a pedagogical method for teaching substance abuse education to social welfare students. Dr. Hogan's research focuses on the effects of welfare reform legislation on marginalized populations, with a specific interest in the effects of eliminating welfare benefits to low-income substance abusers. He also has an interest in the history and construction of substance abuse policy in the United States and its relationship to social inequality.

Mary F. Holley, MD, trained at Baylor College of Medicine in Houston, Texas, United States. She is a licensed physician and a former obstetrician-gynecologist in northern Alabama. She is founder and director of Mothers against Methamphetamine, a national nonprofit drug education organization. Dr. Holley is author of *Crystal Meth: They Call It Ice* and has produced several educational videos for drug education and prevention. She serves on the Alabama Attorney General's Methamphetamine Task Force as chairperson of the Education Committee. Her Web site can be seen at http://www.mamasite.net.

Herbert D. Kleber, MD, is professor of psychiatry and director of the Division on Substance Abuse at the Columbia University College of Physicians and Surgeons and the New York State Psychiatric Institute in New York, United States. Before founding the Division in 1991, he served for two and a half years as the deputy director for Demand Reduction at the Office of National Drug Control Policy in the White House under President George H. W. Bush and director William Bennett. Prior to that, Dr. Kleber was professor of psychiatry at Yale University School of Medicine (1966–1989) where he founded the Substance Abuse Treatment Unit. He currently oversees research on new medications to treat cocaine, heroin, prescription opioids, and marijuana problems. Dr. Kleber is the author or co-author of more than 250 papers, chapters, and books dealing with all aspects of substance abuse, and the co-editor of the *American Psychiatric Press Textbook of Substance Abuse Treatment*. He has received numerous awards, is listed as one of the "Best Doctors in America,"

and was elected in 1996 to be a member of the Institute of Medicine of the National Academy of Science.

Rev. Linda Mercadante, PhD, Straker Professor of theology at the Methodist Theological School in Delaware, Ohio, United States, helps train ministers who understand addiction issues and licensed counselors with a focus on spirituality. She specializes in theology and culture, exploring the theological implications of such issues as addiction, victimization, gender imagery for God, human dysfunction, reproductive loss, film, and memoir. Author of five books and nearly 50 articles, her works include *Victims and Sinners: Spiritual Roots of Addiction and Recovery* (1996), *Bloomfield Avenue: A Jewish- Catholic Jersey Girl's Spiritual Journey* (2006), and *Gender, Doctrine and God: The Shakers and Contemporary Theology* (1990). She lectures internationally on addiction and victimization to therapists, researchers, and recovering persons. She also gives workshops and retreats on these topics as well as on spiritual memoir, faith and film, and other issues in theology and culture.

See Ching Mey, PhD, MEd, is a professor in the School of Educational Studies, Universiti Sains Malaysia in Pulau, Pinang, Malaysia. Her areas of specialization are educational psychology, counseling psychology, counselor education, special education (emphasis on autism), psychological testing, and mental health. She is a registered and licensed counselor. Ching Mey conducts seminars and workshops and is consulted on topics related to motivation, mental health, strategies and techniques in counseling, psychological testing, stress management, grief and bereavement, counseling special populations, screening and diagnosis of special children, teaching and learning of special children, behavior management, and parenting skills. She has published five academic books and about 200 international and national academic journal articles. She has received 16 research grants. In recent years, she has been invited to give keynote addresses, plenary presentations, talks, and workshops by international organizations such as PACERS (Philippines), A WIFI (Philippines), University of San Carlos (Philippines), SAARC (India), Department of Mental Health (Thailand), University of Surabaya (Indonesia), Seoul National University (Korea), iT21-International (Singapore), Charles University (United Kingdom), California State University (United State), Asia Pacific Association of Psychotherapists, and APECA. She has received several awards recognizing her contributions, including several Outstanding Leadership and Service Awards from the Lions Clubs International, the Pingat Jasa Kebaktian (Meritorious Service Award) from the Penang State Government, the Maal Hijrah Excellence Award (for Academic Category) from the University Science Malaysia, and the Sin Chew

Da Ai Award (Sin Chew Kind Heart Award) and Alumni Citizenship Award 2007 from the Ohio State University Alumni Association.

Kathryn Meyer, PhD, is associate professor in the Wright State University Department of History in Dayton, Ohio, United States. Her areas of expertise include Asian narcotics trafficking, Asian crime, and Chinese and Japanese history. She authored the book, *Webs of Smoke: Smugglers, Warlords, Spies and the History of the International Narcotics Traffic*. (1998).She earned her doctorate at Temple University in Philadelphia, Pennsylvania, United States.

Changiz Mohiyeddini, MD, PhD, obtained his undergraduate, MA, and PhD degrees in psychology from Trier University (Germany) and his postdoctoral degree (professorial qualification in Germany) from the University of Tuebingen (Germany). He held numerous academic positions at Luxembourgian, German, and Swiss universities before joining Roehampton University, London, England, in 2006 as a reader in psychology. His research has focused on understanding the process of emotion regulation that can lead children, adolescents, and adults to have serious emotional and behavioral problems (e.g., depression, anxiety, alcohol and drug abuse, rumination).

Jean O'Callaghan, PhD, is a principal lecturer at Roehampton University, London, England, where she coordinates an undergraduate degree program in psychology and counseling. She is an integrative psychotherapist and an academic psychologist with research interests in self-regulation and the psychology of disability. Her therapeutic expertise integrates diverse perspectives of clients' meaning making that variously employ cognitive behavioral and narrative poststructuralist therapeutic techniques to empower clients to reframe a presenting problem such as alcohol addiction.

Ramandeep Pattanayak, MD, is currently working as a senior resident in the Department of Psychiatry, All India Institute of Medical Sciences (AIIMS) in New Delhi, Delhi, India. She obtained her postgraduate degree in psychiatry from the same institute in 2005. During her residency, she has also been trained at the National Drug Dependence Treatment Centre, Department of Psychiatry, AIIMS, and has gained sufficient clinical and research exposure in the field of drug dependence. She has also gained experience as a research officer in a phase 3 drug trial in the Department of Psychiatry, AIIMS. She was awarded a medal in community medicine during her graduation.

Regina Pauli, BSc, PhD, obtained her undergraduate degree in psychology from Royal Holloway, London, and her PhD from the University of Surrey. She has been a lecturer at Roehampton University in London, England, since 1995 and is presently principal lecturer in the School of Human and Life Sciences

there. She has research interests in teamwork, especially in higher education; evaluation of teaching quality; medical image interpretation; and emotion and cognition.

Martha Romero Mendoza, PhD, received her bachelor's degree in clinical psychology at the Universidad Iberoamericana in Mexico City, Mexico, her master's degree in clinical psychology at the National Autonomous University of Mexico, and her doctorate in anthropology at the National Autonomous University of Mexico. Owing to her outstanding studies, she was distinguished with the Medal Alfonso Caso. Since 1985, Romera Mendoza has worked as a researcher of medical sciences and professor at the National Institute of Psychiatry in Mexico City, Mexico, and as professor of qualitative methodology at the Medical School at the National Autonomous University of Mexico. Owing to her interest in gender-related problems as part of the International Research Group on Gender and Alcohol of the Kettil Brunn Society, she has been an advisor of the Word Health Organization in Geneva, Switzerland. Romero Mendoza has participated in different multinational research projects and congresses coordinated by the World Health Organization and the Pan-American Health Organization dealing with addiction problems. She has published more than 70 research articles, 15 book chapters, and 3 research books. Romero Mendoza is presently developing a harm reduction program designed for women who abuse alcohol and drugs and who are situated in high-risk environments. Her main interests are in addictions, gender issues, violence, comorbidity problems, and qualitative research.

Clete Snell, PhD, is assistant professor of criminal justice. He received his PhD in 1999 from Sam Houston State University, in Houston, Texas, United States. Dr. Snell teaches in the master of science in juvenile justice online degree program and is currently chair of the PVAMU Distance Learning Council. His research interests include juvenile delinquency theory and juvenile justice policy. He has an ongoing grant with the Texas Department of Health to evaluate the enforcement of Texas tobacco laws. He recently published a book titled *Neighborhood Structure, Crime, and Fear of Crime: Testing Bursik and Grasmick's Neighborhood Control Theory*. He is currently writing another book titled *Big Tobacco and Youth Smoking*.

Va Nee L. Van Vleck, PhD, received her doctorate in economics from the University of Iowa, in Iowa City, Iowa, United States; she also holds a MA in economics from Columbia University in New York City, United States. Her present research emphases are in applied microeconomics, focusing on issues of labor, health, and the health care sector. She was a postdoctoral fellow in Public Health and Alcohol Studies, funded by the National Institute

on Alcohol Abuse and Alcoholism, at the Prevention Research Center and the Alcohol Research Group in Berkeley, California. During the fellowship, Dr. Van Vleck was mentored in the Drug and Alcohol Research Team in the Division of Research (northern California), Kaiser Permanente, in Oakland. Dr. Van Vleck's most recent research has focused on the adverse effects of alcohol abuse on individuals' incomes and likelihood of employment, positive third-party incentives to combat drunk driving, and the impact of uncompensated care on the financial status of San Joaquin Valley hospitals. Having taught in a wide variety of undergraduate settings, she is now an assistant professor at California State University, Fresno.

Meera Vaswani, PhD, earned her doctorate from Delhi University, a leading university in India. She is a member of the All India Institute of Medical Sciences, a prestigious and internationally recognized institute in India, where she was first a postdoctoral fellow, subsequently became a lecturer, and rose to be a professor in the same institute. She is now at the National Drug Dependence Treatment Centre, department of psychiatry, and at the All India Institute of Medical Sciences, in New Delhi, Delhi, India. During this time, she was elected as a fellow of the Royal Society, London, and as a member of the National Academy of Medical Sciences, India. She was selected for a United Nations fellowship, for which she worked at the University of Glasgow in the United Kingdom. She was one of the three scientists in the world selected for the Distinguished International Scientist Collaborative Award from the National Institute on Drug Abuse, National Institutes of Health, in 2007. She has completed several projects on addiction (alcohol and heroin addiction) and published many papers in both national and international journals. She has been a guest speaker at several national and international forums. She was the only delegate to be invited by Japan to represent India in the Asia Pacific Society of Biological Research in Alcoholism. Subsequently, she was elected to the board of directors of the Asia Pacific Society for Alcohol and Addiction Research.

The Praeger International Collection on Addictions

Recent Titles in
Abnormal Psychology

Mental Disorders of the New Millennium, Volumes 1–3
Thomas G. Plante, editor

Bleeding to Ease the Pain: Cutting, Self-Injury, and the Adolescent
Lori G. Plante

Understanding and Treating Depression: Ways to Find Hope and Help
Rudy Nydegger

THE PRAEGER INTERNATIONAL COLLECTION ON ADDICTIONS

Volume 2
Psychobiological Profiles

Edited by Angela Browne-Miller

Praeger Perspectives

Abnormal Psychology

Thomas G. Plante, Series Editor

PRAEGER

Westport, Connecticut
London

Library of Congress Cataloging-in-Publication Data

The Praeger international collection on addictions / edited by Angela Browne-Miller.
v. cm. — (Abnormal psychology, ISSN 1554–2238)
Includes index.
Contents: v. 1. Faces of addiction, then and now—v. 2. Psychobiological profiles—v. 3. Characteristics and treatment perspectives—v. 4. Behavioral addictions from concept to compulsion.
ISBN 978–0–275–99605–5 (set : alk. paper)—ISBN 978–0–275–99607–9 (v.1 : alk. paper)—ISBN 978–0–275–99609–3 (v.2 : alk. paper)—ISBN 978–0–275–99611–6 (v.3 : alk. paper)—ISBN 978–0–275–99613–0 (v.4 : alk. paper) 1. Substance abuse. 2. Alcoholism. 3. Compulsive behavior. 4. Addicts. I. Browne Miller, Angela, 1952–
HV4998.P72 2009
616.86—dc22 2008051468

British Library Cataloguing in Publication Data is available.

Library of Congress Catalog Card Number: 2008051468
ISBN: 978–0–275–99605–5 (set)
978–0–275–99607–9 (Vol. 1)
978–0–275–99609–3 (Vol. 2)
978–0–275–99611–6 (Vol. 3)
978–0–275–99613–0 (Vol. 4)
ISSN: 1554–2238

First published in 2009

Praeger Publishers, 88 Post Road West, Westport, CT 06881
An imprint of Greenwood Publishing Group, Inc.
www.praeger.com

Printed in the United States of America

The paper used in this book complies with the Permanent Paper Standard issued by the National Information Standards Organization (Z39.48–1984).

10 9 8 7 6 5 4 3 2 1

Contents

PART II: BODIES

PART III: PSYCHOBIOLOGIES

This page intentionally left blank

Series Foreword

Tragically, most people across the globe have either struggled with a health- and relationship-damaging addiction or know someone who has. Addictions, broadly defined, have touched the lives of the majority of people in multiple cultures and locations. For centuries, numerous people have suffered with their addictions to alcohol and drugs as well as with other addictions, with often devastating outcomes. Sadly, important relationships, jobs and careers, and many lives have been lost due to the destructive power of addiction. These tragedies not only occur for those who suffer from addiction, but for their loved ones, coworkers, and community members, and for innocent victims who are perhaps in the wrong place at the wrong time when an addiction-related accident, crime, or violence occurs. The enormous cost of addiction in health care, traffic accidents, crime, violence, loss of workplace productivity, and broken families is too large to quantify. The global spread and success of organizations such as Alcoholics Anonymous (as well as related organizations such as Narcotics Anonymous, Sexoholics Anonymous, and Overeaters Anonymous) is a testament to the numerous people trying to recover from their addictions. Sadly, for every person seeking help for his addiction problem, there are likely to be many more people who never do. Clearly we need help to better understand, evaluate, treat, and cope with those who suffer from addictions.

In this remarkable four-volume set, *The Praeger International Collection on Addictions,* Angela Browne-Miller, PhD, DSW, MPH, has assembled an all-star and diverse team of leading experts from across the globe to provide a state-of-the-art understanding of the various facets of addiction. Each chapter

is written in a manner that is suitable for professionals working in the field as well as educated lay readers and those who either struggle with addictions or live or work with someone who does. What is especially remarkable about the four-volume set is its emphasis on addiction from around the globe, examining multicultural and international issues in addiction, as well as its coverage of so many multifaceted aspects of diverse addictions. For example, it certainly makes sense to cover fully addiction topics such as alcohol abuse and illegal drug use of, say, cocaine and heroin, yet chapters are also offered that examine addictions to caffeine, Internet pornography, work, television, intimate relationship abuse, and shopping. The chapters highlight biological, psychological, social, spiritual, and public health perspectives, with chapter authors who are psychologists, psychiatrists, other physicians, nurses, social workers, counselors, clergy, and other professionals. Dr. Browne-Miller is uniquely qualified to assemble this project as she is someone who has worked in the field of addiction for many years and has training in a unique blend of both the policy and the clinical sides of psychology, social work, education and public health.

The set is complete, state of the art, and highly informative and engaging. There is something for everyone interested in the field of addiction for professional or personal reasons. It is hoped that professionals and lay readers will greatly benefit from this important work and, in doing so, will find a way to improve the lives of those touched by addiction. It is my hope that both research and practice in the field of addiction will be greatly improved thanks to this set. The lives of those who either struggle with addiction or live with those who do will ultimately be improved thanks in part to this critical series. I am grateful to Dr. Browne-Miller and her assembled contributors for providing us all with such important and high-quality volumes that are now available to the public and professional communities. If only one life is saved or improved thanks to this set, it will be a great success in my view; yet I expect that many lives will ultimately be saved or greatly improved thanks to *The Praeger International Collection on Addictions*.

Thomas G. Plante, PhD, ABPP
Santa Clara University and Stanford University School of Medicine,
Praeger Series Editor, Abnormal Psychology

Preface

Angela Browne-Miller, PhD, DSW, MPH

Welcome to *The Praeger International Collection on Addictions,* addressing the insidious, pervasive, worldwide problem of human addiction. Addiction is clearly a global issue, touching every population, every nation, and every age group, people from all walks of life everywhere, directly or indirectly. Indeed, we are talking about an affliction of epic and epidemic proportions. We cannot look away. This is the health of the human species we are talking about.

The World Health Organization (WHO, 2008, p. 1) reports that "psychoactive substance use poses a significant threat to the health, social and economic fabric of families, communities and nations. The extent of worldwide psychoactive substance use is estimated at 2 billion alcohol users, 1.3 billion smokers and 185 million drug users" (p. 1). The WHO has estimated there to be at least 76.3 million persons with alcohol use disorders worldwide, and at least 15.3 million persons with drug use disorders worldwide. Alcohol use and abuse as well as the use and abuse of other psychoactive substances contributes to substantial individual and public health costs. Alcohol is but one substance playing a major role in this global addiction epidemic, but clearly there are many others, despite efforts to prevent new addictions and addicts, and to contain world drug markets (UN, 2008, p. 7).

For example, cocaine shares the stage with other abused drugs. Its prevalence is estimated to be up to 3 percent of the population in developing countries, with severe medical, psychological, social, and economic consequences including, but not limited to, the spread of infectious diseases (e.g., AIDS, hepatitis, and tuberculosis), plus crime, violence, and neonatal drug exposure.

Amphetamine-type stimulant (ATP) abuse is more widespread than cocaine abuse in at least 20 countries. Methamphetamine is presumed to lead in ATP addiction rates, with massive meth epidemics affecting several whole countries and entire regions of others. Social and public health costs of methamphetamine production and use via smoking, sniffing, inhaling, and injecting are staggering and growing in many regions. Additionally, there has been a global increase in the production, transportation, and use of opioids, especially heroin, with worldwide heroin production doubling or even tripling since the mid-1980s. Global estimates are that 13.5 million persons consume opioids, with 9.5 million of these being heroin users who face health risks including hepatitis, HIV, and death. And cocaine, meth, and heroin are just one piece of the picture.

The hotly debated drug cannabis—or the *Cannabis* family of drugs with the euphoric tetrahydrocannabinols, or THCs, including marijuana and hashish preparations—is said to be the most widely abused drug. Research is now suggesting the risk for acute health effects of long-term, chronic cannabis use, including potential impairment of cognitive development, learning, memory, recall, attention, and coordination. (Certainly the presence and extent of long-term effects of casual, of regular and of chronic use are as yet not entirely ascertained.) Both casual use of marijuana and medical use of forms of what is termed medical marijuana (e.g., dronabinol sold as Marinol, the cannabidiols, or CBDs) are subsets of all forms of cannabis use. There are legitimate therapeutic uses of this substance, and these uses make it all the more difficult to regulate marijuana drugs fairly and effectively.

We have here, and in the use of any psychoactive medication for therapeutic purposes, a gray area in which illicit and licit use overlap and can confuse many adult and youth consumers, researchers, and policy makers, among others. In the emergence (or reemergence in history, some will argue) of cannabis as medicine, we have a model for asking which, if any, abused substances may, and perhaps should, be repurposed for medicinal or treatment purposes, and how this is best done against the backdrop of the global addiction epidemic.

Regarding marijuana, we are confronted with the age-at-first-use issue, which suggests that early onset of regular cannabis use may affect not only the academic and social performance of children and teens, but also their future susceptibility to addictions. It was in the 1960s that the hotly debated label "gateway drug" was applied to marijuana, perhaps to scare off its use, and only in the decades since have we understood better what this might actually mean to us. It may not be that marijuana provides the training wheels for drug addiction, but rather that it may serve as an indicator of future use of the same or other drugs. Of course, today, with so many young people having access, and taking advantage of their access, to the whole range of psychoactive substances,

the question of which drug might be a gateway to which other drug dissolves into the fury of the countless addiction conundrums of our constantly changing times.

There is always a new, or rediscovery of an old, addiction on the horizon. There is also always a new (or rediscovered) psychoactive substance for exploratory, research, and perhaps even treatment purposes emerging (or re-emerging). Labeling all of these substances as addictive right out the gate may or may not serve science or even humanity itself. How can we be certain the approach we take will be a constructive one? With new legal (where licensed for development and experimentation) and illegal (where not being utilized under protection of law) so-called designer drugs emerging at a staggering rate, we must admit that we cannot know what is coming, nor whether the new compound will be addictive, or popular, or of medicinal value, or even accessible. We can only imagine what the brave new world of chemistry will continue to bring and whether any benefits can be made available without accompanying risks and detriments.

Moreover, the desire to explore and achieve various altered states of consciousness in religious, spiritual, ritual, and perhaps even treatment settings, is unfolding into debates about rights (Browne-Miller, 1989, pp. 258–260). When there is no demonstrated risk to self or others, we have to ask ourselves whether this right should be protected, especially in circumstances of traditional uses for religious purposes. Again, this dilemma arises against the backdrop of the global and runaway epidemic of substance addiction. How do we balance pressures from opposite directions (freedom protecting the right to use versus control to stop injury and costs of using), when these pressures are not balancing themselves?

Also against the backdrop of global addiction levels, is the massive level of addiction to legal drugs, many of which are heavily marketed to consumers. The legal drug tobacco is said to be the substance causing the most damage globally, with at least one-third of the global population smoking. While smoking rates may be dropping in some countries, the reverse is true globally. As just one of its effects, smoking accounts for some 90 percent of all lung cancer in men and 70 percent of all lung cancer in women. And yet tobacco use is overwhelmingly viewed as being "the single most avoidable cause of disease, disability and death" in the United States (CDC, 2008, p. 2).

And perhaps nothing here has touched so many lives as the regularly consumed, legal drug caffeine, perhaps because coffee drinking is considered so very normal and acceptable, even necessary, in everyday life. However, we must ask whether there is a level of caffeine use that is abuse—or perhaps self-abuse. Surely we do not want to throw caffeine use onto this list of substance abuses

and addictions. Still, a collection on addiction would not be complete without at least touching on this matter, and therefore we do address caffeine herein.

And then there are also the addictions to prescription drugs (such as Vicodin, Percocet, OxyContin, and Darvon), which we find increasing rapidly and already a worldwide phenomenon, with the most commonly abused prescription drugs being opiates. The U.S. National Institute of Mental Health characterizes prescription drug addiction as the second most common illegal use of drugs in the United States, second only to marijuana.

We must also note that unusual, virtually invisible psychoactive substances are working their way into our everyday lives. Household and workplace products contain many volatile substances, exposure to which can be not only damaging, but also intoxicating, and perhaps addicting. Although this domain of substance use and abuse is not specifically addressed herein, we must acknowledge the severe and perhaps largely unmeasured effects of this domain of even routine, legal substance use as well as unintentional and intentional abuse.

So as not to exclude nonsubstance addictions in this overview of addiction today, the fourth volume in this collection on addiction reminds us that work, television, shopping, food (with its particularly difficult-to-call-addiction nature), intimate partner relationship, gambling, Internet, and even pornography addictions make their marks in our lives, either indirectly or directly. These behavioral, nondrug addictions, which occur alone and co-occur with each other, also do co-occur with substance uses, abuses, and addictions. Every human being is in some way affected by the prevalence of behavioral addictions, either directly or indirectly. The study of behavioral addictions teaches us a great deal about addiction itself.

All this suggests the picture of an addiction-prone and largely chemically dependent human species. And this is just the tip of the iceberg. With this truly incomplete laundry list of human fallibilities—or better stated, perhaps, human *vulnerabilities*—this four-volume collection on addiction is truly that: a collection of perspectives, approaches, and findings. Each chapter is a snapshot of the work and thinking taking place in many fields of addiction. Contributors to this collection work with addiction on the various social, philosophical, psychological, spiritual, policy, political, economic, biological, and even cellular levels, all places where this thing we call "addiction" lives. Certainly this collection would have to comprise hundreds of volumes, rather than the four that it does, to address addiction in all its iterations.

Here we give voice to a diverse cross section of perspectives on addiction. This is in no way an exhaustive cross section (of either perspectives or addictions); rather, this collection suggests the diversity of perspectives, theories, practices, and types of addiction in the field—or better stated, *fields*—of addiction. The four volumes of this collection represent the voices of those who have

graciously and even bravely stepped forward from their numerous countries and arenas of work to contribute their ideas, research, and experiences. Certainly there are many others out there, many other aspects of addiction, and many other drugs and objects of addiction not addressed in these volumes.

This work is divided into four volumes, with the first three addressing addictions to substances and the fourth addressing behaviors that show characteristics of addiction. Volume 1, *Faces of Addiction, Then and Now,* offers a sampling of the depth and breadth of addiction today and in the past; volume 2, *Psychobiological Profiles,* surveys some of the interlinked psychological and biological aspects of addiction; volume 3, *Characteristics and Treatment Perspectives,* samples the range of addiction treatment perspectives and approaches; and volume 4, *Behavioral Addictions from Concept to Compulsion,* gives the reader a glimpse of behavioral addictions other than substance addictions.

Readers will observe that the content of these volumes is indeed diverse and in no way represents any one view or theory of addiction. There are many other voices out there who must also be heard, and only in the interest of time and space are we stopping here, at these volumes. The content of these volumes in no way expresses the opinion of this editor, nor of this publisher, regarding what is right, best proven, or even most en vogue in the addiction world; rather, this *International Collection on Addictions* seeks a display of, a sampling of, the diversity of effort to quell the detrimental effects of addiction on individuals, families, communities, societies, economies, and international relations; on ecologies; and in fact, on the human population of planet Earth.

REFERENCES

Browne-Miller, Angela. (2008, reprint of 1989). Synaptic rights. In *Working dazed: Why drugs pervade the workplace and what can be done about it* (pp. 258–260). Houston, Texas: Questia Media. Also available at http://www.questia.com/library/book/working-dazed-why-drugs-pervade-the-workplace-and-what-can-be-done-about-it-by-angela-browne-miller.jsp

Centers for Disease Control (CDC). (2008). *Targeting tobacco use: The nation's leading cause of preventable death* (March 2008 revision). Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

United Nations (UN). (2008). 2008 world drug report. UNODC (United Nations Office on Drug Control), Vienna, Austria: Vienna International Centre, Full report in English available at http://www.unodc.org/documents/wdr/WDR_2008/WDR_2008_eng_web.pdf

World Health Organization, Management of Substance Abuse, Facts and Figures. (2008). "The Global Burden," 2008, (Accessed 9-01-08), p. 1 http://www.who.int/substance_abuse/facts/global_burden/en/index.html. **See Also:** http://www.who.int/substance_abuse/facts/en/index.html

This page intentionally left blank

Introduction to Volume 2: Where Psychology and Biology Meet

Angela Browne-Miller, PhD, DSW, MPH

This is Volume 2 of *The Praeger International Collection on Addictions: Psychobiological Profiles*. Here we sample the array of understandings of addiction as we know it today. The intent is to share a sampling from a wide range of material, not to hone in on any one drug addiction, or any one mechanism of addiction, but rather to establish the multilevel (social, psychological, biological) omnipresence of addiction, its reality and dangers. As our scientific understanding of the psychological, neuropsychological, molecular, genetic, and related, even inextricably linked, bases of addictions and interacting conditions is rapidly increasing, there is hope that augmented and new approaches to addiction can turn the tide of this mounting global condition.

We begin "Part I: Issues" with chapter 1, "Addiction and Health Promotion," by Kathleen Bradbury-Golas, DNP, RN, APN, of the Richard Stockton College of New Jersey, in Pomona, New Jersey, United States, and also a nurse in private practice in New Jersey. Bradbury-Golas reminds us that substance abuse is harmful both directly and via the negative lifestyle behaviors that accompany it. Relapse prevention therapy and other treatments surely can decrease the conditions which would invite relapse. Primary care practitioners must recognize what is involved here, and collaborate with addiction specialists to achieve positive health outcomes. Clearly, collaborating or at least coordinating to treat and curb addiction is going to be more effective than working in isolation. This collaboration flows in both directions. Bradbury-Golas also notes that what is most hopeful in the primary prevention of addiction—treating it before it occurs—is the addiction vaccine. According to the U.S. National Institute on

Drug Addiction (NIDA), this vaccine is likely to be available within from one to 10 years. One vaccine in this genre is TA-CD, which prevents the cocaine metabolite from ever reaching the user's brain. Like any other vaccine, TA-CD causes the immune system to create antibodies that attach to cocaine molecules, which in turn are broken down by enzymes found in the bloodstream, with the result that both the desire for and the "pleasure" of cocaine use is prevented.

Next, in chapter 2, Ted Goldberg, PhD, at the department of Social Work at Stockholm University, Stockholm, Sweden, and also at the department of Caring Sciences and Sociology at the University of Gävle, in Gävle, Sweden, shares his insights into the process of "Becoming a Problematic Consumer of Narcotics." Based on studies in Stockholm, Sweden over a four-and-a-half-year period, Goldberg presents a four-stage life-history model indicating why drug use begins and continues. Goldberg applies a life development or life-history career model to explain what brought his subjects to, and led them to continue, their drug use. Discussion of the "future problematic consumer" of narcotic drugs includes a review of symptoms that precede first consumption of narcotics. Goldberg notes the "destructive life-project" he has seen in his subjects: "The problematic consumers of illicit substances I studied are extremely self-destructive people who lead lives that slope steeply toward extermination." Those self-destruction and self-extermination tendencies that may be present in drug addiction behavior warrant our direct attention. We must ask what these behaviors may be telling us about ourselves and about our species.

Ann N. Dapice, PhD, of T. K. Wolf, Inc., an American Indian-focused addiction treatment program in Tulsa, Oklahoma, United States, contributes chapter 3, "Adaptation and Addiction." Have we outlived the usefulness of once-essential survival traits? Do we therefore walk around with antiquated brains and nervous systems that are not adapting rapidly enough to our changing environments—or are we more than ever in need of these age old basic functions, such as the fight or flight response? Addiction is nothing new, and in a species scenario in which the environment may change more rapidly than we do, addiction, that enduring and self-exacerbating human behavior, persists. Dapice writes: "Food and tobacco are connected to other addictive substances in important evolutionary and physiological ways. Understanding the co-development of mammalian brains and ancient psychotropic plants, as well as the implications of ancient psychotropic substance abuse in altering mammalian brains, helps assess the causes and effects of addiction in today's world." Dapice also points out that some things can be used addictively by some people while not addictively by others.

Scott E. McClure, PhD, at the University of California San Diego, department of Psychiatry, Center for Criminality and Addiction Research, Training,

and Application (CCARTA) in San Diego, California, United States, authors chapter 4, "Co-occurring Trauma and Substance Use Disorders with Criminal Offenders." McClure points out that individuals who have been abused physically and/or sexually, and those who have "criminally involved" families, have increased chances of being incarcerated and of being traumatized. Hence, post-traumatic stress disorder (PTSD) is common among this group. McClure also notes that the majority of persons in substance abuse treatment programs have experienced traumas, and that there is a significant overlap between "criminogenic" risk factors and those for substance abuse, trauma, and incarceration itself. McClure adds that: "Safety and coping should be the primary focus for individuals in corrections-based treatment. This is especially true for individuals with severe forms of PTSD and other co-occurring disorders who may decompensate upon addressing trauma symptoms. After clients have successfully developed skills to regulate emotions and cope with trauma symptoms without substance use, they may progress to processing the trauma through exposure therapy, and eventually shift their focus on reconnecting to a functional life."

Joan Mathews-Larson, PhD, LADC, and Mark K. Mathews, LADC, BCCR, both of the Health Recovery Center in Minneapolis, Minnesota, United States, add another aspect of addiction, in chapter 5, "The Role of Allergies in Addictions and Mental Illness." Mathews-Larson and Mathews bring together the sciences of human ecology (which says, among other things, that human behavior and mental health can be profoundly affected by substances in our environment) and orthomolecular medicine (which concentrates on particular vitamin and mineral molecules present in the human body to treat particular diseases). Mathews-Larson and Mathews say that both human ecology and orthomolecular medicine must be involved in addressing allergies that affect mental health to reduce inflammatory responses destabilizing the brain, and related tendencies toward addiction. Special attention to "delayed onset allergies" is important, as these allergies can produce numerous symptoms and affect every organ in the body. As Mathews-Larson and Mathews explain: "Unlike the quick response . . . immediate-onset allergy, [delayed onset] symptoms come on slowly—from two hours to several days. Ingesting allergy-provoking foods prompts the gradual formation of immune complexes until finally they overload the immune system's ability to clear them out. That's when symptoms are felt. Unfortunately, the time differential makes symptoms difficult to link to the foods that cause them. And because the initial response to allergy-provoking foods is often a pleasurable, endorphin-like effect, it becomes even more difficult to accept that they are a problem. In the end, a pattern, initiated and sustained by these foods, contributes to an addictive progression of disease." Here we have a model for the pathogenesis of alcoholism, proceeding the way delayed onset

allergic reactions proceed: "Whether responding to alcohol, drugs, or allergy foods, allergic/addictive chemistry follows the same course because the body recognizes all these substances as toxins, and turns to the same system—the immune system—to clear them. It's when the toxic load exceeds the immune system's ability to clear it that inflammation begins its destructive process."

"Part II: Bodies" begins with consideration of something that is oft overlooked in discussions of addiction—the all-too-common effects of caffeine, the world's most popular psychoactive drug. Kyle M. Clayton, MS, and Paula Lundberg-Love, PhD, both of the University of Texas at Tyler (UTT), in Tyler, Texas, United States, address caffeine as addictive in chapter 6, "Caffeine: Pharmacology and Effects of the World's Most Popular Drug." In the United States, about 90 percent of all adults consume caffeine on a daily basis. Caffeine withdrawal brings on a range of symptoms, and caffeine use affects health in a number of ways. Caffeine intoxification, something most of us rarely think about in daily life, is indeed possible when the level of 250 mcgs is consumed, and doses of caffeine over 5 to 10 grams can be lethal.

Dirk Hanson, MA, of the blog, Addiction Inbox, and freelance science writer based in Ely, Minnesota, United States, offers another commonly overlooked aspect of addiction in terms of its potential severity and symptoms in chapter 7, "Marijuana Withdrawal: A Survey of Symptoms." With approximately 14 million Americans smoking marijuana regularly, we cannot avoid noting the presence of withdrawal symptoms among many heavy users. In fact, there is growing evidence that there is a "marijuana withdrawal syndrome" characterized by restlessness, anxiety, hostility, depression, sleeplessness, loss of appetite, and a mental state described as "inner unrest." (Additional withdrawal systems are being detected every day.) Clearly, marijuana withdrawal has gone unnoticed or less noticed as withdrawal from other substances is so much more severe, and better documented.

We then move into three chapters that delve into what is considered a "more serious" addiction, methamphetamine addiction. Mary F. Holley, MD, of Mothers Against Methamphetamine in northern Alabama, United States, and the Alabama State Attorney General's Methamphetamine Task Force, shares her look at "Marijuana Interaction with Methamphetamine Addiction" in chapter 8. Holley informs us that marijuana affects the brain in many of the same areas that methamphetamine does. Injury to the hippocampus and frontal lobes arising in heavy marijuana users can be amplified by use of methamphetamine. Holley refers to the endocannabinoid system here, noting that early use of marijuana can affect adolescent neurodevelopment, as can early meth use. Together, marijuana and meth set the stage for severe effects on development.

Herbert C. Covey, PhD, of the Adams County, Colorado, (United States) Social Services Department, and instructor at the College of Continuing Education, University of Colorado at Boulder, Colorado, United States, asks "What Is Methamphetamine and How and Why Is It Used?" in chapter 9. Covey explains that "Meth abusers typically take the drug early in the morning and in two- to four-hour intervals, similar to being on a medication. In contrast, cocaine abusers typically take the drug in the evening and take it over a period of several hours that resembles a recreational-use pattern. They typically continue using until all of the cocaine is gone." Covey reminds us that a certain amount of meth use, and meth addiction, is hidden while taking place right before our eyes: "Some meth addicts use at levels that allow them to maintain jobs, homes, some money, and at least the appearance of being in control."

In chapter 10, Kathryn M. Wells, MD, at the Kempe Center for the Prevention and Treatment of Child Abuse and Neglect and the Health Sciences Center, both at the University of Colorado at Denver, in Denver, Colorado, United States, reports on "The Short- and Long-Term Medical Effects of Methamphetamine on Children and Adults," which are marked and costly to individual lives and to societies, in fact, to entire nations—with global ramifications. She states, "Meth use and abuse in this country [United States] has far-reaching ramifications for not only the user but for society." Wells calls for "continued collaborative efforts" as these "are critical to advancements in understanding the medical effects of meth on the users and children who are exposed."

In a look at the dangers of "vapors," Jace Waguspack, BS, and Paula K. Lundberg-Love, PhD, at the University of Texas at Tyler (UTT), Tyler, Texas, United States, contribute their research in chapter 11, "Vapors May Be Dangerous If Inhaled: An Overview of Inhalants and Their Abuse." Waguspack and Lundberg-Love tell us that the word *inhalant* is used to describe a "heterogeneous group of chemicals that share a common route of administration," reporting that inhalant use is a "serious problem, particularly among young people. The incomplete knowledge of the mechanisms of action for some of these substances, combined with insufficient resources for the treatment of the abusers and the complexity of the issues surrounding inhalant abuse, make it difficult for these individuals to receive the help that they need."

This is followed by chapter 12, "The Effects and Abuse Potential of GHB: A Pervasive 'Club Drug,'" submitted by Bethany L. Waits, BA, at the University of Texas at Tyler (UTT), Tyler, Texas, United States, and Paula K. Lundberg-Love, PhD, also at UTT. Gamma-hydroxybutyrate (GHB) is a widely used drug—in its purest form, a white powdery substance soluble in water, sometimes found in tablet or a capsule form, sometimes injected into the

bloodstream, and distributed, both legally and illegally, as a colorless, odorless, and tasteless solution. Use of GHB has undergone a marked increase since its discovery in the late 1960s. It affects the brain as Waits and Lundberg-Love explain: "Once ingested, GHB acts by depressing or downregulating the activity of the central nervous system (CNS), including the brain and the spinal cord, and its effects are similar to other CNS depressants such as alcohol and benzodiazepines (BZDs)." GHB offers an example of the novel drug model: "Although GHB was initially synthesized for use as an intravenous anesthetic for surgical procedures, it has since been marketed therapeutically as a dietary supplement, an anabolic agent, and a drug for the treatment of narcolepsy." Additionally, GHB has been studied for its relief of withdrawal symptoms, including those from alcohol. Against the backdrop of treatment uses of GHB is the disturbing undercurrent of illegal GHB abuse by adults and teens, and even children.

In chapter 13, "HIV and Addiction from an African Perspective: Making the Link," we have the work of Mary Theresa Webb, PhD, of the Global Outreach for Addiction Leadership and Learning (GOAL) Project based in Aliquippa, Pennsylvania, United States; also of the International Substance Abuse and Addiction Coalition (ISAAC) based in the UK; and of the OPORA Training Center in Moscow, Russia. ("Opora" is the Russian word for "support." Moscow based nongovernmental OPORA works with the Russian government to fight addiction, has trained over 3,000 people in addiction and Twelve-Step programs, and has implemented 60 recovery groups in 31 cities in Russia.) Webb writes this chapter with Donald Omonge, BA, of the Substance Abuse Recovery and HIV/AIDs (SARAH) Network, a Kenyan faith-based organization. Webb and Omonge advocate that it is essential that special programs be suited specifically to the African cultures and customs in order to effectively combat the spread of HIV/AIDS. They state that "implementing model programs specifically suited to African cultures and customs to combat the spread of HIV/AIDS must include training components that cross both disciplines (HIV/AIDS and alcohol and other drugs) [and] . . . must be geared to those within the culture who are most effective as change agents." Webb and Omonge also note that "unless GBV [gender-based violence] and rape of vulnerable women and children is curtailed through establishing shelters and a legal protection system, very little hope exists for curbing the spread of the HIV virus in Kenya as well as in other African countries." Also note that injected heroin was determined to be the major risk factor for the spread of HIV; however, now alcohol consumption has became a greater risk than injecting heroin.

And, finally, closing Part II of this volume, in chapter 14, "Drug Abuse–Related HIV/AIDS Epidemic in India: Situation and Responses," Atul

Ambekar, MD, and Meera Vaswani, PhD, at the National Drug Dependence Treatment Center, Department of Psychiatry, All India Institute of Medical Sciences, in New Delhi, Delhi, India, insist that we take heed of a serious aspect of HIV/AIDS in India: "The first case of HIV was detected in India in 1986. In the past two decades since then, the HIV epidemic continues to grow unabated in India." Yet, there is "still no generalized epidemic of HIV in India at the national level. . . . The national level data should, however, be interpreted with caution. India, being a vast and heterogeneous country, has a heterogeneous HIV situation. It has often been commented that there is not one, but many, simultaneous HIV epidemics currently spreading in India," Ambekar and Vaswani write. Certainly, there are harm-reduction services in India; however, they are not enough: "There is a visible and obvious gap in terms of the number of services available and the requirements of these services. Without a rapid scale-up in the number and the quality of the services, [it will be] very challenging to realize the goal of halting and reversing the HIV epidemic."

"Part III: Psychobiologies" moves deeper into various psychological and biological aspects of addition, beginning with chapter 15, "Alcohol Abuse: Impact on Vital Brain Functions and Societal Implications" by Mary Theresa Webb, PhD. In this chapter, Webb notes that the European Union countries and the United States have sounded the global alarm that we are destroying our brains as well as our bodies with the high level of alcohol use around the world. Webb reports on the damaging effects of alcohol on vital brain functions, and the related profound effects on the societal level. Webb notes that neuroimaging study data reveal an alarming increase in heavy alcohol use, with related risks in older adults of loss of cognitive abilities, increased risk of cardiovascular incidents, and dementia.

Chapter 16, "Addiction and Cognitive Control," contributed by Vicki W. Chanon, PhD, and Charlotte A. Boettiger, PhD, both at the Behavioral Neuroscience Program at the University of North Carolina at Chapel Hill in Chapel Hill, North Carolina, United States, characterizes substance use disorder as a neurobehavioral disorder, influenced by disruptions in the cognitive control functions of the brain. With the advent of cognitive neuroscience tools, we are now in a position to make great strides in understanding the brain mechanisms of addiction. Chanon and Boettiger detail the functions of cognitive control and explain that "addicts disproportionately direct their attentional resources toward stimuli associated with their abused substance," referring to "abnormalit[ies] in attentional allocation to drug cues in addicts." Further research along these lines can lead to new treatments, and effectiveness in these treatments, of substance abuse disorders. We have just begun to understand the effect of cognitive regulation on addiction etiologies.

Next, Giuseppe Carrà, MD, MSc, PhD, Consultant Psychiatrist and Research Fellow at the University College London, in London, England, and Sonia Johnson, MSc, MRCPsych, DM, Clinical Lecturer at the University College London, share their work on "Schizophrenia and Substance Misuse" in chapter 17. Comorbid substance abuse problems and schizophrenia or other severe psychoses are associated with poor treatment outcomes. The numbers of those affected by this dual condition are significant: U.S. data show that it may be as much as half of persons with schizophrenia who have co-occurring substance abuse disorders. There is a significant overlap between mental illness and substance abuse, one that insists we do not forget it lest we miss a major subpopulation of persons who are addicted.

Mary F. Holley, MD, of northern Alabama, United States, returns in chapter 18, with her look at the "Neural Basis for Methamphetamine Addiction—Rethinking the Definition of Dependence." As indicated by the title, Holley moves for the rethinking of the very definition of dependence. Motivated by her great concern regarding the high usage of methamphetamine and the severe consequences of this, Holley reminds us that low availability of treatment for meth addiction places the burden on the corrections system. Holley writes, "addiction is a brain disease and not merely a moral failure." Rethinking addiction to fully incorporate this perspective, this reality in fact, will shift the entire field of addiction.

This is followed by chapter 19, "Neurobiological Mechanisms and Cognitive Components of Addiction," written by Jorge Juárez, PhD, and Olga Inozemtseva, PhD, both at the Universidad de Guadalajara in Guadalajara, Jalisco, Mexico. Juárez and Inozemtseva note that the boundaries between the concept that addiction is a public health concern and the concept that it is a problem of inadequate social adaptation are blurry. Furthermore, the pleasure center of the brain, the mesolimbic-cortical system, is central in that several potentially addictive substances act upon it. To effectively address addiction, we must take all of this and more into account from a highly interdisciplinary perspective.

And Mary F. Holley, MD, speaks to us again, in chapter 20, with her report on "Fetal, Neonatal, and Early Childhood Effects of Prenatal Methamphetamine Exposure." Methamphetamine, unlike cocaine, is used by women in equal rates to men, and is far more toxic than cocaine, with a far more intense addiction, and a far more rapid disintegration of personal and family life. Children exposed to methamphetamine in utero are highly likely to show learning disabilities and neurologic dysfunction, which is indeed a public health disaster.

Emmanuel S. Onaivi, MSc, PhD, at William Patterson University in Wayne, New Jersey, United States, also of the U.S. National Institute of

Health (NIH), headquartered in Bethesda, Maryland, United States, writes on the "Endocannabinoid Hypothesis of Drug Addiction" in chapter 21. Onaivi argues that pharmacological treatment of drug addiction is disappointing for the most part, and that new approaches are very much needed. Onaivi tells us that, at least to some extent, misconceptions of addiction as a willpower and morality problem have receded, and recognitions that addiction is a brain disease have surfaced. However, misconceptions holding that dopamine is released in the brain's reward system with all drug use linger. Onaivi reports that many substances, or "agents" as they are also called, do not activate midbrain dopamine-mediated transmission and therefore this popularly applied marker (the dopamine marker) is no longer supporting the perception that drugs are merely activating the brain's reward system. Onaivi contends that in the central nervous system the role of marijuana-like substances produced by the human body, endocannabinoids, must be addressed.

Chapter 22, "Regulation of μ-Opioid Receptor Desensitization in Sensory Neurons," is contributed by Cui-Wei Xie, MD, PhD, at the Department of Psychiatry and Biobehavioral Sciences, David Geffen School of Medicine, and the Semel Institute for Neuroscience and Human Behavior, University of California Los Angeles (UCLA), Los Angeles, United States. Here, Xie calls for further study to "identify and characterize interactions between endogenous receptors in native neurons." Such research may reveal to us mechanisms of "control of opioid receptor signaling" facilitating the strength of "treatment of opiate tolerance and dependence." While most studies to date focus on systems where receptors are "overexpressed," Xie writes that "emerging evidence indicates that formation of hetero-oligomers [between receptors] permits cross regulation of receptor signaling and trafficking, which in some cases, leads to the development of cross desensitization." Such research is key to advancing the power and focus of the treatment of addiction on the cellular level.

Each of the contributors to this volume and the other volumes in the *Praeger International Collection on Addictions* is working on one of the many frontiers of modern addiction research and treatment. And all presented herein is surely just the tip of the iceberg, a sampling of what we know and what we think we know about how addictions work as these addictions feed this global epidemic we call *addiction*. The global picture is both one of grim need and one of unwavering hope. If somehow the goal of massive international collaboration to stop both addiction and the conditions it results in can be realized, perhaps hope will triumph.

This page intentionally left blank

Part I

ISSUES

This page intentionally left blank

Chapter **1**

Addiction and Health Promotion

Kathleen Bradbury-Golas, DNP, RN, APN

Substance abuse interferes with a person's life purpose and interpersonal relationships. Health care researchers expect that the overuse of alcohol and illicit medications will impact upon society's health for decades. The impact of substance abuse, addiction, and relapse is far-reaching. Cardiovascular disease, stroke, cancer, human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), hepatitis, and lung disease can all be affected by drug abuse (National Institute on Drug Abuse [NIDA], 2005). Some of these effects occur when drugs are used at high doses or after prolonged use; however, some may occur after just one use.

Substance abuse harms the body not only from the use of the substance itself but from the negative lifestyle behaviors in which the user participates. Poor nutrition, tobacco smoking, insomnia, and lack of exercise all impact on the physical well-being of a person. Pregnant drug users not only have poor self-care, they also bear lower birth weight and addicted babies who must go through withdrawal soon after birth. These negative lifestyle practices create an environment of continued addiction by decreasing the energy level and self-esteem of the abuser (Apovian, 2006). Recovery becomes almost impossible. If an addict manages to achieve a recovered state, he may believe that the other habits don't matter. For example, smokers who are in recovery believe that smoking cessation will threaten the other substance recovery. However, people are more likely to die of tobacco-related diseases than from alcohol-related problems (American Academy of Family Physicians, 2006).

To add to the problem, the 2005 Monitoring the Future and the Centers for Disease Control and Prevention's (CDC) Youth Risk Behavior Surveillance

System (YRBSS) found heroin use beginning as early as 8th grade. The 2005 YRBSS reported 2.4 percent of 8th- to 12th-grade students using heroin at least once in their lifetime. Early-age heroin use will not only complicate the physical and mental health of the individual, but can lead to lost time in the person's most productive years. They are at risk of their educations being interrupted or negatively affected, which in turn, may influence the ability to make necessary lifestyle changes later in life.

The user's obsession with substance use disrupts family life and creates a destructive pattern of a co-dependency within the family; family members supply the user with money to purchase the substance, thereby denying the problem. Families may split apart, either from separation or divorce due to spousal addiction or children being placed in foster care, group homes, or with other relatives due to parental addiction. Employed drug abusers are more likely to have occupational accidents, affecting either themselves or others. These accidents cost their employers two times more in medical and worker compensation claims than their drug-free workers (NIDA, 1999). About 70 percent of crime is drug related and the judicial system in several states has had to resort to drug court (Herbert & Shaw, 2007).

In order to maintain recovery, addicts not only have to resist the use of mood-altering substance(s) but return to a healthy physical state, repair interpersonal relationships, and develop some sense of peace or spirituality. To accomplish this, substance abusers move through a process in which decisions are made to not only initiate the behavior change but to maintain it (Bandura, 1986; Miller & Rollnick, 1991; Ritter, 2002). Many factors, such as increased self-efficacy and educational level, have been identified as important to the recovery process. Other factors, particularly depression, have been shown to increase the likelihood of relapse, preventing recovery from occurring (Compton, Cottler, Jacobs, Ben-Abdallah, & Spitznagel, 2003; Stein, Solomon, Herman, Anderson, & Miller, 2003; Subramaniam, Lewis, Stitzer, & Fishman, 2004). Research has not shown definitive evidence on what triggers relapse or what changes recovering abusers have made in their lives to decrease the incidence of relapse, thereby maintaining a healthy, productive life. Once recovering abusers have not used drugs, do they practice healthy lifestyle behaviors? No studies have focused on substance abusers health behaviors while in recovery.

RESEARCH FINDINGS

Maslow's theory of human motivation (1970) suggests that as persons experience higher levels of need satisfaction, there is less lower-level tension and they are motivated to make better decisions about health promotion. Much of

the early research regarding health and illness used theoretical models that primarily centered on illness prevention, not health promotion. These prevention models include but are not limited to the Health Belief Model (Rosenstock, 1960), Theory of Reasoned Action and Planned Behavior (Ajzen, 1991), and the Relapse Prevention Model (Marlatt & Gordon, 1985).

Drug relapse prevention treatment models have long been based on social cognitive and behavioral theories that transition the addict through the withdrawal phase to identifying and preventing high-risk situations for relapse (Witkiewitz & Marlatt, 2004). All acknowledge self-efficacy as essential in behavior change. Self-efficacy is defined as the degree to which an individual feels confident and capable of performing a certain behavior in a specific situation (Bandura, 1977). Increased self-efficacy is a strong predictor of outcomes in all types of addictive behavior, including gambling (Sylvain, Ladouceur, & Boisvert, 1997), smoking, and drug use (Sklar, Annis, & Turner, 1999). Not only has self-efficacy been a major determinant in relapse prevention, it is also a key factor in maintaining any health-promoting behavior.

Therefore, once substance abusers stop using and have found the motivation to abstain, they not only begin altering their brain chemistry but build new identities (Spriggs, 2003; Wilcox & Erikson, 2004). With improved self-efficacy and relapse prevention skills, the substance abuser reaches a nonusing state, a chosen behavior change. This builds a firm foundation for future health behavior changes. However, presently, health promotion practices of substance users who are maintaining a substance-free lifestyle have not been described in the literature.

Though much of the research on health promotion has described various populations of interest, few studies have been done that utilized an intervention to achieve a behavior change. Nola Pender's revised Health Promotion Model (HPM) combines numerous constructs from expectancy-value and social cognitive theories within a nursing holistic perspective (Pender, Murdaugh, & Parsons, 2006). The model proposes a positive and humanistic definition of health and though disease is present, it is not the most important element. Once a person commits to the behavior change, his affect toward the behavior increases self-efficacy, which in turn, increases the likelihood of enacting the behavior. As the addict attains substance abstinence, his confidence to maintain the behavior increases, which in turn, gives the addict strength to continue the recovery state. Another proposition of the model states that as self-efficacy increases, the number of perceived barriers, such as the loss of a high and withdrawal symptoms, to a specific health behavior decreases. However, in the end, if addicts do not believe that they can succeed at quitting, they will relapse and continue to use. Measuring health promotion can be completed through use of

the Health Promotion Lifestyle Profile II. This reliable and valid instrument includes the six subscales of health promotion: nutrition, physical activity, health responsibility, spirituality, interpersonal relations, and stress management. Much research utilizing this model has centered on adopting improved nutrition and physical activity into a healthy lifestyle.

Many other factors affect the adoption of health-promoting behavior, such as unavailability of primary prevention facilities (Wilson, 2005), obesity (Nies, Buddington, Cowan, & Hepworth, 1998), and internal motivation (Weitzel & Waller, 1990). Internal motivation and recovery engagement have been shown to be major determinants of relapse prevention (Brown, 2003; Zeldman, Ryan, & Fiscella, 2004). Internally motivated study participants had lower relapse rates and higher self-efficacy. Therefore, educational level and increased self-efficacy are the strongest predictors of health promotion in any population (Ahluwalia, Mack, & Mokdad, 2005; Gillis, 1993; Holloway & Watson, 2002; Kawabata, Cross, Nishioka, & Shimai, 1999; Kim, Jeon, Sok, & Kim, 2006; Martinelli, 1999a, 1999b; McCleary-Jones, 1996; Washington, 2001). In one of the few studies using intervention to change health behavior, Sorensen et al. (1998) conducted a WellWorks Study at one of four intervention centers that were participating in the Working Well Cooperative. After assessment for and education regarding weight management and smoking cessation, results showed that those who received ongoing education and assessment reduced their fat consumption significantly. However, smoking cessation was not statistically changed. The researchers found that health promotion programs are most effective among white-collar workers, verifying the previous research findings that education is a strong predictor of lifestyle behavior change.

Much research has been completed on the importance of interpersonal relationships and health. Social support has contributed to physical health by changing diet and exercise regimens, reducing smoking and alcohol use, promoting better sleep and adherence to medical regimens, thereby improving health (Cohen, Underwood, & Gottlieb, 2000). Berkman and Syme (1979) found during a nine-year period that those people who had no social or community ties were more likely to die of all causes than those who maintained family, friend, and community relationships. Allgower, Wardle, and Steptoe (2001) found that low social support was directly related to sedentary lifestyle, poor sleep, and risky behavior such as not wearing seatbelts and not using sunscreen. Numerous other studies have linked poor social support with excessive alcohol consumption, tobacco use, and drug use (Barnes, Reifman, Farrell, & Dintcheff, 2000; Fisher & Feldman, 1998; Shedler & Block, 1990). Other study results demonstrated that intrapersonal determinants accounted for 52 percent of drug use relapses and a person's perception of setting risk for relapse

was the most important element in determining future relapse (Cummings, Gordon, & Marlatt, 1980; Walton, Reischl, & Ramanthan, 1995). Jackson's (2006) study found that depressive symptoms and perceived social support contributed significantly to the prediction of healthy nutritional practices, exercise, substance abuse, and adherence to medical regime in women but not in men.

Lastly, stress management and spirituality are an important part of wellness and are indispensable to maintaining health. Behavioral coping skills have a positive effect to manage work-related stress (Gardner, Rose, Mason, Tyler, & Cushway, 2005). Spirituality, or spiritual growth, is a component of health promoting self-care behavior, or health responsibility. It is a concept that has long been addressed as essential in chronic illness, quality of life and death. A study by Daaleman and Kaufman (2006) found that those participants who reported greater spirituality ($p < 0.01$) were more likely to report less symptoms of depression. Callaghan (2005) investigated the relationship between self-care behaviors and self-care efficacy and found that spiritual growth is significantly related to an adolescent's initiative and health responsibility. This is consistent to existing literature regarding the direction of similar associations with spirituality (National Institute of Mental Health, 2006).

Only one research study has explored health promotion and drug users. Branagan (2006) evaluated a nurse-led education program on preventing overdose among 23 methadone treatment clinics in Ireland. The education program consisted of leaflet and poster education promoting overdose prevention and how to deal with an overdose should it occur. The posters and leaflets were placed in high visual impact locations in 15 clinics in Ireland. A total of 200 questionnaires were distributed and 81 percent of the participants responded positively to reading the poster or leaflet. Of those who responded, 71 percent indicated that they would make changes in their lifestyles based on what they read in the leaflet and recommended that this information be distributed to other agencies that have contact with drug users, such as prisons, and general practice surgeries. Branagan showed positive response to the education; however, the study did not indicate any statistics on overdose knowledge prior to the education program. Being that 16–43 percent of opiate addicts in the United States suffer from multiple overdoses or die from suicide, most frequently the result of overdose, repetition of this educational endeavor may be worthwhile (Hickman et al., 2003; Pfab, Eyer, Jetzinger, & Zilker, 2006).

Regarding addiction, tobacco smoking has been the most extensively studied in relation to health. Smoking increases risks for multiple causes of illness and death, and studies show that 71 percent of those who use illicit drugs also smoke (Ritchie, Ahluwalia, Mosier, Nazir, & Ahluwalia, 1997). Tobacco

smoking has been shown to increase mortality from other health-related illnesses and increase the number of risky lifestyle practices (U.S. Department of Health and Human Services [USDHHS], 2004a; Perkins et al., 1993). Positive relationships between self-efficacy and not initiating smoking, not increasing smoking, and environmental tobacco avoidance was evidenced by Martinelli (1999a, 1999b) and Kawabata et al. (1999). McCleary-Jones (1996) found that age, educational level, and income modified health-promoting practices in smoking and nonsmoking black women. This study found that educational level was the strongest predictor of practicing health promotion, a finding consistent with other studies (Lusk, Kerr, & Ronis, 1995; Foulds et al., 2006).

HEALTH PROMOTION INTERVENTIONS AND SUBSTANCE ABUSE

In January 2000, the U.S. Department of Health and Human Services launched Healthy People 2010, a comprehensive, nationwide health promotion and disease prevention agenda. The National Center for Health Statistics/Centers for Disease Control and Prevention (CDC) quantified the cost of substance abuse (i.e., smoking, illegal drug use, and alcohol) to the United States at more than $484 billion per year, treating such adverse effects as infectious diseases, crime, unintentional injuries, teenage pregnancies, and chronic psychiatric disorders (USDHHS, 2004b). Twenty-five percent of patients seen in primary care clinics have alcohol or drug disorders (Jones, Knutson, & Haines, 2003). Healthy People 2010 has proposed that opioid-dependent patients be able to receive treatment services and opiate antagonist medication (buprenorphine) in outpatient settings, such as primary care offices. In addition, evidence is suggesting that access to primary care will improve not only general health but also drug abuse–related outcomes (Alba, Samet, & Saitz, 2004). The possibility of improved outcomes has created Current Procedural Terminology (CPT) codes for the screening and counseling of alcohol or substance abuse in primary care offices (Hughes, 2008).

Recently, studies have examined common medical problems in patients who are recovering from chemical dependency. Alba et al. (2004) found that chemically dependent individuals without primary care suffered from increased hospitalization, reported chronic medical conditions, and practiced unhealthy lifestyle choices. Friedmann, Zhang, Hendrickson, Stein, and Gerstein (2003) found that the availability of primary care services at onsite substance treatment programs significantly improved addiction severity at follow-up by 14 percent, but did not change the severity of medical outcome. Another alternative is to deliver behavior health services within primary care. In this type of treatment

service, the primary care practitioner addresses the health care issues related to substance abuse whereas the behavioral health and/or addiction specialist services provide management of the substance abuse issues (Ernst, Miller, & Rollnick, 2007).

Substance abuse patients are returning to primary care follow-up sooner than the expected 90 days. With insurance restrictions, patients are often discharged from addiction specialty treatment centers within 7 days and are expected to be monitored for continued use and any other comorbid conditions by primary care providers. The first 90 days postaddictions specialty discharge is the highest risk period for relapse (National Clearinghouse Guidelines, 2005). It requires primary care health providers to be knowledgeable of when to contact the addiction specialist or mental health care provider, assessing the patient for signs of abuse or adherence to the recovery plan, monitoring for adverse effects of addiction-focused pharmacotherapy and consistent motivational support to maintain abstinence. It is imperative that primary care providers work with patients to become more engaged in their own health.

Additional demands are placed on the body while achieving or maintaining recovery. There is a need for more energy, improved psychological well-being, and stabilization of organ function. Proper nutrition supplies the body with the adequate building blocks by which the person can maintain proper health and fight off infection. For cocaine users, the loss of appetite and weight loss from overstimulation can lead to malnutrition; whereas marijuana increases appetite and many long-term users become overweight. Opiate abusers suffer from the constipation effects of the drug class. Diet recommendations include but are not limited to regular mealtimes, portioned low-fat nutritious meals, to prevent overeating and increased vitamins, protein, and fiber. Dehydration is common during recovery, therefore, encouraging adequate fluid intake is essential (Apovian, 2006).

Exercise not only improves mood and reduces mood swings, it adds the internal strength to deal with negative stressful states, which often are triggers that lead to lapses or relapses (Gross, 2006). Stress management techniques assist the substance abusers in preventing lapses and improve their interpersonal communication skills. These interventions create a circle of positive outcomes, thereby leading to a healthier lifestyle, both physiologically and psychosocially. As recovery progresses and the user improves health promotion activities, self-esteem and motivation increase to maintain substance recovery and health. However, the most important element of health promotion is for the person to avoid returning to substance use, before changing diet or quitting smoking.

Pharmacologic agents may assist in achieving recovery. Gamma–vinyl-GABA (Vigabartin) has shown positive effects in blocking the craving for

cocaine. When combined with counseling, 30 percent of patients successfully kept off cocaine during a nine-week study (Interlandi, 2008). Another medication, Acamprosate calcium (Camparal), already on the market for alcoholism, has the potential to reduce cravings and help prevent relapses, though the effect is individual. Naltrexone extended release injectable suspension (Vivitrol) works on blocking the action of the alcohol directly, thereby blocking the feel-good effect. Nothing has been as successful as varenicline (Chantix) for nicotine addiction. Varenicline studies have shown that 44 percent of tobacco addicts were able to quit smoking after 12 weeks. However, none of these medications will work without extensive behavioral-cognitive group therapy and counseling and they are not to be a permanent part of the treatment regimen. These medications remain only tertiary prevention in the early phases of detoxification.

It is the potential of vaccines that is the most hopeful for primary prevention in addiction. Worldwide research is developing vaccines to inoculate people against addictive substances such as cocaine, heroin, and methamphetamine. The National Institute of Drug Abuse states that these vaccines may be available within 1 to 10 years. The most promising vaccine TA-CD, a vaccine for cocaine abuse, prevents the addictive substance from ever reaching the user's brain. It will work like any other vaccine. The immune system is prompted to create antibodies and when an individual takes cocaine the next time, the body mounts an automatic defense. Antibodies attach to the cocaine molecules, which are then broken down by enzymes in the bloodstream. This in turn eliminates the high and desire to have more cocaine (Hylton, 2008). Unfortunately, it has taken almost 10 years to develop this vaccine.

Research on depression as a factor contributing to addiction and relapse has been inconclusive in the literature. The question is not if the depression actually exists since co-occurrence of a mood disorder and substance abuse ranges from 42.6 to 60.3 percent in the United States (Grant, Stinson, & Dawson, 2004; Kessler et al., 1996). However, the question is really "Does depression cause substance abuse?" or "Does substance abuse cause a depressive state?" Depression has a greater impact on overall health than other chronic medical conditions. The underdiagnosis of co-occurring disorders affects clinical interventions, medications, and the psychosocial treatments that can improve patient outcomes, particularly relapse prevention and health promotion behaviors. Without detection and appropriate treatment, the physical and mental health of the individual fails.

Studies have shown that there is a relationship between substance abuse recovery and spirituality. Spirituality in recovery has been limited to the 12 Step–based spiritual practices, whereby a person accepts the existence of a higher power and develops faith, gratitude, and humility that govern the recovery process. Beyond this 12 Step method, however, other spiritual interventions,

such as transcendental mediation and therapeutic touch, have been shown to have a significant positive effect on the recovery process, though not always at a significant level. Unfortunately, many of these studies were limited due to limited funding and allocated resources. Additional research in complementary spiritual practices is needed.

Harm-reduction strategies are presently very controversial in health philosophy, as they are misinterpreted as drug legalization tools. These approaches are to reduce the negative consequences of drug abuse to an individual or harm done to the addict's family or community. Sometimes these strategies ignore the needs of the individual addict as a trade-off for the betterment of the community at large. Needle-exchange programs' primary goal is to reduce the risk of spreading infectious diseases such as HIV/AIDS and hepatitis by taking infected needles out of circulation. Yet, these programs require local funds to operate and are not consistently available throughout the United States. Even the use of agonist medications for heroin addicts is not widely accepted practice, though research has shown that the use of these medications helps in restoring the individual to functionality in the family, at work, and in the community (Kosten, 2005). These strategies have been shown to be helpful in the adolescent age group through behavioral modification, which may eliminate substance use (Canadian Medical Association, 2008).

Patient care is affected by policy. Policy determines who gets what kind of care from whom and when (Mason, Leavitt, & Chaffee, 2007). A major role of the health care providers dealing with substance abusers is to recognize the needs of the client/patient and work toward attaining the resources required to provide high-quality patient care. Caring for the addicted and recovering patient in the outpatient primary care setting is a goal of Healthy People 2010. Discrimination has been shown to have a negative impact on both mental and physical health. Young, Subere, Ahern, and Galea (2005) found that discrimination was significantly associated with poor mental health, depression, and the number of chronic physical health conditions in illicit drug users. Negative emotional states and stress have been shown to have a direct effect on patterns of behavior that affect physical health. Yet, these underserved and misunderstood patients are most often treated negatively and inconsistently when compared to other widely publicized medical conditions, such as cardiovascular and pulmonary disease. Even pharmaceutical companies are reluctant to develop products for drug addicts.

With better health promotion, health care costs in future years could potentially decrease. In addition, earlier initiation of health promotion in the abusing population may assist those individuals in avoiding major disease complications and help in substance abuse recovery. However, the initiation of health promotion education and services is not often covered under insurance or Medicaid

and requires money to enact. Discrimination due to drug use may limit available resources, access to social welfare, and compliance with medications and health care regimens (Young et al., 2005). Obtaining the financial resources for this endeavor would necessitate creating or changing policy at the insurance company, state, or federal level. Primary care and addictions specialty providers are empowered to make these changes occur. Grassroots lobbying efforts from the local to federal level enable these policies to come to fruition.

SUMMARY

Healthy lifestyle practices or behaviors have been defined by many groups, yet perception of health, benefits of a health-promoting behavior, and barriers to the behavior are all individual. The person must have the internal motivation and develop the self-efficacy or confidence to adopt the behavior change and then to maintain it. As addicts attain substance abstinence, their confidence to maintain the behavior increases which in turn gives the addict strength to continue the recovery state. Research has shown internal motivation and self-efficacy as the best predictors of behavior change, including relapse prevention and recovery from drug use. Other factors, such as perceptions of risk and control, also play crucial roles in the recovery process. Barriers to abstinence, such as craving, depression, and withdrawal symptoms, prevent many substance abusers from adopting a permanent lifestyle change. This in turn decreases their chance of life without multiple medical problems. Increasing participation in drug abuse treatment can decrease the spread of these diseases by reducing risky behaviors, such as sharing injection equipment and having unprotected sex.

Relapse prevention therapy, along with other adjunctive treatments, to reduce stress, improve nutrition, decrease depression and physical symptoms, increase the addicted person's emotional and spiritual states, energy level, and overall well-being (Brooks, 2006). With the rise of rapid three-day addiction detoxification programs in health care institutions, addicted individuals will be relying on primary care practitioners to address their addiction and other health needs. To maintain the recovery process, collaboration between primary care and addictions specialists is pivotal in evaluating and treating these patients to ensure positive health outcomes.

REFERENCES

Ahluwalia, I., Mack, K., & Mokdad, S. (2005). Changes in selected chronic disease-related risks and health conditions for nonpregnant women 18–44 years old. *Journal of Women's Health, 14*(5), 382–386.

Ajzen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes, 50,* 179–211.

Alba, I., Samet, J., & Saitz, R. (2004). Burden of medical illness in drug- and alcohol-dependent persons without primary care. *American Journal on Addictions, 13,* 33–45.

Allgower, A., Wardle, J., & Steptoe, A. (2001). Depressive symptoms, social support and personal health behaviors in young men and women. *Health Psychology, 20,* 223–227.

American Academy of Family Physicians. (2006). *Smoking cessation in recovering alcoholics.* Retrieved April 4, 2008, from http://familydoctor.org/online/famocen/home/common/addictions/tobacco/269.printview

Apovian, C. (2006). Diet and substance abuse recovery. *Medical encyclopedia.* Retrieved April 4, 2008, from http://www.nim.nih.gov/medlineplus/ency/article/002149.htm

Bandura, A. (1977). Self efficacy: Toward a unifying theory of behavioral change. *Psychological Review, 84*(2), 191–215.

Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory.* Englewood Cliffs, NJ: Prentice Hall.

Barnes, G., Reifman, A., Farrell, M., & Dintcheff, B. (2000). The effects of parenting on the development of adolescent alcohol misuse: A six wave latent growth model. *Journal of Marriage and the Family, 62,* 175–186.

Berkman, L., & Syme, S. (1979). Social networks, host resistance and mortality: A nine year follow up study of Alameda County residents. *American Journal of Epidemiology, 109,* 186–204.

Branagan, O. (2006). Providing health education on accidental drug overdose. *Nursing Times, 102*(6), 32–33.

Brooks, A., Schwartz, G., Reece, K. and Nangle, G. (2006). The effect of *Johrei* healing on substance abuse recovery: A pilot study. *Journal of Alternative and Complementary Medicine, 12*(7), 625–631.

Brown, N. (2003). Relapsing, running and relieving: A model for high-risk behavior in recovery. *Journal of Addictions Nursing, 14,* 14–17.

Callaghan, D. (2005). The influence of spiritual growth on adolescents' initiative and responsibility for self-care. *Pediatric Nursing, 31*(2), 91–97.

Canadian Medical Association. (2008). Youth substance use and abuse: Challenges and strategies for identification and intervention. *Canadian Medical Association Journal, 178*(2), 145–148.

Cohen, S., Underwood, L., & Gottlieb, B. (2000). *Social support measurement and intervention.* London: Oxford University Press.

Compton, W., Cottler, L., Jacobs, J., Ben-Abdallah, A., & Spitznagel, E. (2003). The role of psychiatric disorders in predicting drug dependence treatment outcomes. *American Journal of Psychiatry, 160*(5), 890–895.

Cummings, C., Gordon, J., & Marlatt, G. (1980). Relapse strategies of prevention and prediction. In W. R. Miller (Ed.), *The addictive disorders: Treatment of alcoholism, drug abuse, smoking, and obesity.* New York: Pergamon.

Daaleman, T., & Kaufman, J. (2006). Spirituality and depressive symptoms in primary care outpatients. *Southern Medical Journal, 99*(12), 1340–1345.

Ernst, D., Miller, W., & Rollnick, S. (2007). Treating substance abuse in primary care: A demonstration project. *International Journal of Integrated Care, 7*(10), 1–8.

Fisher, L., & Feldman, S. (1998). Familial antecedents of young adult health risk behaviors: A longitudinal study. *Journal of Family Psychology, 12*, 66–80.

Foulds, J., Gandhi, K., Steinberg, M., Richardson, D., Williams, J., Burke, M., et al. (2006). Factors associated with quitting smoking at a tobacco dependence treatment clinic. *American Journal of Health Behavior, 30*(4), 400–412.

Friedmann, P., Zhang, Z., Hendrickson, J., Stein, M., & Gerstein, D. (2003). Effect of primary medical care on addiction and medical severity in substance abuse treatment programs. *Journal of General Internal Medicine, 18*, 1–8.

Gardner, B., Rose, J., Mason, O., Tyler, P., & Cushway, D. (2005). Cognitive therapy and behavioural coping in the management of work-related stress: An intervention study. *Work and Stress, 19*(2), 137–152.

Gillis, A. (1993). Determinants of a health-promoting lifestyle: An integrative review. *Journal of Advanced Nursing, 18*, 345–353.

Grant, B., Stinson, P., & Dawson, D. (2004). Prevalence and co-occurrence of substance user disorders and independent mood and anxiety disorders. *Archives of General Psychiatry, 61*, 807–816.

Gross, S. (2006). *Relapse prevention*. Retrieved April 24, 2008, from http://www.helphorizons.com/library/search_details.htm?id=77

Herbert, K., & Shaw, J. (2007, January 2). Drug courts are battling addictions behind crime. *Philadelphia Inquirer, 178*(216), pp. A1, A8.

Hickman, M., Carnwath, Z., Madden, P., Farrell, M., Rooney, C., Ashcroft, R., et al. (2003). Drug related mortality and fatal overdose risk: Pilot cohort study of heroin users recruited from specialist drug treatment sites in London. *Journal of Urban Health, 80*, 274–287.

Holloway, A., & Watson, H. (2002). Role of self efficacy and behaviour change. *International Journal of Nursing Practice, 8*, 106–115.

Hughes, C. (2008). CPT 2008: A glimpse of the future of family medicine. *Family practice management*. Retrieved April 17, 2008, from http://www.aafp.org/fpm

Hylton, H. (2008). *A drug to end drug addiction*. Retrieved April 12, 2008, from http://www.time.com/time/health/article/0,8599,1701864,00.html

Interlandi, J. (2008, March 3). What addicts need. *Newsweek*, 36–43.

Jackson, T. (2006). Relationships between perceived close social support and health practices within community samples of American women and men. *Journal of Psychology, 140*(3), 229–246.

Jones, E., Knutson, D., & Haines, D. (2003). Common problems in patients recovering from chemical dependency. *American Family Physician, 68*(10), 1971–1978.

Kawabata, T., Cross, D., Nishioka, N., & Shimai, S. (1999). Relationship between self-esteem and smoking behavior among Japanese early adolescents: Initial results from a three-year study. *Journal of School Health, 69*(7), 280–284.

Kessler, R., Nelson, C., McGonagle, K., Edlund, M., Frank, R., & Leaf, P. (1996). The epidemiology of co-occurring addictive and mental disorders: Implications for prevention and service utilization. *Annuals of Orthopsychiatry, 66*, 17–31.

Kim, S. Y., Jeon, E. Y., Sok, S., & Kim, K. B. (2006). Comparison of health-promoting behaviors of noninstitutionalized and institutionalized older adults in Korea. *Journal of Nursing Scholarship, 38*(1), 31–35.

Kosten, T. (2005). What are America's opportunities for harm reduction strategies in opiate dependence? *American Journal of Addictions, 14*, 307–310.

Lusk, S., Kerr, M., & Ronis, D. (1995). Health promoting lifestyles of blue-collar, skilled trade, and white-collar workers. *Nursing Research, 44*(1), 20–24.

Marlatt, G., & Gordon, J. (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York: Guilford Press.

Martinelli, A. (1999a). An explanatory model of variables influencing health promotion behaviors in smoking and nonsmoking college students. *Public Health Nursing, 16*(4), 263–269.

Martinelli, A. (1999b). Testing a model of avoiding environmental tobacco smoke in young adults. *Image: Journal of Nursing Scholarship, 31*(3), 237–242.

Maslow, A. H. (1970). *Motivation and personality*. New York: Harper and Row.

Mason, D., Leavitt, J., & Chaffee, M. (2007). *Policy and politics in nursing and health care*. Philadelphia: Elsevier.

McCleary-Jones, V. (1996). Health promotion practices of smoking and non-smoking black women. *ABNF Journal, 7*(1), 7–10.

Miller, W. R., & Rollnick, S. (1991). *Motivational interviewing: Preparing people to change addictive behaviors*. New York: Guilford Press.

National Clearinghouse Guidelines. (2005). Medication-assisted treatment for opioid addiction in opioid treatment programs: Approaches to providing comprehensive care and maximizing patient retention. Retrieved April 24, 2008, from http://www.guideline.gov/summary/summary.aspx?doc_id=8352&nbr=004675&string=substance+AND+abuse

National Institute on Drug Abuse. (1999). *InfoFacts—Workplace trends*. Retrieved January 6, 2007, from www.nida.nih.gov/infofacts/workplace.html

National Institute on Drug Abuse. (2005). *InfoFacts—Hospital visits*. Retrieved January 6, 2007, from www.nida.nih.gov/infofacts/hospitalvisits.html

National Institute on Drug Abuse (NIDA) and University of Michigan. (2005). *Monitoring the future 2005: Data from in-school surveys of 8th, 10th and 12th grade students*. Retrieved January 23, 2007, from http://www.monitoringthefuture.org/pubs/monographs/overview2005.pdf

National Institute of Mental Health. (2006). *NIMH, the numbers count*. National Institutes of Health. Retrieved March 4, 2007, from http://nimh.nih.gov/publicat/numbers.cfm

Nies, M., Buddington, C., Cowan, G., & Hepworth, J. (1998). Comparison of lifestyles among obese and nonobese African American and European American women in the community. *Nursing Research, 47*(4), 251–257.

Pender, N. J., Murdaugh, C. L., & Parsons, M. A. (2006). *Health promotion in nursing practice* (5th ed.). Upper Saddle River, NJ: Prentice Hall.

Perkins, K., Rohay, J., Meilahn, E., Wing, R., Matthews, K., & Kuller, L. (1993). Diet, alcohol, and physical activity as a function of smoking status in middle aged women. *Health Psychology, 12*(5), 410–415.

Pfab, R., Eyer, F., Jetzinger, E., & Zilker, T. (2006). Cause and motivation in cases of non-fatal drug overdoses in opiate addicts. *Clinical Toxicology, 44*, 255–259.

Ritchie, K., Ahluwalia, H., Mosier, M., Nazir, N., & Ahluwalia, J. (1997). A population-based study of cigarette smoking among illicit drug users in the United States. *Addiction, 97*, 861–869.

Ritter, A. J. (2002). Naltrexone in the treatment of heroin dependence: Relationship with depression and risk of overdose. *Australian and New Zealand Journal of Psychiatry, 36*, 224–228.

Rosenstock, I. (1960). What research in maturation suggests for public health. *American Journal of Public Health, 50*, 295–301.

Shedler, J., & Block, J. (1990). Adolescent drug use and psychological health: A longitudinal inquiry. *American Psychologist, 45*, 612–630.

Sklar, S., Annis, H., & Turner, N. (1999). Group comparisons of coping self efficacy between alcohol and cocaine abusers seeking treatment. *Psychology of Addictive Behaviors, 13*, 123–133.

Sorensen, G., Stoddard, A., Hunt, M. K., Hebert, J., Ockene, J., Avrunin, J., et al. (1998). The effects of a health promotion-health protection intervention on behavior change: The WellWorks Study. *American Journal of Public Health, 88*(11), 1685–1690.

Spriggs, M. (2003). Can we help addicts become more autonomous? Inside the mind of an addict. *Bioethics, 17*(5–6), 542–554.

Stein, M., Solomon, D., Herman, D., Anderson, B., & Miller, I. (2003). Depression severity and HIV injection risk behaviors. *American Journal of Psychiatry, 160*, 1659–1662.

Subramaniam, G., Lewis, L., Stitzer, M., & Fishman, M. (2004). Early treatment time course of depressive symptoms in opiate addicts. *Journal of Nervous and Mental Disease, 179*(4), 215–221.

Sylvain, C., Ladouceur, R., & Boisvert, J. (1997). Cognitive and behavioral treatment of pathological gambling: A controlled study. *Journal of Consulting and Clinical Psychology, 65*, 727–732.

U.S. Department of Health and Human Services. (2004a). *Health effects of cigarette smoking*. Retrieved June 25, 2006, from http://www.cec.gov/tobacco/factsheets/HelathEffectsofCigaretteSmoking_Factsheet.htm

U.S. Department of Health and Human Services. (2004b). *Progress review: Substance abuse*. Retrieved June 19, 2006, from http://www.healthypeople.gov/data/2010prog/focus26/

Walton, M., Reischl, T., & Ramanthan, C. (1995). Social settings and addiction relapse. *Journal of Substance Abuse, 7*(2), 223–233.

Washington, O. (2001). Using brief therapeutic interventions to create change in self-efficacy and personal control of chemically dependent women. *Archives of Psychiatric Nursing, XV*(1), 32–40.

Weitzel, M., & Waller, P. (1990). Predictive factors for health-promotive behaviors in white, Hispanic, and black blue-collar workers. *Family and Community Health, 13*, 23–34.

Wilcox, R., & Erikson, C. (2004). Prevention of relapse to addiction: Information for the practitioner. *Texas Medicine, 100*(2), 52–61.

Wilson, M. (2005). Health-promoting behaviors of sheltered homeless women. *Community Health, 28*(1), 51–63.

Witkiewitz, K., & Marlatt, G. A. (2004). Relapse prevention for alcohol and drug problems. *American Psychologist, 59*(4), 224–235.

Young, M., Subere, J., Ahern, J., & Galea, S. (2005). Interpersonal discrimination and the health of illicit drug users. *American Journal of Drug and Alcohol Abuse, 31*, 371–391.

Zeldman, A., Ryan, R., & Fiscella, K. (2004). Motivation, autonomy support, and entity beliefs: Their role in methadone maintenance treatment. *Journal of Social and Clinical Psychology, 23*(5), 675–696.

This page intentionally left blank

Chapter **2**

Becoming a Problematic Consumer of Narcotics

Ted Goldberg, PhD

This chapter is based on participant observation studies of problematic consumers of narcotics in Stockholm, Sweden, over a period of four and a half years. When doing fieldwork, a researcher meets many people with different characteristics, personalities, strengths, and weaknesses. The wealth of data is so great that it is easy to drown in a sea of details. To formulate the theory presented here, it has been necessary to look beyond individual differences and concentrate on what I have come to see as a major underlying pattern common to problematic consumers of narcotics, that is, people who made illicit substances the central element in their lives. I have developed a dynamic developmental model, or a life-history "career model," to illuminate what brought my research subjects to "the drug scene" and why they elect to remain on it.

As narcotics consumption is a complex phenomenon regulated by cultural norms and definitions, place, time, age, gender, and so on, I am unable to determine the extent to which my model can be generalized. It is my hope that readers will be inspired to test its applicability on other populations.

In this chapter the terms *narcotics*, *illicit substances*, and *illicit drugs* will be used as synonyms.

ASSUMPTIONS

All knowledge of humans and society, scientific or otherwise, is based on unproved assumptions. With postulates as a foundation, we build our theoretical understanding (Israel, 1984, p. 16). Differences of opinion in the drug

debate can often be traced to different suppositions. Unfortunately, assumptions in many texts are implicit, leaving it to the reader to unmask them. I shall therefore begin by making explicit the major assumptions upon which my model is based.

Assumptions about Consumers

I assume that the intellectual capacities of those who use narcotics show approximately the same variation as in the population at large, and that those who use illicit drugs understand the consequences of their actions to approximately the same extent that nonconsumers understand their own behavior. Furthermore, I do not agree with those who postulate that it is primarily the chemical effects of the narcotics themselves that determine consumption. Bejerot (1979, p. 90), for instance, writes: "That the individual has become chemically controlled and has serious deficiencies or completely lost self-control vis-à-vis intoxicants is the essential nature of narcotics addiction." Instead, my starting point is that drug consumers should not be seen merely as objects. They are also subjects who actively choose both to use narcotics and to take the consequences thereof.

Assuming that a person is competent to make decisions leads us to ask such questions as: Why does he choose as he does? What alternatives does he believe he has and what alternatives actually are at his disposal? To what extent is he conscious of the motives behind his decisions? The model presented in this chapter will address these kinds of questions.

Nature and Nurture

Certainly, most serious researchers would agree that human behavior is a product of both biochemical and psychosocial processes. However, the relative importance of these two roots in conjunction with drug consumption is the subject of great and ongoing controversy. So even if the term *biopsychosocial* is being used more and more by those who participate in the drug debate, we really aren't in agreement. The question is: How much of human drug behavior should/can be explained by biochemical variables and what is psychosocially determined? This can be illustrated with the help of a continuum. While almost nobody is at either end of the continuum, a person who places himself toward the left side of the continuum is saying something very different from somebody who is near the other end.

Those who assume that human behavior is primarily of biochemical origin see deviance first and foremost as a result of some combination of genetic dispo-

Continuum 1

Behavior	Behavior
biochemically	psychosocially
determined	determined

sition, metabolic disturbance, and/or drug-induced malfunction in vital organs such as the brain or other parts of the central nervous system. For instance, Robinson and Berridge (1993, p. 249) assume that "addictive behavior is due largely to progressive and persistent neuroadaptations caused by repeated drug use." Those who emphasize biochemical determinants are of the opinion that the root of problematic drug consumption lies principally within the individual and/or the narcotics themselves, and that her social experiences are of secondary importance. Solutions are sought by attempting to find substances that will maintain a normal chemical balance within the individual, detoxification, trying to prevent access to illegal psychoactive substances, information, and so on.

By defining the behavior exhibited after drug consumption as a problem lying within the individual and/or the drugs, the biochemical perspective is of major political significance. The implication is that problematic consumption is not an indication of a need for social change.

Researchers emphasizing psychosocial variables analyze either individual characteristics (i.e., personality, life experiences, underlying needs) and/or social structures. On the micro-level, they ask how the individual became such as she is, and look for answers in, for instance, her family and/or other small groups she currently belongs to and/or has belonged to in the past. On the macro-level, they ask what factors in society create problems for parts of the population as a result of their position in the larger social system. Answers are sought in society's social institutions, culture, and history.

The basic assumption among those who place themselves toward the right-hand side of the above continuum is that humans are social beings and their behavior is to a great extent the result of social relationships. Without denying that people differ in their biological make-up, these differences are given subordinate roles. Biology is seen as a framework that sets limits for what we can achieve; social factors are looked upon as determining the extent to which an individual's capabilities will develop. From this point of view, it follows that behavior cannot be analyzed solely in an individual context. We must also look at macro-level processes and political decisions that create possibilities and obstacles both for the individual and the intimate small groups that form her (Mills, 1997).

Complexity and Nonlinear Thinking

Differences of opinion in the drug debate can also be traced to assumptions as to what are causes and what are effects. Some arguments are based on the assumption that biochemical changes induced by illicit drugs produce (undesirable) behavior, such as lack of achievement in school (Nordegren & Tunving, 1984, p. 143), delayed maturation (Dole & Nyswander, 1967, p. 477), and criminality (see Inciardi, 1992, p. 22ff. for some examples of the drastic kinds of statements that have been made and how they have been used politically). Levine (2003, p. 147ff.) uses the term *drug demonization* for the tendency to see illicit drugs as the major cause of just about any behavior we don't approve of in any individual who takes them.

The arguments presented in this chapter will be based on an alternative assumption: neither narcotics nor any other single factor explain human behavior. Instead, we should try to uncover the latticework of multiple factors that increase the probability that some people will choose to take illicit drugs in a problematic way. This implies that rather than principally concentrating on the illicit substances themselves, considerable effort must also be directed toward understanding other factors, such as the consumer's life experiences, her motives, the conditions of her existence, the situations in which illicit drugs are consumed, the society in which her life is being/has been led, and so on.

An assumption upon which the model presented in this chapter is based is that as a result of his social relationships and life experiences, the individual gains knowledge about himself, the society he lives in, and his roles in that society. All of these factors greatly influence the way he will relate to illicit substances, which in turn, has repercussions for his future relationships to others and to society. In other words, there are feedback relationships between the individual, other people, narcotics, and society.

The model presented here is based upon the assumption that narcotics are not the major culprits. While I do not deny that the biochemical effects of psychoactive substances are a part of the equation, human relationships and the societal conditions from which they originate are seen as playing a major role as determinants of both illicit drug consumption and the behavior displayed in conjunction with it.

From this starting point, I ask: What roles do narcotics play in relation to other aspects of the consumer's life? How do illicit substances relate to her past experiences, her present life situation, and her future possibilities? How did she come to contemplate starting to take psychoactive substances? What made her transform these thoughts into action? Once having started taking illicit drugs,

why does she choose to continue? One of my fundamental assumptions is that if drug experiences (both chemical and social) are not relevant for a person's life seen as a whole, she will not make them the major focus of her existence.

Therefore, it is an important task for drug research to uncover and describe the relationships between illicit drug consumption and other life experiences.

Simultaneous and Process Models

Characteristic for simultaneous models is the assumption that all of the factors that operate to produce a phenomenon operate concurrently (Becker, 1963/1973, p. 22–23). In such models, we lose sight of the history of both the individual and the society in which he lives. An example of simultaneous thinking is the desperate parent who says: "My son was a wonderful kid until he fell in with the wrong crowd and was enticed into smoking a joint, which lead to his downfall." In this kind of explanation, all of this adolescent's previous life experiences are deemed irrelevant.

To my way of thinking, it is unreasonable to believe that a single event (how traumatic or euphoric it may have been) could eradicate the effects of all of the experiences a person has had in her life. I therefore attempt to develop a sequential model that analyzes processes that start in the past and continue through the present and into the future. To help develop a sequential model, Becker (1963/1973, p. 24) uses the term *career*. Similar to athletes who begin their careers in a sand lot and work their way up through different leagues before becoming professionals, the "deviant" has a career consisting of different stages, where earlier experiences become a fundament upon which later stages rest. In this chapter, I will briefly present the stages in the deviant career model I developed to explain the data I collected during my fieldwork. (The model is expounded upon in Goldberg, 1999, pp. 79–150.)

DIFFERENT KINDS OF CONSUMERS

It is not difficult to understand why we must use different concepts and variables to comprehend why a connoisseur drinks $500 wines with gourmet dinners than we need for an analysis of the drinking patterns of skid-row alcoholics. Both are using the same psychoactive drug but it is obvious that the underlying meanings and motives are very different. A common way to illuminate this distinction is to differentiate between "users" and "misusers."[1] However, I have found that these two concepts are understood in so many different ways in the literature and in everyday speech that they no longer convey meaning in a precise way. We therefore need other terms.

Continuum 2

Abstainers ..... Recreational consumers ..... Problematic consumers

drugs play ..... drugs first priority
no role .....

My conceptualization is a continuum with those for whom drugs mean nothing at one end and people who make drugs their first priority at the other. As already noted, *problematic consumers* are people who allow psychoactive substances to play a dominant role in their lives, that is, those who approach the far end of my continuum. *Substantial consumer* will be used as a synonym.

Recreational consumers, on the other hand, consider drugs as one aspect among many in their lives. Borrowing an idea from Norman Zinberg, Stanton Peele (1998, p. 8) writes that nonaddicted drug users "subordinate their desire for a drug to other values, activities, and personal relationships, so that the narcotic or other drug does not dominate their lives. When engaged in other pursuits that they value, these users do not crave the drug or manifest withdrawal on discontinuing their drug use." It is in this sense that I use the term *recreational consumers*.

Although frequency of consumption is a significant aspect in my definitions, it isn't a sufficient criterion. Under certain conditions, it is possible to take drugs regularly without allowing them to govern one's activities. In societies where psychoactive substances are readily accessible, socially accepted, and inexpensive, they can be used in this way. In the Netherlands, for instance, cannabis is more or less socially accepted and can be readily and affordably purchased in any number of coffee shops. It is therefore possible to take this drug on a daily basis with a minimum of social and economic cost. Some people smoke cannabis in a manner comparable to those whose alcohol consumption primarily consists of taking a drink after work or before going to bed. To my way of thinking, people who take illicit substances in this manner shouldn't be considered problematic consumers.

Problematic consumers and recreational consumers may well take the same psychoactive substances, but the meanings behind the act and their motives are very different. A central question in conjunction with all drug consumption is: *What is the individual attempting to accomplish when she takes these substances?*

A number of studies based on different populations have found statistical differences in the psychosocial backgrounds of problematic consumers as compared to recreational consumers. For instance, research on more than 50,000 military conscripts in Sweden (Solarz, 1990) shows that the backgrounds of

recreational consumers did not significantly differ from those of nonconsumers. On the other hand, both this and a host of other studies (i.e., Chein, Gerard, Lee, & Rosenfeld, 1964; Puhakka, 2006; Shanks, 2008; Shedler & Block, 1990) clearly indicate that problematic consumers have had unusually negative life experiences predating their initial experience with narcotics. It appears that problematic consumers in postindustrial societies are primarily recruited from those who have many of the following factors in their backgrounds:

- poverty
- at least one parent with a high level of alcohol consumption
- corporal punishment
- serious conflicts in the home
- broken homes
- if the father was physically present, he tended to take little interest in the family
- spoiled or severely frustrated
- subjected to unclear demands
- inconsistent use of punishment
- chronic physical and/or mental illness in the family
- sexual abuse
- overcrowded living conditions
- multiproblem family
- run away from home on multiple occasions
- subjected to discrimination.

These factors play an important role in the development of the individual's self-image, and are major components in the labeling process to be described in this chapter.

The aforementioned studies also indicate that future problematic consumers of illicit substances showed some of the following symptoms prior to their first narcotics consumption:

- depression
- severe headaches
- stomach problems
- low tolerance for stress
- lack of sleep
- anguish
- nervousness
- excessive aggression.

Furthermore, their childhood years were characterized by:

- lack of initiative
- giving up when faced with resistance

- maladjustment in school (i.e., they have felt themselves mistreated, have received poor grades in both academic subjects and conduct, have been left back and/or placed in a remedial class, have been truant)
- extensive consumption of tobacco
- sniffing organic solvents
- frequent hangovers and/or starting to take pick-me-ups at an early age
- criminal offenses such as shoplifting, assault and battery, theft, vandalism
- being registered by the police and/or social authorities
- not participating in organized leisure time activities
- having feelings of inferiority
- a negative self-image.

The prevalence of extremely negative experiences early in the lives of problematic consumers indicates that problematic consumption should not be considered to be primarily an individual predicament. Macro-analysis is necessary for understanding the roots of the problem.

However, people with similar psychosocial backgrounds are not predestined to a given pattern of drug consumption. Individual social experiences and personal characteristics differentiate people from comparable social environments. Therefore, we must also direct our attention to drug consumers as individuals.

Accordingly, explanations for different types of drug consumption should principally be sought:

- on the macro-level, where people are molded by the society in which they live/have lived
- on the micro-level, where individuals are shaped through interaction with other people in intimate small groups
- on the biological level as we differ genetically.

Instead of attempting to cover all of this in one short chapter, I shall concentrate on my fieldwork and the model I developed to explain what I consider to be the most important underlying patterns behind recruitment to the drug scene in Stockholm. Before beginning, I wish to emphasize that the model concerns only problematic consumers and is not relevant for recreational consumers.

THE NEGATIVE SELF-IMAGE AND SELF-DESTRUCTIVE BEHAVIOR

Early in my fieldwork, I was given the following advice by one of my research subjects: "Don't feel sorry for junkies when they're kicking cold turkey. Addicts are masochists who want to suffer and die." At the time, I found this advice absurd, as the people I had previously associated with wanted to make their

lives as satisfactory as possible, and I assumed that everybody had similar aspirations. Furthermore, from the literature I had read about narcotics, I had been given the impression that addicts, if anyone, sought euphoria. At the time, it seemed beyond question that everyone who takes illicit drugs does so because it makes them feel good.

In retrospect, the advice given by this problematic consumer was the key to understanding the essential nature of what I would be observing while in the field. But the idea was so remote from my "preunderstanding" (the ideas I brought with me into the field) that it took a long time before I could accept it.

Few, if any, doubt that problematic consumers of narcotics risk dying young. That they also ruin their health, are disdained by others, can't trust anyone in their surroundings, are obliged to commit crimes or prostitute themselves daily, sometimes "freak out," periodically become "paranoid," and so on is also well known.[2] Why would anybody subject herself to all of this?

A common answer is some variation of the following theme: "Normally both men and animals are steered by the pleasure-pain principle: some degree of effort is required both to obtain pleasure and to avoid discomfort. . . . In drug addiction . . . a 'short circuit' occurs in the biological system and the normal pleasure-pain principle no longer functions" (Bejerot, 1970, pp. 22–23). "When addiction supervenes . . . the drug hunger takes on the strength and character of instinctive drives" (p. 22). "The drug effects take on the strength of libidinal desires and outweigh all mental, physical, social and economic complications arising from the abuse" (p. xvii). In other words, the individual is no longer in control of her actions. All the negative aspects are outweighed by the euphoric effects of drugs that now steer the individual's behavior (see also Bejerot, 1979, p. 28–31, 37–38, 95).

While it is true that some people (but not all) experience moments of euphoria from psychoactive substances, those who start to use drugs on a daily basis quickly discover that there is a flip side to the coin. The proportion between pleasure and pain increasingly tilts toward the latter as the quantity and frequency of narcotics consumption increases.

If one questions them, even those who believe that euphoria is at the root of problematic consumption will usually agree that such is the case. But they argue that the euphoric experiences of the early stages were so powerful that substantial consumers continue in the hope of reexperiencing them, and/or that by the time they discover the negative effects of narcotics it is too late, as they are caught up in a biochemical dependency and can no longer use their own willpower to control their lives.

However, upon closer examination of what the problematic consumers in my study actually did and experienced, I found that "seeking euphoria" does not

explain what was taking place. In fact, quite the opposite was true—they were destroying themselves. In saying this, I am not claiming that everything problematic consumers do is directly or even primarily directed at self-destruction. But the long-term effect of their lifestyle points in this direction.

All of the substantial consumers I observed while in the field were self-destructive—but to different degrees. Some took drastic measures such as "cleansing" one's syringe with water from the toilet bowl in a public restroom when there is a sink right next to it, borrowing other people's "works" in spite of the risk of contamination with HIV and/or hepatitis, asking others to smuggle alcohol into the hospital (instead of a drug that is milder on the liver) while being treated for hepatitis, finding a pill on the street and eating it without having the slightest idea what it contains, and so on.

But acts as drastic as these do not constitute the major part of the self-destructiveness on the scene. We get a better understanding of what is going on by observing less dramatic activities that are repeated as a part of everyday life: that is, improper diet, lack of sleep, unsanitary living conditions, physical and/or sexual abuse, prostitution, not brushing one's teeth, not attending to sores, paranoia, poverty, and so forth. All of these are integral parts of the drug scene. In fact, I find it very unlikely that the word *euphoria* would come into anyone's mind while observing the drug scene. Indeed, it would not be an exaggeration to say that little or nothing in the lives of substantial consumers of illicit substances is as much as pleasant.

Another way to try to get around the obvious self-destructiveness of problematic consumers is to presuppose that they do not understand what they are doing. Some assume that substantial consumers must either be intellectually lacking, or are so influenced by drugs that they cannot think straight, or have an exceptional ability to delude themselves. However, my observations do not lend support to these assumptions. Admittedly, it can be difficult to think clearly while under the influence of psychoactive substances, and just about everybody (including problematic consumers of illicit drugs) engages in a certain amount of self-deception. Many substantial consumers put up a front in their contacts with the "straight world," which can easily reinforce the impression that they are fooling themselves, but if one manages to penetrate behind this facade, as I did during my field research, it becomes obvious that while they might not be conscious of all of the consequences of every particular choice they make, sooner or later just about every problematic consumer comes to understand that in the long run the substances they are taking and the life they are leading will destroy them. While narcotics sometimes (but far from always) give a measure of short-term relief, substantial consumers eventually come to an awareness that their lives are rapidly deteriorating.

Since problematic consumers eventually come to understand that both they themselves and everybody else who remains on the scene are on the road to extinction, yet do not change their way of life, there must be a strong self-destructive side in their personalities. People who aren't extremely self-destructive do not remain on the drug scene very long. That is, there are clear differences between problematic and recreational consumers. Recreational consumers experiment with drugs. They seek certain experiences, such as an alternate state of consciousness, sexuality, new sensations and impressions, relaxation, and so on, and are prepared to make some sacrifices to achieve these ends. But they are not willing to destroy their lives. When economic, social, psychological, and/or physiological costs reach an excessive level, recreational consumers either stop taking drugs altogether or greatly reduce consumption. Few people wish to annihilate themselves and this explains why only a small minority of those who experiment with illicit substances maintain a high level of consumption for long periods of time.

But for people with an extremely self-destructive side to their personalities, the negative effects inherent in the lifestyle on the drug scene become an important reason for continuing with narcotics. At least for my research subjects, *problematic consumption of narcotics is a major component in a self-destructive life project*.

However, it is an oversimplification to divide humanity into two categories: those who are self-destructive and those who aren't. Clearly there are degrees of self-destructiveness. The problematic consumers of illicit substances I studied are extremely self-destructive people who lead lives that slope steeply toward extermination. Not quite as self-destructive people might well follow negative paths, but most likely ones that incline at a lesser angle.

A PROCESS MODEL

The model presented here can be seen as a variation of labeling theory and will identify a series of stages that problematic consumers of illicit substances pass through during their careers. However, before I begin, I must define a few concepts.

The *self-image* is an individual's conception of himself in relation to the ideals of his culture. People do not have a self-image at birth and we are not free to choose our self-image at will. Instead the self-image should be seen as developing through social relationships. Those people whose responses contribute to the formulation of an individual's self-image are his *significant others*. Parents (the people who raise the child) are usually his first and the most important significant others. It is through the parents that the child begins to develop

his self-image. As he grows older and comes into contact with society outside of the home, some of the people he meets will become significant others and they too will influence his self-image. However, the basis of the self-image has already been established by that time.

A person with a *negative self-image* has learned through interaction with significant others that there is a low degree of correspondence between his personal characteristics and/or behavior and cultural ideals as to how one should be.

Labeling is a process, not a few exceptional events. In the sense that the concept is used here, *labeling* is a course of events consisting of a large number of negative reactions from significant others, which taken as a whole, cause the individual to redefine his self-image in a negative direction.[3] Labeling occurs in many different places, such as in the family, in relationships with contemporaries, in school; and it can be evoked by many different things such as the individual's behavior, appearance, beliefs, sexual preferences, and so on. It is important to keep in mind that labeling is an ongoing process during an extended period of time, and even if one can identify dramatic events in the individual's life, these do not constitute a sufficient explanation. The self-image develops from the sum of large numbers of both prominent and seemingly insignificant experiences and reactions during the entire lifetime of the individual. Therefore, the older we get, the more well established it becomes and the longer it takes to change its essential content, even if adjustments can be achieved in a shorter time frame.

Parental Labeling

With the help of the concept *primary deviance*, I can intimate the course of events that have constituted the beginning of the labeling process for my research subjects. *Primary deviance* consists of acts that do not comply with societal norms but that are not committed with malice aforethought. They are not the product of a well-established negative self-image but rather the result of a lack of understanding of the plausible consequences of one's actions. In the words of a famous anthropologist, the newborn child is "a barbarian invader from another planet"; that is, a baby knows nothing of the culture it is born into. In order to learn societal norms (right from wrong), significant others must respond to the child's spontaneous behavior. *Everybody begins their lives with primary deviance. What distinguishes one person from the other are the ways their significant others react*. Somewhat simplified, one can say that some children learn that they themselves are good, even if what they are doing for the moment is unacceptable. Others learn, after a sufficient number of "unsuitable"

reactions, that it is not simply what they are doing for the time being that is unsatisfactory, but rather that there is something basically wrong with them as human beings. This is *parental labeling* and the *first stage* in the deviant careers of my research subjects.

Some parents appear to be unable to help their children develop a positive self-image, and it is reasonable to ask why this is so. Where one seeks answers depends to a large extent upon one's assumptions regarding the relative importance of nature and nurture (see earlier section).

From my point of view, sociological explanations are important as they reveal the societal pressures people in different socioeconomic groups are subjected to, and the increased probability of certain types of behavior. Statistically, such factors as belonging to a minority group, having uneducated parents, living in a slum, and so on, greatly reduce one's choices in life. But societal pressure is only a framework, not an inescapable process that forces people to act (or not act) in a particular way. Indeed, all people in similar social situations do not respond in the same way. Why Smith, but not his neighbor Jones, succumbs to societal pressure and labels his child cannot be explained sociologically. We must therefore also look at psychological and biological variables. However, it is reasonable to assume that if we can reduce societal pressures the number of people who cannot cope should be diminished, thereby decreasing the number of people who label their children.

Societal Labeling

As children grow older, they spend an increasing amount of time out of the home and they come into contact with society, that is, neighbors, day care personnel, elementary school teachers, and so on. During this period, children continue to learn social norms through reactions from others. In their initial contacts with society, the behavior of children is principally characterized by primary deviance, but as they grow older and gain social experience, their actions become increasingly influenced by societal norms. All children exhibit primary deviance in their initial contacts with society. Once again, the most important element is not the behavior but the reactions from significant others. Most children will learn that they are O.K. even if certain things they do are not. But some will learn that the problem lies not in what they are doing, but with them. This is *societal labeling*, the *second stage* in my deviant career model.

However, it is important to keep in mind that not all negative reactions are a part of a labeling process. *Labeling theory does not imply laissez-faire*. People need reactions in order to learn how to function in society. But the reactions must not equate the actions of the moment with the person herself.

Most often, it is not merely a matter of chance if a child is labeled in her contacts with society. Children who have been labeled by their parents run a far greater risk of being subjected to societal labeling. To explain why this is so, I must introduce another concept. *Behavioral incongruity* arises when an individual's behavior isn't in harmony with her self-image. She will react by trying to diminish the incongruity either by changing the behavior or her self-image. Figure 2.1 gives a picture of behavioral incongruity.

However, the picture conveyed by this figure is greatly oversimplified. Firstly, it gives the impression that people are static, when in reality they change. Some of the more important factors influencing changes in my research subjects will be discussed shortly.

Secondly, the self should be regarded as multiple and pluralistic rather than unitary. In, for instance, Robert Ornstein's (1986, 1991) multimind model we have many "small minds." This is because we participate in various social situations and we do not receive the same responses from all of our significant others. This awakens contradictory desires and ambivalent feelings in us. But Ornstein also postulates that there is a "governing self" that decides which of our small minds we are cognizant of at any given time. That is, even if we have

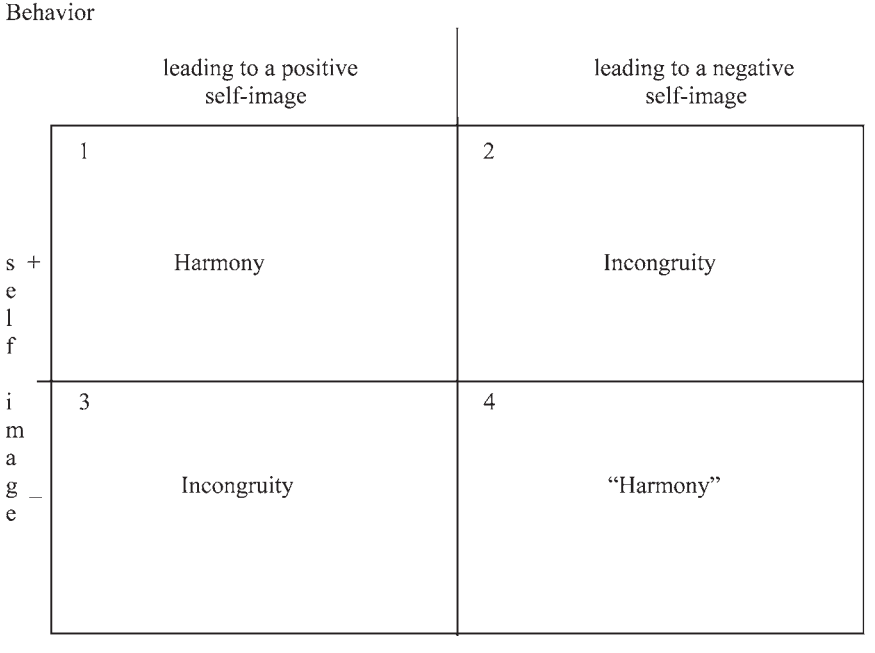

FIGURE 2.1 Behavioral incongruity.

many different "selves" and even if our "identity" changes depending upon the demands of the situation we are in for the moment, all of our identities are not equally important. The *self-image*, as I use the term, can be seen as the *governing self*.

A third complication with the figure is that there are degrees of positive and negative self-images. We can think of the matter as a continuum between those who look upon themselves as a short step under the deity, and those who regard themselves as Satan reincarnated. Depending on where a person stands on this continuum, a certain range of behavior will be acceptable to her; that is, her behavior will not cause incongruity as long as she keeps herself within this domain. However, if she should overstep these boundaries in either direction, that is, either act more positively or more negatively than her self-image will allow for, she will experience incongruity. She must then either discontinue the incongruous activity, place it within the framework of one of her small minds and therefore only be conscious of it in special circumstances, or incorporate it into her governing mind and thereby change her self-image to one that allows for such behavior. Figure 2.2 is a somewhat oversimplified model of these relationships.

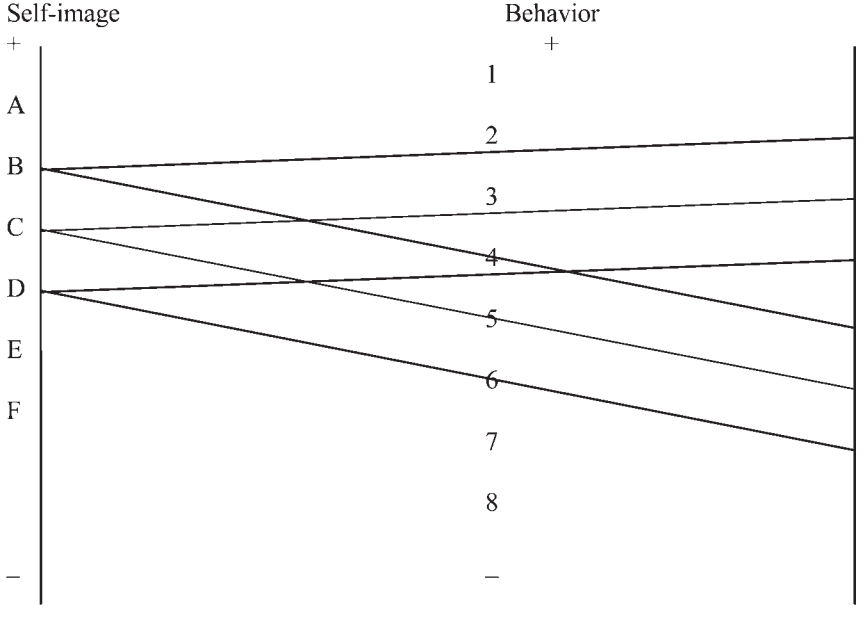

FIGURE 2.2 The relation between self-image and behavior.

An individual with a self-image at position C can behave between points 3 and 6 on the behavior scale without causing incongruity. If he should start acting according to point 2 on the behavior scale, he will either have to change his self-image in a positive direction at least as far as point B, or place the behavior in one of his small minds, or desist from such actions. For instance, upon becoming parents some of my research subjects tried to take care of their children. In doing so, they improved their self-images and this helped to somewhat regulate both their drug consumption and their behavior in general. Indeed, parenthood can be an important factor in getting off the drug scene entirely. But this is certainly not the case for all. Some research subjects who tried to fulfill their roles as parents saw parenthood belonging to a category separate from the rest of their lives. That is, successes in this role were placed in a small mind and not generalized to the self-image. These people tended to limit the amount of time they spent with their children, but when they were with their kids, they exerted themselves. However, it was difficult to see positive effects of this in other situations. And finally, there were those who after a period of time abandoned their children altogether. When I got to know these people well enough for them to confide in me, it became evident that they thought the worse of themselves for having done so. That is, failing to fulfill one's duties as a parent is so important that they didn't merely resume their previous negative self-image, they "devaluated" it further.

Correspondingly, if an individual should start behaving according to point 7 on the behavior scale above, he will either have to change his self-image in a negative direction at least as far as point D, or incorporate the behavior in one of his small minds, or discontinue such activity. Note that the behavior ranges for neighboring points on the self-image scale overlap. This is because people don't change their entire behavioral pattern at once, but rather a little at a time.

An individual with a negative self-image tends to behave so as to provoke others to confirm what he "knows" about himself rather than cause incongruity by acting in a way that would give responses leading to a more positive self-image. For this reason, it is difficult to change an established negative self-image. Negative reactions from significant others tend to modify the behavior of those who basically have a positive self-image, because these reactions are incongruous. For those who already have a negative self-image, admonishments are usually interpreted as a confirmation of what they already believe to be true, and therefore do not lead to behavior more in line with societal norms.

Yet another complication with Figure 2.1 involves the nature of the harmony in cell 1, as compared with the "harmony" in cell 4. In spite of their negative self-images, my research subjects grew up under the influence of mainstream

society and have internalized societal norms. Although some authors refer to the drug scene as a *subculture*, I don't find the concept relevant for my observations in Stockholm.[4] For a deviant subculture to arise and remain in existence, its members would have to neutralize the societal norms they have been bombarded with since birth. Although some researchers have gone so far as to itinerate specific methods for achieving this (e.g., see Sykes & Matza, 1957, p. 667ff.), I was unable to distinguish any significant degree of neutralization in my research subjects. When one merely listens to what problematic consumers say in interviews, it is easy to get the impression that they have different norms from the rest of society. But participation observation allows the researcher to establish relationships based on trust, and once this is achieved, one hears entirely different stories. Neither I nor a host of other researchers who have achieved a deeper contact with their informants report any significant degree of neutralization of societal norms (e.g., see Bourgois, 2003; Jonsson, 1973; Yablonsky, 1962). Not only do problematic consumers accept societal norms, but on the whole they tend to have very traditional values. This can be clearly seen, for instance, in what female problematic consumers have to say about sex roles (Lander, 2003, p. 155ff.) and motherhood (Trulsson, 2003, p. 80, 98).

Among the many tragedies in problematic consumers lives is that every day they break rules they themselves feel they should be following. Consequently, their actions cause self-reproach, which is added to all the negative reactions they get from other people. Therefore, the threat of having to devaluate one's self-image is ever present. Deviance bears within itself a certain degree of incongruence. I have indicated this by placing quotes around the word *harmony* in cell 4 in Figure 2.1. As a result of their inability to neutralize societal norms, problematic consumers experience a definite pull toward society; and almost all of those I met during my fieldwork did in fact take tentative steps in this direction on occasion. An example of this is attempting to find a job. If this tentative step is well received, it can initiate a reevaluation of the self-image. If the endeavor fails, it can be more difficult to try again.

In their early contacts with society, children who have been labeled by their parents exhibit the beginning stages of the provocative behavioral patterns easily recognized by everyone who has worked with marginalized people. Although the child's self-image is not firmly established, he can consciously break rules he is aware of, and in doing so provoke negative reactions that serve as a confirmation of what he believes he knows about what kind of person he is. The more frequently a child receives confirmation of his developing negative self-image, the more strongly established it becomes. It is therefore of primary importance that adults who interact with these children have a clear theoretical and/or emotional understanding of what the child is trying to convey with his

behavior, so that their reactions do not confirm what the child suspects (that there is something wrong with him) or even worse, become a part of the labeling process (that is, teach the child that he is even worse than he previously believed). Once again, the goal is to teach the child norms without labeling him; to react to what he is doing for the moment, but not against the child himself.

In recent years, politicians in practically all industrialized countries have been making budget cuts. To the extent that they decide to "save money" by withdrawing support from institutions and organizations where children and adolescents establish deeper relationships with adults other than their parents, politicians weaken a possible counterbalance to labeling responses in the home. Examples of such measures are increasing the ratio of children to personnel in community-run day care centers, cramming more pupils into school classes, reducing the number of adults working in schools, withdrawing economic support to youth organizations, cultural organizations, recreation centers, athletic clubs, and so on. With reduced resources, it becomes all the more difficult for the personnel to avoid labeling reactions when responding to children with provocative behavioral patterns, and it is therefore reasonable to expect that more of the children who run the risk of proceeding deeper into a deviant career will in fact do so.

Secondary Deviance

When a child has matured to the point where she has a reasonably clear understanding of societal norms, and has established a self-image based on a substantial number of life experiences, she can enter the *third stage* of the deviant career, *secondary deviance*. The age at which this ensues varies depending upon such factors as the amount and the severity of labeling in the earlier stages. For most of my research subjects, this third stage was established within a few years after becoming a teenager, but in extreme cases it occurred earlier.

Secondary deviance is characterized by acts contrary to societal norms that the individual is cognizant of. Secondary deviance is a product of a relatively well-established negative self-image and is a means of attempting to deal with manifest and/or latent problems arising from labeling. The child has become an adolescent. She has a relatively clear comprehension of right and wrong, and her behavior is increasingly influenced by her negative self-image.

Once a person has accepted that she is not accepted by others, normal social control does not function. People with a positive self-image can be brought to desist from certain behavior by negative reactions because these threaten both their view of themselves and their position as members of a group. But

a person with a negative self-image already knows that she is different and an outsider. She doesn't see negative reactions as a guideline to help her gain acceptance because her experiences have taught her that people like her cannot be accepted. So she interprets negative reactions as a further confirmation of how inferior she is. When a person has established a negative self-image, others cannot socially control her by threatening to withdraw love, friendship, or support, since she doesn't feel that she has any of this to begin with. As Bob Dylan succinctly put it: "When you ain't got nothing, you've got nothing to lose."

By defining certain acts as commendable and others as reprehensible, society shows its citizens how to maintain their self-images. For instance, the consumption of certain psychoactive substances is illegal (that is, deplorable). Those who have negative self-images know from their own experiences—the responses they have received from others—that people like them do unacceptable things. They are therefore attracted to illicit drugs. On the other hand, it becomes difficult for people with more positive self-images to consume these substances without experiencing behavioral incongruity. If these people choose to attempt to deal with life's problems by using chemicals, they will go to a doctor to get a prescription for legal drugs. They can do this without causing behavioral incongruity because it is legitimate to take medicine (even if the prescribed drugs should happen to have biochemical effects that are similar to those of narcotics).

If we were to remove current social and legal stigma from illicit substances, the pattern of consumption of these drugs would change. On the one hand, more people would be able to take them as doing so would no longer be experienced as incongruent. On the other hand, those who have a strong negative self-image would either have to take them in an unacceptable manner and/or find new ways to confirm their self-images.

Problematic consumers of narcotics have at least some, and most often all, of the following objectives when they take illegal substances:

1. confirmation of the deeply rooted negative self-image,
2. escape from their own and other people's expectations,
3. self-destruction,
4. revenge.

Due to labeling, problematic consumers of narcotics have drastically negative self-images, initiated prior to their starting to take illicit drugs. Others have deemed them inferior and they have accepted that such is the case. They try to flee, for instance, with the help of psychoactive substances, but they have already internalized the condemnation, and they can't escape from what they bear within. Due to all the negative experiences problematic consumers endure as an integral part of

the life they lead on the drug scene, they confirm for themselves that they deserve to be severely punished. After all, they destroy for others and have devastated their own lives. As time passes and the magnitude of negative life experiences escalates, they become all the more convinced that they are reprehensible and that they do not deserve to exist. Increasingly, their life-pattern becomes a process of ensuring that justice is done. Others have condemned them, they have accepted the verdict, and they become their own executioners. But at the same time, by stealing from them, frightening them, giving them a bad conscience, and so on, problematic consumers wreak revenge on those who have passed judgment.

People establish a self-image through others, and they must have help from others to modify it. If we want an individual to change her behavior, we must be willing to take the role of significant other and not only refuse to confirm the negative self-image but also give responses necessary to help her embark upon and endure the long process that leads to a more positive self-image. But this is easier said than done when the labeling process has gone so far that her experiences have taught her to expect negative responses from others. If she is suddenly given positive reactions, the world becomes incomprehensible to her. If, for instance, a social worker or some other significant other does not react as expected, she is looked upon as either being ignorant or simply not having understood what kind of person she is dealing with. This calls for extremely provocative behavior from the problematic consumer to generate the "correct" responses.

If significant others understand that they are being provoked, and are able to find responses that do not label the individual, this can initiate a reevaluation of the negative self-image. But if the provocations are "successful," which sooner or later they usually are, it leads to more labeling and little by little the individual embarks upon the fourth stage in the deviant career.

The Deviance Spiral

The *fourth stage* is called the *deviance spiral* and consists of the following elements, shown in Figure 2.3.

The problematic life experiences that constitute the labeling process create the negative self-image, which in turn leads to secondary deviance, which constitutes failure according to cultural definitions, which induces more labeling, and so on. Each new failure is partially the result of earlier failings but also contributes to future deviance. In this fourth stage, the individual's behavior is usually secondary deviant in several different areas, that is, consuming drugs, selling drugs, theft, prostitution, unemployment, not providing for one's children, homelessness, and so on. As a result, she is subjected to labeling reactions from many different sources.

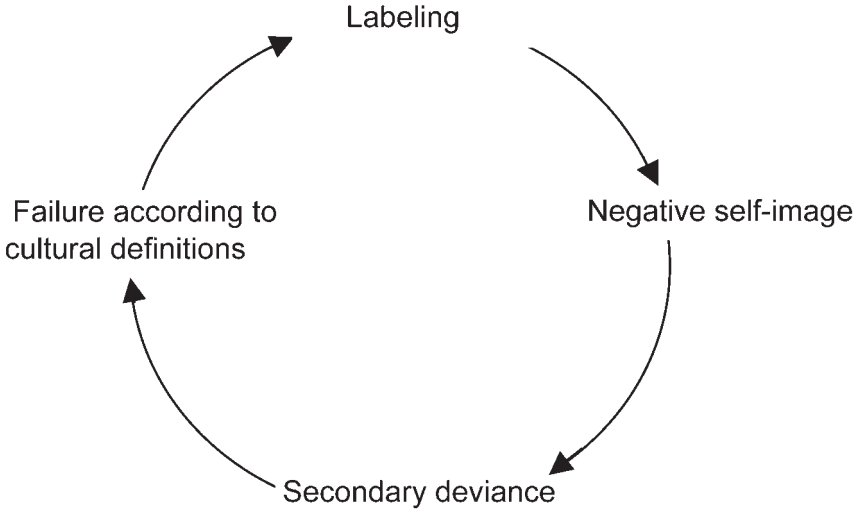

FIGURE 2.3 The deviance spiral.

The deviance spiral is a deterioration of the third stage. As is evident from the name and from Figure 2.3, the problematic consumer of narcotics is now caught in the vortex of a vicious spiral, drawing him downward. This occurs because, when failures and negative reactions accumulate, it becomes all the more difficult to maintain one's self-image at the same (low) level. The individual devalues her self-image still further, which leads to more serious secondary deviance, which other people define as even greater failure and respond to by further labeling, which leads to another devaluation of the self-image, which makes still more serious deviance possible, and so on.

Sooner or later, the deviance spiral leads to everybody who comes into contact with the drug scene getting to the point where they feel that they have had enough. Those who lack self-destructive tendencies usually come to this conclusion quickly and leave the scene shortly after arriving because they don't wish to subject themselves to the rampant negative experiences that surround them. For some people, it takes a little longer before they decide to leave. Only the most self-destructive people stay on the scene so long that they hit rock bottom. That is, they follow the deviance spiral downward to the point where they feel that only two choices remain: either get off drugs, or more directly, commit suicide (e.g., by taking an overdose or by "burning" a pusher who is both willing and able to have them killed). The following example from my fieldwork illustrates the deviance spiral.

When I met R. he was one of the biggest drug dealers in Stockholm. He was unemployed, took narcotics on multiple occasions every day, associated almost exclusively with problematic consumers, and regardless of what the subject of a conversation was initially, it was almost invariably narcotics before the discussion ended. Illicit substances were the hub around which just about everything in his life revolved.

One morning I received a telephone call from him. He was extremely agitated and asked me to come to his apartment. As most problematic consumers do on occasion, he had decided to reappraise his relationship to society. In this particular instance, he was going to try to get a job. When I got to his apartment, he was pacing back and forth nervously, smoking hasch. He told me he had applied for a position as a mailman and that he had an interview at 1:00 P.M. He was certain that nobody would ever employ him, and he was extremely nervous. (In the short run, it was a no-win situation for him. On the one hand, if he was not offered a job, the rejection would have been a blow to his already lacerated self-image. On the other hand, getting a job would have been incongruent to this same self-image.) R. spent several hours smoking one joint after the other and repeating that he wasn't going to get the job. By the time he left for his interview, he was hardly able to communicate. My interpretation of his behavior is that he put himself into a position where if he didn't get the job, he could protect his self-image by claiming that it was because he inadvertently had gotten too stoned.

Amazingly enough, he was given the position, presumably because at the time there was a drastic shortage in the work force in Sweden and the postal authorities were probably desperate.

The most common scenarios when problematic consumers manage to get a job are that they either don't go to it at all, or they show up only sporadically, or they do the job so poorly that they are fired, or they get into conflicts with coworkers and are let go for that reason. Put simply, having a job and doing it well is drastically incongruent to their self-images. With the help of provocation, they get themselves fired and confirm for themselves and others that they are not worthwhile members of society. To illustrate these points I return to R.

On R.'s postal route there were many older four-story walk-ups with only two apartments on each floor (in Sweden the mail is put through a slot in the door of each apartment). One day R. started a conversation with me by saying something to the effect that one of the biggest problems in Sweden is that people are lonely. As he is something of an amateur sociologist, R. presented a long analysis of, among other things, the breakdown of social relationships within the extended family as a result of urbanization. When he finally got to the point, he told me that he had taken it upon himself to do something to help people break their social isolation. In short, he delivered all the mail for each building to just one of the apartments (on the ground floor, of course). That way, he explained,

neighbors would have an excuse to ring each other's doorbells and get to know one another.

On several occasions R. told me that some days he couldn't be bothered delivering the mail at all and just threw it into the bushes. As I never actually saw him at this job, I don't really know to what extent he was exaggerating when he spoke of his way of performing his duties. However, I do know that when he had a large shipment of drugs to sell, he didn't go to work at all. He claimed that he usually just didn't show up without calling in sick.

If some of all this is true, R.'s behavior can be seen as an example of the way problematic consumers use provocation to get others to confirm their negative self-images. Normally, an employee who acts the way R. said he did would be asked to leave. However, R. was not fired.

About six months after he started working as a mailman, R. sold an exceptionally large amount of narcotics and had more cash than he'd ever had before. He became concerned about keeping so much money in his apartment while he was waiting for his suppliers to return to Sweden to collect what he owed them. In his own words, he was getting paranoid about the police being on to him, so he made a deal with a problematic consumer he considered a friend that this person would be paid to keep the cash in his apartment. To make a long story short, two of R.'s "friends" who knew about the money stole almost all of it and went off on a spree in southern Europe. R. was left holding the bag and had to pass on the burn. Luckily for him, his suppliers accepted his explanation, because if they hadn't, he would have been killed, as he could not possibly have gotten hold of so much money. However, his problems were far from over. As R. explained it, he now either had to have the two people who burned him murdered or stop dealing; because if word got around that you could get away with cheating him, R. would be ripped off time and again, and he would have to pass on the burns. Sooner or later (presumably sooner), his suppliers would no longer accept any excuses and they would have him eliminated.

In our conversations during this period, R. repeatedly told me that he was many (bad) things but he wasn't a murderer. However, to maintain his position on the drug scene, he now had to become one. To have somebody killed was drastically incongruent with his self-image and he didn't want to do it, but what would/could he do if he were to leave the scene? R. was tormented and frightened but he could not bring himself to sentence two people to death. Instead he fled to the Far East. He had been out there before but this time it wasn't the same. Even this dream was tarnished, and he returned to Stockholm after about a month.

Before leaving Sweden, R. quit his job as a mailman—but resigning is a very different matter than being fired. He had kept a job for more than half a year and could have continued even longer. Becoming a member of society was evidently not as impossible as it once seemed. At the same time, resuming his position on the scene was not really an option as he would literally have to kill to do so. Upon returning to Sweden, R. quickly found a new job and this was one of the major

steps in the long process of getting off drugs. Instead of continuing downward in the deviance spiral, R. managed to reverse the process. But if he hadn't already established that he could hold a job, his choices may well have been reduced to killing others or himself. To choose the former would be a major step downward in the deviance spiral. The latter is a choice made by all too many problematic consumers.

When he has hit rock bottom, the individual's small mind that wants to join society but that has been held back, is given higher priority. But if he fails now, all that remains is death. It is therefore not advisable to let the deviance spiral go this far. By not confirming the problematic consumer's negative self-image (e.g., by understanding his provocations and finding nonlabeling responses to them), we can help him break the negative spiral much earlier, preferably before it starts to gain momentum.

As it may be difficult to understand how it is practically feasible to do this, an example may be illuminating. A. S. Neill, former headmaster of Summerhill, a school in England that had among its pupils many "problem children" who had been expelled from other schools, and that used very unusual pedagogical methods, writes: "If I should be painting a door and Robert came along and threw mud on my fresh paint, I would swear at him heartily, because he has been one of us for a long time and what I say to him does not matter. But suppose Robert had just come from a hateful school and his mud slinging was his attempt to fight authority, I would join with him in his mud slinging because his salvation is more important than the door. I know that I must stay on his side while he lives out his hate in order for him to become social" (Neill, 1960, p. 119).

We can summarize my career model by comparing *two extreme kinds of careers*. Both begin with primary deviance, but the reactions received make the careers go in diametrically different directions.

Culturally successful career	Deviant career
1 Parental acceptance	1 Parental labeling
2 Societal acceptance	2 Societal labeling
3 Normative behavior	3 Secondary deviance
4 Personality enhancement	4 Deviance spiral

The stages in the careers should not be considered completely separate. They can be in progress concurrently and can either reinforce or counteract each other. For instance, parental labeling begins before societal labeling, but can continue for years after the child has made many societal contacts. It is also not necessarily so that an individual will be subjected to the same kinds of reactions from both parents and society, as is the case in the two extreme careers outlined

above. Labeling reactions from certain significant others can be counteracted by acceptance from others; in which case, the emerging self-image will neither be extremely positive nor extremely negative (see Figure 2.2). In other words, the number of possible careers far exceeds the two mentioned above. In a welfare state such as Sweden, few people experience the kind of extreme deviant career described here, which makes its prevalence among problematic consumers of narcotics all the more noteworthy.

NOTES

1. Obviously, one can choose to nuance even further. For instance, a report from a governmental commission in the United States distinguished between five different patterns of consumption (see Wurmser, 1995, p. 7).
2. *Paranoid*, as it is used on the drug scene, is slang and should not be equated with its scientific namesake. As in all slang, the concept is not clearly defined. Paranoid denotes vague fears, terrifying experiences, and feelings of persecution. The word is frequently used by problematic consumers because being *paranoid* is a prevalent part of their everyday existence.
3. Logically, one could also think of a positive self-image as being the result of a positive labeling process. However, in this chapter, labeling denotes a negative process.
4. *Subculture* may possibly be a relevant term to describe, for instance, people from eastern Africa who continue to chew *khat* while living in Europe, or Iranians who after emigrating, continue to smoke an opiate on special occasions. While this behavior is not accepted in the majority culture, it is within the bounds of the individual's culture of birth. So in the context of the majority culture the individual is now living in, this might be considered a drug subculture. However, these types of culturally regulated consumption have little to do with the behavior of problematic consumers. If a Somali should chew *khat* or an Iranian should smoke heroin at the expense of his other duties, he would no longer be acting within the framework of his culture and his fellow-countrymen would find his behavior unacceptable.

REFERENCES

Becker, H. S. (1963/1973). *Outsiders*. New York: Free Press.

Bejerot, N. (1970). *Addiction and society*. Springfield, IL: Charles C. Thomas.

Bejerot, N. (1979). *Missbruk av alkohol, narkotika och frihet* [Abuse of alcohol, narcotics and freedom]. Stockholm: Ordfront.

Bourgois, P. (2003). *In search of respect: Selling crack in El Barrio*. Cambridge, UK: Cambridge University Press.

Chein, I., Gerard, D. L., Lee, R. S., & Rosenfeld, E. (1964). *The road to H: Narcotics, delinquency and social policy*. New York: Basic Books.

Dole, V. P., & Nyswander, M. E. (1967). Rehabilitation of the street addict. *Archives of Environmental Health, 14*, 477–480.

Goldberg, T. (1999). *Demystifying drugs: A psychosocial perspective*. New York: St. Martin's Press.

Inciardi, J. A. (1992). *The war on drugs II*. Mountain View, CA: Mayfield.

Israel, J. (1984). *Sociologi: Inledning till det kritiska samhällsstudiet* [Sociology: Introduction to the critical study of society]. Stockholm: BonnierFakta.

Jonsson, G. (1973). *Att bryta det sociala arvet* [Stopping social inheritance]. Stockholm: Folksam.

Lander, I. (2003). *Den flygande maran: En sudie om åtta narkotikabrukande kvinnor i Stockholm* [The floating hag: Ethnography of eight drug using women in Stockholm]. Stockholm: Kriminologiska inst., Stockholms Universitet, Avhandlingsserie nr 11.

Levine, H. G. (2003): Global drug prohibition: Its uses and crises. *International Journal of Drug Policy, 14*, 145–153.

Mills, C. W. (1997). *Den sociologiska visionen* [The sociological imagination]. Lund: Arkiv.

Neill, A. S. (1960). *Summerhill: A radical approach to child rearing*. New York: Hart.

Nordegren, T., & Tunving, K. (1984). *Hasch: Romantik och fakta* [Hasch: Romanticism and facts]. Stockholm: Prisma.

Ornstein, R. (1986). *Multimind*. London: Macmillan.

Ornstein, R. (1991). *The evolution of consciousness: Of Darwin, Freud and cranial fire: The origins of the way we think*. New York: Prentice Hall.

Peele, S. (1998). *The meaning of addiction: An unconventional view*. San Francisco: Jossey-Bass.

Puhakka, O. (2006). *Missbrukare på Sergels Torg 2005* [Misusers in Sergels Torg, 2005]. Stockholm: Stockholms stadsledningskontor, Forsknings- och utvecklingsenheten, FoU-rapport 2006:1.

Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Research Review, 18*, 247–291.

Shanks, E. (2008). *Utländsk bakgrund och tungt narkotikamissbruk—En kartläggning av levnadsförhållanden* [Foreign background and problematic consumption—An investigation of life experiences]. Stockholm: Rapport i Socialt Arbete nr 125, Institutionen för Socialt Arbete—Socialhögskolan, Stockholms Universitet.

Shedler, J., & Block, J. (1990). Adolescent drug use and psychological health: A longitudinal inquiry. *American Psychologist, 45*, 612–630.

Solarz, A. (1990). *Vem blir drogmissbrukare? Droger, kriminalitet och kontroll* [Who becomes a drug misuser? Drugs, criminality and control]. Stockholm: Brårapport 1990:3, Brottsförebyggande Rådet.

Sykes, G. M., & Matza, D. (1957). Techniques of neutralization: A theory of delinquency. *American Sociological Review, 22*, 664–670.

Trulsson, K. (2003). *Konturer av ett kvinnligt fält: Om missbrukande kvinnors möten i familjeliv och behandling* [Contours of a female domain: On female misuser's experiences of family life and treatment]. Lund: Lund dissertations in Social Work 12, Socialhögskolan, Lunds Universitet.

Wurmser, L. (1995). *The hidden dimension: Psychodynamics in compulsive drug use*. New York: Jason Aronson.

Yablonsky, L. (1962). *The violent gang*. New York: MacMillan.

This page intentionally left blank

Chapter **3**

Adaptation and Addiction

Ann N. Dapice, PhD

Addiction has been a problem for thousands of years. It involves lasting changes in brain function that may be difficult to reverse. The numbers of "altered brains" include nearly 2 million heroin and cocaine addicts, some 15 million alcoholics, and tens of millions of cigarette smokers in the United States alone (Harvard Mental Health Letter, 2004). Globally, the use of drugs has reached all-time highs (Saah, 2005). As bad as these numbers are, they do not include a far larger array of addictions and numbers of the addicted worldwide.

Research increasingly demonstrates the similarity of, and connections to, a variety of conditions that appear addictive in nature. Some addictions are to external substances (e.g., alcohol, tobacco, cocaine). Some are to external behaviors that stimulate pleasure pathways in the brain (e.g., gambling, online pornography, stalking, and shopping). Some addictions are legal (e.g., food, tobacco, alcohol, prescription drugs, and shopping). Some are not (e.g., cocaine, methamphetamines, stalking). Some substances such as food, alcohol, and prescription painkillers can be used nonaddictively by some people but not by others, and some activities such as gambling, sex, and shopping can be engaged in nonaddictively by some but not others. The consequences of addictions on the addicted and those who are negatively affected by these addictions vary greatly—from chronic disease and slow death, to immediate and sometimes violent death. The social and financial costs of these addictions eventually affect us all. Meanwhile, prevention, intervention, and treatment for addictions, as well as punishment for illegal addictions also vary greatly. Activities such as eating and sexual intercourse are necessary for human survival. In fact, addictions

can be seen as originating in behaviors and substances that were once adaptive for the survival of humankind (Dapice, 2006; Fisher, 2004; Popkin, 2007).

Organisms that develop beneficial adaptations increasing the likelihood of their survival are more likely to pass on adaptive genes. The brain's reward system reinforces important behaviors—eating, drinking, sleeping, engaging in sex, but it is easily misled. Computers, the Internet, slot machines, and money can all activate the same regions of the brain as psychoactive drugs (Breiter, Aharon, Kahneman, Dale, & Shizgal, 2001). A recent brain scan study showed that when men were shown erotic pictures, they were more likely to make a larger financial gamble than if they were shown something frightening like a snake, or something neutral like a stapler. The arousing pictures would light up the same area of the brain that lights up when financial risks are taken. This was described as related to the evolutionary need that men have for both money and women. The study was designed to determine cause and effect. The erotic pictures lit up the area in the brain and risk taking followed. When photos of snakes and spiders were shown to the participants, the portion of the brain that is associated with pain, fear, and anger lit up and people were likely to keep their bets low (Knutson, Wimmer, Kuhnen, & Winkelman, 2008). People have different sets of risk factors: neurobiological, psychological, and social. Regardless of the risks to addiction, it is necessary to have exposure to an object of addiction that stimulates a positive experience in the user—relief of discomfort, or creation of pleasure (Walsh, 2007). What causes relief or pleasure varies greatly with the individual and thus the kinds of addictives vary as well.

Food and tobacco are connected to other addictive substances in important evolutionary and physiological ways. Understanding the co-development of mammalian brains and ancient psychotropic plants, as well as the implications of ancient psychotropic substance abuse in altering mammalian brains, helps assess the causes and effects of addiction in today's world. It allows us to come closer to treating the root of the addiction, not the symptoms alone (Saah, 2005). Processes formerly necessary for our safety, well-being, and survival may now serve to make and keep us ill. These include (1) the so-called "thrifty gene" (Popkin, 2007, p. 91), (2) mechanisms for fight and flight (Dapice, 2006; Dapice, Inkanish, Martin, & Brauchi, 2002), and (3) mechanisms for continuation of the species (Fisher, 2004). Our bodies and brains continue to function according to hunting and gathering needs appropriate thousands of years ago.

THRIFTY GENE

Contrary to expectations, skeletons exhumed from 10,000 years ago on the American continent were seen to be healthier than those found since horti-

culture began (Steckel & Rose, 2002). Early humans stored food substances through the production of insulin in response to the ingestion of carbohydrates. This allowed for fat storage in the body to protect people during winter and times of famine. Similarly, the body and mind sought to limit the amount of energy expanded through physical activity. One ate large amounts when foods were ripe and available, and exercised only as needed for existence (Dapice, 2006, p. 253; Flier & Maratos-Flier, 2007, p. 72; Ozelli, 2007, pp. 84–85).

Food sources became drugs to prevent "decreased fitness" due to starvation and death. Addictive drugs share with "palatable food" the property of increasing extracellular dopamine (Di Chiara & Bassareo, 2007, p. 233). Nicotine, cocaine, and ephedrine sources were first mixed with an alkali substance, most often wood or lime ash, creating a "free base to facilitate diffusion of the drug into the blood stream" (Saah, 2005). Traditionally, American Indians processed corn in a variety of ways using lye or lime ash. Science has since discovered that this process used with corn was required to release complete amino acids and to provide the niacin required to regulate blood sugar. Different tribes had different recipes for treating corn using this method (e.g., Muskogee Creek "softkey," Cherokee "hominy," etc.). Some tribes still have official "corn lyers" (Dapice, Inkanish, Martin, & Montalvo, 2001).

The benefits of civilization have given us mass production and preservation of foods, tobacco, and other substances through agriculture, the industrial revolution, and global transportation. Substances such as tobacco, once rare and localized to a particular geographical area, required first slave labor and then industrialized technologies to produce in amounts sufficient for addiction. Tobacco, once scarce and used for ceremony only, is now plentiful, used in addictive ways, and kills in large numbers worldwide. The World Health Organization estimates that smoking kills more than 4 million people a year. This figure may rise to 10 million per year by 2030 because of rapidly growing tobacco use in developing countries (HealthCentral.com, 2000). As it happens, preservation of tobacco, which includes large amounts of sugar, adds to its addictive qualities by appealing to the brain's natural survival need for carbohydrates (Bennett, Howell, & Doll, 1970).

While tobacco use remains in first place with respect to fatal forms of substance abuse, obesity is rapidly moving ahead as the number-one killer (Tanner, 2004). Research demonstrates that brain circuits involved in drug addiction are also activated by the desire for food. The right orbital-frontal cortex is involved in compulsive behaviors characteristic of addictive states and this same brain region is activated when addicted individuals crave drugs such as cocaine. Food stimulation increases levels of dopamine and when obese individuals were examined, they were found to have fewer dopamine receptors—as has been found

in addiction to other substances (Brookhaven National Laboratory, 2004). It is, of course, important that food be satisfying for the survival of the species. Having fewer receptors increases the level of food intake, when possible, and this was once adaptive during winter and famine (Dapice, 2006; Ozelli, 2007). However, in a new understanding of how people become addicted to food, researchers note that taste and palatability are not the only factors since, when the ability to taste is removed, the same dopamine pathways are stimulated by calories (Andrews & Harvath, 2008; De Araujo et al., 2008). Popkin notes that the rapid globalization of obesity has been encouraged by marketing of unhealthy foods worldwide and the food industry has insisted that governments "should not restrict an individual's dietary choices" (2007, p. 93).

The American Cancer Society notes that while a third of the nation's 50 million smokers attempt to quit each year, not unlike statistics on treatment effectiveness in alcohol and drug addiction (Mathews-Larson, 1991; National Institute on Alcohol Abuse and Alcoholism [NIAAA], 1996; Polick, Aarmor, & Bracker, 1980), fewer than 5 percent succeed (Bartosiewicz, 2004). It has been observed and reported (Morgan, 2003) that smokers who try to stop tobacco use through patches and gum become addicted to these as well (Bartosiewicz, 2004). Research has demonstrated that drinking alcohol improves the enjoyment of tobacco. On the average, more than 85 percent of adults with a history of alcohol abuse smoke, and they may be more addicted to nicotine than are smokers without a history of drinking (Abrams, Monti, Niaura, Rohsenow, & Colby, 1996). Carl Anderson, researcher at Harvard University Medical School, reported in a personal communication (September 2004) that the amino acid, L-Glutamine, is missing in the brains of smokers, but not in the brains of those who can take or leave tobacco products. L-Glutamine also helps regulate blood sugar and related cravings for carbohydrates and alcohol (Mathews-Larson, 1991). Again, the cravings can be seen to be adaptive when it is desirable that one take in more of a substance than is needed at the time.

It is known that EEG brain waves are shaped genetically and researchers have demonstrated that reduced amplitude correlates with alcohol dependence. They have identified the chromosomal region that affects the P300 electrical brain wave, previously correlated with alcohol craving and predisposition to relapse. These EEG abnormalities are true both of alcoholics as well as their "alcohol-naïve" (never used) offspring (Begleiter & Porjesz, 1988; Propping, Kruger, & Mark, 1981; Tabakoff & Hoffman, 1988; Volavka, Pollock, Gabrielli, & Mednick, 1985). Studies at the University of Connecticut by Lance Bauer show that relapse to alcohol, cocaine, and opioid dependence can be predicted by brain waves. The high-frequency activity on EEGs was found to far outweigh clinical and demographic variables as a predictor of relapse (Bauer,

2001). Alcoholic P300 brain waves appear as "jagged mountains" compared to the normal appearance of gentle "foothills."

Alcoholism among some groups is partly related to lack of adaptation to certain foods. Alcoholism is highest among peoples who received grains such as wheat, barley, and oats relatively late—Russians and northern Europeans. American Indians have had even less time to adapt to these foods (Mathews-Larson, 1991). Research also shows that hypoglycemia affects up to 95 percent of alcoholics, causing them to become irritable, angry, depressed, hostile, and crave carbohydrates in the form of food or alcohol. Consuming carbohydrates in various forms serves to relieve symptoms temporarily, but with surges of insulin the cycle continues. In the alcoholic community, this phenomenon is known as "dry drunk" and may be observed whether or not individuals have ever used alcohol. Diabetics often experience these same hypoglycemic symptoms and are taught how to respond appropriately with diet (Bell & Martin, 2002; Mathews-Larson, 1991). The cravings are the key to this once-adaptive behavior.

FIGHT/FLIGHT

The fight/flight physiological process protected our ancestors from predators. The mammalian drive to escape danger is driven by the ability to feel negative emotions (Saah, 2005). However, fight/flight responses become destructive when such actions are inappropriate to the situation or when the stress or danger becomes chronic. Cortisol, produced during these times, becomes toxic to cells in the body and the brain, killing brain cells and leaving depression in its wake (Sapolsky, 1996). Especially important is the relationship of stress, cortisol, and predisposition to self-medication. As noted by the National Institute of Drug Abuse (NIDA) in a special issue on stress and drug abuse (2002), studies in the *Journal Psychoneuroendocrinology* indicate (1) stress and cortisol sensitize animals for drug-seeking behaviors and facilitate self-administration, (2) animals that are underaroused and have low levels of cortisol are more prone to develop drug-seeking behaviors, (3) severe stress early in life induces a series of physiological, neurobiological, and hormonal events that result in dysregulation of biological reward pathways in the central nervous system and in stress response systems; these changes seem to prompt self-administration of drugs and alcohol later in life, (4) prenatal exposure to stress and drugs predispose animals to drug-seeking behaviors in adulthood, (5) posttraumatic stress disorder (PTSD) is a risk factor for substance abuse, and (6) the administration of cocaine to humans causes similar physiological reactions such as secretion of adrenalin and cortisol, and psychological reactions similar to arousal caused

by stress. Researchers at the Scripps Research Institute in California (Koob, 1999) observed that heavy drinking not only depletes the brain's supplies of neurotransmitters necessary for feelings of well-being and pleasure (dopamine, serotonin, GABA, and opioid peptides), but it also promotes the release of cortisol. This release of cortisol causes tension and depression, which in turn causes the individual to drink more, leading to an ongoing vicious cycle.

Child abuse, neglect, sexual abuse, and verbal abuse cause damage to the cerebellar vermis in the brain, causing electrical irritability that the brain attempts to quell physiologically and the individual attempts to alleviate by abuse of alcohol and drugs. This area of the brain is extremely sensitive to stress hormones (Anderson et al., 1999). Brain imaging technology (Teicher, 2002) demonstrates that there are three major changes observed in the brains of adults who were abused as children: (1) limbic irritability with increased incidence of clinically significant EEG abnormalities, (2) deficient development of the left hemisphere of the brain (throughout the cerebral cortex and hippocampus), and (3) deficient integration of the left and right hemispheres of the brain with diminished development of the middle portions of the corpus callosum that serves as a bridge connecting the left and right brain. These lateralization changes in the brain are similar to those found by many other PTSD researchers. Essentially, the right brain takes control with negative affect and related behaviors (Bremner et al., 2000; Dapice et al., 2002; Sapolsky, 2000). However, the cerebellum has the potential architecturally to continue communication between the left and right brain with the administration of electrical stimulation (C. M. Anderson, personal communication, September 2003). Anderson (2001) has also noted a convergence of data suggesting that abnormalities in the cerebellar vermis may be involved in a wide array of psychiatric disorders, including depression, substance abuse, and ADHD. In *Nearness of Grace*, Arnold Mandell writes that exaggerated pruning of unused neural connections as a result of high levels of stress hormones leads to an extremely reduced range of potential behavior, which results in individuals "who lie without reason, get drunk, binge on promiscuity, steal unneeded things, or withdraw into interpersonal isolation" (2005, p. 30).

The relationship between stress and addiction is not limited to humans. Recent research among primates by Michael Nader of Wake Forest University has demonstrated the impact of unequal power on the one with power—and the ones without. Socially dominant male monkeys showed a brain chemistry change that encouraged resistance to using drugs such as cocaine. This alteration actually increased the number of dopamine receptors. Male monkeys at the bottom of the pecking order displayed *no* boost of the dopamine receptors and readily self-administered large amounts of cocaine (2002, p. 53).

CONTINUATION OF THE SPECIES

An example of yet another normal adaptive response gone awry can be seen in the case of romantic love (Fisher, 2004) and stalking behavior. Unlike addictive substances that are taken into the body, it is an example of external events that stimulate internal responses leading to addiction (Fisher, 2004; Meloy & Fisher, 2006). As an addiction, stalking shows all the symptoms—"tolerance, dependency/craving, withdrawal and relapse" (Meloy & Fisher, 2006, p. 364). Because it serves as a paradigm example of addictions that are not substances taken into the body, because more than 1 million women and nearly 400,000 men are stalked every year in the United States alone, and because stalkers are the most lethal of all criminals (Meloy, 2006, p. 172), space will be taken here to discuss how such an addiction works in the brain.

Stalking seems to originate with once-adaptive mechanisms for mating and reproduction—the sex drive or lust (testosterone), attraction or romantic love (dopamine), and attachment or companion love (vasopressin and oxytocin). The sex drive motivates people to consider a variety of possible partners for survival of the species. Attraction causes people to focus their energies on a specific individual. Attachment motivates people to remain in a relationship long enough to raise their offspring. However, these mechanisms in an individual with personality disorders such as narcissism or borderline personality combine in dangerous and often lethal ways. Stalking perpetrators often have attachment problems from early in life that may be the result of parental loss, neglect, abuse, or abandonment, and may also be related in some cases to genetic defect (Meloy, 2006, p. 278).

Brain imaging studies of stalkers demonstrate elevated activity of dopamine in the reward/motivation system. This activity produces focused attention and unwavering motivation and goal-directed behaviors. These are associated with other feelings, including "exhilaration, increased energy, hyperactivity, and sleeplessness." As noted above, this system can be stimulated by a number of phenomena, including money and cocaine. Activation of these pathways is most likely related to several traits of the "spurned or unrequited" stalker. This response includes heightened energy and intense motivation to "pursue the victim" (Meloy & Fisher, 2006, p. 357). Deactivation of other brain responses may be part of the problem. The right amygdala, involved in fear and other negative emotions, is deactivated and this may cause stalkers to be unable to pay attention to the dangers of their actions.

Another shared characteristic of lovers and stalkers is their obsessive thoughts about the loved one. They report that they cannot get the obtrusive thoughts out of their minds. This is linked to the suppressed activity of central

serotonin since research links low serotonin to obsessive thoughts. Low serotonin is also linked to another characteristic of lovers and stalkers—impulsivity. There seems to be a negative feedback loop between dopamine and serotonin. Low serotonin elevates dopamine activity and elevated dopamine suppresses serotonin. As the stalker feels energy, attention, and motivation to pursue the victim, rising levels of dopamine suppress serotonin leading to more obsession and dysphoria. As obsession continues, dopamine further lowers serotonin. Other brain systems combine with these processes to produce the symptoms of "energy, impulsivity, dysphoria, fearlessness, and obsession" (Meloy & Fisher, 2006, p. 359). Observers note that these individuals often appear to be on methamphetamines—even when they are not.

Failed in attachment from childhood, caught in the negative feedback loop of increasing dopamine and decreasing serotonin, along with increasing levels of stress cortisol, continued rejection by the victim, all this sets in motion a frustration-attraction response that may increase and sustain the stalker's ability to stalk. They may also experience abandonment rage, which happens when an expected reward is in doubt or unobtainable, stimulating the amygdala in the brain and triggering rage. Both romantic love and rage have a great deal in common. Both produce obsessive thinking, focused attention, motivation, and goal-directed behaviors desiring union or revenge (Meloy & Fisher, 2006, p. 361). When cortisol has been stimulated over time, the levels become abnormally low and unlike previously thought, it is low cortisol, not testosterone, that is related to violence in bullies in school and prisoners in jails (MacKeen, 2000).

Fisher and Meloy point out that stalkers are in a state of addiction to their own chemicals. They are seen to relapse in the same way as addicts to cues such as people, events, and songs (Meloy & Fisher, 2006, pp. 364–365). One perpetrator said, "She was like a drug . . . that I needed . . . my high was being with her. . . . I felt like dying when not with her." After the victim obtained a protective order, he murdered her. He said he let his obsession ruin his life. "I lost it all because of my obsession. . . . This obsession was bad. . . . It was like being in heaven and in hell at the same time" (Kienlen, Birmingham, Solberg, O'Regan, & Meloy, 2006, p. 140). It is important to notice the narcissism that focuses only on what happened to him—not the woman he killed. In spite of the fact that stalkers may be one of the worst offenders due to their intelligence and violence, they are rarely arrested for their crimes (Meloy, 1998, p. 3; Meloy, Cowet, Parker, Hofland, & Friedland, 2006, p. 143). Stalking behaviors sometimes continue for years, leaving victims in chronic acute traumatic stress where the impact of cortisol damage to victims' brains and other organs is only now beginning to be realized. Stalking behavior needs to be stopped for the sake of both perpetrators and victims.

OTHER BEHAVIORAL ADDICTIONS

Necessary risk taking, mating, and exchange of needed goods are all important to continuation of the species. In "Behavioral Addictions: Do They Exist?," Constance Holden (2001) noted that gamblers get high, show tolerance, have withdrawal symptoms—like drug addicts. Holden gave as example the work of Anna Rose Childress whose brain imaging showed sex addicts to resemble cocaine addicts. Further, Internet abuse is seen as the country's fastest-growing addiction with people addicted online to the same things they are addicted to offline—gambling (including short-term trading), pornography, and shopping, Holden referred to psychiatrist Susan McElroy's statements that the form addictions take has a lot to do with gender. "Men are overwhelmingly represented among sex 'addicts' and outnumber women by about 2 to 1 in gambling and substance abuse." Women are prone to what she calls the "mall disorders"—eating, shopping, and kleptomania—where the ratio of females to males in kleptomania is 2 or 3 to 1 and perhaps 90 percent of compulsive shoppers are women. Bulimia, which is characterized by binging and vomiting, is also seen to be an addiction, unlike anorexia, which involves rigidly controlled behavior and no "high" (Holden, 2001, pp. 980–982). The bulimia-anorexia difference is similar to distinctions between battering and stalking, where stalking is a form of addiction, while brain imaging of batterers demonstrates a misperception of cues causing batterers to mistakenly feel under attack and respond in misplaced attempts to protect themselves (George et al., 2000). Other behaviors such as working and jogging are normally adaptive, but in exaggerated form can take over with the same compulsivity as any other addiction.

A curious example of substance-interaction with addictions thought to be behavioral is the relatively new finding that a prescription drug for "restless leg syndrome" (RLS) can result in a variety of behavioral addictions. Television commercials can be heard regularly listing "side effects" such as pathological gambling, compulsive eating, increased alcohol consumption, and sexual obsession (Mayo Clinic, 2007). What is more interesting is that it is not so widely recognized that over-the-counter diphenhydramine (Benadryl), found in a number of drug combinations for sleep and colds, can elicit RLS (Rye, 2005). The "addictive" behaviors will end with the removal of the RLS medication—and may not begin if diphenhydramine is not taken in the first place.

DISCUSSION

Behaviors once critical to the survival of our species relied on the limits of the environment for regulation. With these limits removed by modern agriculture, transportation, and global trade, we are left with no internal controls

(Saah, 2005) to regulate behaviors where often endless excess is available. In essence, the brain has no brakes. Technology has changed more rapidly than evolutionary adaptations (Walsh, 2007). Drugs of abuse not only stimulate areas of the brain that have evolved to encourage adaptive behaviors, they stimulate these areas more effectively than the survival behaviors themselves (Di Chiara & Bassareo, 2007). In nature, rewards usually come only with effort and after a delay (Harvard Mental Health Letter, 2004). Addictive substances and behaviors provide shortcuts to feeling "fit." Meanwhile, nature and nurture are related in that fight/flight stress makes the brain think famine is coming and so substances are taken in excessively to prepare for the coming "famine." Stress may also exaggerate behaviors that were once adaptive such as sex and risk taking. It is these once-adaptive and interrelated behaviors that now threaten our worldwide health and survival at ever increasing rates.

The understanding of addiction then is complicated and requires both breadth and depth of knowledge from a variety of disciplines if we are to begin to understand its nature. Addiction is no longer about a limited number of substances and people. Many of us, if not most, are addicted to one substance or behavior or another—including the substances and behaviors that are the essence of living itself. The processes that evolved to protect us often do not. It is a new world of addiction—literally and figuratively. Our human "civilization" has made us successful beyond all imagination. First, we learned to gain at least some control of our food supply by planting seeds into the ground so we would not be limited to gathering what Mother Nature provided alone. Instead of hunting untamed animals, we would eventually learn to tend, fence, and breed animals we would depend upon for meat. These actions alone would not prevent hunger from times of famine, but we would also learn to trade with other groups for what we needed but did not have. The movement from hunter-gatherer societies to ones that were pastoral and horticultural required larger families to do the work—as it also provided more food than might once have been available. Larger families would move people from hunter-gatherer egalitarian groups to hierarchical systems where some had more power than others. Eventually, this "civilization," across large societies worldwide, would find slaves helpful in accomplishing even greater amounts of work—allowing others to assume special privilege of class and power. The industrial revolution would come to make slavery no longer necessary, but class and unequal power would generally remain.

Thus the poor may have more abundance than in the past, but it is generally of low quality, full of bad fats and refined carbohydrates that lead to obesity, Type 2 Diabetes, cardiovascular disease, and cancer. Therefore, in what the Food and Agriculture Organization of the United Nations has called the

"double burden" of malnutrition, undernutrition exists side by side with rapid rise in overweight people and obesity and related chronic diseases (Spotlight, 2006). Based on a recent study, Dr. Michele Companion of the University of Colorado describes the same phenomenon occurring with American Indians in the United States—as well as in the developing nations of the world (*Native American Times*, 2008).

Human brains seem not to have evolved much from hunter-gatherer times—they continue to depend on a limiting environment for brakes. For most people worldwide, the environment no longer provides these limits. In the modern world, we use cell phones, e-mail, text messaging, wireless computers, and computer games "24/7." Have our electronics, and their messages, made us more attuned to possible dangers from which we must flee or be ready to fight? There seems to be a constant need to be in touch, to know what is happening, a fear of what will happen if we do not stay continuously "tuned in."

CONCLUSION

Not only do we have "old brains," but with new brain imaging, we are able to understand that the brains of people who are addicted are seriously damaged by the drugs of abuse—and may have been damaged by genetic predisposition, or child abuse, or both, long before self-medication began. Science not only can provide us with new information, it can also tell us of past activities such as the use of lye and lime in treating foods and psychotropic plants for nourishment.

The marketing of tobacco and processed foods worldwide, especially to developing countries, has changed the nature of health problems in the world. Tremendous amounts of money are involved in this global trade. New meaning has been given to "international drug traffic." We have tremendous numbers of addicted and a larger array of addictions worldwide. The material cost and human suffering is beyond imagination. Our "old" brains that are capable of developing the technology that can look inside our brains will need to find new ways to stop the damage.

REFERENCES

Abrams, D., Monti, P., Niaura, R., Rohsenow, D., & Colby, S. (1996, March). Intervention for alcoholics who smoke. *Alcohol Health and Research World*, *20*(2), 111–117.

Anderson, C. M. (2001). The integrative role of the cerebellar vermis in cognition and emotion. *Consciousness and Emotion*, *2*(2), 284–299.

Anderson, C. M., Polcari, A. M., McGreenery, C. E., Maas, L. C., Renshaw, P. F., & Teicher, M. H. (1999). Cerebellar vermis blood flow: Associations with psychi-

atric symptoms in child abuse and ADHD. *Society for Neuroscience Abstracts, 25* (pt. 2), 1637.

Andrews, Z., & Harvath, T. (2008, March). Tasteless food reward. *Neuron, 57,* 806–808.

Bartosiewicz, P. (2004, May). A quitter's dilemma: Hooked on the cure. *New York Times,* Retrieved March 18, 2008, from http://www.nytimes.com/2004/05/02/business/yourmoney/02smok.html?pagewanted=all&position=

Bauer, L. (2001, September). EEG shown to reliably predict drug and alcohol relapse potential. *Neuropsychopharmacology, 25*(3), 332–340.

Begleiter, H., & Porjesz, B. (1988). Potential biological markers in individuals at high risk for developing alcoholism. *Alcoholism: Clinical and Experimental Research, 12*(4), 488–493.

Bell, L., & Martin K. (2002). A natural prescription for addiction. *Counselor, 3*(4), 40–44.

Bennett, A. E, Howell, R. W., & Doll, R. (1970, May). Sugar consumption and cigarette smoking. *Lancet, 1*(7655), 1011–1014.

Breiter, H. C., Aharon, I., Kahneman, D., Dale, A., & Shizgal, P. (2001). Functional imaging of neural responses to expectancy and experience of monetary gains and losses. *Neuron, 30*(2), 619–639.

Bremner, J., Narayan, M., Anderson, E., Staib, L., Miller, H., & Charney, D. (2000). Hippocampal volume reduction in major depression. *American Journal of Psychiatry, 157,* 115–127.

Brookhaven National Laboratory. (2004). *Exposure to food increases brain metabolism.* Retrieved September 21, 2004, from http://www.bnl.gov/ bnlweb/pubaf/pr/2004/bnlpr041904.htm

Dapice, A. (2006). The medicine wheel. *Journal of Transcultural Nursing, 17*(3), 251–260.

Dapice, A., Inkanish, C., Martin, B., & Brauchi, P. (2002, September). Killing us slowly: When we can't fight and we can't run. *Native American Times*. Retrieved May 25, 2004, from http://www.dlncoalition.org/related_issues/killing_us_slowly.htm

Dapice, A., Inkanish, C., Martin, B., & Montalvo, E. (2001, June). Killing us slowly: The relationship between type II diabetes and alcoholism. *Native American Times*. Retrieved May 25, 2004 from http://vltakaliseji.tripod.com/Vtlakaliseji/id20.html

De Araujo, I., Oliveira-Maia, A., Sotrikova, T., Gainetdinov, R., Caron, M., Nicolelis, M., et al. (2008, March). Food reward in the absence of taste receptor signaling. *Neuron, 57,* 930–941.

Di Chiara, G., & Bassareo, V. (2007). Reward system and addiction: What dopamine does and doesn't do. *Current Opinion in Pharmacology, 9*(2), 233. Retrieved March 10, 2008, from http://www.sciencedaily.com/releases/2004/04/040420214434.htm

Fisher, H. (2004). *Why we love: The nature and chemistry of romantic love*. New York: Henry Holt.

Flier, J., & Maratos-Flier, E. (2007, September). What fuels fat. *Scientific American, 297*(3), 72.

George, D., Hibbeln, J., Ragan, P., Unhau, J., Phillips, M., Doty, L., et al. (2000). Lactate-induced rage and panic in a select group of subjects who perpetrate acts of domestic violence. *Biological Psychiatry, 47,* 804–812.

Harvard Mental Health Letter. (2004). *The addicted brain.* Retrieved March 1, 2008, from http://www.health.harvard.edu/newsweek/The_addicted_brain.htm

HealthCentral.com. (2000). *WHO accuses tobacco companies.* Retrieved March 10, 2008, from http://medicalnewstoday.com/articles/9706.php

Holden, C. (2001, November). Behavioral addictions: Do they exist? *Science, 294,* 980–982.

Kienlen, K., Birmingham, D., Solberg, K., O'Regan, J., & Meloy, J. R. (2006). A comparative study of psychotic and nonpsychotic stalking. In J. R. Meloy (Ed.), *The scientific pursuit of stalking* (pp. 121–142). San Diego: Specialized Training Services.

Knutson, B., Wimmer, G. E., Kuhnen, C., & Winkelman, P. (2008, March). Nucleus accumbens activation mediates the influence of reward cues on financial risk taking. *NeuroReport, 19*(5), 509–513.

Koob, G. (1999). Alcohol stimulates release of stress chemicals. *Scripps Research Institute.* Retrieved March 10, 2008, from http://www.atforum.com/SiteRoot/pages/addiction_resources/webvol_4.pdf

MacKeen, D. (2000). *Hormonal rages: A new study links decreased levels of cortisol with aggressive behavior in boys.* Retrieved September 10, 2002, from http://www.salon.com/health/log/2000/01/14/cortisol

Mandell, A. (2005). *Nearness of grace: A personal science of spiritual transformation.* Retrieved September 15, 2005, from www.cieloinstitute.org/pages/357835/page357835.html?refresh=1111958880143

Mathews-Larson, J. (1991). *Seven weeks to sobriety.* New York: Villard Books.

Mayo Clinic (2007, February 9). Medical therapy for restless legs syndrome may trigger compulsive gambling. *ScienceDaily.* Retrieved March 31, 2008, from http://www.sciencedaily.com/releases/2007/02/070208222800.htm

Meloy, J. R. (1998). *The psychology of stalking: Clinical and forensic perspectives.* San Diego: Academic Press.

Meloy, J. R. (2006). *The scientific pursuit of stalking.* San Diego: Specialized Training Services.

Meloy, J. R., Cowet, P. Y., Parker, S. B., Hofland, B., & Friedland, A. (2006). Domestic protection orders and the prediction of subsequent criminality and violence to protectees. In J. R. Meloy (Ed.), *The scientific pursuit of stalking* (pp. 143–162). San Diego: Specialized Training Services.

Meloy, J. R., & Fisher, H. (2006). Some thoughts on the neurobiology of stalking. In J. R. Meloy (Ed.), *The scientific pursuit of stalking* (pp. 347–372). San Diego: Specialized Training Services.

Morgan, K. (2003, March). More than a kick. *Science News, 163*(12), 184.

Nader, M. (2002, January). Biology of rank: Social status sets up moneys' cocaine use. *Science News, 161*(4), 53.

National Institute of Drug Abuse. (2002, April 8). *NIDA addiction research news.* Retrieved May 25, 2004, from http://www.drugabuse.gov/MedAdv/02/NS-04.html

National Institute on Alcohol Abuse and Alcoholism. (1996). *NIAAA reports Project Match main findings.* Retrieved May 25, 2004, from http://www.niaaa.nih.gov/NewsEvents/NewsReleases/match.htm

Native American Times. (2008, April). International Relief and Development releases new study of double burden of malnutrition/obesity facing American Indians. Retrieved April 10, 2008, from http://www.nativetimes.com/index.asp?action=displayarticle&article_id=9461

Ozelli, K. L. (2007, September). This is your brain on food. *Scientific American, 297*(3), 84, 85.

Polick, V., Aarmor, D., & Bracker, H. (1980). *The course of alcoholism, four years after treatment* (pp. 169–170). Santa Monica, CA: Rand Corp.

Popkin, B. (2007, September). The world is fat. *Scientific American, 297*(3), 91–93.

Propping, P., Kruger, J., & Mark, N. (1981). Genetic disposition to alcoholism: An EEG study in alcoholics and their relatives. *Human Genetics, 59,* 51–59.

Rye, D. (2005). Restless leg syndrome. Business briefing: *US Neurology Review.* Retrieved March 5, 2008, from http://www.touchbriefings.com/pdf/1239/rye.pdf

Saah, T. (2005). The evolutionary origins and significance of drug addiction. *Harm Reduction Journal, 2*(8). Retrieved March 5, 2008, from http://bmc.ub.uni-potsdam.de/1477–7517–2-8/

Sapolsky, R. (1996). Why stress is bad for your brain. *Science, 273,* 749–750.

Sapolsky, R. (2000). Glucocorticoids and hippocampal atrophy in neuropsychiatric disorders. *Archives of General Psychiatry, 57,* 925–935.

Spotlight. (2006, February). *The double burden of malnutrition: Fighting hunger—and obesity.* Retrieved April 10, 2008, from http://www.fao.org/Ag/Magazine/0602sp1.htm

Steckel, R. H., & Rose, J. C. (2002). *The backbone of history: Health and nutrition in the Western Hemisphere.* New York: Cambridge University Press.

Tabakoff, B., & Hoffman, P. L. (1988). Genetics and biological markers of risk for alcoholism. *Public Health Reports, 103*(6), 690–698.

Tanner, L. (2004). *Study: We're eating ourselves to death.* Associated Press. Retrieved September 21, 2004, from www.cbsnews.com/stories/2004/03/09/health/main604956.shtml

Teicher, M. H. (2002, March). Scars that won't heal: The neurobiology of child abuse. *Scientific American,* 68–75.

Volavka, J., Pollock, V., Gabrielli, W. F., & Mednick, S. A. (1985). The EEG in persons at risk for alcoholism. *Recent Developments in Alcoholism, 3,* 21–36.

Walsh, E. (2007, May). Addiction and technology—From sex to drugs: Considering evolution and addiction. *Addiction and the Humanities, 3*(4). Retrieved March 5, 2008, from http:www.basisonline.org/2007/05/addiction_the_h.html

Chapter **4**

Co-Occurring Trauma and Substance Use Disorders with Criminal Offenders

Scott E. McClure, PhD

It was a moonlit Saturday morning in the slums of London on February 17, 1872, when an event happened that had an indirect yet permanent influence on the English language. Dr. William Chester Minor was a trained physician who served at the Battle of the Wilderness in May 1864 during the American Civil War, a war noted for its gruesomeness and casualties. Dr. Minor's military duties involved the typical medical procedures used to aid the continuous stream of injured soldiers in need of medical treatment. Many of the wounded soldiers needs were far beyond the capabilities of medicine at that time and treatment often resulted in numerous amputations and deaths. In addition to standard medical procedures, Dr. Minor's duties included branding a letter D on the faces of war deserters, many of whom were Irish. These events would later have a profound impact on Dr. Minor's psychological well-being, especially branding Ds on the faces of Irish deserters, a duty he was always reluctant to do. Dr. Minor knew the long-term impact it would have on the Irish, both in the United States and in Ireland where many Irish military veterans planned to return with their newly learned combat skills to fight in the Irish revolts. The branded D would forever label these individuals in the United States as war deserters and would also become an identification mark in Ireland for soldiers of the revolution, thus resulting in rejection by both countries.

Following the Civil War, Dr. Minor's behaviors became impulsive and reckless, often involving alcohol and prostitutes. By 1867 his erratic behavior, accompanied by uncontrolled fits of rage, severe headaches, nightmares, paranoia, and delusions of persecution led to a military discharge and eventually admittance into St. Elizabeth's Lunatic Asylum in Washington, D.C. Upon

release from the asylum, Dr. Minor, still haunted by his paranoid delusions that the Irish were after him, moved to London in an attempt to start a new life. Unfortunately, his symptoms of psychological trauma followed him and eventually precipitated the murder of a complete stranger on this winter morning of February 17, 1872. In an ironic turn of events for history, Dr. Minor's assault resulted in his admittance into the English asylum in 1872 where he spent the majority of his remaining life providing thorough and decisive definitions and literary examples for the first comprehensive English dictionary, the *Oxford English Dictionary*.

In today's modern media, a news report titled "Disgruntled Veteran Murders Innocent Family Man" would be a typical headline for the events that happened on February 17, 1872. If the individual was intoxicated at the time of the event, his fate would fare worse, as society generally lacks empathy for violent crime and substance misuse. But, given the entire background and context, provided by Winchester's (1998) book *The Professor and the Madman*, in which Dr. Minor's offense occurred, it is obvious that he was an outstanding citizen and veteran military doctor, who as a result of psychological trauma triggered by the Civil War acted violently in response to his psychiatric symptoms.

In the 1800s we lacked the scientific knowledge and sophistication to provide adequate treatment for trauma symptoms. In modern psychology and psychiatry, we have made major progress in trauma treatment, yet as with Dr. Minor, we still fail to provide this treatment for many individuals who may need it the most, such as criminal offenders.

INMATE POPULATION PROFILE IN THE UNITED STATES

Violence, sexual victimization, racial segregation, substance abuse, and organized crime are prevalent in jails, state, and federal prisons in the United States. This is concerning as more than half of state and federal inmates are nonviolent offenders (Bureau of Justice Statistics, 2001), many of whom are entering prison for the first time. Hence, in order to reduce the odds of victimization and survive prison culture, inmates face the choice to participate in prison politics, which often involves the cost of witnessing and participating in illegal activities and violent behaviors, or to chance not participating in prison politics, which increases the likelihood of victimization.

The number of incarcerated individuals in the United States has nearly quadrupled since 1980. The deinstitutionalization of mental health care and the criminalization of drug policy ("war on drugs") have substantially increased sentencing for nonviolent offenders. Regardless of a decrease in violent and

property crimes since the 1990s, our state, federal, and jail inmate populations have steadily risen to 2,245,189 in 2006, which constitutes a 2.8 percent increase since 2005 (William, Todd, & Paige, 2007).

Compared to the general population, prisoners disproportionately come from economically and socially disadvantaged environments. According to the 2002 Bureau of Justice Statistics special report on inmate profile statistics, nearly 70 percent of jail inmates reported regular use of alcohol and drugs, 56 percent of jail inmates grew up in a single-parent home, 1 in 9 lived in a foster home, 31 percent grew up with a parent or guardian who abused alcohol or drugs, and 46 percent reported having a family member who had been incarcerated (Bureau of Justice Statistics, 2004). In addition, more than 50 percent of females and 10 percent of incarcerated males reported being either physically or sexually abused in the past. Abuse rates for male inmates may be underreported. This is particularly true for male offenders victimized while in prison, who are often required to play an ultramasculine role to avoid further victimization and to pertain to the prison cultural values of secrecy and loyalty, which are often needed to survive in this hierarchical society (Goff, Rose, Rose, & Purves, 2007).

Individuals who come from past histories of physical and sexual abuse, who have criminally involved families, and participate in substance misuse, have an increased chance of being incarcerated and experiencing traumatic events. Many incarcerated individuals come from neighborhoods inundated with illicit substances and gang activity. As in prison, to survive in economically disadvantaged neighborhoods, individuals often participate in street politics. As a result, it is nearly impossible to avoid witnessing and at times participating in physical violence and various other illegal activities, which often result in increased vigilance, emotional numbing, increased substance use, and other means of coping with those chaotic environments. Many of these coping skills can become symptoms of posttraumatic stress disorder (PTSD), which will be discussed later in this chapter. Even though many individuals will experience traumatic events in their lifetimes, the development of PTSD is the exception and not the norm.

HISTORICAL DEVELOPMENT OF POSTTRAUMATIC STRESS DISORDER (PTSD)

Prior to the Vietnam War, there were few scientific studies that examined the psychological effects of trauma. Though few studies existed, descriptions of the psychological impact of combat trauma are numerous throughout historical literature. The *Iliad* describes the cold and detached nature of Achilles, who fre-

quently partook in reckless acts of courage fighting hundreds of enemy soldiers without regard for his own life. Ernest Hemingway's short story *Soldier's Home*, which is arguably semiautobiographic, describes in great detail the psychological effects of war trauma on the individual and his surrounding life. This story ends with the main character feeling disconnected and emotionally numb to his current life and lacking motivation and excitement about his future.

During the 1800s military doctors began to diagnose soldiers with "exhaustion" due to a mental shutdown following combat trauma. In the late 1800s Dr. Mendez DaCosta described "Soldier's Heart" as a diagnosis for Civil War combat veterans. Psychological symptoms included hypervigilance and increased startle response, and physiological symptoms included fatigue, heart palpitations, sweating, and tremors. During World War I and World War II, the diagnosis "shell shock" and "combat neurosis" labeled the psychological and physiological symptoms associated with war trauma (Herman, 1992). It was not until the Vietnam War that the scientific examination of the psychological effects of trauma in general and during combat began (Friel, White, & Hull, 2008). The post-Vietnam examination of psychological trauma paved the way for our modern-day classification of PTSD, which first appeared as a diagnosis in the *Diagnostic and Statistical Manual of Mental Disorders Version III (DSM–III)* in 1980 (American Psychiatric Association [APA], 1980).

The *DSM–III* required the following criteria for a PTSD diagnosis: The person experienced or witnessed a distressing event that is outside the range of usual human experience such as a serious threat to one's life or physical integrity. In addition to experiencing this event, the individual must also present with the symptom triad of reexperiencing the event, numbing and/or avoidance, and hyperarousal. These symptoms must persist for more than one month following the event (APA, 1980). The *DSM–III* diagnostic definition of trauma was an important step in the evolution of trauma identification and treatment. The new symptomatic PTSD diagnosis allowed researchers to generalize the traumatic experience beyond victims of war and paved the way for new research regarding the traumatic experiences of incest, rape, domestic violence, child abuse, and other trauma-related topics.

Fourteen years after the publication of the *DSM–III*, the *DSM–IV* was published, to be followed shortly by the evidence-based text revision (*DSM–IV–TR*) in 2000 (APA, 2000). Significant advances in PTSD theory and research enabled the *DSM–IV–TR* PTSD committee to change the primary diagnostic criteria to include a response of intense fear, helplessness, or horror in reaction to the traumatic event as well as to improve upon the original symptom triad.

DSM–IV–TR DIAGNOSTIC CRITERIA FOR PTSD

The *DSM–IV–TR* states that for a diagnosis of PTSD an individual must be confronted with, experience, or witness an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of others. The person's response to this event must involve intense fear, helplessness, or horror. In addition to the first two criteria, the individual must experience one or more symptoms of *recurrence* (distressing recollections, distressing dreams, flashbacks, distress or physiological reactivity triggered by internal or external cues associated with the event), three or more symptoms of *avoidance/numbing* (avoid thoughts, feelings, or conversation; avoid activities, places, or people that trigger memories of the event; inability to recall important details associated with the event; diminished interest in activities, feeling detached or estranged from others, restricted affect, foreshortened sense of future), and two or more symptoms of *increased arousal* (sleep difficulties, irritability or anger outbursts, difficulty concentrating, hypervigilance, exaggerated startle response). These symptoms must persist for more than one month following the event and cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (APA, 2000).

TRAUMA AND SUBSTANCE USE DISORDERS IN THE GENERAL POPULATION

Nearly all individuals who participate in substance use treatment programs have experienced psychological trauma. Trauma exposure rates for individuals in both inpatient and outpatient, voluntary and mandated drug treatment programs are near 100 percent (Farley, Golding, Young, Mulligan, & Minkoff, 2004). Approximately 40 percent of clients in drug treatment programs meet the diagnostic criteria for PTSD (Brown, Recupero, & Stout, 1995; Dansky, Roitzsch, Brady, & Saladin, 1997; Farley, et al., 2004; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Triffleman, Marmar, Delucchi, & Ronfeldt, 1995). This is exceptionally high considering that the estimated lifetime prevalence of PTSD among adult Americans is 7.8 percent, with women (10.4%) being twice as likely as men (5%) to have PTSD at some point in their lives (National Center for Posttraumatic Stress Disorders, 2005). It is known that PTSD and other trauma-related problems among substance-abusing populations are associated with many physical, emotional, and interpersonal problems. These problems include an increased risk for chronic health problems, use of "harder" drugs, greater consumption of drugs and alcohol, more frequent relapses, poorer retention in treatment, and more inpatient hospitalizations

than having a substance use disorder without trauma exposure (Brown et al., 1995; Jacobson, Southwick, & Kosten, 2001; Cronkite, Henson, Prins, Gima, & Moos, 2004; Ouimette, Finney, & Moos, 1999; Ouimette et. al., 2004). In addition, individuals with PTSD and trauma-related problems are more likely to be unemployed and have less social support than those without (Ouimette et al., 1999).

The physical, emotional, and interpersonal problems associated with individuals who have co-occurring substance use and trauma disorders are alarming. This becomes even more alarming when the overlap between trauma correlates and the criminogenic risk and needs principles used to identify an individual's potential for success in treatment and risk of criminal recidivism are compared.

Marlowe (2007) defines *criminogenic risks* as the characteristics of criminal offenders that increase the likelihood of a relapse to drug misuse and decrease the likelihood for success in treatment, thus increasing the chance of recidivism. The most notable high-risk factors include an earlier onset of substance abuse (especially prior to age 14) or crime (especially prior to age 16), attempting rehabilitation at a younger age (especially before 24 years of age), a recidivist criminal record, previous unsuccessful attempts at rehabilitation, and a co-existing diagnosis of antisocial personality disorder. More strikingly similar are an individual's *psychosocial/criminogenic needs*, which are an individual's areas of dysfunction that, if improved, can considerably reduce the likelihood of continued involvement in substance misuse and criminal behaviors (Marlowe, 2007). Notable high-need factors include compulsive addiction to drugs or alcohol, psychiatric pathology, emotional trauma, brain injury, chronic medical conditions, and illiteracy.

Considering the overlap between criminogenic risk/need factors and those associated with co-occurring trauma and substance use issues, it is fair to assume that many individuals who have not yet participated in the criminal justice system are at substantial risk for incarceration. This is especially true for individuals whose addiction worsens and their need for more and harder substances increase as their tolerance and physiological withdrawal symptoms increase. This progression of addiction usually leads to decreased social functioning and increased criminal behaviors. For example, to achieve the desired level of intoxication needed to numb the emotional pain of past traumas and relieve physical withdrawal symptoms, an individual will likely be in possession of larger amounts of substances and need to participate in other illegal activities such as prostitution, drug sales, theft, and at times violent crimes to financially support his addiction. Hence, for many individuals who go untreated for

co-occurring substance misuse and trauma, it is only a matter of time before they enter the revolving doors of the criminal justice system.

CO-OCCURRING TRAUMA AND SUBSTANCE USE DISORDERS WITH CRIMINAL OFFENDER POPULATIONS

Rates of PTSD for both male and females are higher in prison settings than in the general population (Goff et al., 2007; Kubiak, 2004; Trestman, Ford, Zhang, & Wiesbrock, 2007). The PTSD rates are likely to be higher for incarcerated individuals for several reasons. Most incarcerated individuals have participated in substance use, criminal behavior, and come from areas of extreme poverty, all of which increase the risks of trauma exposure (Hochstetler, Murphy, & Simons, 2004). In addition to being at risk prior to incarceration, prison can be the source of new traumas and a trigger for old ones (Kubiak, 2004). As with individuals who experience traumatic events in war or civilian life, individuals who experience traumatic events in prison will have different reactions and varying degrees of adaptation and/or recovery from the event. The effect of a traumatic event while incarcerated will likely vary dependent upon an inmate's genetics, history prior to incarceration, and resources for overcoming and coping with the experience (Hochstetler et al., 2004).

The assessment and treatment of PTSD and other trauma-related symptoms rarely occurs for criminal offender populations. This is disturbing given the disproportionately high incidence of PTSD in this population, the high comorbidity between PTSD and substance use, and the alarmingly high rates of substance use disorders in criminal offender populations.

Inmates are more likely to have histories of substance abuse, mental health problems, and to have witnessed or been victimized by acts of violence than noninmates (Hochstetler et al., 2004). Prevalence rates for PTSD among incarcerated men have been shown to be over four times greater than those of the general population (Ehlers, Maercker, & Boos, 2000) and two times greater for incarcerated women than in the general population (Jordan, Schlenger, Fairbank, & Cadell, 1996). Even these astonishing rates may be an underestimate. Trestman et al. (2007) examined the lifetime prevalence rates of PTSD in a sample of 2,196 incarcerated male and female inmates not identified as acutely mentally ill at intake in Connecticut jails. In this sample, lifetime PTSD prevalence rates for men reached 20 percent and 41.8 percent for women. These findings are alarming as they suggest that many incarcerated individuals who suffer from PTSD often go undetected and untreated.

In addition, Kubiak (2004) explored the differences in treatment adherence, substance relapse, and criminal recidivism in a sample of 199 incarcerated men (n = 139) and women (n = 60) who volunteered to participate in two residential in-prison substance abuse treatment programs. Fifty-five percent of the treatment population met the criteria for a lifetime prevalence of PTSD with no statistically significant difference in prevalence rates for men (53%) and women (60%). Not surprisingly, due to the aggressive hierarchical nature of men's prisons, men were more likely to report a traumatic experience within the past 12 months while incarcerated than women were. Only one woman reported a traumatic event in the past 12 months compared to 75 percent of men. Most important, statistical analysis identified that both men and women with co-occurring PTSD and a substance use disorder were more likely to recidivate and relapse than those with only a substance use disorder were. This is highly suggestive of the need to address co-occurring substance use and trauma symptoms for incarcerated individuals, as they appear to be highly associated with relapse and criminal recidivism.

Regardless of the high incidence of co-occurring PTSD and substance use with incarcerated individuals, the effects of psychological trauma on substance relapse and criminal recidivism rates are rarely addressed (Kubiak, 2004). This is concerning as several studies have reported high incidence of trauma history among men and women who enter prison (Jordan et al., 1996; Kupers, 1996; Teplin, Abram, & McClelland, 1996), and there is documented evidence of exposure to as well as participation in violence within institutions (Kupers, 1996; Toch, 1998; Websdale & Chesney-Lind, 1998), including sexual victimization for both men and women (Beck & Harrison, 2008; Wolf, Blitz, & Shi, 2007). In fact, an estimated 4.5 percent of state and federal inmates have experienced sexual victimization while incarcerated, with the highest prison rates ranging from 9.3 to 15.7 percent (Beck & Harrison, 2008). This presents a serious problem as 80 percent of federal and state inmate convictions were due to either a drug-related crime, being under the influence during the crime, or having committed a crime to support drug use (Belenko & Peugh, 1998). Approximately 40 percent of clients in drug-treatment programs have PTSD (Brown et al., 1995; Dansky et al., 1997; Farley et al., 2004; Kessler et al., 1995; Triffleman et al., 1995) and up to 50 percent of released inmates who enter community-based treatment programs are thought to have a co-occurring PTSD and substance use disorder and tend to demonstrate poorer long-term treatment outcomes than those without (Ouimette et al., 1999). Hence, in order to reduce recidivism rates with criminal offender populations, the relationship between trauma and substance use disorders needs to be addressed prior to release.

TREATMENT OF CO-OCCURRING SUBSTANCE USE DISORDERS AND TRAUMA

Individuals with PTSD often use substances in an effort to cope with overwhelming emotions, unwanted thoughts and memories, sleep disturbances, anxiety, depression, irritability, and other symptoms associated with trauma. When abstinent from substances, many of these symptoms reoccur with overwhelming intensity and often result in relapse only to further exacerbate PTSD symptoms (Kubiak, 2004). This process makes PTSD different from other mental health issues that co-occur with substance misuse in that trauma symptoms may worsen upon abstinence (Najavits, 2005). Another characteristic that differentiates PTSD from other psychiatric diagnosis is that it is the only mental health diagnosis with a directly identifiable cause that is external from the impacted individual (Najavits, 2005). Fortunately, an identifiable cause makes recovery from PTSD possible. Though recovery is possible, the success of trauma treatment will depend largely on the characteristics of the treated individual (biological vulnerability, genetics, socioeconomic status, single trauma, or chronic exposure), and the selected treatment intervention.

Most addiction treatment programs, whether community- or prison-based, do not assess for trauma history or offer any trauma-related treatment (Dansky et al., 1997). For individuals diagnosed with PTSD, substance use treatment without mental health treatment is less effective than for individuals without PTSD (Kubiak, 2004). For example, studies that have compared individuals in treatment for substance use disorders with and without co-occurring PTSD found higher relapse rates during treatment (Kubiak, 2004), at three-month (Brown, Stout, & Mueller, 1996) and one-year (Ouimette, Ahrens, & Moos, 1997) posttreatment follow-up for individuals with PTSD.

Research has demonstrated better treatment outcomes, for both PTSD symptoms and reduced substance use, when substance use disorders and trauma are addressed simultaneously (Cocozza et al., 2005; Kubiak, 2004; Ouimette et al., 1999). Regardless of this knowledge, most corrections-based addiction treatment programs do not address trauma symptoms. This unfortunate gap between the scientific evidence for effective treatment and its application in frontline treatment delivery may be due to several reasons. For example, trauma assessment rarely occurs in jails or prisons, corrections-based addiction counselors generally lack the knowledge and skills necessary to implement trauma treatment, and most prison-based treatment programs do not utilize clinically licensed supervision to monitor treatment quality.

Currently, there is little empirical research on effective treatment approaches for incarcerated populations. Heckman, Cropsey, and Olds-Davis (2007) con-

ducted an extensive literature review of 156 published empirical articles that addressed trauma treatment in correctional institutions. Only seven articles met the criterion for being empirically oriented and pertaining to PTSD in the criminal justice system. Of those seven, only two studies showed promising results and only one included treatment for co-occurring substance use and trauma disorders (Valentine & Smith, 2001; Zlotnick, Najavits, Rohsenow, & Johnson, 2003). The first study examined an exposure-based approach that utilized repetitive guided imagery of the traumatic event to reduce sensitivity to the event and cognitive restructuring to eliminate irrational beliefs associated with it (Valentine & Smith, 2001). Though the exposure-based treatment had positive results, it did not address substance use and required a trained clinician. This makes it impractical for large substance treatment programs in which the counselors are not clinicians. In addition, exposure-based treatments do not account for the risks associated with doing invasive trauma treatment with substance-abusing individuals whose symptoms may worsen while reliving their traumatic memories.

The second effective treatment intervention utilized *Seeking Safety* (Najavits, 2002), a noninvasive, present-focused, skill-based treatment designed to help individuals cope with both PTSD and substance use disorders (Zlotnick et al., 2003). The strengths of Seeking Safety include a positive current focus, practical coping skills, being manual-based, and noninvasive. Seeking Safety also produced positive outcomes as evidenced by reduced trauma and substance misuse symptoms. Unfortunately, no known empirical studies exist that examine the effectiveness of Seeking Safety treatment with incarcerated men, though it has shown effectiveness with men in the general population (Najavits, 2005).

Presently, there is a substantial need for more research on Seeking Safety's effectiveness with incarcerated individuals, especially for men. Regardless, Seeking Safety appears to be the strongest evidence-based approach to treat co-occurring substance use and trauma disorders with incarcerated individuals. It is present-focused, can be implemented by trained addiction counselors, and is coping skills driven, which makes it a safer intervention for individuals who are at high risk for relapse and may have limited abilities to regulate emotions and impulsivity.

Despite significantly elevated trauma rates and increased exposure to trauma compared to the general population, PTSD treatment for incarcerated individuals is minimal (Heckman et al., 2007). There is limited research on effective treatments for co-occurring PTSD and substance use disorders in the general population (Najavits, 2005) and even fewer for criminal offender populations (Heckman et al., 2007). Hence, there is great need for trauma assessment,

treatment, and empirical research for this underserved population (Goff et al., 2007; Heckman et al., 2007; Kubiak, 2004; Trestman et al., 2007).

IMPLEMENTING CO-OCCURRING TRAUMA AND ADDICTION TREATMENT FOR CRIMINAL OFFENDER POPULATIONS

Clients are best served if both trauma and substance use disorders are treated simultaneously, preferably by the same individual or agency (Najavits, 2005). The time has passed where individuals are required to have a sustained period of abstinence prior to treatment. We now better understand the vicious cycle of substance use and PTSD by which substance use reinforces psychological avoidance, a primary symptom of PTSD, which in turn reinforces substance use. The utilization of an integrated treatment approach to address both substance use and trauma symptoms simultaneously may allow for the improvements in one domain to influence improvements in the other (Najavits, 2005). Thus, as the individual develops improved skills to cope with trauma, the individual will be less inclined to utilize substances as a means of coping, and reduced substance use may in turn reduce the potential for subsequent traumas.

In addition to integrated substance use and trauma treatment, individuals with trauma histories may benefit from stage-based treatment interventions (Herman, 1992; Najavits, 2005). Stage-based trauma interventions generally address safety and coping at the first stage, followed by mourning (processing) the traumatic experience, and finally reconnection, the process by which the individual focuses on functioning in work and relationships (Herman, 1992; Najavits, 2005). Safety and coping should be the primary focus for individuals in corrections-based treatment. This is especially true for individuals with severe forms of PTSD and other co-occurring disorders who may decompensate upon addressing trauma symptoms. After clients have successfully developed skills to regulate emotions and cope with trauma symptoms without substance use, they may progress to processing the trauma through exposure therapy, and eventually shift their focus on reconnecting to a functional life. Corrections-based treatment may be the appropriate place to begin the initial phase of trauma treatment, but referral upon release to individual therapy or advanced group therapy by a licensed clinician trained in trauma treatment is recommended for the advanced stages of trauma treatment that utilize processing and exposure techniques.

Successful treatment implementation will depend upon several factors. First and foremost, the positive benefits of treatment must outweigh any negative

consequences. If trauma treatment elicits negative thoughts and emotions beyond the treated individual's ability to cope and beyond the addiction counselor's skill level, the client's safety will be violated. Safety is fundamental for successful treatment, if violated individuals may relapse into harmful behaviors such as substance use, physical aggression, and even suicide.

In corrections-based addiction treatment, where safety, security, and trust are of the utmost concern, only the safest and most effective trauma treatments should be applied. Regressive-based treatments such as exposure therapy, Eye Movement Desensitization and Processing (EMDR) therapy, and psychoanalytical treatments, though effective if performed by qualified individuals, are not the best modes of treatment for offenders while incarcerated. One point of concern in regard to treating trauma symptoms in corrections-based addiction treatment programs is the lack of qualified clinicians to address the complex issues that arise when clients discuss and/or relive their trauma histories. Most treatment providers are not equipped with the knowledge, skills, or formal education to work with trauma-related issues and can inadvertently induce harm on the inmate client. Unskilled addiction treatment counselors may mistakenly allow clients to disclose specific trauma details. The disclosure of trauma details without a skilled clinician can be harmful during individual therapy, can elicit trauma symptoms for others during group treatment, and can produce vicarious traumatization for both inmates and treatment staff (Baird & Kracen, 2006). Though disclosure can be effective for regressive- and exposure-based treatments, handling trauma disclosure is beyond the skill level of most addiction treatment counselors.

Even if noninvasive skills-based treatment is applied, adequately trained staff should always deliver it. Trained, licensed clinicians are the optimal choice for treatment delivery, but well-trained addiction counselors supervised by a trained licensed clinician may be as effective and more economically practical for corrections-based treatment. In addition to training frontline treatment staff, all staff, including administrative, corrections, and program management can help improve the treatment atmosphere if trained in *trauma-informed* services (Najavits, 2005).

It is very important that addiction treatment programs understand the difference between *trauma-informed* treatment and *trauma-competent* treatment (Najavits, 2006). Trauma-informed treatment includes giving basic trauma education to all staff at all levels. The Substance Abuse and Mental Health Services Administration (SAMHSA) Women, Co-occurring Disorders and Violence Study (WCDVS) has identified 10 principles that define trauma-informed services (Elliot, Bjelajac, Fallot, Markoff, & Reed, 2005). Though the WCDVS study is specific to women, it seems fair to generalize its principles to men.

The 10 principles include:

1. Trauma-informed services (TIS) recognize the impact of violence and victimization on development and coping strategies.
2. TIS identify recovery from trauma as a primary goal.
3. TIS employ an empowerment model.
4. TIS strive to maximize an individual's choices and control over her recovery.
5. TIS are based in a relational collaboration.
6. TIS create an atmosphere that is respectful of survivors' need for safety, respect, and acceptance.
7. TIS emphasize strengths, highlighting adaptations over symptoms and resilience over pathology.
8. The goal of TIS is to minimize the possibilities of retraumatization.
9. TIS strive to be culturally competent and to understand each individual in the context of her life experiences and cultural background.
10. Trauma-informed agencies solicit consumer input and involve consumers in designing and evaluating services.

While trauma-informed services aim to educate the entire workforce in the basic knowledge, skills, and understanding of the needs of individuals with histories of trauma, trauma-competent treatment seeks to train fewer, carefully selected individuals with the skills necessary for trauma treatment (Najavits, 2006). These individuals need to become educated on manual-based interventions for co-occurring substance and trauma disorders, require clinical supervision, and need formal training on trauma-based interventions. In addition, not all addiction treatment counselors are a good fit for delivering trauma-focused treatment (Najavits, 2006). Individuals who have not learned to cope with their own trauma histories, those who have poor boundaries, and those who are particularly confrontational in their treatment approach may jeopardize the quality of treatment, and at times may cause more harm than good. Fortunately, with proper training, the appropriate staff selection, and clinical supervision, it appears that we can begin to address the ever-evolving problem of co-occurring trauma and substance use disorders with criminal offenders.

CONCLUSION

Clearly, there is a need to improve the assessment, treatment, and research on criminal offender populations with co-occurring trauma and substance use disorders (Goff et al., 2007; Heckman et al., 2007; Kubiak, 2004; Trestman et al., 2007).

Treating both substance use and trauma disorders simultaneously can improve the effectiveness of treatment, as both disorders impact one another. To date, there is limited research on effective trauma treatment interventions for criminal offenders, and even less for criminal offenders with co-occurring PTSD and substance use disorders. Even though there is limited empirical research, it appears that present-focused, noninvasive, standardized, skills-based treatment that emphasizes coping and safety, such as the Seeking Safety treatment design (Najavits, 2002), is the best fit for trauma treatment in correctional institutions.

Until there is increased scientific evidence for effective treatment interventions for criminal offenders with co-occurring trauma and substance use disorders, it seems logical to utilize the most efficient and safest interventions possible. It is this author's opinion that corrections-based treatment is not the place to experiment with treatment modalities that are not evidence-based and scientifically driven. The utilization of nonscientifically driven trauma treatment in correctional institutions, especially by staff that are not trained clinicians, may be borderline unethical behavior as criminal recidivism rates continue to fall between 70 and 80 percent, which indicates that treatment as usual is not highly effective. In addition, there is no room for experimentation in this environment where safety and reduced recidivism is the primary concern. Therefore, the benefits of simultaneously treating trauma and addiction is evident and highly needed, and if done correctly, may substantially reduce criminal recidivism and help formally incarcerated individuals become productive members of society.

REFERENCES

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. text revision). Washington, DC: Author.

Baird, K., & Kracen, A. C., (2006). Vicarious traumatization and secondary traumatic stress: A research synthesis. *Counseling Psychology Quarterly, 19*, 181–188.

Beck, A. J., & Harrison, P. M. (2008). Sexual victimization in state and federal prisons reported by inmates, 2007. Washington, DC: *Bureau of Justice Statistics Report*.

Belenko, S. & Peugh, C. (1998). *Behind bars: Substance abuse and America's prison population*. New York: Center on Addiction and Substance Abuse at Columbia University.

Brown, P. J., Recupero, P. R., & Stout, R. (1995). PTSD substance abuse comorbidity and treatment utilization. *Addictive Behavior, 20*, 251–254.

Brown, P. J., Stout, R. L., & Mueller, T. (1996). Post-traumatic stress disorder and substance abuse relapse among women. *Psychology of Addictive Behavior, 10,* 124–128.

Bureau of Justice Statistics. (2001). Criminal Offender Statistics. *U.S. Department of Justice: Office of Justice Programs.* Retrieved May 28, 2008, from http://www.ojp.usdoj.gov/bjs/crimoff.htm

Bureau of Justice Statistics. (2004). Profile of jail inmates, 2002. *U.S. Department of Justice: Office of Justice Programs.* Retrieved May 28, 2008, from http://www.ojp.usdoj.gov/bjs/abstract/pji02.htm

Cocozza, J. J., Jackson, E. W., Hennigan, K., Morrissey, J. P., Reed, B. G., & Fallot, R., et al. (2005). Outcomes for women with co-occurring disorders and trauma: Program-level effects. *Journal of Substance Abuse Treatment, 28*(2), 109–120.

Dansky, B. S., Roitzsch, J. C., Brady, K. T., & Saladin, M. E. (1997). Posttraumatic stress disorder and substance abuse: Use of research in a clinical setting. *Journal of Traumatic Stress, 10,* 141–148.

Ehlers, A., Maercker, A., & Boos, A. (2000). Posttraumatic stress disorder following political imprisonment: The role of mental defeat, alienation, and perceived permanent change. *Journal of Abnormal Psychology, 109,* 45–55.

Elliott, D. E., Bjelajac, P., Fallot, R. D., Markoff, L. S., & Reed, B. G. (2005). Trauma-informed or trauma-denied: Principles and implementation of trauma-informed services for women. *Journal of Community Psychology, 33*(4), 461–477.

Farley, M., Golding, J. M., Young, G., Mulligan, M., & Minkoff, J. R. (2004). Trauma history and relapse probability among patients seeking substance abuse treatment. *Journal of Substance Abuse Treatment, 27,* 161–167.

Friel, A., White, T., & Hull, A. (2008). Posttraumatic stress disorder and criminal responsibility. *The Journal of Forensic Psychiatry & Psychology, 19,* 64–85.

Goff, A., Rose, E., Rose, S., & Purves, D. (2007). Does PTSD occur in sentenced prison populations? A systematic literature review. *Criminal Behavior and Mental Health, 17,* 152–162.

Heckman, C. J., Cropsey, K. L., & Olds-Davis, T. (2007). Posttraumatic stress disorder treatment in correctional settings: A brief review of the empirical literature and suggestions for future research. *Psychotherapy: Theory, Research, Practice, Training, 44,* 46–53.

Herman, J. L. (1992). *Trauma and recovery: The aftermath of violence from domestic abuse to political terror.* New York: Basic Books.

Hochstetler, A., Murphy, D. S., & Simons, R. L. (2004). Damaged goods: Exploring predictors of distress in prison inmates. *Crime and Delinquency, 50,* 436–457.

Jacobson, L. K., Southwick, S. M., & Kosten, T. R. (2001). Substance use disorders in patients with posttraumatic stress disorder: A review of the literature. *American Journal of Psychiatry, 158,* 1184–1190.

Jordan, K., Schlenger, W., Fairbank, J., & Cadell, J. (1996). Prevalence of psychiatric disorders among incarcerated women. *Archives of General Psychiatry, 53,* 513–519.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry, 52,* 1048–1060.

Kubiak, S. P. (2004). The effects of PTSD on treatment adherence, drug relapse, and criminal recidivism in a sample of incarcerated men and women. *Research on Social Work Practices, 14*(6), 424–433.

Kupers, T. (1996). Trauma and its sequelae in male prisoners: Effects of confinement, overcrowding, and diminished services. *American Journal of Orthopsychiatry, 66,* 190–196.

Marlowe, D. B., (2007, August 23) *Section on criminal justice research, Treatment Research Institute*. Written testimony to the Commission.

Najavits, L. M. (2002). *Seeking safety: A treatment manual for PTSD and substance abuse*. New York: Guilford Press.

Najavits, L. M. (2005). Theoretical perspective on posttraumatic stress disorder and substance use disorder. *Australian Psychologist, 40,* 118–126.

Najavits, L. M. (2006). Managing trauma reactions in intensive addiction treatment environments. *Journal of Chemical Dependency, 8,* 153–161.

National Center for Posttraumatic Stress Disorders. (2005). *National Comorbidity Survey*. Retrieved June 2, 2008, from http://www.ncptsd.va.gov/ncmain/ncdocs/fact_shts/fs_epidemiological.html

Ouimette, P. C., Ahrens, C., & Moos, R. (1997). Post traumatic stress disorder in substance abuse patients: Relationships to 1-year posttreatment outcomes. *Psychology of Addictive Behaviors, 11,* 34–47.

Ouimette, P. C., Finney, J. W., & Moos, R. H. (1999). Two-year posttreatment functioning and coping of substance abuse patients with posttraumatic stress disorder. *Psychology of Addictive Behaviors, 13,* 105–114.

Ouimette, P. C., Cronkite, R., Henson, B. R., Prins, A., Gima, K., & Moos, R. H. (2004). Posttraumatic stress disorder and health among female and male medical patients. *Journal of Traumatic Stress, 17,* 1–9.

Sabol, W. J., Minton, T. D. & Harrison, P. M. (2007). Prison and jail inmates at midyear 2006. *U.S. Department of Justice: Office of Justice Programs*. Retrieved November 22, 2008, from http://www.ojp.usdoj.gov/bjs/pub/pdf/pjim06.pdf

Teplin, L., Abram, K., & McClelland, G. (1996). Prevalence of psychiatric disorders among incarcerated women. *Archives of General Psychiatry, 53,* 505–512.

Toch, H. (1998). Hypermasculinity and prison violence. In L. H. Bowker (Ed.), *Masculinities and violence* (pp. 168–178). Thousand Oaks, CA: Sage.

Trestman, R. L., Ford, J., Zhang, W., & Wiesbrock, V. (2007). Current and lifetime psychiatric illness among inmates not identified as acutely mentally ill at intake in Connecticut's jails. *Journal of the American Academy of Psychiatry and Law, 35,* 490–500.

Trifflemann, E. G., Marmar, C. R., Delucchi, K. L., & Ronfeldt, H. (1995). Childhood trauma and posttraumatic stress disorder in substance abuse inpatients. *Journal of Nervous and Mental Disease, 183,* 172–176.

Valentine, P. V., & Smith, T. E. (2001). Evaluating incident reduction therapy with female inmates: A randomized controlled clinical trial. *Research on Social Work Practice, 11,* 40–52.

Websdale, N., & Chesney-Lind, M. (1998). Doing violence to women: Research synthesis on the victimization of women. In L. H. Bowker (Ed.), *Masculinities and violence* (pp. 55–81). Thousand Oaks, CA: Sage.

Winchester, S. (1998). *The professor and the madman*. New York: HarperCollins.

Wolf, N., Blitz, C. L., & Shi, J. (2007). Rates of sexual victimization in prison for inmates with and without mental disorders. *Psychiatric Services, 58,* 1087–1094.

Zlotnick, C., Najavits, L. M., Rohsenow, D. J., & Johnson, D. M. (2003). A cognitive-behavioral treatment for incarcerated women with substance abuse disorder and posttraumatic stress disorder: Findings from a pilot study. *Journal of Substance Abuse Treatment, 25,* 99–105.

This page intentionally left blank

Chapter **5**

The Role of Allergies in Addictions and Mental Illness

Joan Mathews-Larson, PhD, LADC, and
Mark K. Mathews, LADC, BCCR

Understanding allergies as they pertain to addictions and mental health issues requires an analysis of multiple genetic factors. Most agree allergy symptoms are the result of hypersensitive receptors on mast and basophil cells responding to various antigens by triggering the release of inflammatory mediators—particularly histamine. Standard treatment protocols address this underlying flaw with allergy medications, which block the release of histamine, but do nothing to abolish the underlying problem. The consequence of this approach has fueled a rise in addictive patterns with a progressive decline in the health for many allergy-afflicted people. This chapter will examine a genetic basis for allergies more carefully, and present evidence that the pathogenesis of addictions and many mental health problems are actually manifestations of IgG (immunoglobulin G, a serum protein antibody) delayed-onset allergies progressing toward more serious degenerative diseases.

Although the cause of allergies can be explained as an unregulated degranulation of histamine from mast and basophil cells due to gene malfunction, other complex genetic factors contributing to allergy processes need to also be considered (Akdis & Blaser, 2003, p. 15). More than 30 years ago, scientists suspected a genetic connection involving an immune system disorder existed for many diseases, based on structural anomalies in cells. Researcher Chang (1975) states:

> The view that diseases such as cancer, systemic lupus erythemotosus, and agammaglobulinemia can be successfully treated by unilateral manipulation of the

> humeral or the cell-mediated immune response per se is probably too simplistic. It has become increasingly clear that many of these disorders of the immune system seem to arise from flaws in cell differentiation, resulting in a hyper- or hypofunction of a particular cell type. (p. 82)

Chang's statement suggests gene-influenced diseases might occur in various ways, and even though unique genetic disorders manifest into different diseases, the process is often accompanied by inflammation (i.e., it incorporates an allergy response).

Geneticist Dr. Chris Reading's insightful reasoning expounds on flaws in cell differentiation, as proposed by researcher Chang. He believes maladapted genes causing allergies must be corrected or prevented from being expressed to prevent the progression of disease. Dr. Ravikovich (2003) aptly states Dr. Reading's concerns:

> A slight genetic defect, mostly inherited, occasionally acquired, may remain unnoticed for years. However, time itself and/or cumulative effects of the hazards [allergy provoking substances] may "break the back of the camel." In other words, the initial small defect in the genes may become magnified, and as a result, the operation guided by these genes may cause cells to respond [express] with a pathological reaction to what they would have perceived as harmless before. (p. 7)

Dr. Reading concedes, substances that provoke recurrent allergic responses are especially hazardous for people with underlying genetic defects; but more important, he offers insights into understanding how these structural genetic defects are formed before getting switched on later in life.

Dr. Reading focuses on rogue genes: those present at birth, and distortions that may develop later in life to initiate diseases. Genetic distortions are expressed both structurally and functionally, and with more than 30,000 genes in the nucleus of a single cell, and trillions of cells in the human body, intervening to correct the structural genetic defects causing allergies is impractical, if not impossible (Ravikovich, 2003, p. 8). Instead, Dr. Reading (2002) urges preventative tactics to stop irritating the hypersensitive immune system.

> Hypersensitivity reactions result from repeated exposure to a particular substance or to its chemically related substances. . . . The substance, if it is a large polypeptide, acts as an antigen and stimulates the body to form antibodies. Otherwise the substance acts as a hapten and combines with proteins in the body to form antigens. The reaction between an antigen from a later exposure and the corresponding antibodies results in the release of histamine. (Lu & Kacew, 2002, p. 49)

Like Dr. Ravikovich, Dr. Reading recognizes hypersensitive reactions stem from repeated exposures to allergy-provoking foods. But rather than focusing

on correcting the genetic malfunction causing the exaggerated release of histamine, Dr. Reading recommends avoiding allergy foods (Reading, 2002, p. 143). A delayed allergy response, although clearly tied to the release of histamine, might not respond to histamine corrective protocols; in fact, the classic exaggerated histamine release, used to define an allergic response, may not even be the primary problem in an IgG delayed response. After all, cell degranulation (histamine release) is how healthy tissue should respond when foreign substances (e.g., polypeptides/allergens) come into contact with it (Rocklin, 1982, pp. 49–70; Sirois & Borgeat, 1982, pp. 205–206).

Recognizing substances that provoke allergy responses may compromise the health of the host in more ways than just the release of histamine, Dr. Reading augments previous allergy concepts with a thought-provoking theory that elucidates the pathological progression of diseases, and provides insight into how such great disparities can exist in people's susceptibility to contract them.

To follow his reasoning, Reading (2002) first offers some clinical facts to be reviewed:

1. During pregnancy the mother and fetus interact chemically. Dehydroepiandrosterone (DHEA) secreted from the adrenal cortex of the fetus stimulates estrogen hormones from the mother's placenta to affect the formation (differentiation) of fetal tissues. An adequate supply of vitamins and minerals from the mother is crucial for the process to work.
2. Secondly, the propensity of the most common offending allergy-causing foods (grain, milk, egg, beef, and yeast) to trigger allergies is actually caused by components of the foods called subfractions: proteins such as gluten, a-gliadin, a-casein, and so on.
3. Thirdly, when people consume allergy-provoking foods, they do not properly absorb vitamins and minerals, exacerbating deficiencies in many important nutrients (p. 140).

Dr. Reading reasons when pregnant women consume allergy foods, their allergy-induced vitamin and mineral deficiency has two deleterious effects on the forming fetus: it hinders the release of hormones from the mother's placenta, and it lowers levels of vitamins and minerals supplied to the fetus. This reduces the amount of DHEA being released from the fetus's adrenal cortex, which further interrupts the supply of estrogens from the placenta, resulting in an abnormal balance of hormones, vitamins, and minerals. Additionally, damage caused by the toxic subfractions in the foods to which the mother is allergic may also damage the fetus if the sensitivity to them is passed on genetically. Dr. Reading (2002) states:

> These subfractions, I believe, cross the placenta and can actually take the place of some of the hormones that are [for the reasons outlined above] in short supply.

> In other words, the toxic subfractions may actually have a hormone-like action on the fetus. The net result of the faulty hormone-vitamin-mineral balance, plus the presence and hormone-like action of the toxic food fractions, is that some of the fetus's tissues are laid down abnormally. Formed before the immune system has matured, these tissues have a special metabolism that makes them different from normal cells. They are, in fact, premalignant . . . likely to go wild and proliferate in later life, if exposed again to the toxic food fractions and faulty hormone-vitamin-mineral environment that originally helped to form them. (p. 141)

Biochemists recognize cells have receptors for various hormones, proteins, enzymes, and other substances needed to maintain normal growth and division. Each receptor is specially "tuned" to a specific hormone, protein, or enzyme; however, "Cells forming pre-malignant tissue also have receptors for fractions of toxic foods because they took the place of some natural hormones when fetal tissues were forming in the womb" (Reading, 2002, pp. 141–142). Since genes determine the development and activity of all cellular receptors, it is logical to conclude these problems are structural, not functional (Ravikovich, 2003, p. 2). Therefore, to prevent these genes from being switched on, the food fractions originally present when the anomalous genes first formed must be avoided.

Dr. Reading (2002) notes, when allergy-sensitive people ingest allergy-triggering foods, toxic food fractions cross into their bloodstream and stimulate cells with receptors for them. Additionally, these subfractions suppress the immune system by upsetting the balance of hormones, vitamins, and minerals—basically recreating the anomalous environment that was present when the abnormal tissue first formed.

> [Cells of abnormal] tissue take in toxic food fractions just as they did in the womb. And just as they did in the womb, the fractions stimulate the cells to proliferate and reproduce—only this time the uterus is not present to provide growth-regulating hormones to keep the process in check. So cells proliferate wildly and uncontrollably. And when they do so, cancer has begun. (p. 142)

Whenever someone puts forth a theory to explain the disease of cancer, inevitably eyebrows will rise; however, this time we might not want to dismiss Dr. Reading's theory too quickly. His track record for arresting and reversing cancer using allergy elimination diets is impressive.

Reading (2002) believes cancer and many other diseases are actually congenital disorders: "begun by the effect of food allergies on the developing fetus, and exploding out of control when the faulty pre-birth environment reoccurs." He also notes "such cancers are actually mimicking the rapid proliferation of fetal tissues. Or, if you like, they [cancerous cells] are really abnormal fetal tissues starting to proliferate all over again" (p. 142). What makes his argument

so compelling is that many cancer cells do in fact release fetal proteins, which are normally seen *only* in unborn babies (Akira et al., 1972, pp. 1–7; Reading, 2002, p. 142).

Addicts and alcoholics, unaware of the biochemical damage stemming from allergic subfractions, are often fooled into thinking they will regain their health and start feeling better by simply being abstinent or avoiding their "primary" addictive substances. However, in abstinence they usually feel worse. So, to counter the misery of withdrawal, alcoholics instinctively and unknowingly indulge their allergies by ingesting copious quantities of foods from which their alcohol was fermented—grains, sugars, yeast, and so forth. Consumption of these allergy-provoking foods sustains an ongoing load of toxins, and biochemical imbalances (Randolph & Moss, 1980, p. 23). Until they break away from their allergy foods, addicts and alcoholics will continue to experience physical and psychological pain. Confused, they'll turn to psychological and spiritual treatment, without understanding the source of the problem is their allergy-riddled diet.

Although restoring proper histamine function, as Dr. Ravikovich directed, may be well suited to address functional problems characterizing IgE immediate onset allergies, countering an IgG delayed allergic reaction is a different, more complex allergy problem. We believe it is most effectively dealt with by avoiding subfraction-containing foods that trigger the formation of immune complexes, and the proliferation of disease-causing cells. In other words, these are structural genetic defects that cannot be corrected, but can be controlled by avoiding the triggers (allergy foods) that switch them on. Unfortunately, foods that switch on allergies are the foods people love to eat again and again, their comfort foods—foods with addictive, drug-like qualities. Why do some foods cause drug-like reactions? A survival mechanism allows the body to override pain by releasing "feel-good" chemicals called endorphins (Braly & Holford, 2006, p. 90). Many analgesic drugs are based on similar chemical structures; all of them, including endorphins, are peptides—small groups of bound amino acids (Terenius, 2000, p. 1). When protein is ingested, it is broken down to peptides, and then, if digestion is working well, those peptides get broken down further to individual amino acids. Unfortunately, allergy foods impair digestion and damage the GI tract, impairing the complete digestion of proteins.

> In the laboratory, endorphin-like peptides have been made from wheat, milk, barley, and corn using human digestive enzymes. These peptides have been shown to bind to endorphin receptor sites. Preliminary research shows that certain foods, most commonly wheat and milk, may induce a short-term positive feeling, even if, in the long term, they are causing health problems. (Holford, 2002, p. 73)

Once these peptides reach the bloodstream, they become a problem for the immune system to gather and clear, setting the stage for a classic delayed IgG allergic response.

An IgG delayed-onset food allergy can produce more than 100 allergic symptoms and affect almost every organ or tissue in the body. It is estimated one in three (children and adults), and more than 70 percent of people battling chronic conditions, who are unresponsive to conventional medicine, are also struggling with IgG allergies. In addition, more than 100 medical diseases and conditions have been connected to IgG allergies (Braly & Holford, 2006, p. 16).

> These food allergies occur when your immune system creates an over abundance of IgG antibodies to a particular food allergen. The antibodies, instead of attaching to mast cells like their IgE counterparts, bind directly to the food particles as they enter your bloodstream, creating "immune complexes." The more of these you have floating around the bloodstream, the more on edge your immune system becomes, sending out phagocytes to gobble the complexes up. Basically your immune system gradually goes into red alert. (Braly & Holford, 2006, pp. 15–16)

Unlike the quick response, characteristic of an IgE immediate-onset allergy, IgG symptoms come on slowly—from two hours to several days. Ingesting allergy-provoking foods prompts the gradual formation of immune complexes until finally they overload the immune system's ability to clear them out. That's when symptoms are felt. Unfortunately, the time differential makes symptoms difficult to link to the foods that cause them. And because the initial response to allergy-provoking foods is often a pleasurable, endorphin-like effect, it becomes even more difficult to accept that they are a problem. In the end, a pattern, initiated and sustained by these foods, contributes to an addictive progression of disease.

The starting point of most diseases is in the gut. Allergy foods factor heavily in the etiology of diseases because they damage the GI tract, and impair digestion. In that respect, the deleterious effects from allergies and addictions is the same (Rubin, 2003, pp. 48–55). Furthermore, combining allergy foods with alcohol heaps more stress on the immune system by doing more damage to the gut.

> [For those who drink alcohol], hypersensitivity will continue indefinitely because ethanol greatly increases the permeability of intestinal membranes, making it more likely that macromolecules [polypeptides] will be absorbed into the bloodstream. Alcohol also contributes to immune system hypersensitivity by creating nutritional deficiencies. (Bates, 1987, p. 35)

The problem with alcohol is that it compounds the damage caused by food allergies by increasing permeability in the gut membrane. This destructive effect can be tolerated by the average person, but for the person who already has a food sensitivity problem, just a little bit of alcohol every day will overwhelm the immune system's ability to fight disease and maintain biochemical stability.

> Even a quite moderate use of alcohol will prove to be such an obstacle to healing that the GI tract will never repair itself, and allergies will never cool. Yet these people will turn to alcohol for relief from depression, and it will give a temporary feeling of happiness, giving the impression that it is good medicine. The more you drink, the worse your GI tract will leak allergens into your bloodstream and the more depressed you will become. (Bates, 1987, p. 57)

Bates describes the pathogenesis of alcoholism the same way a food addiction could be described. Whether responding to alcohol, drugs, or allergy foods, allergic/addictive chemistry follows the same course because the body recognizes all these substances as toxins, and turns to the same system—the immune system—to clear them. It's when the toxic load exceeds the immune system's ability to clear it that inflammation begins its destructive process.

Inflammation is a slow progression in some allergic reactions. Unlike quick IgE allergy reactions, where allergens settle directly into tissues to initiate cellular degranulation, many IgG delayed-onset allergies come on slowly. The process begins with antigens clumping to antibodies to form free-floating immune complexes in the blood (Suen & Gordon, 2003, pp. 134–135). Macrophages are then deployed to devour these complexes and take them out of circulation. It's when the immune complexes exceed the immune system's ability to clear them that inflammation occurs—a delayed reaction. Charles Bates (1987) states:

> The body has many ways of dealing with immune complexes. They are large enough to be filtered out of the blood in the liver. Wherever they congregate, they activate the degranulation of mast cells, which release prostaglandins, histamines, leukotrienes, and other inflammatory chemicals called the complement cascade. The result could be heat, pain, or anything from a runny nose to the destruction of healthy tissue. When the immune system destroys healthy tissue in the pancreas, this is called diabetes. Arthritis is the immune system attacking bone and connective tissue. Many diseases of the GI tract follow this pattern. (p. 91)

Recalling the lessons of both Dr. Ravikovich and Dr. Reading, and acknowledging these immune complexes (in delayed-onset food allergies) are clumps of food fractions, the complexity of the biochemical dilemma becomes more

obvious. On the one hand, excessive inflammation will lead to tissue destruction and disease—Ravikovich's concern; on the other hand, clumps of food fractions congregating in healthy tissue will recreate an anomalous environment conducive for the proliferation of premalignant cells—Dr Reading's concern. It is not a choice of one or the other. Under stressful conditions of an IgG allergy, the body must deal with these problems simultaneously. In support of Dr. Reading's advice: the best approach to avert this dilemma is to avoid the foods that put the immune system in such a precarious position.

Clearing allergy-provoking foods can be a challenging task. IgG allergies are perpetuated no differently than any other kind of chemical addiction. If the allergic addictive person is deprived of the offending allergen long enough, he or she will go into withdrawal. Philpott and Kalita (2000a) state:

> Adaptive addiction can be described as a state of relative freedom from symptoms, occasioned when the addictive substance is contacted frequently enough and the biological homeostatic state is in good repair. It is, however, a state of chronic stress, precariously balanced, and paves the way for the emergence of an "illness." (p. 28)

Of course, the stress from an ongoing battle with toxic allergens in foods will weaken the immune system. Then, adding seasonal antigens, environmental stresses, physical stresses, or even emotional stress can finally deplete the body's defenses enough so that illness becomes a frequent state.

The similarities of allergies, alcoholism, and addictions are undeniable (i.e., they are the same problem based on similar molecules, following the same etiology). People with IgG allergies and those with other drug/alcohol addictions are engaged in a similar pattern of use—stimulated by the release of endorphin-like substances, and perpetuated by the need to defer the symptoms of withdrawal. Philpott and Kalita (2000a) state:

> The state of partial and temporary relief by contact with allergens is termed "addiction." Understanding addiction as an extension of a maladaptive allergy state is necessary if one is to understand the seriousness of addiction to frequently eaten foods and commonly met chemicals. (p. 28)

Chemically dependent, allergic addictive people may continually submit their bodies to immune-suppressing, disease-initiating chemicals and toxic food fractions for many years before they finally succumb to the fact that it's robbing them of their health, and taking years off their lives.

The truth about allergies is that they are the foundation of addictions. Correcting the genetic malfunction driving IgE immediate-onset allergies (Dr. Ravikovich's work) does not go far enough to break the hold addictive sub-

stances have on those unfortunate enough to be genetically predetermined or biochemically altered for IgG delayed-reaction allergies and addictions. As Dr. Reading noted, addictions stem from biochemical anomalies shaped by an endless variety of genetic factors—prenatally and later in life. The scope of allergies can also be expanded to elucidate many mental health disorders. Because they too are rooted in biochemical disruptions tied to allergies, preventative interventions can be incorporated before allergies manifest into more serious diseases.

The brain is a very metabolically active organ. Although it composes only 2 percent of the body's weight, it consumes 20 percent of the body's available oxygen and 25 percent of the body's glucose (Atavistik Pictures, 2006). Transporting all that oxygen and glucose to the brain requires a great deal of blood—the same blood that carries allergy-inciting molecules to other organs in the body. You would think this obvious potential for problems would be of great concern to modern medicine.

> Although allergic mental illness is encountered daily in every doctor's office around the world, it is rarely recognized by physicians because they don't realize allergic reactions often appear as depression, anxiety, irritability, confusion, paranoia, hyperactivity, autism, catatonia, or schizophrenia. Unfortunately, most cases of brain allergies are misdiagnosed as doctors search in vain for emotional causes, while allergic brain malfunctions remain unsuspected. (Mandell & Scanlon, 1979, pp. 90–91)

Compounding the problem of medical ignorance, Dr. Russell Blaylock states:

> Whole foods pass through the intestine, get into the bloodstream and bring forth an immune response. . . . The brain's immune system is also activated, releasing toxic components including glutamate, and that causes neurological dysfunction. You can get all kinds of symptoms triggered from immune reactions: lethargy, stupor, disorientation, paranoia, delusions, hallucinations, agitation, rage, panic attacks, criminal behavior, and even seizures. (Atavistik Pictures, 2006)

In spite of various organic risks to stable brain function, conventional medicine and psychiatry mostly refuses to acknowledge and address the brain's vulnerability to allergens and food subfractions ushered in via the vascular transport system.

Along with food fractions from allergens, many viruses also live and thrive in the blood. The body requires a robust immune system to keep them at bay. A key component in that defense system is endothelial-relaxing factor, or nitric oxide, a natural molecule manufactured in healthy blood vessels that works to destroy invading pathogens. "Nitric oxide is used by the immune system to

stave off infectious bacteria, viruses, and parasites, and it even curtails the proliferation of certain types of cancerous cells" (Ignarro, 2005, p. 48). However, as vessels harden over time, or if they are poisoned by toxic heavy metals (lead, mercury, etc.), they lose the ability to manufacture this crucial molecule needed to protect the brain and body from pathogens in the blood. One way of restoring elasticity to the veins and production of nitric acid is through chelation therapy. It is believed that by chelating out heavy metals, blood vessels resume normal production of nitric oxide (Gordon Research Institute, 2000).

But how do viruses contribute to mental illnesses? Philpott and Kalita (2000b) implicate the origin of *all organic brain disorders* to viral infections from the herpes family (i.e., Epstein-Barr, cytomegallo, and human herpes virus #6). If a pregnant mother ingests foods that suppress her immune system (allergy foods), infections flare up, and may get passed on to her developing fetus. In addition, infectious viruses might also invade the brain of a young child, particularly because the blood brain barrier is much more vulnerable in the early years. Then, as that young brain develops, these viruses infect neurons, causing them to swell. Swelling injures the developing brain, including the temporal lobe, and particularly the frontal part of the brain, resulting in damage that impairs perception, judgment, and the ability to concentrate (pp. 148–149).

We believe most mental health disorders can be tied to a symbiotic relationship of allergies (primarily food allergies) and viruses. Unfortunately, viruses do not fade away or die off. Studies reveal Epstein-Barr, cytomegallo, and human herpes #6 viral infections remain in the body and the brain for a lifetime, causing fluctuating symptoms in response to varying levels of stress—and, of course, allergies are great promoters of stress. When stress flares, these viruses become more active, which further stresses the immune system. Philpott and Kalita (2000b) state:

> These are lymphotropic viruses that infect the immune system. B-lymphocytes become disordered from the viral infection, producing antibodies and an autoimmune response inappropriately. As a result, the child's immune system becomes compromised. The child becomes more reactive to chemicals and inhalants in the environment. This additional stress makes the child *much more prone to maladaptively react to specific foods* [authors' emphasis]. (p. 149)

Interestingly, those specific foods are the ones that help viruses to flourish, and disrupt mental stability—allergy foods. In addition, they are the ones that get chosen—even after the infected host has been warned of the gravity of the situation. It is a hallmark of addictive behavior, but it begs the question: Who's in charge—the person, or the pathogens? Perhaps addiction is a struggle for survival between microorganisms that thrive in an acidic, hypoxic environment

and normal cells and tissues that thrive in a more alkaline-hyperoxia environment (Young & Young, 2002, pp. 12–37).

But mental health disorders have been linked to many biochemical abnormalities, not just allergies. Schizophrenia is a branch of mental disorders, in the extreme. Years ago, Dr. Carl Pfeiffer and colleagues (1970) recognized schizophrenia as a syndrome, not a single disease. To his credit, he identified different biochemical deficiencies, which created symptoms interpreted as schizophrenia. For example, he determined many schizophrenics were pyroluric, due to metabolic anomalies, which contributed to a zinc and B6 deficiency (pp. 139–145). Likewise, his contemporary, Dr. Abram Hoffer (2005), saved many "schizophrenics" from wasting away in mental institutions by elucidating the true cause of pellagra—a B3 niacin deficiency (p. 70). Dr. Chris Reading (2002) observed, "While many, or even most, cases of so called schizophrenia are really allergic conditions or hereditary metabolic disorders, a significant minority remains that doesn't appear to fit into either category. These can be called the true schizophrenias" (p. 180). Nevertheless, many symptoms labeled as schizophrenia can be traced back to biochemical deficiencies or excesses altering metabolic processes to create distorted expressions of the brain.

So why do these deficiencies or excesses occur in the first place? If it were just a question of poor nutrition, mental illness would be easy to correct; however, look deeper and you'll find diet is subordinate to the allergies that drive them. Indeed, allergies perpetuate cravings for the very foods that sustain the allergic inflammatory conditions that drive degenerative diseases. Again, let's ask, "Who's in charge, up there in the brain?" Foods that promote inflammation, hypoxia, and acidity—ideal conditions for viruses to thrive in—are the ones schizophrenics and allergic/addictive mentally ill patients want to eat, not diets of nonallergic foods, which cool the inflamed brain, and give support to the immune system (Young & Young, 2002, pp. 12–37). But why should inflammation of the brain (i.e., chronic encephalitis) result for only some who indulge their allergic impulses? Are "nonschizophrenics" only spared a miserable fate of mental anguish because they are not infected with viruses that capitalize on repeated exposures to allergy foods?

Historically, there have been times when schizophrenics were unable to obtain foods that would aggravate their allergies, and intensify their viral infections. During World War II, rations of wheat and rye became scarce in Scandinavian countries. Based on the unavailability of these gluten-containing foods, estimates from Finland figured "the mean annual *decrease* in the number of first admissions [for schizophrenia] from the prewar mean was estimated to be about 19 percent in 1940–1942 and about 45 percent in 1943–1945" (Dohan, 1966). This intriguing study shows a strong relationship between

gluten-containing foods and schizophrenia. Unless steady provisions of allergy foods are ingested, the virus cannot maintain its influence over the brain. Famine for the virus equates to sanity for the host.

Allergies are at the foundation of the problem because they distort the terrain of tissues by forming a more acidic environment conducive for viruses to flourish, and microforms to change into more virulent strains (Young & Young, 2002, pp. 21–23). In other words, allergies work hand-in-hand with disease-causing viruses to suppress the immune system, and influence thought processes of the brain. In their clinical experience, Philpott and Kalita (2000b) note:

> Schizophrenia is a state of disordered brain function in areas of perception, mood, thought, and motor function. Acute mental symptoms can be triggered by maladaptive reactions to foods, chemicals, or inhalants to which the subject is allergic, addicted, or otherwise hypersensitive. Several studies have demonstrated that maladaptive reactions to environmental substances, especially foods, are significant factors in evoking mental illnesses. (p. 149)

These "maladaptive reactions to environmental substances" help to promote, sustain, and expand an internal environment (terrain) that fortifies a viral presence in the tissues of the brain. Viral influence affects the way mood, thought, and personality is expressed; finally, even reason is compromised, resulting in irrational behavior and a demeanor perceived as mentally ill.

Fortunately, there is a way to restore stability and give back control to a "mentally ill" brain. The two branches of medicine best equipped to deal with mental health problems brought on by organic-based infections and allergies are ecology medicine and orthomolecular medicine. Both serve to reduce the inflammatory response from allergies, and stabilize the brain. Ecology medicine works by identifying and abstaining from allergy-causing foods, essentially laying siege to viruses and virulent pathogens by keeping out chemicals and foods fractions that help perpetuate the hypoxic, acidic environment they thrive in. The other approach, orthomolecular medicine, works by providing nutrients in optimum amounts needed to restore proper metabolism and health at a cellular level.

Human ecology acknowledges that substances in our environment can have a profound effect on our mental health and behavior. "Evidence suggests that the basic organic forces behind many chronic physical and mental illnesses are addictive reactions to frequently eaten foods and commonly met chemicals" (Philpott & Kalita, 2000a, p. 57). Identifying and avoiding symptom-inducing substances is fundamental to restoring mental stability. Ecology medicine holds the potential for stopping and reversing the pathology of many mental health diseases.

Orthomolecular medicine strives to treat infectious and degenerative diseases by varying the concentration of "right molecules" (i.e., vitamins, minerals, etc.), which are normally present in the human body (Lawson, 2003). Cells inadequately provided with the nutrients needed for proper metabolism and health create a condition for disease. When a cell becomes deficient in even one nutrient, its entire function will be seriously impaired. Multiply that by millions of cells, and tissues and organs may be affected. The brain is an organ composed of million of cells. When its metabolism becomes disordered due to allergic responses, viral attacks, and nutritional deficiencies, it falls into a state of disease. The brain expresses a diseased state with changes in perceptions and cognitive function, resulting in illusions, hallucinations, and delusions (Philpott & Kalita, 2000a, p. 73).

Orthomolecular psychiatry can repair the nutritional imbalances to restore mental health, but that repair is fragile (i.e., it cannot be sustained if *first-cause* ecological factors are not also stopped). The power of ecological and orthomolecular medicine together gives increased therapeutic value beyond either approach alone. Satisfactory clinical results often cannot be achieved unless both systems are combined. Ongoing research into how nutritional factors and ecology medicine support each other in cooling allergies will improve future treatment of mental health problems.

REFERENCES

Akdis, C., & Blaser, K. (2003). Histamine in the immune regulation of allergic inflammation. *Journal of Allergy and Clinical Immunology, 112*, 15–22.

Akira, Y., Kawaharada, M., Ikebe, M., Takahashi, A., Anzai, T., & Wada, T. (1972). Diagnostic significance of two fetal proteins, {alpha}f and rßs, in hepatoma and other neoplastic diseases. *Japanese Journal of Clinical Oncology, 2*, 1–7.

Atavistik Pictures (Producer). (2006). *Nutrition and behavior: A lecture by Russell Blaylock* [DVD]. Ridgeland, MS: Atavistik Pictures.

Bates, C. (1987). *Essential fatty acids and immunity in mental health.* Tacoma, WA: Life Sciences Press.

Braly, J., & Holford, P. (2006). *Hidden food allergies: The essential guide to uncovering hidden food allergies—and achieving permanent relief.* Laguna Beach, CA: Basic Health Publications.

Chang, Y. (1975). Introduction: Prospectives on new immunoregulants. In M. Rosenthale & H. Mansmann (Eds.), *Immunopharmacology* (pp. 79–83). Holliswood, NY: Spectrum Publications.

Dohan, F. C. (January, 1966). Wheat "consumption" and hospital admissions for schizophrenia during World War II: A preliminary report. *American Journal of Clinical Nutrition, 18*, 7–10. Retrieved December 1, 2007, from www.ajcn.org

Gordon Research Institute (Producer). (2000). *Detoxification with the new EDTA program* [Videotape]. New York: Gordon Research Institute.

Hoffer, A. (2005). *Adventures in psychiatry: The scientific memoirs of Dr. Abram Hoffer*. Caledon, ON: KOS Publishing.

Holford, P. (2002). *Improve your digestion*. London: Judy Piakus Publishers.

Ignarro, L. (2005). *No more heart disease: How nitric oxide can prevent—even reverse—heart disease and strokes*. New York: St. Martin's Griffin.

Lawson, S. (2003). *What is orthomolecular medicine?* Retrieved December 10, 2005, from http://lpi.oregonstate.edu/f-w99/orthomolecular.html

Lu, F., & Kacew, S. (2002). *Lu's basic toxicology: Fundamentals, target organs and risk assessment* (4th ed.). New York: Taylor and Francis.

Mandell, M., & Scanlon, L. W. (1979). *Dr. Mandell's 5-day allergy relief system*. New York: Pocket Books.

Pfeiffer, C., Mailloux, R., & Forsythe, L. (1970). *The schizophrenias: Ours to conquer*. Wichita, KS: Bio-Communications Press.

Philpott, W., & Kalita, D. (2000a). *Brain allergies: The psychonutrient and magnetic connections*. Chicago: Keats Publishing.

Philpott, W., & Kalita, D. (2000b). *Magnetic therapy: An alternative medicine definitive guide*. Boulder, CO: Alternative Medicine.com.

Randolph, T., & Moss, R. (1980). *An alternative approach to allergies*. New York: Lippincott & Crowell.

Ravikovich, F. (2003). *The plot against asthma and allergy patients: Asthma, allergies, migraine, chronic fatigue syndrome are curable, but the cure is hidden from patients*. Alton, ON: KOS Publishing.

Reading, C. (2002). *Trace your genes to health*. Ridgeville, CT: Vital Health Publishing.

Rocklin, R. E. (1982). Modulation of inflammatory and immune responses by histamine. In P. Sirois & M. Rola-Pleszczynski (Eds.), *Immunopharmacology: Research monographs in immunology* (pp. 49–74). New York: Elsevier Biomedical Press.

Rubin, J. (2003). *Patient, heal thyself*. Topanga, CA: Freedom Press.

Sirois, P., & Borgeat, P. (1982). Mediators of immediate hypersensitivity. In P. Sirois & M. Rola-Pleszczynski (Eds.), *Immunopharmacology: Research monographs in immunology* (pp. 201–222). New York: Elsevier Biomedical Press.

Suen, R., & Gordon, S. (2003, August–September). A critical review of IgG immunoglobulins and food allergy—implications in systemic health. *Townsend Letter for Doctors and Patients*, 241–242.

Terenius, L. (2000). From opiate pharmacology to opioid peptide physiology. *Upsala Journal of Medical Sciences, 105*, 1.

Young, R. O., & Young, S. R. (2002). *The ph miracle: Balance your diet, reclaim your health*. New York: Warner Wellness.

Part II

BODIES

This page intentionally left blank

Chapter **6**

Caffeine: Pharmacology and Effects of the World's Most Popular Drug

Kyle M. Clayton, MS, and Paula K. Lundberg-Love, PhD

Caffeine is the most commonly used psychoactive drug in the world (Julien, 2005). A naturally occurring substance derived from more than 60 plants worldwide, caffeine's availability permeates most cultures. In the United States, it is estimated that up to 90 percent of adults consume caffeine on a daily basis, with an average daily intake of 200 to 400 mg per day (Meyer & Quenzer, 2005). The most common sources for daily caffeine intake include coffee, tea, soft drinks, and chocolate. While various factors, including serving size and method of preparation, affect the amount of caffeine found in particular products, the average cup of coffee contains approximately 100 mg of caffeine, while average servings of tea, soft drinks, and chocolate are slightly lower, 50 mg, 40 mg, and 20 mg, respectively. Caffeine tablets of 50–200 mg are available without a prescription (e.g., Vivarin, Nodoz), and various over-the-counter pain relievers, migraine medications, and antihistamines also contain caffeine. Currently, the Food and Drug Administration lists caffeine as safe for consumption, and its use as an additive is not restricted. However, the pervasiveness of caffeine's use, along with the volume of recent research findings on the subject, facilitates the need for exploring the effects of caffeine on the human body.

HISTORY

People have been consuming caffeine in various forms for thousands of years, with early cultures having discovered that the chewing and ingestion of caffeine-containing plants or seeds caused specific positive mood-altering and stimula-

tory effects. Ancient Chinese legend asserts that Shennong, Emperor of China in approximately 3000 B.C., discovered tea after leaves accidentally fell into boiling water resulting in a fragrant restorative drink (Lu & Yu, 1995). Coffee originated in Africa around A.D. 575, where coffee beans were used for food as well as currency. During the tenth century, coffee was restricted primarily to Ethiopia where its native beans were first cultivated. As Arabic trade expanded, coffee beans moved into other regions including northern Africa, where they were mass-cultivated. Coffee beans eventually entered the European markets where historical sources indicate that coffee initially arrived in Venice as a result of the trade between Europeans and those from North Africa and the Far East.

The appreciation of coffee in Europe was evident in the sixteenth century, and became increasingly popular in the decades to follow. During the seventeenth century, the first "coffee houses" were established, with openings in Britain, Paris, and Venice. Following their inception, coffee houses became popular throughout Western Europe, where they served as a common forum for business and social relations as well as intellectual exchange. Coffee was not widely accepted as the caffeinated drink of choice throughout colonial America until Revolutionary Americans made a nationwide switch from tea to coffee in the eighteenth century. This change occurred as a protest to the Tea Act, in which heavy taxes were levied by the British government on tea imported by Americans. This revolt culminated in the Boston Tea Party, in which large quantities of tea were disposed of in Boston Harbor.

Similar to the coffee bean and tea leaf, the kola nut (cola) also appears to have been rooted in ancient cultural tradition. West African cultures use the kola nut as a euphoric stimulant to restore vitality and ease hunger pangs. Cola was an original additive to many soft drinks, and became the focus of a health scare in 1911 when the U.S. government seized portions of Coca-Cola syrup in Chattanooga, Tennessee, alleging that the product was unsafe due to its caffeine content (Benjamin, Rogers, & Rosenbaum, 1991). Following this seizure, the government initiated litigation (*The United States v. Forty Barrels and Twenty Kegs of Coca-Cola*), in an attempt to force Coca-Cola to remove caffeine from its formula. The public attention brought by the suit led to congressional action, which eventually resulted in the amendment of the Pure Food and Drug Act in 1912, including caffeine on the list of "habit-forming" and "deleterious" substances that must be listed on a product's label.

The earliest evidence of cocoa use comes from residue found inside ancient Mayan pots dated from 600 B.C. to A.D. 250 (Gorman, 2002). Made from the beans of the tropical plant *Theobroma cacao*, cocoa was a favorite drink of ancient Maya and Aztec people in Mesoamerica. Chocolate was consumed in a bitter and spicy beverage called *xocoatl*, often seasoned with vanilla, chile

pepper, and achiote. Xocoatl was believed to fight fatigue, a belief possibly attributable to the theobromine and caffeine content. Chocolate was a valuable commodity during this time as cocoa beans were often used as currency in trade. Chocolate was introduced to Europe in the 1500s, and became popular as a beverage by the late seventeenth century. In the latter part of the eighteenth century, chocolate production began in North America, with the establishment of a cocoa bean grinding mill in Massachusetts.

In 1819, pure caffeine was isolated for the first time by German chemist Friedrich Ferdinand Runge (Weinberg & Bealer, 2001). The structure of caffeine was elucidated near the end of the nineteenth century by Hermann Emil Fischer, who was also the first to achieve its total synthesis (Théel, 1902). During this time, cola products began to appear around the world and became one of the most prominent caffeinated beverages. Along with traditional sources of caffeine, including coffee, tea, and chocolate, the twentieth century saw the rise of caffeine intake through energy drinks, additives, and analgesic and appetite suppressant medications. Today the world population consumes more than 100,000 tons of caffeine annually, the equivalent of one caffeinated beverage per person per day.

PHARMACOKINETICS OF CAFFEINE

Taken orally, which is the typical method of drug administration, caffeine is rapidly and completely absorbed. Significant blood levels of caffeine are reached within 30 to 45 minutes of ingestion, with complete absorption occurring within the next 90 minutes (Julien, 2005). Caffeine's absorption occurs within the gastrointestinal tract, beginning in the stomach but occurring primarily within the small intestine. Following absorption, caffeine is distributed freely throughout the body and the brain. Caffeine is highly lipid-soluble, and higher concentrations are found within the brain as compared to plasma (Paton & Beer, 2001). Specifically, animal studies have demonstrated a significant disparity in brain/plasma concentration, with brain levels of caffeine as high as 80 percent (Kaplan, Greenblatt, Leduc, Thompson, & Shader, 1989).

Caffeine is metabolized to paraxanthine, theobromine, and theophylline, each with distinct effects on the body, including the breaking down of stored fat, blood vessel dilation, increased urine volume, and relaxation of bronchi muscles. These metabolites account for virtually all caffeine excretion, with only a small percentage of the original substance being eliminated as nonmetabolized caffeine.

The half-life of caffeine at modest levels of intake is approximately four to six hours in most adults. This period increases with higher levels of intake and

for the elderly and those with impaired liver function. Caffeine's half-life is extended significantly in women during the late stages of pregnancy, and in instances of long-term use of oral contraceptive steroids (Chawla & Suleman, 2006). Active cigarette smokers experience a shortened half-life for caffeine, but half-life increases following smoking cessation. Research has suggested that increased levels of caffeine in plasma following smoking cessation may contribute to cigarette withdrawal symptoms in heavy coffee drinkers (Feldman, Meyer, & Quenzer, 1997).

MECHANISM OF ACTION OF CAFFEINE

Caffeine acts through multiple mechanisms to exert a variety of effects on the central nervous system. Caffeine's principal mechanism of action is as an adenosine receptor antagonist (Julien, 2005). Caffeine readily crosses the blood brain barrier, and its similarity in structure to adenosine, a naturally occurring substance in the brain, allows the caffeine molecule to bind to adenosine receptors (particularly adenosine A_1 & A_{2a} receptors) on the surface of cells and block the access of adenosine to its receptors. Recent research suggests that the blockade of adenosine receptors and its ensuing reduction in adenosine activity is responsible for the behavioral stimulation associated with caffeine (Fisone, Borgkvist, & Usiello, 2004). Adenosine plays a key role in the transfer of energy within the body, and its inhibitory function promotes sleep and suppressed arousal, while also producing anti-inflammatory effects as well as acting as a cardiac antiarrhythmic agent. Adenosine also serves as a neuromodulator that produces an overall inhibitory effect by reducing the effects of acetylcholine, dopamine, and glutamate within the central nervous system (CNS). In particular, adenosine inhibits ascending acetylcholine projections to the thalamus and cortex, while inhibiting dopamine activity within the hippocampus, striatum, nucleus accumbens, and prefrontal cortex. In addition, adenosine mediates the effects of glutamate release in the hippocampus during excitotoxicity.

In humans, there are four types of adenosine receptors, including A_1, A_{2a}, A_{2b}, and A_3. Each receptor is encoded by a particular gene and has a specific function. The adenosine A_1 receptor is found throughout the entire body, and generally has an inhibitory function. For example, adenosine A1 receptors found in the brain slow metabolic activity. The adenosine A_1 and A_{2a} receptors both act to regulate myocardial oxygen consumption and coronary blood flow, with the A_1 receptor causing a decrease in heart rate, force of contraction, and responsivity to adrenaline, while the A_{2a} receptor increases blood flow through vasodilatation of coronary arteries. Caffeine antagonism of adenosine receptors prohibits adenosine activity within cells, thus altering their function.

Caffeine's blockade of adenosine receptors increases stimulatory activity, producing changes in mood, increased vigilance, and heightened mental acuity. For example, increased release of acetylcholine associated with adenosine inhibition contributes to caffeine's behavioral arousal effects. The positive stimulatory effects of caffeine are primarily due to the blockade of adenosine receptors that stimulate GABAergic neurons, which in turn typically inhibit the release of dopamine in the reward system of the striatum (Mandel, 2002). This inhibition of GABAergic neurons and the resulting increase in dopaminergic activity within certain areas of the brain may explain many of the behavioral effects associated with caffeine use, including heightened alertness, elevated energy, and increased concentration (Garrett & Griffiths, 1997). In particular, elevated dopamine levels in the prefrontal cortex produced by caffeine appear to contribute to an increase in alertness. In contrast, caffeine does not stimulate the release of dopamine in the nucleus accumbens, which is consistent with the mild behavioral reinforcing properties commonly noted (Acquas, Tanda, & Di Chiara, 2002).

In addition to adenosine antagonistic properties, caffeine is a competitive inhibitor of the enzyme cAMP-phosphodiesterase (cAMP-PDE), which converts cyclic AMP (cAMP) in cells to its noncyclic form, allowing cAMP to build up in cells. Cyclic AMP participates in the messaging sequence produced by cells in response to stimulation by epinephrine. Thus, by blocking the removal of cAMP, caffeine intensifies and prolongs the effects of epinephrine and epinephrine-like drugs such as amphetamine, methamphetamine, and methylphenidate.

The metabolites of caffeine also contribute to caffeine's effects. Theobromine is a caffeine metabolite that serves as a vasodilator, increasing the amount of oxygen and nutrient flow to the brain and muscles. Theophylline, the second of the three primary metabolites, acts as a smooth muscle relaxant that increases heart rate and efficiency. The third metabolic derivative, Paraxanthine, is responsible for an increase in the lipolysis process, which releases glycerol and fatty acids into the blood to be used as a source of fuel by the muscles (Dews, 1987).

BEHAVIORAL AND PHYSIOLOGICAL EFFECTS

The effect of caffeine on humans is largely a function of dosage, frequency of use, and individual sensitivity. Effects are often consistent with those reported by users of other CNS stimulants with very different mechanism of action, including cocaine and amphetamines. At low to moderate daily doses (50–300 mg), commonly reported subjective effects include arousal,

increased concentration, elevated mood, increased motivation to work, and decreased sleepiness. The most typical mood-altering effects include increases in energy, alertness, and feelings of well-being (Garrett & Griffiths, 1997). In comparison to the positive effects often reported by respondents consuming low to moderate doses, consumers of higher doses of caffeine increasingly report feelings of anxiety, nervousness, restlessness, and insomnia. The mechanism by which caffeine produces its subjective effects is yet to be determined. Caffeine has not shown to induce dopamine release in the nucleus accumbens of the brain, an action common to drugs with addictive properties, but more likely, exerts effects on mood through indirect stimulation of dopamine in the striatum. Recent research has shown that there is a direct correlation between the experience of positive effects and level of daily use (Attwood, Higgs, & Terry, 2006). Those consuming higher daily doses of caffeine (> 200 mg) were more likely to report positive effects, which may account for their high levels of use. In addition, it was found that while some moderate users did not report significant positive effects, they maintained their schedule of daily consumption, supporting the possibility that low to moderate use may be maintained by other factors, including environmental reinforcement and expectancy. There is controversy regarding the mechanism by which caffeine has shown to elevate mood, as well as performance on tasks utilizing alertness, speed, vigilance, and reaction time. While some have argued that the positive impact of caffeine can be directly attributed to the removal of withdrawal symptoms (James, 2005), more recent research has found improved performance and elevated mood to be a direct result of the stimulatory properties of caffeine, unrelated to effects of withdrawal (Christopher, Sutherland, & Smith, 2005; Hewlett & Smith, 2007; Smith, Sutherland, & Christopher, 2005).

Caffeine-induced anxiety is recognized in the *DSM–IV-TR* as a substance-related disorder characterized by the presentation of prominent anxiety, panic attacks, or obsessions or compulsions directly resulting from the physiological effects of caffeine (American Psychiatric Association [APA], 2000). Moderate daily caffeine consumption (> 200 mg) has been demonstrated to increase anxiety ratings in normal participants (Graham, Schultz, Mayo-Smith, Ries, & Wilford, 2003), while doses of 750 mg have been shown to induce panic attacks (Paton & Beer, 2001). People with a high disposition toward anxiety or those diagnosed with anxiety disorders are often very sensitive to the anxiogenic properties of caffeine, making them particularly vulnerable. Although there is some evidence that those with anxiety disorders tend to limit their exposure to caffeine (Kruger, 1996), it is generally recommended that those with anxiety-related disorders be advised to abstain completely from caffeine use.

Sleep appears to be the physiological process most sensitive to the effects of caffeine. Exposure to caffeine has the ability to produce cerebral stimulatory effects, especially in areas controlling locomotor activity and the sleep-wake cycle, and acute doses exceeding 200 mg are capable of producing significant effects on sleep (Chawla & Suleman, 2006). The *DSM–IV* recognized caffeine-induced sleep disorder as a significant disturbance in sleep brought on by the physiological effects of caffeine (APA, 1994). Caffeine typically disrupts sleep by prolonging sleep latency, shortening total sleep duration, and increasing nocturnal awakenings. Similar to its anxiogenic properties, caffeine's effect on sleep is determined by many factors, including dosage, tolerance, individual sensitivity, and time between ingestion and attempted sleep (Graham et al., 2003). Elevated doses correlate specifically to delays in sleep onset when ingested immediately prior to attempted sleep, and individual differences, such as slower metabolic rate, can increase instances of sleep disruption by extending drug availability. While partial tolerance can decrease caffeine's stimulatory effects, complete tolerance does not occur, leaving even the most habitual users at risk for sleep disturbances. Overall, caffeine's ability to disrupt sleep at high doses is well established, while difficulties at lower to moderate doses appear to be more attributable to individual differences, including sensitivity and schedule of use.

Caffeine also exerts significant effects on cardiovascular function. Research on caffeine's effect on the heart and heart disease is ongoing, and both positive and adverse effects have been demonstrated. Caffeine increases cyclic adenosine monophosphate (cAMP) in heart cells, which simulates the action of epinephrine, causing a slight stimulant effect. This stimulation increases heart rate, cardiac contractility (force of contraction), and cardiac output. Caffeine also has been shown to increase blood pressure, especially in those disposed to hypertension. Repeated blood pressure elevation in habitual users might contribute to an increased risk of heart disease (Lane, Pieper, Phillips-Butte, Bryant, & Kuhn, 2002). In contrast to negative effects, a recent study found that increased caffeine intake provided protection against the risk of heart disease mortality among nonhypertensive elderly participants (Greenberg, Dunbar, Schnoll, Kokolis, & Kassotis, 2007). A conclusive finding regarding the means by which caffeine provides such protection was unavailable; however, Greenberg et al. (2007) suggested that there was evidence to support the hypothesis that caffeine's ability to increase blood pressure counteracts postprandial hypotension. Other possible explanations for caffeine's ability to decrease the risk of heart disease among nonhypertensive elderly patients included commonly demonstrated cardiovascular effects such as increased myocardial contractility (strength of heart contraction), reduced fibrinolysis time (faster clot

breakdown), and inhibition of baroreflex activity (increases blood pressure). Also, caffeine's ability to dilate coronary arteries and increase blood flow to the heart has been demonstrated (Rachima-Moaz, Peleg, & Rosenthal, 1998), which might serve to mitigate negative effects in some individuals. With respect to increased risk for the typical adult population, it is possible that caffeine's ability to increase blood pressure is more prominent in coffee consumption than that of tea, as a recent study has indicated that the amino acid theanine (commonly found in tea) may reduce elevated blood pressure (Rogers, Smith, Heatherley, & Pleydell-Pearce, 2008). While there is not a consensus on the effects of caffeine consumption on heart disease, it is generally recommended that individuals with hypertension or heart disease minimize consumption.

As opposed to caffeine's effect on arteries of the heart, where dilation increases oxygen supply, caffeine has the opposite effect on the blood vessels of the brain; cerebral blood vessels are constricted, which decreases blood flow and reduces pressure within the brain (Julien, 2005). This action can prove beneficial for people suffering from headaches, and more specifically, treatment of migraines. Nonprescription medications containing a combination of acetaminophen, aspirin, and caffeine (e.g., Excedrin Migraine) have demonstrated effectiveness in reducing migraine-related symptoms (Chawla & Suleman, 2006).

Additional physiological effects of caffeine include changes in respiratory, urinary, and gastrointestinal function. Caffeine increases both gastric acid secretion and urine output, while producing antiasthmatic effects on lung function. Caffeine causes bronchial relaxation and is commonly used in the treatment of newborn infants who display apneic episodes (periodic suspension of breathing) or bronchopulmonary dysplasia, which is characterized by inflammation and scarring in the lungs. These conditions often occur in low-weight premature babies, and in such cases, caffeine has shown to be an essential aid in normalizing respiration and preventing death.

TOXICITY

Caffeine intoxication is recognized by both the *ICD–10* (World Health Organization [WHO], 1992) and *DSM–IV–TR* (APA, 2000) as a distinct syndrome associated with excessive caffeine use. Caffeine intoxication is defined by a number of clinical features that emerge in response to recent consumption of caffeine. The *DSM–IV* indicates that the diagnosis of caffeine intoxication is dependent on recent daily consumption, generally exceeding 250 mg (more than 2–3 cups of coffee), along with the presentation of intoxicated related symptoms following caffeine use. Common features of caffeine intoxication in adults are similar to the overdose effects of other central nervous system

stimulants (e.g., cocaine, amphetamine), and include anxiety, restlessness, excitement, insomnia, rambling flow of thought and speech, irritability, tremor, diuresis, flushed face, gastrointestinal disturbance, psychomotor agitation, and irregular or rapid heartbeat. Caffeine intoxication symptoms in infants include rapid heartbeat, rapid breathing, tremors, shock, muscle tension, nausea, and vomiting. In adult cases of highly elevated doses, symptoms such as fever, hallucinations, delusions, and loss of consciousness have occurred (Medline, 2006). Caffeine intoxication, due to its relatively short half-life (4–6 hours), typically resolves rather quickly with cessation of consumption and supportive care. However, individual reactions differ as related to overall health and caffeine sensitivity. Life-threatening complications, generally related to cardiac dysrhythmias, can occur and immediate medical care may be needed to restore cardiovascular stability. Peritoneal dialysis, hemodialysis, or hemofiltration may also be required in cases of severe caffeine overdose. Extreme dose toxicity can result in death, but such instances are very rare. Lethal dosage is dependent on weight and individual sensitivity to caffeine. However, doses exceeding 5 to 10 grams taken within a limited time frame are generally considered a significant risk. Such a sizeable dose is difficult to achieve under conventional methods (e.g., 50 to 100 average cups of coffee), but can be more readily administered with the ingestion of caffeine tablets. In cases of lethal overdose, the typical cause of death is often described as ventricular fibrillation (Holmgren, Nordén-Pettersson, & Ahlner, 2004).

TOLERANCE, DEPENDENCE, AND WITHDRAWAL

Tolerance refers to a decrease in responsiveness to a drug following repeated exposure in which the dose necessary to achieve the initial reinforcing effect is increased. Tolerance can also refer to the decline of aversive effects typically associated with large doses of the drug. Each of these altered responses to drug exposure can promote the use of increased dosage and repeated administration. Partial tolerance has been shown to develop among chronic caffeine users consuming levels as low as 100 mg per day (Evans & Griffiths, 1999). Caffeine tolerance can develop rather quickly among high-dose consumers. Complete tolerance to the sleep disruption effects of caffeine has been reported after consuming 400 mg of caffeine three times a day for seven days, while complete tolerance to subjective effects such as nervousness, tension, jitters, and elevated energy were observed to develop after consuming 300 mg three times per day for 18 days, and it is possible that such tolerance can occur within a shorter period of time (Griffiths & Mumford, 2000). With respect to chronic users, it should be noted that while tolerance has been shown to develop concerning

certain subjective measures (including mood and energy level), tolerance is more prevalent among physiological effects such as respiratory and cardiovascular function, along with sleep disruption.

Substance dependence due to caffeine is recognized as a clinical diagnosis in the *ICD–10* (WHO, 1992). Substance dependence refers to the presentation of cognitive, behavioral, and physiological symptoms related to an individual's continued use of a substance despite significant adverse consequences. Clinical diagnoses of substance dependence can be specified as with or without physiological dependence (evidence of tolerance or withdrawal). While caffeine dependence is not currently recognized as a clinical disorder in the *DSM–IV–TR* (APA, 2000), current research is focusing on whether the diagnosis of caffeine withdrawal or caffeine withdrawal syndrome is warranted for inclusion in future editions of this and other diagnostic texts. Caffeine withdrawal has been documented in numerous empirically validated double-blind experiments (Evans & Griffiths, 1999; Rogers, Martin, Smith, Heatherley, & Smit, 2003; Tinley, Yeomans, & Durlach, 2003). In a recent review of caffeine withdrawal studies, the following symptom categories were indicated as valid: headache, fatigue, decreased energy/activity, decreased alertness, drowsiness, decreased contentment, depressed mood, difficulty concentrating, irritability, and foggy/not clearheaded. It was also noted that additional flu-like symptoms, including nausea/vomiting, tremors, and muscle pain/stiffness are often reported. The most commonly reported symptom in caffeine withdrawal is headache, which occurs in approximately 50 percent of people reporting withdrawal symptoms (Juliano & Griffiths, 2004). Withdrawal symptoms generally begin within 12–24 hours following cessation of caffeine, and reach peak intensity at approximately 20–48 hours with an overall duration of two to seven days. Severity and occurrence of symptoms appear to relate to increases in dosage. However, withdrawal symptoms have been exhibited in those consuming as little as 100 mg/day (approximately one cup of coffee).

REPRODUCTIVE AND PRENATAL CONCERNS

The effect of maternal caffeine consumption on miscarriage and prenatal development remains undetermined, as credible scientific studies have reported a variety of findings. Concerning miscarriage, past studies have shown an increased risk for those consuming high daily doses (> 600 mg) of caffeine, while moderate doses did not increase the risk (Klebanoff, Levine, DerSimonian, Clemens, & Wilkins, 1999). A more recent study found that daily doses exceeding 200 mg were sufficient to increase the risk of miscarriage. In contrast, a recent study by Savitz, Chan, Herring, Howards, and Hartmann

(2008) demonstrated little evidence for risk of miscarriage among women with modest daily caffeine intake. Similar to the issue of miscarriage, caffeine's ability to affect fetal growth is a topic of current debate. A Norwegian study comparing caffeine intake of mothers of small-for-gestational-age (SGA) infants with mothers of non-SGA infants found that high caffeine intake during the third trimester correlated with an increased risk for growth retardation in male fetuses (Vik, Bakketeig, Ulla Trygg, Lund-Larsen, & Jacobsen, 2003). A separate European study reported that caffeine intake was not responsible for increased risk of SGA, and noted that many of the previous studies reporting such an effect had failed to account for other known contributory factors, including smoking and alcohol consumption (Parazzini et al., 2005). In addition to increased risk of SGA, instances of stillbirth also have been studied. In a study by Wisborg, Kesmodel, Hammer Bech, Hedegaard, and Brink Henriksen (2003) containing more than 18,000 pregnant women, those who consumed four to seven cups of coffee a day had an 80 percent increased risk of stillbirth, while women consuming more than eight cups a day had a 300 percent increased risk. While further research is needed to clarify the effects of caffeine on prenatal health and development, current medical recommendations generally advise pregnant women to minimize its consumption.

DRUG INTERACTIONS AND TREATMENT CONSIDERATIONS

Acute administration of high doses of caffeine to individuals diagnosed with panic disorder has been shown to increase cortisol to levels similar to those found in clinically depressed individuals (Paton & Beer, 2001). These elevated levels of cortisol can sometimes mimic the symptoms of depression, leading to false diagnosis. Similarly, caffeine can also interfere with medications prescribed for the treatment of insomnia. Benzodiazepines such as temazepam (Restoril) and triazolam (Halcion) are medications that bind to GABA-A receptors in the brain that enhance the function of GABA and create sedative and hypnotic effects. Caffeine can inhibit the binding of benzodiazepines to their specific receptors on the GABA-A receptor sites, therefore neutralizing the effects of such medications and inhibiting their sedative hypnotic effects (Sawnock, 1995). Such interactions should be considered when evaluating the effectiveness of medications used to treat insomnia. Caffeine screening and monitoring schedules can be an effective tool for proper diagnosis and medication management.

Caffeine also exerts a significant effect on other medications. For example, caffeine additives have demonstrated the ability to increase the effectiveness of

pain relievers in the treatment of headaches by as much as 40 percent while helping the body absorb headache medications more quickly, contributing to faster symptom relief. Thus, augmenting such medications with caffeine allows for dose reduction of the analgesic agent, which can reduce the risk for potential side effects and possible drug addiction (WebMD, 2008). With respect to the treatment of smoking cessation, the effect nicotine has on the metabolism of caffeine should be considered. In regular smokers, nicotine increases the metabolism of caffeine, while smoking cessation often decreases the rate of caffeine metabolism. Slower metabolism leads to increased levels of caffeine in blood plasma, and high levels of caffeine following smoking cessation could contribute to nicotine withdrawal symptoms (Julien, 2005).

Caffeine consumption is very common among patients with schizophrenia (Rihs, Muller, & Bauman, 1996). Heavy caffeine users may require higher doses of antipsychotic medication in treatment. The possible explanations for the elevated therapeutic dose requirements include the following: (1) caffeine may increase the severity of psychotic symptoms due to an increase in catecholamine release, which could result in an increase in medication to treat such symptoms; or (2) caffeine may interfere with the effectiveness of antipsychotic medication. Caffeine's blockade of adenosine receptors can result in increased dopamine binding to dopamine D2 receptors, which competes with the action of many antipsychotic agents that target the blockade of dopamine D2 receptors (Paton & Beer, 2001). Paton and Beer (2001) also suggest that caffeine may decrease the effectiveness of iron, lithium, and zinc, and that increases in caffeine could heighten the possibility of clozapine, olanzapine, and tricyclic antidepressant toxicity.

CONCLUSION

Caffeine is readily available around the world and consumed by most adults in the United States. Caffeine is most often ingested in coffee or tea, but can be found in a wide variety of foods, drinks, supplements, and medications. Taken orally, which is the typical method of drug administration, caffeine is rapidly and completely absorbed within the gastrointestinal tract and then metabolized and excreted with a typical half-life of four to six hours. Caffeine functions primarily as a central nervous system stimulant, with its principal mechanism of action as an adenosine A1 and A2a antagonist. Typical stimulatory effects include elevated arousal and concentration, reduced fatigue, and sleep disturbances. Caffeine also produces significant physiological effects on the cardiovascular, respiratory, and gastrointestinal systems, including increased heart rate and blood pressure, antiasthmatic effects, and increased gastric acid secretion and diuresis.

Caffeine intoxication is generally dependent on recent daily consumption exceeding 250 mg, and presents in adults with symptoms such as anxiety, restlessness, insomnia, tremor, gastrointestinal disturbance, and irregular or rapid heartbeat, with rare instances of hallucinations, delusions, and loss of consciousness following highly elevated doses. Caffeine intoxication symptoms in infants include rapid heartbeat, rapid breathing, tremors, shock, muscle tension, nausea, and vomiting. Extremely high doses of caffeine (5–10 g) can be lethal with the cause of death typically attributed to ventricular fibrillation. Tolerance and withdrawal symptoms have been demonstrated in caffeine users with the most prominent symptoms consisting of headache, fatigue, decreased alertness, and irritability. Withdrawal symptoms generally begin within 24 hours following cessation of caffeine, with an overall duration of two to seven days. Severity of symptoms is often a function of elevated doses or individual sensitivity, but caffeine tolerance has been shown to occur in users consuming as little as 100 mg a day, which is equivalent to one average cup of coffee.

Certain clinical uses for caffeine have been demonstrated, including treatment for migraine headaches, treatment of newborn infants who display apneic episodes (periodic suspension of breathing) or bronchopulmonary dysplasia, and as a possible protection against the risk of heart disease mortality among nonhypertensive elderly participants. Caffeine displays significant drug interactions with medications typically prescribed for anxiety, depression, insomnia, pain relief, and schizophrenia. Such interactions should be considered in the treatment of relevant disorders.

There is debate over caffeine's role in heart disease, prenatal and reproductive function, as well as its classification as an addictive substance. Current research is inconclusive, and further exploration regarding the role of caffeine in these and other conditions is warranted. In general, while caffeine appears safe in moderation, certain individuals, including pregnant women, those with anxiety-related disorders, cardiovascular disorders, or insomnia, are advised to limit its consumption.

REFERENCES

Acquas, E., Tanda, G., & Di Chiara, G. (2002). Differential effects of caffeine on dopamine and acetycholine transmission in brain areas of drug-naïve and caffeine-pretreated rats. *Neuropsychopharmacology, 27*(2), 182–193.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Attwood, A. S., Higgs, S., & Terry, P. (2006). Differential responsiveness to caffeine and perceived effects of caffeine in moderate and high regular caffeine consumers. *Psychopharmacology, 190*, 469–477.

Benjamin, L.T., Jr., Rogers, A. M., & Rosenbaum, A. (1991). Coca-Cola, caffeine, and mental deficiency: Harry Hollingworth and the Chattanooga trial of 1911. *Journal of the History of the Behavioral Sciences, 27*(1), 42–55.

Chawla, J., & Suleman, A. (2006). Neurologic effects of caffeine. *eMedicine,* Retrieved May 5, 2008, from http://www.emedicine.com/neuro/topic666.htm

Christopher, G., Sutherland, D., & Smith, A. (2005). Effects of caffeine in non-withdrawn volunteers. *Human Psychopharmacology: Clinical and Experimental, 20,* 47–53.

Dews, P. B. (Ed.). (1987). *Caffeine: Perspectives from recent research.* Berlin: Springer.

Evans, S. M., & Griffiths, R. R. (1999). Caffeine withdrawal: A parametric analysis of caffeine dosing conditions. *Journal of Pharmacology and Experimental Therapeutics, 289,* 285–294.

Feldman, R. S., Meyer, J. S., & Quenzer, L. F. (1997). *Principles of neuropsychopharmacology.* Sunderland, MA: Sinauer Associates.

Fisone, G., Borgkvist, A., & Usiello, A. (2004). Caffeine as a psychomotor stimulant: Mechanism of action. *Cellular and Molecular Life Sciences, 61*(7/8), 857–872.

Garrett, B. E., & Griffiths, R. R. (1997). The role of dopamine in the behavioral effects of caffeine in animals and humans. *Pharmacology Biochemistry and Behavior, 57*(3), 533–541.

Gorman, J. (2002, July 20). The original cocoa treat: Chemistry pushes back first use of the drink. *Science News, 162*(3), 38.

Graham, A. W., Schultz, T. K., Mayo-Smith, M. F., Ries, R. K., & Wilford, B. B. (Eds.). (2003). *Principles of addiction medicine* (3rd ed.). Chevy Chase, MD: American Society of Addiction.

Greenberg, J. A., Dunbar, C. C., Schnoll, R., Kokolis, S., & Kassotis, J. (2007). Caffeinated beverage intake and the risk of heart disease mortality in the elderly: A protective analysis. *American Journal of Clinical Nutrition, 85,* 392–398.

Griffiths, R. R., & Mumford, G. K. (2000). *Caffeine: A drug of abuse?* American College of Neuropsychopharmacology. Retrieved April 28, 2008, from http://www.acnp.org/g4/GN401000165/Default.htm

Hewlett, P., & Smith, A. (2007). Effects of repeated doses of caffeine on performance and alertness: New data and secondary analysis. *Human Psychopharmacology: Clinical and Experimental, 22,* 339–350.

Holmgren, P., Nordén-Pettersson, L., & Ahlner, J. (2004). Caffeine fatalities—four case reports. *Forensic Science International, 139,* 71–73.

James, J. E. (2005). Caffeine-induced enhancement of cognitive performance: Confounding due to reversal of withdrawal effects. *Australian Journal of Psychology, 57*(3), 197–200.

Juliano, L. M., & Griffiths, R. R. (2004). A critical review of caffeine withdrawal: Empirical validation of symptoms and signs, incidence, severity, and associated features. *Psychopharmacology, 176,* 1–29.

Julien, R. M. (2005). *A primer of drug action: A comprehensive guide to the actions, uses, and side effects of psychoactive drugs* (10th ed.). New York: Worth Publishers.

Kaplan, G. B., Greenblatt, D. J., LeDuc, B. W., Thompson, M. L., Shader, R. I. (1989). Relationship of plasma and brain caffeine metabolites to benzodiazepine receptor binding and locomotor activity. *Journal of Pharmacology and Experimental Therapeutics 248,* 1078–1083.

Klebanoff, M. A., Levine, R. J., DerSimonian, R., Clemens, J. D., & Wilkins, D. G. (1999). Maternal serum paraxanthine, a caffeine metabolite, and the risk of spontaneous abortion. *New England Journal of Medicine, 341*(22), 1639–1644.

Kruger, A. (1996). Chronic psychiatric patients' use of caffeine: Pharmacological effects and mechanisms. *Psychological Reports, 78,* 915–923.

Lane, J. D., Pieper, C. F., Phillips-Butte, B. G., Bryant, J. E., & Kuhn, C. M. (2002). Caffeine affects cardiovascular and neuroendocrine activation at work and home. *Psychosomatic Medicine, 64,* 595–603.

Lu, Y., & Yu, L. (1995). *Classic of tea: Origins and rituals.* New York: Ecco Press.

Mandel, H. G. (2002). Update on caffeine consumption, disposition and action. *Food and Toxicology, 40,* 1231–1234.

Medline. (2006). *Caffeine overdose.* Retrieved May 1, 2008, from http://www.nlm.nih.gov/medlineplus/ency/article/002579.htm

Meyer, J., & Quenzer, L. (2005). *Psychopharmacology: Drugs, the brain, and behavior.* Sunderland, MA: Sinauer Associates.

Parazzini, F., Chiaffarino, F., Chatenoud, L., Tozzi, L., Cipriani, S., Chiantera, V., et al. (2005). Maternal coffee drinking in pregnancy and risk of small for gestational age birth. *European Journal of Clinical Nutrition, 59,* 299–301.

Paton, C., & Beer, D. (2001). Caffeine: The forgotten variable. *International Journal of Psychiatry in Clinical Practice, 5,* 231–236.

Rachima-Moaz, C., Peleg, E., Rosenthal, T. (1998). The effect of caffeine on ambulatory blood pressure in hypertensive patients. *American Journal of Hypertension 11,* 1426–1432.

Rihs, M., Muller, C., & Bauman, P. (1996). Caffeine consumption in hospitalized psychiatric patients. *European Archives of Psychiatry and Clinical Neuroscience, 246,* 83–92.

Rogers, P. J., Martin, J., Smith, C., Heatherley, S. V., & Smit, H. J. (2003). Absence of reinforcing, mood and psychomotor performance effects of caffeine in habitual non-consumers of caffeine. *Psychopharmacology (Berlin), 167,* 54–62.

Rogers, P. J., Smith, J. E., Heatherley, S. V., & Pleydell-Pearce, C. W. (2008). Time for tea: Mood, blood pressure and cognitive performance effects of caffeine and theanine administered alone and together. *Psychopharmacology, 195,* 569–577.

Savitz, D. A., Chan, R. L., Herring, A. H., Howards, P. P., & Hartmann, K. E. (2008). Caffeine and miscarriage risk. *Epidemiology, 19*(1), 55–62.

Sawnock, J. (1995). Pharmacological rationale for the clinical use of caffeine. *Drugs, 49,* 37–50.

Smith, A., Sutherland, D., & Christopher, G. (2005). Effects of repeated doses of caffeine on mood and performance of alert and fatigued volunteers. *Journal of Psychopharmacology, 19,* 620–626.

Théel, Hj. (1902). *Presentation speech by professor Hj. Théel, president of the Swedish Royal Academy of Sciences on December 10, 1902*. Retrieved May 16, 2008, from http://nobelprize.org/nobel_prizes/chemistry/laureates/1902/press.html

Tinley, E. M., Yeomans, M. R., & Durlach, P. J. (2003). Caffeine reinforces flavour preference in caffeine-dependent, but not long-term withdrawn, caffeine consumers. *Psychopharmacology (Berlin), 166*, 416–423.

Vik, T., Bakketeig, L. S., Ulla Trygg, K., Lund-Larsen, K., & Jacobsen, G. (2003). High caffeine consumption in the third trimester of pregnancy: Gender specific effects on fetal growth. *Paediatric and Perinatal Epidemiology, 17*, 324–331.

WebMD. (2008). *Migraines, headaches, and caffeine*. Retrieved May 1, 2008, from http://www.webmd.com/migraines-headaches/guide/triggers-caffeine?page=2

Weinberg, B. A., & Bealer, B. K. (2001). *World of caffeine: The science and culture of the world's most popular drug*. New York: Routledge.

Wisborg, K., Kesmodel, U., Hammer Bech, B., Hedegaard, M., & Brink Henriksen, T. (2003). Maternal consumption of coffee during pregnancy and stillbirth and infant death in first year of life: Prospective study. *British Medical Journal, 326*, 420–422.

World Health Organization. (1992). *International statistical classification of diseases and related health problems* (10th ed.). *Classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.

Chapter **7**

Marijuana Withdrawal: A Survey of Symptoms

Dirk Hanson, MA

More than 14 million Americans smoke marijuana regularly, making it the most commonly used illicit drug in America. In 2006, marijuana was the only drug used by 52.8 percent of illegal drug users (U.S. Department of Health and Human Services, 2006). Over the past 15 years, as addiction researchers have been busily mapping out the chemical alterations in the human nervous system caused by alcohol, cocaine, nicotine, heroin, and tranquilizers, America's most popular illegal drug has remained largely a scientific mystery. Marijuana, the drug millions of Americans have been using regularly for years, is the least studied drug of all.

Why has cannabis research lagged behind that of other drugs of abuse? For decades, the prevailing belief among users and clinical researchers alike was that marijuana did not produce dependency and therefore could not be responsible for major withdrawal symptoms. This thinking is based, quite understandably, on the widespread observation that most marijuana users do not have difficulty going without marijuana, either by choice or by necessity. However, marijuana withdrawal effects are frequently submerged in the welter of polyaddictions common to active addicts. The withdrawal rigors of, say, alcohol or heroin tend to drown out the subtler manifestations of cannabis withdrawal. As Barbara Mason, director of the Laboratory of Clinical Psychopharmacology at Scripps Research Institute, has explained: "People are deciding every day whether to use or not to use marijuana, for medical purposes or otherwise, and there is little scientific information to advise this decision" (2008, 1).

Marijuana withdrawal, which typically affects only heavy smokers, has not been well characterized by the research community. Until recently, there was scant evidence in animal models for marijuana *tolerance* and *withdrawal*, the classic determinants of addiction. Now, however, several researchers have identified the existence of symptoms brought on by the abrupt discontinuation of regular marijuana use in both animal and human studies (de Fonseca et al., 1997). A growing body of evidence supports the existence of a clinically significant marijuana withdrawal syndrome in a subset of marijuana smokers. The syndrome is marked by irritability, restlessness, generalized anxiety, hostility, depression, difficulty sleeping, excessive sweating, loose stools, loss of appetite, a general "blah" feeling, and a mental state that has been described as "inner unrest."

Recent clinical research, combined with anecdotal field reports collected by the author, demonstrate the existence of marijuana withdrawal and the consistency of the most common symptoms of withdrawal and detoxification.

BACKGROUND

In 1992, molecular biologists identified the elusive brain receptor where tetrahydrocannabinol (THC), the primary active ingredient in marijuana, did its work. Shortly after that discovery, researchers at Hebrew University in Jerusalem identified the body's own form of THC, which uses the same CB1 receptors as THC (CB1 is cannabinoid receptor type 1). They christened the internally manufactured substance *anandamide*, after the Sanskrit *ananda*, or "bliss" (Fackelmann, 1993).

Anandamide has a streamlined three-dimensional structure that THC mimics. Both molecules slip easily through the blood brain barrier. Some of the mystery of marijuana's effects was resolved after researchers demonstrated that marijuana definitely increased dopamine activity in the limbic area of the brain. Tanda, Pontieri, and Di Chiara demonstrated that dopamine levels in the nucleus accumbens doubled when rats received an infusion of THC (1997). It appears that marijuana raises dopamine and serotonin levels through the intermediary activation of opiate and Gamma-aminobutyric acid (GABA) receptors (Wilson & Nicoll, 2001). (GABA is the chief inhibitory neurotransmitter in the mammalian central nervous system. The opiate receptor and other brain receptors are proteins located on the surfaces of nerve cells, or neurons.) THC may perform a signaling function in neurons containing GABA and glutamate.

THC and its organic cousin, anandamide, make an impressive triple play in the brain: They effect movement through receptors in the basal ganglia, they

alter sensory perception through receptors in the cerebral cortex, and they impact memory by means of receptors in the hippocampus. It is clear that some of the effects of cannabis are produced in much the same way as the effects of other addictive drugs—by means of neurotransmitter alterations along the limbic system's reward pathway.

A great deal of the early research was marred by inconsistent findings and differing definitions of addiction and withdrawal. Most recreational marijuana users find that too much pot in one day makes them lethargic and uncomfortable. Self-proclaimed marijuana addicts, on the other hand, report that pot energizes them, calms them down when they are nervous, or otherwise allows them to function normally. Heavy marijuana users claim that tolerance does build. And when they withdraw from use, many report strong cravings.

Work by Jones, Benowitz, and Herning had helped establish certain baseline symptoms—irritability, insomnia, and lack of appetite—as early as 1981. Studies by Budney, Novy, and Hughes in 1999 further outlined the syndrome in heavy daily marijuana smokers. But the abstinence effects were often inconsistent, and frequently hard to measure. Moreover, their clinical relevance was not always evident.

For marijuana withdrawal to be considered a clinical fact, several criteria had to be met. First, the typically transient pattern of withdrawal effects must be distinguishable from rebound effects. (A rebound effect is defined as the reappearance of a preexisting symptom, and is thus not considered a true withdrawal effect.) In addition, the symptoms must occur reliably, as demonstrated by comprehensive prospective studies (Budney, Hughes, Moore, & Vandrey, 2004). The symptoms under consideration must also be considered clinically significant. Finally, there needs to be a clear and repeatable timeline in evidence for the withdrawal effects.

It has been suggested that the reported symptoms of abrupt marijuana cessation do not rise to the level of withdrawal typically associated with drug detox. It is now possible to lay out the neurochemical basis of marijuana withdrawal, and to demonstrate that marijuana acts on the brain in a fashion similar to other addictive drugs.

There is solid experimental evidence that chronic, heavy cannabis users develop tolerance to its subjective and cardiovascular effects. "In summary," Budney et al. write, "cannabis withdrawal effects clearly occur in the majority of heavy, daily users" (2004, p. 1974). As a rough estimate, approximately 10 percent of marijuana users are at risk for dependence and withdrawal, the classic determinants of drug addiction (Joy, Watson, & Benson, 1999). There is clinical and epidemiological evidence that some heavy cannabis users experience problems in controlling their cannabis use, and continue to use the drug despite

experiencing adverse personal consequences of use (Hall, Solowij, & Lemon, 1999). Moreover, there is strong clinical evidence that some users experience a withdrawal syndrome upon the abrupt cessation of cannabis use. The timeline is similar to withdrawal from other addictive drugs.

In 2004, a group at the University of Vermont funded by the US National Institute of Drug Abuse (NIDA), undertook a critical review of all major relevant studies of the validity and clinical significance of marijuana withdrawal (Budney et al., p. 1967). The review of studies demonstrated with certainty that there are people with a propensity for heavy marijuana use who suffer a clearly delineated, verifiable, and frequently vivid set of withdrawal symptoms when they try to quit. One of the most striking pieces of evidence for this is the similarity of symptom sets emerging from the clinical studies to date. The most common "reliable and clinically significant" effects of abrupt withdrawal in heavy pot smokers, according to the University of Vermont research group, included "severity of craving and sleep difficulty, decreased appetite, and increased aggression, anger and irritability" (Budney, et al., p. 1967; Kouri, 2002, p. 30).

As another study author concluded: "Marijuana withdrawal doesn't include dramatic physical symptoms such as the pain, nausea, heavy sweating, and cramps associated with opiate withdrawal. Nevertheless, the symptoms of marijuana withdrawal appear clinically significant" (Zickler, 2002, p. 3). A recent comprehensive outpatient study (Kouri & Pope, 2000, p. 483) with prewithdrawal baselines showed greater levels of anxiety, negative mood, physical discomfort, and decreased appetite during abstinence but not at baseline, compared with two control groups. Moreover, in a "home environment" study, researchers worked with marijuana users who provided self-ratings during marijuana withdrawal; these users smoked an average of 3.6 times daily, did not use other drugs or abuse alcohol, and were free of major psychiatric disorders. The same symptoms predominated, and onset of symptoms occurred reliably within 48 hours of cessation. Moreover, "telephone interviews with collateral observers living with the participants confirmed participants' reports of increased irritability, aggression, and restlessness during abstinence. . . . [T]he validation of symptoms by home-based observers suggested that the effects were of a clinically significant magnitude" (Budney et al., 2004, p. 1971).

Other studies by Budney and colleagues expanded on the list of symptoms that changed significantly from baseline during withdrawal: "anger and aggression, decreased appetite, irritability, nervousness, restlessness, shakiness, sleep difficulty, stomach pain, strange dreams, sweating, and weight loss" (2003, p. 393; 2004, p. 1972). Although most effects were transient, generally lasting no more than two weeks, "strange dreams and sleep difficulties showed significant elevations throughout the study" (2003, p. 394). Budney et al. conclude

that, since most symptoms returned to baseline levels in the former users, "these findings were not rebound effects indicative of symptoms that existed before the use of cannabis" (2004, p. 1972). More recent studies by Haney and others "controlled for potential confounders by using placebo conditions and excluding persons who abused other substances, had an active psychiatric disorder, or were taking psychoactive medication" (Haney et al., 2004, p. 158).

Overall, the research cited above confirms that the most common marijuana withdrawal symptom is low-grade anxiety and dysphoria. Anxiety of this sort has a firm biochemical substrate. A peptide known as corticotrophin-releasing factor (CRF) is primarily responsible. Neurologists at the Scripps Research Institute in La Jolla, California, found that CRF levels in the amygdalas of animals in marijuana withdrawal were as much as three times higher than the levels found in animal control groups (Wickelgren, 1997, p. 1967). Long-term marijuana use alters the function of CRF in the limbic system in a manner similar to other addictive drugs (de Fonseca et al., 1997, p. 2051). (CRF receptors in the amygdala also play a direct role in alcohol withdrawal.)

METHOD

Personal observations and selected case histories of frequent marijuana users were gathered from anonymous, unedited comments posted on a blog site maintained by the author. Punctuation, capitalization, and spelling have been normalized in the excerpts included here. Most of the people who have posted comments thus far (more than 100) arrived at the site by means of the search term *marijuana withdrawal*. This may indicate that a large number of posters are heavy smokers seeking information about abstinence symptoms. The popularity of this search phrase on the Google search engine seems to suggest an interest in, and a need for, scientific information about marijuana withdrawal.

What has surprised many observers is that the idea of treatment for marijuana dependence seems to appeal to such a large and diverse group of people. NIDA has been able to find a cohort of withdrawal-prone smokers with relative ease. According to the principal investigator of one NIDA marijuana study, "We had no difficulty recruiting dozens of people between the ages of 30 and 55 who have smoked marijuana at least 5,000 times. A simple ad in the paper generated hundreds of phone calls from such people" (NIDA, 1999, p. 1). This would be roughly equivalent to 14 years of daily pot smoking.

Comments gathered from anonymous users at an open Web forum created for the discussion of marijuana withdrawal symptoms cannot be controlled for confounding variables such as other addictions or psychological disorders. The comment section of the Web site is open to anyone. What such surveys *can*

accomplish, however, is the demonstration of parallels, or lack of them, between findings in an experimental setting and anecdotal reports from the field. Survey studies cannot offer indisputable proof. Nonetheless, when combined with the results of formal clinical studies, such surveys offer a window into real-world experience, thus complementing the growing scientific data concerning marijuana withdrawal syndrome.

The comments were generated in large part by heavy, regular smokers who either recognized or have begun to recognize in themselves an addictive propensity toward marijuana. As a group, they have great difficulty—and suffer similar symptoms—whenever, and for whatever reason, they choose to abstain. Perhaps, most important, the present survey adds to the growing documentation of the contention that withdrawal symptoms are a frequent cause of relapse in marijuana smokers attempting to achieve abstinence.

RESULTS

All of the following comments can be found at the Web log *Addiction Inbox* (http://addiction-dirkh.blogspot.com/2007/10/marijuana-withdrawal.html). The unnumbered messages on the Web site are dated, and appear in chronological order.

1. Cave. (2008, February 8):
   "Well I just stopped smoking pot after 4 years of everyday use, 5 days ago. I am feeling the withdrawal symptoms ridiculously hard. No appetite, slight nausea, extreme insomnia."
2. Anonymous. (2008, February 26):
   "My boyfriend (of 6 years) has been a smoker for approximately 16 years. He has tried to give up a few times seriously before but has never quite gotten there yet. His behavior is almost unbearable when he does. It really takes a toll on our relationship. I never realized that it could be so bad and that his actions are so exaggerated by withdrawal."
3. Anonymous. (2008, February 26):
   "I'm a 30-year-old man and have been a heavy cannabis user (3 to 4 joints per day, every day) since I was 19. . . . I've been through intense anxiety, depression, restlessness, lack of appetite. I can't sleep for more than a few hours at a time and when I do, I sweat buckets. I have a terrible appetite, I'm cold all the time, like I can't regulate my temperature."
4. Anonymous. (2008, February 27):
   "I thought I was going crazy because all other sites told me that there were no withdrawal symptoms from pot, I can't think or eat and when I do finally get something down my gullet I get the runs straight after. . . . I feel like I have been hit by a truck and it has only been a week since I gave up."

5. Anonymous. (2008, March 1):
"I am 31 and a heavy smoker of 10 years. . . . What is really troubling me, however, is the excessive dreaming. . . . The dreams are vivid and strong, enough to wake me up sometimes."
6. Anonymous. (2008, March 3):
"This idea of 'intense dreaming' is very real and for the first 5 or 6 days after quitting I experienced life-like dreams/nightmares (99% nightmares), which would wake me from my sleep. . . . This idea of breaking out in cold sweat is also very real and quite scary when [it] occurs as [it] got me worried there was something else wrong with me."
7. Scott. (2008, March 3):
"I was blown away when I saw 'excessive sweating' as I have been experiencing that for a few days. . . . If I could cut back drastically, that would be the ideal situation. But I know from experience that I can't just smoke pot 'a little bit.' If I'm going to reduce, it's going to have to be all the way to zero."
8. Anonymous. (2008, March 7):
"I'm on day seven of abstinence and boy, do I feel lousy. Night sweats, anxiety, extreme insomnia, and loads of irritability/anger problems. . . . It's a bit like when you have a bad flu. You plain feel rotten. Anything stress-related is magnified ten-fold."
9. Bob. (2008, March 7):
"I'm 38 years old and have been using weed now daily for almost 21 years. . . . I've been 'clean' now for 4 days and so far it has obviously been difficult, but already I'm showing signs of improvement, the first two days I had no sleep at all. . . . My withdrawal symptoms: Loss of appetite, sweating, irritability, sudden crying fits."
10. Anonymous. (2008, March 8):
"I am a 25-year-old female and I have been smoking pot since I was 13. I have never stopped even a day that I can remember. Not unless I couldn't get it. I have recently started to realize that it is a drug addiction. I was always on the 'it's not addictive' side. I get very anxious if I think I'm not going to have any. . . . It is out of my control I think, and now I'm starting to not feel high. I really wanna stop, but am so scared of the symptoms. I think I need help."
11. Anonymous. (2008, March 18):
"Having read all of these comments and questions I no longer feel so abnormal. I have been experiencing most of these symptoms including vivid dreaming. . . . I have been a smoker since I was 15, every day smoking about 2–3 joints."
12. Anonymous. (2008, March 24):
"I am a 25-year-old female. I started smoking at 18. . . . I quit a few weeks ago. . . . I can't focus on anything. I can't make myself do anything. . . . I snap at everyone, including my boyfriend who has been complaining about my

excessive sweating. I didn't even think of the sweating as a symptom until I read the other posts here."

13. Anonymous. (2008, April 2):
"I just wanted to say I'm glad I found this site because as many people have noted the common wisdom is that there are few, if any, symptoms of withdrawal. . . . I've noticed the irritability and mood swings, which I expected, but didn't make the connection between the vivid and frequent dreams and waking at night until I read all the other comments."
14. Anonymous. (2008, April 8):
"I finally feel sane again after reading these postings. I am a 48-year-old male who has been smoking weed since 1975. Anywhere from 2–6 joints per day of good quality pot for the last four years. Decided to quit about a week ago and my life has been a living hell since. . . . Haven't eaten a full meal in a week, very tired and depressed, stomach in knots."
15. Anonymous. (2008, April 25):
"I quit weed 46 days ago. . . . pretty similar symptoms as everyone else and the most severe anxiety and depression I have ever known. . . . I can't concentrate or focus, I can't seem to forget about what has happened even though I want to, it feels as though my brain keeps reminding me about the 'situation' or some general anxious or negative thought just pops into my consciousness . . . like it's never going to end, like my thoughts are caught in a vicious circle."
16. Richard. (2008, May 3):
"It's not suicidal ideation but it's the feeling that life will just never 'be right'. . . . when you suffer from symptoms that you've been told don't exist, you are left looking for the wrong cause. So, if you're told that marijuana withdrawal does not increase anxiety, anger, or 'hopelessness,' you want to look for a cause of those things. . . . I went through withdrawal periods where I was inappropriately angry at the wrong thing, thinking that specific people were upsetting me when they were not."

DISCUSSION

The U.S. government's essentially unchanged opposition to marijuana research has meant that, until quite recently, precious few dollars were available for research. This official recalcitrance is one of the reasons for the belated recognition and characterization of marijuana's distinct withdrawal syndrome. According to research undertaken as part of the Collaborative Study of the Genetics of Alcoholism, 16 percent of people with a lifetime history of regular marijuana use reported a history of cannabis withdrawal symptoms (Schuckit et al., 1999). In earlier research, Mason discovered that those seeking treatment for

cannabis addiction tended to cluster in two age groups—college age and mid-50s, as reported by Somers (2008).

Budney et al. (2004, p. 1973) write:

> Regarding cross-study reliability, the most consistently reported symptoms are anxiety, decreased appetite/weight loss, irritability, restlessness, sleep problems, and strange dreams. These symptoms were associated with abstinence in at least 70% of the studies in which they were measured. Other clinically important symptoms such as anger/aggression, physical discomfort (usually stomach related), depressed mood, increased craving for marijuana, and increased sweating and shakiness occurred less consistently.

Today, scientists have a much better picture of the tasks performed by anandamide, the body's own form of THC. Among the endogenous tasks performed by anandamide are pain control, memory blocking, appetite enhancement, the suckling reflex, lowering of blood pressure during shock, and the regulation of certain immune responses. This knowledge helps shed light on the wide range of THC withdrawal symptoms, particularly anxiety, chills, sweats, flu-like physical symptoms, and decreased appetite.

Furthermore, we can look to indications for which marijuana is already being prescribed—anxiety relief, appetite enhancement (compounds similar to anandamide have been discovered in dark chocolate), suppression of nausea, relief from the symptoms of glaucoma, and amelioration of certain kinds of pain—for more insight into the common hallmarks of cannabis withdrawal.

What treatment measures can help ameliorate marijuana withdrawal and craving in heavy users who wish to quit? The immediate threat to any decision in favor of abstinence is what might fairly be called the "hair of the dog" effect. Note the findings of a 2004 paper in *Neuropsychopharmacology*: "Oral THC administered during marijuana abstinence decreased ratings of 'anxious,' 'miserable,' 'trouble sleeping,' 'chills,' and marijuana craving, and reversed large decreases in food intake as compared to placebo, while producing no intoxication" (Haney et al., p. 158).

Moreover, "Overall withdrawal severity associated with cannabis alone and tobacco alone was of a similar magnitude. . . . cannabis withdrawal is clinically important and warrants detailed description in the *DSM–V* and *ICD–11*" (Vandrey, Budney, Hughes, & Liguori, 2008, p. 48). It is possible that many more people are trying—and failing—to quit marijuana than researchers have previously suspected. Daily use of marijuana may be driven in part by the desire to avoid or eliminate abstinence symptoms (Haney, Ward, Comer, Foltin, & Fischman, 1999).

To date, there is no effective anticraving medication approved for use against marijuana withdrawal syndrome. More than a decade ago, Ingrid Wickelgren wrote in *Science:* "For instance, chemicals that block the effects of CRF or even relaxation exercises might ameliorate the miserable moods experienced by people in THC withdrawal. In addition, opiate antagonists like naloxone may, by dampening dopamine release, block the reinforcing properties of marijuana in people" (1997, p. 1967). Since stimulation of THC receptors has homologous effects on the endogenous opioid system, various investigators have speculated that naltrexone, the drug used as an adjunct of heroin withdrawal therapy, may find use against symptoms of marijuana withdrawal in people prone to marijuana dependence (Tanda et al., 1997). Further research is needed on the reciprocal relationship between THC and opioid receptor systems.

Serzone (nefazodone), an antidepressant, has been used to decrease some symptoms of marijuana withdrawal in human subjects who regularly smoked six joints per day (Haney et al., 2003). Anxiety and muscular discomfort were reduced, but Serzone had no effect on other symptoms, such as irritability and sleep problems.

Preliminary studies have found that lithium, used to treat bipolar disorder, curbed marijuana withdrawal symptoms in an animal study (Cui, Gu, Hannesson, Yu, & Zhang, 2001). Another drug for mania and epilepsy—Depakote—did not aid significantly in marijuana withdrawal (Haney et al., 2004).

Since difficulty sleeping is one common symptom of withdrawal, common prescription medications might be indicated for short-term use in the case of severe marijuana withdrawal. Some researchers have reported that even brief interventions, in the form of support group sessions, can be useful for dependent pot smokers (Copeland, Swift, & Rees, 2001).

It is also plausible to suggest that the use of marijuana by abstinent substance abusers may heighten the risk of relapse. In a study of 250 patients at a psychiatric/substance abuse hospital in New York, "Postdischarge cannabis use substantially and significantly increased the hazard of first use of any substance and strongly reduced the likelihood of stable remission from use of any substance" (Aharonovich et al., 2005, p. 1507). However, the researchers found that cannabis posed a greater risk to cocaine and alcohol abusers. For heroin, "cannabis use after inpatient treatment did not significantly affect remission and relapse" (Aharonovich, p. 1507).

It is surprising to note the relative paucity of previous clinical data the researchers had to work with in the case of alcohol and marijuana. "The gap in the literature concerning the relationship of cannabis use to the outcome of alcohol dependence was surprising," according to Aharonovich and col-

leagues. "We were unable to find a single study that examined the effects of cannabis use on post-treatment outcome for alcohol dependence, despite the fact that the majority of patients now in treatment for alcoholism dependence also abuse other drugs. Clearly additional studies of this issue are warranted" (2005, p. 1512).

Addiction researcher Barbara Mason of the Scripps Research Institute of La Jolla, California, is, at the time of this writing, overseeing a four-year study of the neurobiology of marijuana dependence under a grant from NIDA. The comprehensive project will involve both animal and human research, and will make use of state-of-the-art functional brain imaging. The federal grant will also be used as seed money for the new Translational Center on the Clinical Neurobiology of Cannabis Addiction at the Scripps Institute ("Scripps Given," 2008).

Above all, it is time to move beyond the common mistake of assuming that if marijuana causes withdrawal in some people, then it must cause withdrawal in everybody. And if it doesn't, it cannot be very addictive. This thinking has been overtaken by the growing understanding that a minority of people suffer a chemical propensity for marijuana addiction that puts them at high risk, compared to casual, recreational drug users. The fact that most people do not become addicted to marijuana and do not suffer from withdrawal is no more revealing than the fact that a majority of drinkers do not become alcoholics.

The idea of marijuana addiction and withdrawal remains controversial in both private and professional circles. For an unlucky few, a well-identified set of symptoms characterizes abstinence from heavy, daily use of pot. In this, marijuana addiction and withdrawal does not differ greatly from alcoholism—the vast majority of recreational users and drinkers will never experience it.

For those that do, however, the withdrawal symptoms of marijuana abstinence can severely impact their quality of life.

REFERENCES

Aharonovich, E., Liu, X., Samet, S., Nunes, E., Waxman, R., & Hasin, D. (2005). Post-discharge cannabis use and its relationship to cocaine, alcohol, and heroin use: A prospective study. *American Journal of Psychiatry, 162*(8), 1507–1514.

Budney, A. J., Hughes, J. R., Moore, B. A., & Novy, P. L. (2001). Marijuana abstinence effects in marijuana smokers maintained in their home environment. *Archives of General Psychiatry, 58*(10), 917–924. Retrieved February 27, 2008, from http://archpsyc.ama assn.org/cgi/content/full/58/10/917?ck=nck

Budney, A. J., Hughes, J. R., Moore, B. A., & Vandrey, R. (2004, November). Review of the validity and significance of cannabis withdrawal syndrome. *American Jour-*

nal of Psychiatry, 161, 1967–1977. Retrieved April 21, 2008, from http://ajp.psychiatryonline.org/cgi/content/full/161/11/1967

Budney, A. J., Moore, B. A., Vandrey, R., & Hughes, J. R. (2003). The time course and significance of cannabis withdrawal. *Journal of Abnormal Psychology, 112,* 393–402.

Budney, A. J., Novy, P. L., & Hughes, J. R. (1999, September 1). Marijuana withdrawal among adults seeking treatment for marijuana dependence. *Addiction, 94,* 1311–1322.

Copeland, J., Swift, W., & Rees, V. (2001, January). Clinical profile of participants in a brief intervention program for cannabis use disorder. *Journal of Substance Abuse Treatment, 20*(1), 45–52. Retrieved April 21, 2008, from http://www.ncbi.nlm.nih.gov/pubmed/11239727

Cui, S. S., Gu, G. B., Hannesson, D. K., Yu, P. H., & Zhang, X. (2001, December 15). Prevention of cannabinoid withdrawal syndrome by lithium: Involvement of oxytocinergic neuronal activation. *Journal of Neuroscience, 21*(24), 9867–9876. Retrieved April 27, 2007, from http://www.jneurosci.org/cgi/content/abstract/21/24/9867

de Fonseca, F. R., Rocío, M., Carrera, A., Navarro, M., Koob, G. F., & Weiss, F. (1997, June 27). Activation of corticotropin-releasing factor in the limbic system during cannabinoid withdrawal. *Science, 276,* 2050–2054.

Fackelmann, K. A. (1993, February 6). Marijuana and the brain: Scientists discover the brain's own THC-delta-9-tetrahydrocannabinol. *Science News.* Retrieved March 28, 2008, from http://findarticles.com/p/articles/mi_m1200/is_n6_v143/ai_13434805/pg_1

Hall, W., Solowij, N., & Lemon, J. (1999). *The health and psychological consequences of cannabis use.* (National Task Force on Cannabis Australia, Monograph Series No. 25). Sydney, NSW: University of New South Wales, National Drug and Alcohol Research Centre. Retrieved February 3, 2008, from http://www.druglibrary.org/schaffer/hemp/medical/home.htm

Haney, M., Hart, C. L., Vosburg, S. K., Nasser, J., Bennetti, A., Zubaran, C., et al. (2004). Marijuana withdrawal in humans: Effects of oral THC or divalproex. *Neuropsychopharmacology, 29,* 158–170.

Haney, M., Hart, C. L., Ward, A. S., & Foltin, R. W. (2003, January). Nefazodone decreases anxiety during marijuana withdrawal in humans. *Psychopharmacology, 165*(2), 157–165.

Haney, M., Ward, A. S., Comer, S. D., Foltin, R. W., & Fischman, M. W. (1999, February). Abstinence symptoms following smoked marijuana in humans. *Psychopharmacology, 141*(4), 395–404.

Hanson, D. (2007, October 17). *Addiction inbox: Marijuana withdrawal.* Retrieved May 3, 2008, from http://addiction-dirkh.blogspot.com/2007/10/marijuana-withdrawal.html

Jones, R. T., Benowitz, N. L., & Herning, R. I. (1981, August–September). Clinical relevance of cannabis tolerance and dependence. *Journal of Clinical Pharmacology,*

8–9(Suppl.), 143–152. Retrieved April 14, 2008, from http://www.ncbi.nlm.nih.gov/sites/entrez

Joy, J. E., Watson, S. J., & Benson, J. A. (1999). *Marijuana and medicine: Assessing the science base*. Institute of Medicine, Division of Neuroscience and Behavioral Health. Washington, DC: National Academy Press. Retrieved March 5, 2008, from http://www.nap.edu/html/marimed/

Kouri, E. M. (2002, February 1). Does marijuana withdrawal syndrome exist? *Psychiatric Times*, *19*(2). Retrieved March 17, 2008, from http://www.psychiatrictimes.com/display/article/10168/54701?pageNumber=3

Kouri, E. M., & Pope, H. G., Jr. (2000, November). Abstinence symptoms during withdrawal from chronic marijuana use. *Experimental and Clinical Psychopharmacology*, *8*(4), 483–492. Retrieved March 17, 2008, from http://www.ncbi.nlm.nih.gov/pubmed/

Mason, B. (2008, March 15). Scripps given $4M grant to study effects of marijuana. San Diego, *North County Times*, 1.

National Institute on Drug Abuse. (1999, April 20). *Chronic marijuana users become aggressive during withdrawal.* (NIDA News Release). Rockville, MD: Author. Retrieved April 9, 2008, from http://www.nida.nih.gov/MedAdv/99/NR-420.html

Schuckit, M. A., Daeppen, J.-B., Danko, G. P., Tripp, M. L., Li, T.-K., Hesselbrock, V. M., et. al. (1999). Clinical implications for four drugs of the *DSM–IV* distinction between substance dependence with and without a physiological component. *American Journal of Psychiatry*, *156*, 41–49.

"Scripps Given $4M Grant to Study Effects of Marijuana." (2008, March 15). *North County Times*, 1. Retrieved March 16, 2008, from http://www.nctimes.com/articles/2008/03/15/news/sandiego/16_02_343_14_08.txt

Somers, T. (2008, March 14). Study aims to clear haze surrounding pot addiction. *San Diego Union*, 1. Retrieved March 16, 2008, from http://www.signonsandiego.com/news/science/20080314–9999–1n14dope.html

Tanda, G., Pontieri, F. E., & Di Chiara, G. (1997, June 27). Cannabinoid and heroin activation of mesolimbic dopamine transmission by a common m1 opioid receptor mechanism. *Science*, *276*, 2048–2050.

U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration. (2006). *Results from the 2006 National Survey on Drug Use and Health: National Findings*. Rockville, MD: Office of Applied Studies. Retrieved March 12, 2008, from http://www.oas.samhsa.gov/NSDUH/2k6NSDUH/2k6results.cfm#Ch2

Vandrey, R. G., Budney, A. J., Hughes, J. R., & Liguori, A. (2008, January 1). A within-subject comparison of withdrawal symptoms during abstinence from cannabis, tobacco, and both substances. *Drug and Alcohol Dependence*, *92*, 48–54.

Wickelgren, I. (1997, June 27). Marijuana: Harder than thought? *Science*, *76*, 1967–1968.

Wilson, R. I., & Nicoll, R. A. (2001, March 29). Endogenous cannabinoids mediate retrograde signalling at hippocampal synapses. *Nature*, *410*, 588–592.

Zickler, P. (2002, October 17). Study demonstrates that marijuana smokers experience significant withdrawal. *NIDA Notes, 17(3)*. Bethesda, MD: National Institutes of Health, National Institute on Drug Abuse. Retrieved March 7, 2008, from http://www.drugabuse.gov/NIDA_notes/NNVol17N3/Demonstrates.html.

Chapter **8**

Marijuana Interaction with Methamphetamine Addiction

Mary F. Holley, MD

The use of marijuana has long been recognized as an antecedent to the use of so-called hard drugs such as methamphetamine. This gateway hypothesis has been roundly debated for many years and attributed to social factors, "peer pressure," and the availability of other drugs in settings in which marijuana is purchased and used. Twin studies have documented the strength of the association between early marijuana use (prior to age 17) and subsequent drug use. In twins discordant for early marijuana use, the marijuana-using twin was more than four times more likely to use and become dependent on stimulants (Lynskey et al., 2003).

The sheer magnitude of marijuana use among adolescents should give us pause to consider the effects of this psychoactive drug on the future of our children. Nearly half of 12th graders have tried marijuana, and 6 percent admit to daily use (Johnston, O'Malley, Bachman, & Schulenberg, 2005), and these are the kids who stayed in school. The rate of marijuana use among high school dropouts is likely to be even higher. The marijuana they are using is also much more potent than the "Iowa Ditch" that grew on the side of the road outside Des Moines in the late 1960s. Marijuana now is grown in high-tech growing labs, with delta-9-tetrahydrocannabinol (TCH) content as high as 15 percent (McLaren, Swift, Dillon, & Allsop, 2008).

This cannabis use is often occurring in adolescence, a time of significant neurologic maturation in areas of executive function, including decision making and impulse control. While multiple predisposing factors predict later drug use, including parental drug use, child abuse, conduct disorder, and novelty

seeking as a personality trait, the use of cannabis in adolescence and early adulthood emerged as the strongest risk factor for later involvement in other illicit drugs (Fergusson, Boden, & Horwood, 2008). Even a small increase in risk of addiction becomes socially significant when 50 percent of high school seniors are trying marijuana.

In addition, marijuana has effects on the brain in many of the same areas that methamphetamine changes, including the hippocampus and many areas of the frontal lobes so that injury to these areas caused by initial use of marijuana may be amplified by subsequent and concurrent use of methamphetamine. While scientists prefer to study isolated drug effects for the intellectual satisfaction of knowing precisely how various systems are affected, human behavior rarely cooperates. We are asked to predict the consequences of drug use based on single drug studies, when single drug use is the exception, rather than the norm.

Medication interactions have often been clinically significant in the context of prescription medications. These interactions are likely to be even more significant in the case of two very popular psychoactive drugs of unknown dose and purity used under conditions of concurrent exposure to alcohol, tobacco, and club drugs in people of variable age and underlying health. This chapter will focus on the interaction between marijuana and methamphetamine on brain function and behavior, with an eye to the development and clinical course of dependence on methamphetamine.

BIOCHEMICAL INTERACTIONS

Recent findings have revealed a vast neurotransmission system dubbed the endocannabinoid system due to its sensitivity to cannabinoid stimulation. The endocannabinoid system has been the subject of vigorous research for many years, not just for its interest as related to a drug of abuse, but also for its importance to understanding neurophysiology in general. The endocannabinoid system is in place not just so that people can get high on pot, but also for numerous neurologic processes of neuromodulation. It influences not only the reward system, but also appetite, learning and memory, and executive functions, including impulse control and decision making. We can expect that use of marijuana, particularly at critical developmental stages, prenatal exposure and adolescent drug use, would exert changes in these functions, perhaps long-lasting changes.

The endocannabinoid system has a complex relationship to the reward system of the brain, changing the sensitivity of the neural structures related to the sensation of pleasure. Interactions between cannabinoid receptors and responsiveness to alcohol (López-Moreno et al., 2008), heroin (Solinas, Yasar,

& Goldberg, 2007), and methamphetamine (Landa, Sulcova, & Slais, 2006) have been delineated, with vast implications for the potential addictiveness of these substances. Since marijuana is often used in the context of concurrent use of other drugs, these interactions must be clearly understood in this time of increasing marijuana use.

Marijuana is also a common drug of abuse in the adolescent age range, from age 10–19, and so its impact on adolescent neurodevelopment must be taken into account as well. Functions such as judgment, impulse control, predicting the future consequences of an action, and delayed gratification, are being established. Such executive functions are processed in the frontal and temporal lobes, in areas that are maturing in adolescence. It is likely the psychoactive drugs have an impact on the maturation of these areas, with profound effects on the mature personality.

IMPACT ON THE REWARD CIRCUIT

Marijuana has, first and foremost, effects on the reward circuit of the brain, resulting in a sensation of euphoria and generalized well-being. Rats treated with a cannabinoid CB1 agonist showed significant increases in dopamine release in the nucleus accumbens as an acute effect (Fadda et al., 2006).

Chronic exposure to TCH has been found to increase the length and number of dendritic branches in the shell of nucleus accumbens and also in the medial prefrontal cortex in rats (Kolb, Gorny, Limebeer, & Parker, 2006). Cannabinoid exposure then seems to increase the sensitivity of the reward circuit to dopamine stimulation at a cellular level. This heightened neural capacity for a dopamine response has its basis in changes in the neuroanatomy of the nucleus accumbens.

Specifically, stimulation of the CB1 cannabinoid receptors primes the reward pathway and sensitizes the system to be more responsive to methamphetamine stimulation (Landa et al., 2006) and to a moderate dose of alcohol (López-Moreno et al., 2008) in a persistent manner. The effect is not just a product of acute intoxication, but is rather a change in the neuroanatomy of the nucleus accumbens. This sensitization could result in a situation in which even low doses of lower-purity methamphetamine would be potently rewarding in individuals who have smoked marijuana prior to, or concurrent with, their first experience with methamphetamine.

This finding alone could help explain the widely differing "addiction rate" among methamphetamine users, depending on the social context of their drug use. Lower addiction rates are often seen in those who use methamphetamine occupationally to enhance their work performance (i.e., military personnel) or

medically (i.e., for obesity) versus those using it recreationally (Eliyahu, Berlin, Hadad, Heled, & Moran, 2007). While still experiencing the alertness, endurance, or anorectic aspects of methamphetamine, these individuals may be much less sensitive to the euphoric effects of the drug in the absence of previous or concurrent marijuana use.

EXECUTIVE FUNCTION

Marijuana has also been found to have significant effects on the functioning of the frontal lobe as it relates to executive function, including decision making and impulse control. This is of concern as methamphetamine also directly impacts many of the same areas. The use of marijuana may set the anatomic stage for the behavioral and personality changes, including the occupational failure and interpersonal strife, so often seen in methamphetamine addiction. These effects would likely be exaggerated in the case of adolescent users who are still in the process of maturing these brain structures. The combined forces of marijuana use and methamphetamine's effects could amplify the frontal lobe injury that so often complicates substance abuse treatment.

Impairment of the frontal lobe areas involved with executive function has been well documented in heavy marijuana users. Bolla, Brown, Eldreth, Tate, and Cadet in 2002 correlated marijuana use to poor performance in problem solving, learning, inhibition, and reaction time in heavy users who were abstinent for 28 days. To delineate the neurologic basis for this finding, Bolla then compared abstinent marijuana users with controls under positron emission tomography (PET) scanning (Bolla, Eldreth, Matochik, & Cadet, 2005) and found that the marijuana users showed less activation in the right lateral orbitofrontal cortex (OFC) and the right dorsolateral prefrontal cortex (DLPFC) than the control group on the Iowa Gambling Task. Heavy marijuana users did not perform as well as nonusers on this measurement of executive function and decision-making skills.

More recent use of marijuana has far more significant effects on the functioning of these executive functions. Pillay et al. (2008) demonstrated anterior cingulate and prefrontal dysfunction in marijuana users at 24 hours' abstinence that partially normalized after 28 days, though anterior cingulate function was still significantly impaired at 28 days' abstinence. Exposure to marijuana, even remotely—after 28 days of abstinence—compromises inhibitory control and executive function. The degree of cognitive impairment is even greater in the presence of acute intoxication, which is the more common context of an individual's first use of methamphetamine.

In tests of inhibition processing, fMRI testing was done at 28 days' abstinence during a Stroop task involving inhibition of the dominant process of reading words to instead give the colors of words printed in incongruent ink. The cortical activation pattern of the heavy marijuana users reflected reduced activation of the anterior cingulate and more widespread dorsolateral prefrontal lobe compensatory activity (Eldreth, Matochik, Cadet, & Bolla, 2004; Gruber & Yurgelun-Todd, 2005). Marijuana users are thus more dependent on dorsolateral prefrontal lobe activity to be successful in inhibitory processing.

Methamphetamine use significantly compromises these "back-up" areas and further impairs decision making and impulse control. The anatomic basis for this reduced impulse control is demonstrated by several fMRI studies involving human methamphetamine users attempting inhibitory control tasks under fMRI monitoring. Anterior cingulate and dorsolateral prefrontal cortex activity has been shown to be essential in response inhibition and impulse control (Garavan, Ross, Murphy, Roche, & Stein, 2002). Studies have demonstrated reduced task-related activation of the anterior cingulate gyrus in methamphetamine users (Hwuang et al., 2006). Paulus et al. (2002) studied methamphetamine addicts in early recovery by fMRI as they did a two-choice prediction task and a two-choice response task. They demonstrated less activation of dorsolateral prefrontal cortex and failure to activate ventromedial prefrontal cortex during this decision-making task.

These changes appear to be more than just a pharmacologic effect of methamphetamine. There appears to be some cellular destruction of a more permanent nature going on as well. The massive releases of neurotransmitter caused by methamphetamine use result in high levels of nitrogen and oxygen free radical formation (Acikgov et al., 2000). These free radicals are formed by the metabolism of methamphetamine, and also by the breakdown of the huge amounts of monoamine neurotransmitters that have been released both intra and extra cellularly. These monoamine neurotransmitters must also be broken down, and MAO, the usual enzyme to do that, is inhibited by methamphetamine. Alternative metabolic routes are used, resulting in the generation of large amounts of hydroxyl free radicals nitric oxide and peroxynitrite, which are extremely toxic to brain cells (Jeng, Ramkissoon, Parman, & Wells, 2006).

Free radical compounds denature proteins, damage DNA, and generally wreak havoc in the areas of the brain in which they are concentrated (Cubells, Rayport, Rajendran, & Sulzer, 1994). Because most of the neurotransmitters are released in the midbrain, nucleus accumbens, and striatum, and in the prefrontal cortex, those areas are disproportionately affected by methamphetamine abuse with progressively worsening cognitive and executive function (Li,

Wang, Qiu, & Luo, 2008). Nordahl et al. (2005) did magnetic resonance spectroscopy (MRS) measures of N-acetylaspartate-creatine and phosphocreatine (NAA/Cr), choline-creatine and phosphocreatine (Cho/Cr), and choline-N-acetylaspartate (Cho/NAA) ratios in the anterior cingulate cortex of abstinent meth users and found evidence of cellular compromise that only partially corrected after prolonged periods (years) of abstinence.

These changes have implications for addiction recovery and relapse as function in crucial frontal lobe areas is compromised. Methamphetamine users had significantly impaired inhibitory control on a Stop Signal Test measuring latency to inhibit a motor response (Monterosso, Aron, Cordova, Xu, & London, 2005). Impulsivity is also related to impaired perception of time intervals, with methamphetamine abusers consistently overestimating time intervals and accelerating fingertaps (Wittmann, Leland, Churan, & Paulus, 2007). This "trigger fingered" impaired capacity for inhibitory processing would reduce the ability to resist impulses, delay gratification, and thus, increase the likelihood of relapse. In a landmark study, Paulus, Tapert, and Schuckit (2005) showed that those addicts who eventually relapsed had markedly reduced activation of the dorsolateral prefrontal cortex and anterior cingulate gyrus compared to addicts who did not subsequently relapse. Subjects were followed for up to three years to observe for relapse, and the predictive power of this functional measure of brain activity in these areas was impressive.

MEMORY AND LEARNING

In addition to its direct effects on the reward circuit and executive function, marijuana has significant effects on the hippocampus and various areas of the frontal lobe that could also affect memory and learning, in a way that could increase the likelihood that a person would use and become dependent on methamphetamine, and increase the neurologic impact of his methamphetamine use. A hippocampus, for instance, that is already compromised by marijuana use might be more vulnerable to the neurotoxic effects of methamphetamine.

Long- and short-term heavy cannabis use leads to impaired verbal memory and fluency, attention, and psychomotor speed at 24 hours after last use (in the absence of intoxication), which has implications for occupational performance and driving safety (Messinis, Kyprianidou, Malefaki, & Papathanasopoulos, 2006). Short-term abstinent cannabis users had deficits in verbal fluency, visual recognition, delayed visual recall, and short- and long-interval prospective memory. There were no differences for immediate visual recall (McHale & Hunt, 2008). Recent users also demonstrated abnormal brain activation pat-

terns during a working memory task, with recruitment of additional regions not typically used for this type of working memory (Kanayama, Rogowska, Pope, Gruber, & Yurgelun-Todd, 2004).

Even after 28 days of abstinence, performance was significantly worse in both long- and short-term memory for heavy users. Nestor, Roberts, Garavan, and Hester (2008) demonstrated that cannabis-using adults had significantly lower activity in the superior temporal gyrus, and several areas of the frontal lobe, compared to controls during learning in a name-face task. Results also showed that cannabis-using adults had significantly lower activity in the frontal and temporal lobes, and higher activity in the right parahippocampal gyrus, during learning. These changes indicate functional deficits and compensatory processes in cannabis users.

Heavy use is defined as daily smoking of at least one joint per day, a condition met by about 6 percent of all high school seniors (Johnston et al., 2005). While moderate recreational users of marijuana had normal performance on tests of working memory and selective attention, cannabis users displayed a significant alteration in brain activity in the left superior parietal cortex after 10 days of abstinence (Jager, Kahn, Van Den Brink, Van Ree, & Ramsey, 2006), indicating that even moderate use has an effect on brain function. But again, those same frontal lobe structures compromised by marijuana use may be more susceptible to the more serious neurotoxic effects of methamphetamine when it is used after or concurrent with marijuana.

The toxicity of methamphetamine is profound, and the cognitive changes seen in methamphetamine addicts are far more pronounced than those associated with marijuana. Methamphetamine abusers have cognitive deficits, abnormal metabolic activity, and structural deficits in frontal, temporal, and para hippocampal cortices, and reduced hippocampal volume. The magnitude of disruption in these areas is correlated with cognitive deficits in attention, memory, and executive function in many domains (London et al., 2005; Thompson et al., 2004). Scott et al. (2007) did a meta-analysis of available data on the neuropsychological effects of methamphetamine abuse/dependence. They revealed deficits in episodic memory, executive functions, information-processing speed, motor skills, language, and visuoconstructional abilities in methamphetamine users that were far more prominent and easily measurable than those found in marijuana users. Primate studies of methamphetamine-exposed animals have demonstrated profound impairment in cognitive function, particularly spatial working memory and long-term associative memory that were related to dopamine deficiencies in the prefrontal cortex, cingulate cortex, and striatum (Castner, Volser, & Goldman-Rakic, 2005). These cognitive deficits greatly impair ability to participate in cognitive behavioral therapy as a com-

ponent of rehabilitation. Memory, attention span, and information processing slowly improve over the course of 12–18 months, requiring prolonged treatment at great cost.

DEVELOPMENTAL CONSIDERATIONS

A large number of the people who are initiating methamphetamine use are in the adolescent age group, with an average age of 19 (Brecht, Greenwell, & Anglin, 2006), and nearly all of them use alcohol, tobacco, and/or marijuana prior to first use of methamphetamine. Adolescents are more likely to smoke or snort meth than to inject it at their first use (Wood et al., 2008) and to do so in the context of marijuana use. Youth and young adults are in a developmental phase in which the rate of maturation and development of the brain is exceeded only by that of infancy and early childhood. In this extremely important developmental window, our teenagers are exposing themselves to neurotoxic drugs that will affect their lives and opportunities significantly.

The vulnerability of the adolescent brain is just beginning to be understood. The endocannabinoid inhibition of synaptic function in the hippocampus was more pronounced in adolescent rats than adults, which may account for the increased sensitivity of adolescent animals to TCH-induced memory impairment (Kang-Park, Wilson, Kuhn, Moore, & Swartzwelder, 2007). Adolescent THC exposure resulted in CB1/G protein uncoupling in the hippocampus that persisted into adulthood (Rubino et al., 2008), suggesting that a cannabinoid-related disconnection can be expressed in adulthood as a developmental deficit. These animal studies support the conclusion that the neurologic changes seen with human marijuana use do not likely precede drug use (as predisposing factors), but are in fact consequences of adolescent marijuana use in normal teens.

Normal adolescent neurodevelopment includes extensive remodeling of the frontal lobes, occurring between the ages of 12 and 21 (Gogtay et al., 2004; Shaw et al., 2008). This process involves myelination of white matter tracts and synaptic pruning in areas of the frontal lobes involved in executive function, including judgment, prediction of future consequences, inhibitory processes, and impulse control. All of these functions are important when resisting peer pressure and controlling alcohol and drug intake.

Tapert et al. (2007) measured brain function by an fMRI technique during a go/no-go task, indicating inhibitory control in adolescent marijuana users compared to controls (controlled for alcohol intake). Marijuana users performed as well as nonusers, but recruited additional cortical areas in the frontal and parietal lobes, in order to perform the task. These changes were noted after 28 days of abstinence and thus reflect a persistent finding. These findings suggest that a key capability of executive function, inhibition of impulses, is

impaired not just when an individual is intoxicated, as classical gateway theories have posited, but for weeks afterward, during a developmental period in which forebrain maturation is taking place.

The effects of marijuana on other cognitive functions have also been found to be more significant in adolescents than in adults. Memory and learning are critical functions for academic and occupational success and have been found to be distorted in adolescent marijuana users. Schweinsburg et al. (2008) showed that marijuana-using adolescents, after one month of abstinence, performed normally on a spatial working memory assessment, but had markedly different cortical activation patterns, reflecting different attention mental strategies used to achieve the same end result. In another fMRI study of marijuana-using adolescents studied in a spatial working memory task, users showed changes in recruitment of the anterior cingulate gyrus, temporal cortex, and hippocampal gyrus, again at 28 days' abstinence (Padula, Schweinsburg, & Tapert, 2007).

Not all of the studies have shown equivalent performance for marijuana-using adolescents. Medina and Tapert's group has done extensive research into the state of the hippocampus in adolescents who use marijuana, alcohol, and both, and found significant differences in hippocampal volumes. They also demonstrated, after a month of monitored abstinence, marijuana users had slower psychomotor speed, poorer complex attention, poorer story memory, and reduced planning and sequencing ability, all correlated with lifetime marijuana exposure and controlling for alcohol use (Medina et al., 2007). Smaller hippocampal volumes in the marijuana-using youth were also associated with more depressive symptoms on the Beck Depression Inventory (Medina, Nagel, Park, McQueeny, & Tapert, 2007).

These studies suggest long-lasting changes in the neuroanatomy of important areas in the frontal lobes related to the acquisition of executive functions. If these areas do not mature properly during adolescence, a developmental window of opportunity is missed. We often find in the course of working with addicted young adults that their emotional maturity matches the chronologic age at which they first began abusing substances. One of the challenges of addiction treatment is encouraging our clients to "grow up." Subsequent maturation can and does occur, but with a great deal more effort on the part of both client and therapist than would be the case with normal adolescence. We must seriously consider such neuroanatomic findings before we blithely accept the fact that 50 percent of our young people are using pot.

PRENATAL EXPOSURE TO POT

Our final concern will be with the issue of prenatal exposure to marijuana and its effects on the unborn child. The young adults and adolescents we have

been describing are predominantly of childbearing age. In Barcelona, Spain, 5.3 percent of newborns had a meconium screen positive for marijuana exposure, while only 1.7 percent of mothers admitted to using it during pregnancy (Lozano et al., 2007). In the United States, 6 percent of newborns in four major cities were positive for marijuana in 2003, an increase from 3 percent in 1993 (Derauf et al., 2007).

Parental substance abuse is a well-known risk factor for the development of substance abuse in children. Children witnessing the substance abuse of a parent are much more likely to view substance abuse as normal and acceptable behavior. They are also likely to have experienced numerous adverse childhood events, including abuse and neglect, which also predispose to addiction as they grow older (Dube et al., 2003).

But there is a little more to it than that. Prenatal exposure to marijuana predicted early use of marijuana by offspring by the age of 14, even after controlling for the child's current alcohol and tobacco use, pubertal stage, sexual activity, delinquency, peer drug use, family history of drug abuse, and characteristics of the home environment, including parental depression, current drug use, and strictness/supervision (Day, Goldschmidt, & Thomas, 2006). Prenatal exposure to marijuana produces changes in the neurologic make-up of the child that independently predispose to later substance abuse.

The endocannabinoid system is key to the early development of the central nervous system, serving as a traffic cop, directing the proliferation, migration, differentiation, and synapse formation of functional neural circuits throughout the developing brain (Harkany et al., 2007). Rats exposed to THC prenatally had increased anxiety scores measure by peeps when removed from the nest, inhibited juvenile social interaction, and play among adolescents, extending into adulthood with impaired elevated maze performance (Trezza et al., 2008). Emotional development has been found to be impaired in humans also. Prenatal marijuana exposure in the first and third trimesters predicted significantly increased levels of depressive symptoms in 10-year-old children (Gray, Day, Leech, & Richardson, 2005).

Prenatal marijuana exposure is also associated with cognitive deficits in children as measured by the Stanford-Binet verbal reasoning and short-term memory scales at ages three, four, and six (Day, Richardson, Goldschmidt, Robles, & Taylor, 1994; Fried & Watkinson, 1990; Goldschmidt, Richardson, Willford, & Day, 2008). Childhood cognitive deficits and learning disabilities represent a nonenvironmental risk factor for subsequent substance abuse. More significantly, prenatal marijuana exposure is associated with impaired executive function and impulse control at age 9–12 (Fried, Watkinson, & Gray, 1998), with poorer Wide Range Achievement Test-Revised (WRAT-R) reading compre-

hension and spelling scores at age 10 (Goldschmidt, Richardson, Cornelius, & Day, 2004), and with impaired higher-order mental function, analysis, and integration at age 13–16 (Fried, Watkinson, & Gray, 2003). These deficits are strong predisposing factors placing children at risk for substance abuse in those vulnerable adolescent years, even if their role models are drug free (Karacostas & Fisher, 1993).

CONCLUSION

Marijuana use is a common antecedent to methamphetamine use, and both drugs affect similar structures in the brain. While methamphetamine is by far the more toxic of the two, causing significant functional disability that may take months or even years of therapy to reverse, marijuana may contribute more to this injury than has been previously acknowledged. Marijuana sensitizes the reward circuit to respond to methamphetamine more robustly, and predisposes to addiction to methamphetamine. Marijuana also appears to aggravate the injury to cognitive structures by impairing the function of the key areas involved in memory and higher-order thinking capacity. These effects are magnified even further when drug use occurs in adolescence, as the brain is going through a major remodeling in preparation for adulthood. When key maturation events fail to occur, there may not be an opportunity for a redo.

Clearly, the scientific findings related to marijuana need to be evaluated whenever the legalization of this drug is considered. The long-term implications of the widespread use of any psychoactive drug should be carefully thought out. If alcohol were up for evaluation by the FDA as a potential addition—and danger—to our society, the data would probably argue for its exclusion. Alcohol causes untold social harm and personal tragedy. Do we really need another intoxicant in our already overly intoxicated society?

REFERENCES

Acikgov, O., Gapnenas, S., Kayatekin, B. M., Pekasetin, C., Uysal, N., Dayi, A., et al. (2000). The effects of a single dose of methamphetamine on lipid peroxidation levels in the rat striatum and prefrontal cortex. *European Neuropsychopharmacology, 10*(5), 415–418.

Bolla, K. I., Brown, K., Eldreth, D., Tate, K., & Cadet, J. L. (2002). Dose-related neurocognitive effects of marijuana use. *Neurology, 59*(9), 1337–1343.

Bolla, K. I., Eldreth, D. A., Matochik, J. A., & Cadet, J. L. (2005). Neural substrates of faulty decision making in abstinent marijuana users. *Neuroimage, 26*(2), 480–492.

Brecht, M. L., Greenwell, L., & Anglin, M. D. (2006). Substance use pathways to methamphetamine use among treated users. *Addictive Behaviors, 32*(1), 24–38.

Castner, S. A., Volser, P. S., & Goldman-Rakic, P. S. (2005). Amphetamine sensitization impairs cognition and reduces dopamine turnover in primate prefrontal cortex. *Biologic Psychiatry, 57*, 743–751.

Cubells, J. F., Rayport, S., Rajendran, G., & Sulzer, D. (1994). Methamphetamine neurotoxicity involves vacuolization of endocytic organelles and dopamine dependent intracellular oxidative stress. *Journal of Neuroscience, 14*(4), 2260–2271.

Day, N. L., Goldschmidt, L., & Thomas, C. A. (2006). Prenatal marijuana exposure contributes to the prediction of marijuana use at age 14. *Addiction, 101*(9), 1313–1322.

Day, N. L., Richardson, G. A., Goldschmidt, L., Robles, N., & Taylor, P. M. (1994). Effect of prenatal marijuana exposure on the cognitive development of offspring at age three. *Neurotoxicologic Teratology, 16*(2), 169–175.

Derauf, C., LaGasse, L. L., Smith, L. M., Grant, P., Shah, R., Arria, A., et al. (2007). Demographic and psychosocial characteristics of mothers using methamphetamine during pregnancy: Preliminary results of the infant development, environment, and lifestyle study (IDEAL). *American Journal of Drug and Alcohol Abuse, 33*, 281–289.

Dube, S. R., Felitti, V. J., Dong, M., Chapman, D. P., Giles, W. H., & Anda, R. F. (2003). Childhood abuse, neglect, and household dysfunction and the risk of illicit drug use: The adverse childhood experiences study. *Pediatrics, 111*(3), 564–572.

Eldrith, D. A., Matochik, J. A., Cadet, J. L., & Bolla K. L. (2004). Abnormal brain activity in prefrontal brain regions in abstinent marijuana users. *Neuroimage, 23*, 914–920.

Eliyahu, U., Berlin, S., Hadad, E., Heled, Y., & Moran, D. S. (2007). Psychostimulants and military operations. *Military Medicine, 172*(4), 383–387.

Fadda, P., Scherma, M., Spano, M. S., Salis, P., Melis, V., & Fattore, L. (2006). Cannabinoid self-administration increases dopamine release in the nucleus accumbens. *Neuroreporter, 17*(15), 1629–1632.

Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2008). The developmental antecedents of illicit drug use: Evidence from a 25-year longitudinal study. *Drug and Alcohol Dependence, 96*(1–2), 165–177.

Fried, P. A., & Watkinson, B. (1990). 36- and 48-month neurobehavioral follow-up of children prenatally exposed to marijuana, cigarettes, and alcohol. *Journal of Developmental and Behavioral Pediatrics, 11*(2), 49–58.

Fried, P. A., Watkinson, B., & Gray, R. (1998). Differential effects on cognitive functioning in 9- to 12-year-olds prenatally exposed to cigarettes and marihuana. *Neurotoxicoogicl Teratology, 20*(3), 293–306.

Fried, P. A., Watkinson, B., & Gray, R. (2003). Differential effects on cognitive functioning in 13- to 16-year-olds prenatally exposed to cigarettes and marihuana. *Neurotoxicologic Teratology, 25*(4), 427–436.

Garavan, H., Ross, T. J., Murphy, K., Roche, R. A., & Stein, E. A. (2002). Dissociable executive function in the dynamic control of behavior: Inhibition, error detection, and correction. *Neuroimage, 27,* 1820–1829.

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., et al. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Science U S A, 101*(21), 8174–8179.

Goldschmidt, L., Richardson, G. A., Cornelius, M. D., & Day, N. L. (2004). Prenatal marijuana and alcohol exposure and academic achievement at age 10. *Neurotoxicologic Teratology, 26*(4), 521–532.

Goldschmidt, L., Richardson, G. A., Willford, J., & Day, N. L. (2008). Prenatal marijuana exposure and intelligence test performance at age 6. *Journal American Academy of Child and Adolescent Psychiatry, 47*(3), 254–263.

Gray, K. A., Day, N. L., Leech, S., & Richardson, G. A. (2005). Prenatal marijuana exposure: Effect on child depressive symptoms at ten years of age. *Neurotoxicologic Teratology, 27*(3), 439–448.

Gruber, S. A., & Yurgelun-Todd, D. A. (2005). Neuroimaging of marijuana smokers during inhibitory processing: A pilot investigation. *Brain Research: Cognitive Brain Research, 23*(1), 107–118.

Harkany, T., Guzmán, M., Galve-Roperh, I., Berghuis, P., Devi, L. A., & Mackie, K. (2007). The emerging functions of endocannabinoid signaling during CNS development. *Trends in Pharmacologic Science, 28*(2), 83–92.

Hwang, J., Lyoo, I. K., Kim, S. J., Sung, Y. H., Bae, S., Cho, S. N., et al. (2006). Decreased cerebral blood flow of the right anterior cingulate cortex in long term and short term abstinent methamphetamine users. *Drug and Alcohol Dependence, 28,* 177–181.

Jager, G., Kahn, R. S., Van Den Brink, W., Van Ree, J. M., & Ramsey, N. F. (2006). Long-term effects of frequent cannabis use on working memory and attention: An fMRI study. *Psychopharmacology (Berlin), 185*(3), 358–368.

Jeng, W., Ramkissoon, A., Parman, T., & Wells, P. G. (2006). Prostaglandin H synthase catalysed bioactivation of amphetamine to free radical intermediates that cause CNS regional DNA oxidation and nerve terminal degeneration. *Federation of American Societies for Experimental Biology Journal, 20,* 638–650.

Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2005). *Monitoring the future national results on adolescent drug use 1975–2004: Vol 1. Secondary school students* (NIH Publication No. 05–5727). Bethesda, MD: National Institute on Drug Abuse.

Kanayama, G., Rogowska, J., Pope, H. G., Gruber, S. A., & Yurgelun-Todd, D. A. (2004). Spatial working memory in heavy cannabis users: A functional magnetic resonance imaging study. *Psychopharmacology (Berlin), 176*(3–4), 239–247.

Kang-Park, M. H., Wilson, W. A., Kuhn, C. M., Moore, S. D., & Swartzwelder, H. S. (2007). Differential sensitivity of GABA A receptor-mediated IPSCs to cannabinoids in hippocampal slices from adolescent and adult rats. *Journal of Neurophysiology, 98*(3), 1223–1230.

Karacostas, D. D., & Fisher, G. L. (1993). Chemical dependency in students with and without learning disabilities. *Journal of Learning Disabilities, 26*(7), 491–495.

Kolb, B., Gorny, G., Limebeer, C. L., & Parker, L. A. (2006). Chronic treatment with Delta 9 tetrahydrocannabinol alters the structure of neurons in the nucleus accumbens shell and medial prefrontal cortex of rats. *Synapse, 60*(6), 429–436.

Landa, L., Sulcova, A., & Slais, K. (2006). Involvement of cannabinoid CB1 and CB2 receptor activity in the development of behavioral sensitization to methamphetamine effects in rats. *Neuro Endocrinology Letters, 27*(1–2), 63–69.

Li, X., Wang, H., Qiu, P., & Luo, H. (2008). Proteomic profiling of proteins associated with methamphetamine-induced neurotoxicity in different regions of rat brain. *Neurochemistry International, 52,* 256–264.

London, E. D., Berman, S. M., Voytek, B., Simon, S. L., Mandelkern, M. A., Monterosso, J., et al. (2005). Cerebral metabolic dysfunction and impaired vigilance in recently abstinent methamphetamine abusers. *Biological Psychiatry, 58*(10), 770–778.

López-Moreno, J. A., Scherma, M., Rodríguez de Fonseca, F., González-Cuevas, G., Fratta, W., & Navarro, M. (2008). Changed accumbal responsiveness to alcohol in rats pre-treated with nicotine or the cannabinoid receptor agonist WIN 55,212–2. *Neuroscience Letters, 433*(1), 1–5.

Lozano, J., García-Algar, O., Marchei, E., Vall, O., Monleon, T., Giovannandrea, R. D., et al. (2007). Prevalence of gestational exposure to cannabis in a Mediterranean city by meconium analysis. *Acta Paediatrica, 96*(12), 1734–1737.

Lynskey, M. T., Heath, A. C., Bucholz, K. K., Slutske, W. S., Madden, P. A., Nelson, E. C., et al. (2003). Escalation of drug use in early onset cannabis users vs co-twin controls. *Journal of the American Medical Association, 289,* 427–433.

McHale, S., & Hunt, N. (2008). Executive function deficits in short-term abstinent cannabis users. *Human Psychopharmacology, 23*(5), 409–415.

McLaren, J., Swift, W., Dillon, P., & Allsop, S. (2008). Cannabis potency and contamination: A review of the literature. *Addiction, 103*(7), 1100–1109.

Medina, K. L., Hanson, K. L., Schweinsburg, A. D., Cohen-Zion, M., Nagel, B. J., & Tapert, S. F. (2007). Neuropsychological functioning in adolescent marijuana users: Subtle deficits detectable after a month of abstinence. *Journal of the International Neuropsychological Society, 13*(5), 807–820.

Medina, K. L., Nagel, B. J., Park, A., McQueeny, T., & Tapert, S. F. (2007). Depressive symptoms in adolescents: Associations with white matter volume and marijuana use. *Journal of Child Psychology and Psychiatry, 48*(6), 592–600.

Messinis, L., Kyprianidou, A., Malefaki, S., & Papathanasopoulos, P. (2006). Neuropsychological deficits in long-term frequent cannabis users. *Neurology, 66*(5), 737–739.

Monterosso, J. R., Aron, A. R., Cordova, X., Xu, J., & London, E. D. (2005). Deficits in response inhibition associated with chronic methamphetamine abuse. *Drug and Alcohol Dependence, 79*(2), 273–277.

Nestor, L., Roberts, G., Garavan, H., & Hester, R. (2008). Deficits in learning and memory: Parahippocampal hyperactivity and frontocortical hypoactivity in cannabis users. *Neuroimage, 40*(3), 1328–1339.

Nordahl, T. E., Salo, R., Natsuaki, Y., Galloway, G. P., Waters, C., Moore, C. D., et al. (2005). Methamphetamine users in sustained abstinence: A proton magnetic resonance spectroscopy study. *Archives of General Psychiatry, 62*(4), 444–452.

Padula, C. B., Schweinsburg, A. D., & Tapert, S. F. (2007). Spatial working memory performance and fMRI activation interaction in abstinent adolescent marijuana users. *Psychology of Addictive Behaviors, 21*(4), 478–487.

Paulus, M. P., Hozack, N. E., Zauscher, B. E., Frank, L., Brown, G. G., Braff, D. L., et al. (2002). Behavioral and functional neuroimaging evidence for prefrontal dysfunction in methamphetamine dependent subjects. *Neuropsychopharmacology, 26*, 53–63.

Paulus, M. P., Tapert, S., & Schuckit, M. A. (2005) Neural activation patterns of methamphetamine dependent subjects during decision making predict relapse. *Archives of General Psychiatry, 62*, 761–768.

Pillay, S. S., Rogowska, J., Kanayama, G., Gruber, S., Simpson, N., Pope, H.G., Yurgelun-Todd, D. A. (2008). Cannabis and motor function: fMRI changes following 28 days of discontinuation. *Experimental and Clinical Psychopharmacology*, 16, 22–32.

Rubino, T., Vigano, D., Realini, N., Guidali, C., Braida, D., Capurro, V., et al. (2008). Chronic delta(9)-tetrahydrocannabinol during adolescence provokes sex-dependent changes in the emotional profile in adult rats: Behavioral and biochemical correlates. *Neuropsychopharmacology, 33*(11), 2760–2771.

Schweinsburg, A. D., Nagel, B. J., Schweinsburg, B. C., Park, A., Theilmann, R. J., & Tapert, S. F. (2008). Abstinent adolescent marijuana users show altered fMRI response during spatial working memory. *Psychiatry Research, 163*(1), 40–51.

Scott, J. C., Woods, S. P., Matt, G. E., Meyer, R. A., Heaton, R. K., et al. (2007). Neurocognitive effects of methamphetamine: A critical review and meta-analysis. *Neuropsychological Review, 17*(3), 275–297.

Shaw, P., Kabani, N. J., Lerch, J. P., Eckstrand, K., Lenroot, R., Gogtay, N., et al. (2008). Neurodevelopmental trajectories of the human cerebral cortex. *Journal of Neuroscience, 28*(14), 3586–3594.

Solinas, M., Yasar, S., & Goldberg, S. R. (2007). Endocannabinoid system involvement in brain reward processes related to drug abuse. *Pharmacologic Research, 56*(5), 393–405.

Tapert, S. F., Schweinsburg, A. D., Drummond, S. A., Martin, P., Paulus, M. P., Brown, S. A., et al. (2007). Functional MRI of inhibitory processing in abstinent adolescent marijuana users. *Psychopharmacology, 194*(2), 173–183.

Thompson, P. M., Hayashi, K. M., Simon, S. L., Geaga, J. A., Hong, M. S., Sui, Y., et al. (2004). Structural abnormalities in the brains of human subjects who use methamphetamine. *Journal of Neuroscience, 24*, 6028–6036.

Trezza, V., Campolongo, P., Cassano, T., Macheda, T., Dipasquale, P., Carrata, M. R., et al. (2008). Effects of perinatal exposure to delta-9-tetrahydrocannabinol on the emotional reactivity of the offspring: A longitudinal behavioral study in Wistar rats. *Psychopharmacology (Berlin), 198*(4), 529–537.

Wittmann, M., Leland, D. S., Churan, J., & Paulus, M. P. (2007). Impaired time perception and motor timing in stimulant-dependent subjects. *Drug and Alcohol Dependence, 90*(2–3), 183–192.

Wood, E., Stoltz, J. A., Zhang, R., Strathdee, S. A., Montaner, J. S., & Kerr, T. (2008). Circumstances of first crystal methamphetamine use and initiation of injection drug use among high-risk youth. *Drug and Alcohol Review, 27*(3), 270–276.

Chapter **9**

What Is Methamphetamine and How and Why Is It Used?[1]

Herbert C. Covey, PhD

WHAT IS METHAMPHETAMINE?

Methamphetamine, also known as *speed*, *crystal*, *ice*, *crank*, or *chalk*, is a powerfully addictive stimulant that chemically affects the central nervous system by producing intoxication through the increased stimulation of the dopamine and norepinephrine receptors in the brain. Dopamine is associated with pain suppression, appetite control, and the brain's self-reward center. Norepinephrine activates the body's fight-or-flight response in emergencies. Meth acts on the brain reward pathway by increasing the release of the neurotransmitters noradrenaline, dopamine, and serotonin and reducing the reuptake of dopamine. These neurotransmitters carry messages from one nerve to another and are critical to the individual's sense of pleasure. Meth provides the user, at least initially, with a tremendous sense of pleasure.

Meth is a synthetic stimulant commonly used as a recreational drug. Physicians legally prescribe the drug as a treatment for attention deficit disorder (ADD) under the brand name Desoxyn, for both children and adults. The illegal form of the drug is made easily in clandestine labs with over-the-counter ingredients. For addicts, it is relatively inexpensive to purchase and has desired effects that last for hours. Some users find it appealing because it causes decreased appetite (resulting in weight loss), heightens energy levels, enhances attention, enables people to be physically (sexually) active for long periods, and provides a general sense of well-being and euphoria similar to that of cocaine. The desired effects of meth use can last between six and eight hours, which are then followed by a coming-down period when the user becomes agitated and

potentially violent. It is particularly addictive for females because of the "benefit" of its corresponding weight loss.

Meth is a Schedule II stimulant in the United States, meaning that it is illegal to buy, sell, or possess without a prescription. Outside of the United States, it is legally controlled in most countries and is only available by prescription. The federal government classifies methamphetamine as a controlled substance. The 1970 Controlled Substances Act placed strict limitations on the importing, manufacture, and retail availability of amphetamine-related drugs. The 2000 Methamphetamine Anti-Proliferation Act applied further limits on the sale of precursor ingredients used in other products.

Criminal sentencing for crystal meth is determined by U.S. sentencing guidelines, which are based on the statutory sentences, including quantity-based mandatory minimum sentences, in the Controlled Substances Act. For meth, the statute and guidelines both set forth alternative formulations for determining quantity-based sentences, because "actual" or "pure" meth is distinguished from "a mixture or substance containing" meth. Under federal law, meth trafficking carries a minimum of five years in prison and fines of $2 million for individuals or $4 million for more than one individual for first offenses involving 50 grams or less. Life imprisonment is the maximum penalty for trafficking with two or more prior offenses. U.S. sentencing guidelines should be consulted for specific penalties for trafficking, purity, quantity, forms, and other legal considerations. Unprescribed meth is illegal in every state.

HISTORY OF METHAMPHETAMINE AND ITS USE

Closely related to meth is the drug amphetamine. Amphetamine was first synthesized in 1887 in Germany by a scientist named L. Edeleano, who named it phenylisopropylamine. Initially, amphetamine had no known medical application. During the 1920s, researchers investigated it as a treatment for depression, as a decongestant, and for other medical purposes. By the 1930s, retailers marketed amphetamine as Benzedrine, which was an over-the-counter inhaler used to treat nasal congestion. By the late 1930s, physicians prescribed amphetamine for narcolepsy, attention-deficit/hyperactivity disorder, and depression. During WWII, military organizations used amphetamines (and methamphetamine) to keep soldiers ready and available for duty. As use of amphetamines spread, so did their abuse. To some, amphetamines became a cure-all for such things as weight control and treating mild depression.

In 1919, a Japanese chemist named A. Ogata produced the first meth. In contrast to amphetamine, meth is more powerful and easier to manufacture. Meth's progenitor is ephedrine, which is naturally found in Mahuang, a Chinese

plant whose stimulant properties have been documented for over 5,100 years (Holthouse & Rubin, 1997a, 1997b). During WWII, the Japanese military used meth to improve military performance. It was also sold over the counter in Japan to increase work performance and endurance during the war (Anglin, Burke, Perrochet, Stamper, & Dawud-Noursi, 2000). Following the war, its use, including intravenously, became epidemic in Japan, as supplies were readily available (Wermuth, 2000). It has been suggested that Adolf Hitler may have been a heavy user.

Following the war, Dexedrine (dextroamphetamine) and Methedrine became readily available in the United States. College students, truck drivers, motorcycle gangs, and athletes used the drug to stay awake and to improve concentration and performance.

In the mid-1960s, people were using meth in San Francisco and parts of the West Coast. In 1967, the first meth lab bust occurred in Santa Cruz, California (Holthouse & Rubin, 1997b). By 1970, use of the drug declined following the 1970 Controlled Substances Act, which restricted the production of injectable meth (Wermuth, 2000). However, meth made inroads in the gay community by the late 1970s (Bonné, 2004) and spread in popularity in California during the late 1980s (Leinwand, 2003). Hawaii, California, and Arizona were some of the earliest and hardest impacted states. California was hit particularly hard because of the smuggling of ephedrine, a critical ingredient in the production of meth, across the Mexican border. With tighter federal controls over ephedrine, pseudoephedrine became a replacement ingredient. In 1996, Congress passed the Methamphetamine Control Act of 1996. This Act doubled the maximum penalties for possession of the drug and increased the penalty for the possession of equipment used to manufacture meth from 4 to 10 years. The Act cracked down on large purchases of the ingredients, such as red phosphorous and iodine, and increased civil penalties for companies that sell precursor chemicals to people that manufacture meth. It did not, however, stop the flow of ephedrine into the United States.

By the 1990s, some young adults found methamphetamine to be a popular alternative to cocaine and heroin. White motorcycle gangs controlled production and distribution of meth before the 1990s (Gibson, Leamon, & Flynn, 2002). Small home labs and Mexican-based criminal organizations eventually took over production and distribution of meth. Mexican-based criminal organizations established "superlabs" in California and Mexico that were capable of producing large amounts of highly pure meth. In congressional testimony to the Senate Judiciary Committee, authority Donnie R. Marshall reported that about 85 percent of all methamphetamine used in the United States in 2000 was produced by these superlabs (Marshall, 2000). During the 1990s and up

to the present day, another shift occurred as cooks started to produce meth in small, home-based clandestine labs.

The meth on the streets today is often more powerful than that available in earlier years. Today, meth cooks have refined recipes to the point that some batches have as much as six times the potency of meth cooked in the 1960s (Mills, 1999). This meth is not always sold on the street, but rather cooks circulate (give or sell) it among friends and acquaintances.

Today, meth is a Schedule II drug that is available only through a highly restricted prescription procedure. Physicians have used meth to treat overeating disorders, depression, Parkinson's disease, obesity, attention deficit disorder, and narcolepsy.

STREET AND SLANG TERMS FOR METHAMPHETAMINE

Users and others have developed a set of terms for meth and its related behaviors similar to those used with other street drugs. These terms can be divided into street terms for the drug itself, the behaviors associated with its use, and the types of users. The terms in Table 9.1 are an extensive compilation of words used to refer to meth (Mills, 1999; Office of National Drug Control Policy, 2003), with the most commonly used terms boldfaced.

STREET AND SLANG TERMS FOR METHAMPHETAMINE ADDICTS

Just as there are different terms for meth, there are terms for its addicts. Many street terms have evolved to describe or refer to meth addicts: Basehead, Battery Bender, Cluckers, Chicken-Headed Clucks, Crack Heads, Crackies, Crankster, Cranker, Doorknobbers, Fienda, Fiends, Fiendz, Gacked, Geek(ers), Geekin, Geeter, Go Go Loser, Jibby, Jibby Bear, Jibbhead, Krista, Loker or Lokers, Neck Creature, Shadow People (due to an aversion of users to light), Sketchpad or Schetchers, Skitzers, Sketch Cookie, Sketch Monster, Speed Freak, Spin Doctor, Spinsters, Tweakers, Tweekin/the Go, or Wiggers.

STREET AND SLANG TERMS FOR BEHAVIORS/FEELINGS

Meth addicts have developed a number of terms they use to describe their state of mind when using meth: Ampin, Amped, Awake, Bache Knock 2 Rock, Bachin, "Bob" (as in discombobulated), Buzzed, Cranked Up, Crank

Table 9.1
Slang Terms for Methamphetamine

Albino Poo, Alffy, All Weekend Long, All Tweakend Long, **Amp**, Anny, Anything Going On, Bache Knock, Bache Rock, Bag Chasers, Baggers, Barney Dope, Batak (Philippine), Bato (Philippine), Batu Kilat (Philippine), Batu or Batunas (Hawaii), Batuwhore, Beegokes, Bianca, Bikerdope, Bikers Coffee, Billy (Great Britain) Bitch, Biznack, Blanco, Blizzard, Blue Acid, Blue Belly, Blue Funk, Blue Meth, Bomb, Booger, Boorit-Cebuano (Filipino), Boo-Yah, Brian Ed, Buff Stick, Buggar Sugar, Buggs, Bumps, Buzzard Dust, Caca, Candy, Cankinstien, CC, Chach, Cha Cha Cha, Chalk, Chalk Dust, Chank, Cheebah, Cheese, Chicken Feed, Chicken Flippin, Chikin or Chicken, Chicken Feed, Chingadera, Chittle, Chizel, Chiznad, Choad, Cinnamon, Clavo, Coco, Coffee, Cookies, Crack Whore, **Crank**, Cri Cri (Mexican border), Criddler, Cringe, Crink, Critty, Cristy, Crizzy, Croch Dope, CR (California), Crow, Crunk, Crypto, **Crystal**, **Crystal Meth**, Crystalight, Cube, Debbie, Tina, and Crissy, Desocsins, Devil Dust, Devil's Dandruff, Devil's Drug, Dingles, Dirt, Dirty, Dizzy D, D-monic or D, Do Da, Doody, Doo-My-Lau, Dope, Drano, Dummy Dust, Dyno, Epimethrine, Epod, Ersar Dust, Ethyl-M, Evil Yellow, Fatch, Fedrin, Fil-Layed, Fizz Whizz, Gackle-a Fackle-a, Gak, Gas, Geep, Gemni, Glass, Go Speed, Glass, Go Fast, Go-ey, Go-Go, Go-Go Juice, Gonzales, Goop, Got Anything, Granulated Orange, Grit, Gumption, Gyp, Hawaiian Salt, Hank, Hanyak, High Speed Chicken Feed, Highten, Hillbilly Crack, Hippy Crack, Homework, Hoo, Horse Mumpy, Hot Ice, Hydro, Hypes, **Ice**, Ice Cream, Icee, Ish, Izice, Jab, Jasmine, Jenny Crank Program, Jet Fuel, Jib, Jib Nugget, Jinga, Juddha, Juice, Junk, Kaksonjac, Kibble, Killer, KooLAID, Kryptonite, L.A., L.A. Glass, Lamer, Laundry Detergent, Lemon Drop, Life, Lily, Linda, Lost Weekend, Love, Low, Lucille, M Man, Magic, **Meth**, Meth Monsters, Methaine, Methandfriend, Methandfriendsofmine, Methanfelony, Methatrim, methmood, Method, Motivation (Colorado), Nazi Dope, Ned, Newday, No Doze, Nose Candy, On a Good One, OZs, Patsie, Peaking, Peanut Butter, Peel Dope, Pepsi (means meth), Pepsi One (Crystal meth), Phazers, Phets, Philopon (East Asia), Pieta, Pink, Poison, Poop, Poo'd Out, Poor Man's Cocaine, Pootanany, Powder, Power Monkeys, Powder Point, Project Propellant, Puddle, Pump, Q'd , Quartz, Quick (Canada), Quill, Racket jaw, Rails, Rank, Redneck Heroin, Richie Rich, Rip, Rock, Rocket Fuel, Rocky Mountain High, Rosebud, Rudy's Rumdumb, Running Pizo, Sack, Sam's Sniff, Sarahs, Satan Dust, Scante, Scap, Schlep Rock, Scooby Snax, Scud, Scwadge, Shab, Shabu, Sha-bang, Shabs, Shabu, **Shards**, Shit, Shiznack, Shiznac, Sciznac, Shiznastica, Shiznit, Shiznitty, Shizzo, Shnizzie Snort, Agua, Shwack, Skeech, Sketch, Ski, Skitz, Sky Rocks, Sliggers, Smiley Smile, Smurf Dope, Smzl, Snaps, Sniff, Snow (Colorado), space Food, Spaceman, Spagack, Sparacked, Sparked, Sparkle, Speed, Speed Racer, Spin Spin Spin, Spinack, Spindarella, Spinney Boo, Spinning Spishak Spook, Spoosh, Sprack, Sprizzlefracked, Sprung (Mississippi), Spun Ducky Woo, Squawk, Stallar, Sto-pid, Stove Top, Styels, Sugar, Super Ice, Sweetness, Swerve, Syabu (Asia), Talkie, Tasmanian Devil, Tenna, Tenner, The New Prozac, The White House, Tick Tick, Tical, Tina, Tish, Tobats, Toots, Torqued, Trash, Trippin Trip, Tubbytoast, Tutu (Hawaii), Twack, Twacked Out, Tweak, Tweedle Doo, Tweek, Tweezwasabi, Twiz, Twizacked, 222 (Chicago Area) Ugly Dust, Vanilla Pheromones, Wake, Way, Wash, WE WE We, Whacked, White Bitch, White Cross (after pill form), White Crunk, White Ink, White Junk, White Lady, White Pony, White, Who-Ha, Wigg, **Working Man's Cocaine**, Xaing, Yaba, Yama, Yammer Bammer, Yank, Yankee, Yay, Yead Out, Yellow Barn, Yellow Powder, Zingin, Zip, Zoiks, and Zoom.

It should be noted that some of these terms are used interchangeably with amphetamine, such as "speed."

Whore Jamie, Feelin Shitty, Foiled, Fried, Gakked, Gassing, Gear or Gear-up, Geeked, Geekin, Gurped, Heated, High, Jacked, Lit, Peaking, Pissed, Pumped, Psychosis, Ring Dang Doo, Riped, Rollin or Rollin Hard, Scattered, Schlep Heads, Sketching, Spin-Jo, Speeding, Sparked, Spracked, Spun, Spun Monkey or Spun Turkey, Stoked, Talkie, Trippin, Twacked, Tweaking, Tweeked, Twisted, Wide Open, Wired, Worked, Woop Chicken, Zipper, and Zoomin. When the user is experiencing the initial rush from using meth, he is "amping." Amping refers to the "amplified" euphoria users feel during the rush.

MISCELLANEOUS METH-RELATED TERMS

The meth drug trade has its own set of terms, including those used by meth addicts and distributors for the sales and distribution of the drug. An *eightball* refers to one-eighth of an ounce of meth. A *teenager, Tina*, or *Teena* refers to one-sixteenth of an ounce, and a *paper* is a term for a quarter gram of meth.

Paraphernalia is a general term for medical supplies or equipment used to make or use meth. Addicts refer to needles as *points, rigs*, or *slammers*. They sometimes call a straw or device used to snort meth a *tooter*. Those involved in making it need to shop or otherwise acquire the precursors (materials) needed to make the drug in a manner and in quantities that do not arouse suspicion. This acquisition of supplies is called *smurfing*.

The meth subculture has developed other terms related to use. For example, *crank, craters, crank bugs*, and *spider bites* refer to sores on the face and body resulting from prolonged meth use. *Meth mouth* refers to the terrible dental conditions addicts have from long-term use.

HOW IS METH USED?

Users can inject, smoke, orally ingest, anally insert, or snort meth. The method the user selects influences how the drug is experienced. Meth is a bitter-tasting powder that easily dissolves in beverages. The powder form of the drug is often snorted, which produces a less intense but much longer-lasting high. In 1993, 42 percent of meth and amphetamine treatment admissions reported they used the drug in this manner, according to the Substance Abuse and Mental Health Services Administration (SAMHSA, 2006). By 2003, only 15 percent of the treatment admissions reported they snorted or inhaled the drug. Recent Treatment Episode Data Set (TEDS) data found that in 2003, 56 percent of primary meth and amphetamine admissions reported smoking the drug, which was up from the 15 percent reported in 1993 (SAMHSA, 2006). In 1993, 29 percent indicated that they injected the drug, which compares to 22 percent in

2003. Smoking or injecting the drug produces a short but more intense and pleasurable "rush." In 1993, oral ingestion represented 13 percent and "other" accounted for 1 percent of the routes of administration. By 2003, oral administration declined to 6 percent and other routes of administration remained unchanged at 1 percent (SAMHSA, 2006).

Powdered meth is a hydrochloride salt form that quickly absorbs water from the air. This form of meth is smokable, as is *crystal meth* or *ice*, which refers to meth grown into crystals. Although some people believe that crystal meth is a freebase form of meth, this is not true. Meth that is grown into crystals is simply easier to smoke. Meth in crystal form, rather than powder, also is more likely to be relatively pure because of the difficulty of growing crystals from impure chemicals. According to SAMHSA (2004), in 1992, 12 percent of meth and amphetamine treatment admissions reported they smoked meth. By 2002, 50 percent of the treatment admissions reported they smoked meth in its crystal form. This represents a major shift from inhaling to smoking over this 10-year span.

Another relatively new way of consuming meth is called "booty bumping." This process involves the user heating meth into liquid form and mixing it with water. The user then draws the fluid into a syringe that lacks a needle. The syringe is then inserted in the anus and meth shot into the user's body. Users rely on this technique because the drug is readily absorbed into the bloodstream.

The crystal form of meth is referred to as *crystal, ice*, or *glass*. If heat is introduced, the user can smoke or inject crystal meth. Smoking it is a much faster and intense way to get high than swallowing the drug. The user places a small amount of crystal in a glass pipe (often called a *tooter*), heats it, and inhales the resulting vapors. Crystal meth or ice melts into a liquid when heated and returns to its crystal form when cooled. Boiling crystal turns it into a semiliquid referred to as *snot*, which can be smoked or placed up the nose. Users view smoking meth as ideal because it can be used almost anywhere, since the vapors are odorless and undetectable.

The rush or high felt by the user is the direct result of the release of dopamine into the section of the brain that controls feelings of pleasure. The rush or high associated with its use is relatively long-lasting compared to other drugs. The effects of meth use can last as long as 12 hours. If it is snorted, the user usually experiences effects within about 5 minutes. If it is orally ingested, the user will feel a rush in around 20 minutes. Oral meth use tends to lack rushing, has less euphoric effects, and tends to cause far less of a feeling of wanting to do it again than the other methods.

The fastest rushes occur when the user smokes or injects meth. The user usually experiences an immediate and powerful response. Smoking and inject-

ing are associated with stronger, faster, and more euphoric effects. While injecting results in a faster reward, it also results in a faster crash. Users learn to manage the rapid crashes by trying to attain another rush by taking more of the drug. These effects are more associated with compulsive or addictive user patterns. The general use trend is more toward smoking meth because of this immediacy and the strength of the initial rush. It should be noted that many addicts have an aversion to using needles and never inject the drug.

Standard-shaped light bulbs are easily converted into meth pipes. Meth pipes lack a screen found with pipes used to smoke cocaine or marijuana. The user places the meth inside the bowl of the glass pipe and then heats it until it turns into a liquid and then emits a gas (vapor). Once it turns to a gas, the user inhales it through the stem of the pipe. Over time, a white milky residue builds up on the sides of the bowl. Because the glass bowl gets very hot, users will sometimes have burn marks on their fingertips from holding the hot bowls or closing off the hole at the top of the pipe to keep the gas from escaping (see Figure 9.1).

After being heated, unused crystal meth returns to its crystal state when cooled by ice or a cool wet rag. Users will cool down unused liquid meth to its crystal form because it is easier to transport and use at another time.

Regardless of the method of use, meth addicts will frequently use it with other drugs such as cocaine, marijuana, and alcohol. Because of their poly-drug use, it is sometimes difficult to sort out the effects of the meth from other drugs. Users rely on other drugs or alcohol to either enhance or supplement the meth high or cushion their withdrawal and depression when coming down.

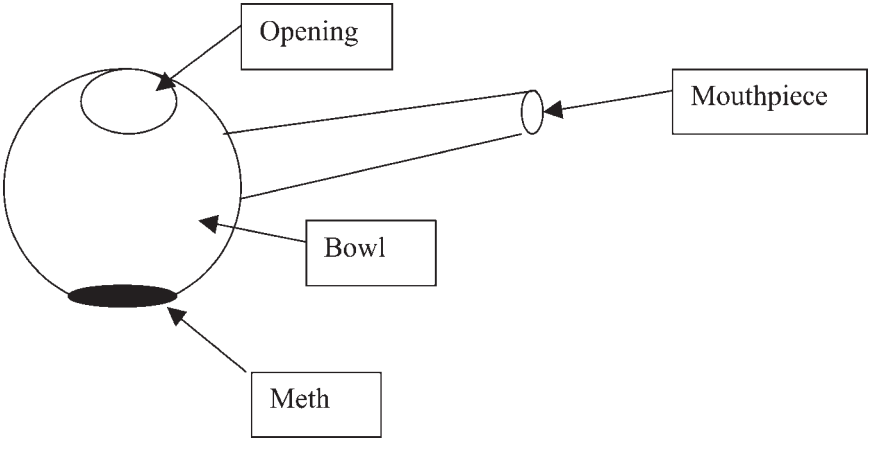

FIGURE 9.1 Typical design of pipe used to smoke methamphetamine.

WHAT DOES METH LOOK LIKE?

Meth is usually found on the street as powder or crystal. Street meth, like many other street drugs, is often diluted or cut with other substances. Powdered meth is cut with a filler or is crushed up with amphetamine or methylphenidate (Ritalin) tablets. Sometimes street meth is not really the drug at all but some other compound. Street meth that is clear and crystal is referred to as *glass* or *crystal*. Meth comes in many colors, including white, yellow, brown, orange, pink, red, or darker colors; it can also be almost transparent. The method of manufacture and chemicals used affects the color meth assumes, but most meth is white. Meth can take such forms as powder, granulated crystals (crystal meth or ice), tablets (pills sometimes called Yaba), or capsules. Crystal meth is made by adding a small molecule group (hydrochloric acid). The Yaba form is a tablet that is often comprised of meth and caffeine and is produced in Asia and Southeast Asia. The tablets are small (they can generally fit through a straw) and most often are reddish orange or green. Yaba tablets sometimes have corporate logos, such as Toyota, MTV, or Calvin Klein, that are popular in the rave scene. Examples of crystal meth can be found in the following photo.

Methamphetamine in crystal ("ice") form. Photo courtesy of the Drug Enforcement Administration.

METH DOSES

When meth is taken in a pill or tablet form, between 0.05 and 0.1 of a gram represents a dose. When it is smoked, the amount needed is smaller—as little as 0.01 of a gram may be all that is needed. The dosage amounts differ based on the purity of the drug, tolerance of the user, frequency of use, and individual reactions that are based on body physiology, metabolism, and method of use. The Erowid (2006) Web site provides some general guidelines for dosage amounts for an infrequent user of pure meth. Caution should be taken with any dosage amount because of the varying individual reactions to the drug. Thus, Table 9.2 lists what some are indicating are dosage estimates and should not be considered a guide for anyone.

The important things to remember about the doses are that a number of factors influence the amount needed to get high—the doses can be very small, and with prolonged use, more of the drug is needed to get the desired results. Erowid (2006) identified vomiting, headaches, dizziness, cold sweats, shaking, and, ultimately, death as possible results from overdosing on meth.

Table 9.2
Approximate Doses by Method of Abuse

	Oral Dosages	Insufflated (Snorted)	Smoked	Injected (IV)
Threshold	5 mg	5 mg	5–10 mg	5 mg
Light Stimulation	5–15 mg	5–15 mg	10–20 mg	5–10 mg
Common	10–30 mg	10–40 mg	10–40 mg	10–40 mg
Strong (some rushing)	20–50 mg	30–60 mg	30–60 mg	30–60 mg
Very Strong (rushing)		501 mg	501 mg	50–100 mg (strong rushing; intense euphoria)
Onset	20–70 minutes (depending on form and stomach contents)	5–10 minutes	0–2 minutes	0–2 minutes
Duration	3–5 hours	2–4 hours	1–3 hours	1–3 hours
Coming Down	2–6 hours	2–6 hours	2–4 hours	2–4 hours
Normal Aftereffects	up to 24 hours	up to 24 hours	up to 24 hours	up to 24 hours

METH USE AND DRUG TESTS

Meth is detectable using standard drug tests of urine from three to five days following use. It is detectable in hair tests for approximately 90 days and in blood samples for one to three days. A number of substances can result in false positives, including ephedrine, pseudoephedrine, and other substances found in such over-the-counter medications as Sudafed, Allerest, Contact, Nyquil, Robitussin, and others. Diet aids, nasal sprays, asthma medications, and several prescription medications may suggest meth use when it has not been used.

WHY DO PEOPLE USE METH?

People use the drug for a number of reasons (Morgan & Beck, 1995). The short-term effects of meth use are desired: the sense of euphoria and pleasure; a high that lasts 8 to 12 hours or more; energy enhancement and alertness; weight loss because of decreased appetite; decreased fatigue; relief from chronic depression; a sense of social bonding with other users; and improved sexual pleasure and drive. Rawson (2005) found that more than 35 percent of the women who used the drug said they did so to lose weight, compared to 10 percent of meth-using men. Rawson also found that about 35 percent of the women used it to relieve depression, compared to about 25 percent of the men. Meth users have reported on what it feels like to use the drug. One indicated, "It made me feel confident, self-assured," then added, "Then it took on a whole new meaning to me. I became a partier" (Bonné, 2004). Another said she felt "this very intense surge of energy through the body." She added, "I felt like I was superhuman because I would think more, I could accomplish more."

Meth users also experience negative short-term effects that are not desired, including increased respiration, higher pulse rate (irregular heartbeat or cardiac arrhythmia), higher blood pressure, increased body temperature (hyperthermia), convulsions, irritability, hyperexcitability, grinding of teeth, nervousness, dilated pupils resulting in an aversion to light, and death, according to the National Institute on Drug Abuse (2002), Anglin et al. (2000), Anglin, Kalechstein, Maglione, Annon, and Fiorentine (1997), and Leshner (2000).

LONG-TERM UNDESIRED EFFECTS OF METH USE

Several agencies, including the Drug Enforcement Administration (DEA) and other authorities (Greenwell & Brecht, 2003; Leshner, 2000; London et al., 2004; National Institutes of Health, 2001, 2004; Volkow et al., 2001a,

2001b) have identified long-term effects of meth use that include severe psychological and physical dependence (addiction); violent behavior that eventually gets coupled with paranoia, making the users even more dangerous; chronic fatigue; talkativeness; overall lifestyle disruption; sleep problems such as insomnia (inability to sleep); cognitive impairments and reduced functioning; confusion; fight-or-flight responses to stimuli; visual and auditory hallucinations; uninhibited sexual functioning with prolonged use; severe depression; picking at the skin and scratching imaginary bugs, which causes open sores and infections. Morals and values are abandoned. The inability to think and act sequentially is impaired and, with heavy, prolonged use, disappears. For example, meth users find it difficult to follow directions or listen to instructions. Heavy use may also lead to homicidal or suicidal thoughts.

Physically, long-term use may result in seizures, chest pain, dry mouth, cardiac valve thickening, death, dramatic weight loss because users lose interest in food and eventually suffer from malnutrition, and brain damage (methamphetamine is neurotoxic). Prolonged use can lead to what is called "amphetamine psychosis," resulting in paranoia, auditory and visual hallucinations, self-absorption, irritability, and aggressive and erratic behavior. Amphetamine psychosis is a disorder similar to paranoid schizophrenia. Individuals with amphetamine psychosis may exhibit bizarre behavior that is sometimes violent.

PATTERNS OF METH USE—LOW INTENSITY, BINGE, AND HIGH INTENSITY

There are three basic patterns of meth use: low intensity, binge, and high intensity. Low intensity is a pattern of use where the user is not psychologically addicted to meth but relies on it for specific perceived benefits, such as a work enhancer or diet-aid drug. Typically, low-intensity users snort or swallow the drug.

Low-Intensity Users

According to Narconon (2002), low-intensity use is characterized by the snorting of powdered meth or ingestion of pills. Users at this level use meth to keep themselves awake and alert for special tasks, or to lose weight. Users at this level are able to hold down jobs, attend school, and otherwise appear to act normal and operate normal lives. Some over-the-road truckers, overtime workers, night-shift workers, stay-at-home parents needing to get several tasks done, and students use it for these purposes. Low-intensity users are unlikely

to come into contact with law enforcement, social service, health, or child welfare caseworkers because of problems resulting from this pattern of use.

Professionals may encounter or be working with low-intensity users and not be aware of their use of meth. It is important to note that chronic low-intensity users often view it as a "functional drug." That is, they see it as helping them get things done, such as lose weight, focus on tasks, or get work done. They believe that they "can stop anytime" or "have it under control." Over the long run, this functionality turns out to be a myth.

Narconon (2002) notes that, while use of meth in this fashion seems to be managed, low-intensity users are a short step away from becoming binge users. Low-intensity users have experienced the rush associated with heavier use and generally only need to smoke or inject to cross over the line into binge-use patterns. When they do so, the problems with their use escalate.

Binge Users

Binge users smoke or inject to experience the euphoria that comes from a more intense use of meth. During a binge, the user will periodically use the drug to maintain the high. It is these strong euphoric experiences that move the user from a low-intensity to a psychologically addicted user. A common and unhealthy use pattern for meth is to re-dose repeatedly for several days in a row. Depending on whether the intention is to stay awake, remain high, or attempt to continue to get "rushing" effects, doses are repeated every 3 to 8 hours to stay awake, or every 30 minutes to 4 hours to remain "high." A user re-dosing often takes the same amount of meth as the first dose. As re-dosing continues beyond 48 hours, the user's dosages tend to increase.

HIGH-INTENSITY USERS

High-intensity users have a goal of never crashing or coming down but maintaining a state of euphoria and the perfect rush. Given the nature of meth and its effects on the body, this becomes an impossible goal. For the chronic user, following the first injection or smoke, each successive rush or high becomes more difficult to obtain, and more of the drug is needed. The user remembers the initial high but can never reach that point again no matter how much of the drug is used.

High-intensity users may start out with a pattern of needing from $20 to $40 a day to support their addiction. This increases several-fold as more and more of the drug is needed to maintain the high they are seeking. The high cost

of heavy use drives many to criminal behavior to support their addiction. For some, crime is the only alternative.

Phases of High-Intensity Use

High-intensity users experience meth use as a series of phases. Depending on how often they use, their history of use, method of use, and dosage amounts, the length and nature of each phase differ but follow a general pattern. In order of occurrence, the general phases follow.

The Rush

The rush is the initial sense of intense euphoria that the user experiences immediately after smoking or injecting meth. Low-intensity users do not experience a rush when snorting or swallowing the drug. During the rush, the user experiences an intense feeling of pleasure and a burning sensation. The initial rush can last between 5 and 30 minutes, which is longer than the rush associated with the stimulant cocaine. Some users, especially injectors, report an initial burning sensation as the meth is introduced into the body. During the rush, the user's metabolic rate increases, blood pressure elevates, and pulse soars. Users compare the experience of the rush as being the equivalent to having multiple sexual orgasms.

The reason for the rush is that meth triggers the adrenal gland to release a hormone called epinephrine (adrenaline), which puts the body in a battle mode—fight or flight. In addition, the physical sensation the rush provides results from the explosive release of dopamine in the pleasure center of the user's brain. Dopamine is released in the brain's pleasure center. Long-term users find that much of their life is devoted to maintaining a constant rush.

The High

The rush is followed by a high that can last between 4 and 16 hours. This is a longer period than that experienced by cocaine users. Some circles refer to being high as the "shoulder." While being high on meth, the user has a sense of being smarter, more focused, argumentative, and aggressive. High users frequently interrupt those around them.

The Binge

When the user continues to use while high, this is known as binging. Binging is an effort by the user to maintain a prolonged high by taking more meth. During the binge the user becomes hyperactive. Because of severe depression

and other negative effects that begin when the drug starts to wear off, users try to avoid crashing or coming down. They may binge to stay high and awake for many days at a time. The law of diminishing returns operates for the user, as the desired effects of meth diminish with each dose. This may cause the user to consume even more to reach the initial rush or stay high. Eventually, there is no rush or high resulting from further consumption, and the user stops.

Tweaking

When the binge ends, a stage of the cycle known as tweaking occurs. The user has a sense of emptiness and dysphoria. Taking more meth will not alleviate the negative feelings experienced by the tweaking user. This stage is not comfortable for the user and some turn to other drugs to self-medicate their feelings. For some, alcohol or other illegal substances are used for self-medications. Tweaking is assumed by many to be the most dangerous stage to professionals, such as law enforcement officers and social workers. Tweakers have not slept for days and as a result are irritable. Think about how moody you get when you don't have enough sleep and then multiply this feeling several times over. Tweakers are unpredictable and short-tempered. Tweakers sometimes are frustrated because they can't find enough of a dose to experience that initial high, and this frustration translates into a sense of unease and aggression. When coupled with the paranoia that results from long-term use, they are loose cannons on the deck able to go off at any time.

Tweakers appear to have rapid and brisk movements. They are overstimulated. Their eyes will rapidly dart around, speech will be rapid, eyes will be clear, and speech concise. A tweaker's eyes may roll back into the head. Tweakers are obsessive about things. For example, they will clean the same thing over and over but ignore other things that need cleaning. The kitchen may be spotless but the house or yard may be full of unfinished projects and filth. In addition, tweakers can become obsessed with dismantling things, such as appliances, with no idea of why they are doing it. They might dismantle a television, washing machine, or any number of objects without a clue about how to put things back together.

Because tweakers hear and see things differently, it can be difficult for law enforcement or caseworkers to predict what, if anything, will set them off. Tweakers have such an altered sense of reality that virtually anybody or anything could set them off into a rage or confrontation. Because many become paranoid, a caseworker, law enforcement officer, family member, or other person could become an unsuspecting target. Because some users fear authorities, they have many weapons around the house. This requires that caseworkers and other professionals be cautious around tweaking individuals.

There is considerable folklore on how long a user can tweak. Reports of 15 to almost 40 days without sleep have been reported to the author. While it is true that addicts do stay awake for very long periods of time, what is likely is they actually take short naps and float in and out of consciousness. They never fall into a deep and replenishing sleep until later in the cycle. While meth is not a hallucinogen or a psychedelic, it is easy to understand how prolonged sleep deprivation would result in bizarre behaviors and thoughts. Sleep deprivation can result in profoundly disturbing hallucinations. For example, many addicts will describe the "shadow people" that seem to appear and be very real to them (Holthouse & Rubin, 1997a).

The Crash

Eventually, because of a lack of sleep and loss of epinephrine, the body becomes exhausted and falls into a deep sleep. This is known as the crash. When high-intensity users stop taking meth they experience depression, anxiety, fatigue, paranoia, aggression, and an intense craving for the drug (London et al., 2004; National Institute on Drug Abuse, 2002). Tweakers sleep like they are dead when the time comes to crash, and they sleep for days afterwards. The crash can last for days, as the body replenishes its supply of epinephrine. Users may try to cushion the crash by using tranquilizers or downer drugs, such as marijuana, heroin, or alcohol (Gibson et al., 2002). Users that are crashing usually pose no real threat to caseworkers, law enforcement officers, or other professionals. However, crashing parents or guardians cannot provide basic care to children and often are neglectful of them and may place them at risk. Protecting, feeding, and overseeing children are not possible for the crashing user, and sometimes their children go without food, sleep, care, and supervision. Some children learn to survive on their own when parents or guardians are crashing. They eat and drink whatever they can find. Crashes may last between one and three days.

Normalcy

Meth addicts eventually return to a state of normalcy for a few days. This stage can last between 2 and 14 days, depending on frequency of use. The high-intensity user never really gets back to complete normalcy because of the physiological damage done to the body, specifically the brain. This is not to say the high-intensity user cannot recover and lead a normal life.

Withdrawal

Users who withdraw from meth experience symptoms of physical distress. Withdrawal from the drug is a prolonged process, and users in withdrawal

experience depression and are initially unable to experience pleasure. They also may experience fatigue, paranoia, and aggression, and have psychotic symptoms that may persist for months or years following use (Office of National Drug Control Policy, 2005). They become lethargic and have no energy. Users, because of poor eating habits, also may experience extreme hunger. If the cravings for meth are strong, some may become suicidal. If more meth is used, their sense of pleasure increases and their depression will temporarily be alleviated. Some suggest that this is a major reason why meth addicts are some of the most difficult to treat and why recidivism rates are high.

Experts agree that meth is a highly addictive stimulant drug (National Institutes of Health, 2001). According to the U.S. Drug Enforcement Administration (2005), "Methamphetamine has a phenomenal rate of addiction, with some experts saying users can get hooked after just one use." Anecdotal accounts and clinical experience suggest that addiction can occur in less than a year. The Center for Substance Abuse Treatment (1999) Tip 33: Treatment for Stimulant Use Disorders notes in chapter 2 that addiction typically occurs after using the drug for two to five years. Prolonged or binge use of meth causes significant tolerance and psychological dependence. Some report addiction after the first use, but this is not typical for most.

THE PHYSICAL APPEARANCE OF METH USERS

It would be inaccurate to stereotype high-intensity meth users as looking a certain way. There is no set appearance for all meth users and addicts. They can assume a variety of appearances depending on a number of factors, including the amount and frequency they are using. Low-intensity meth users can look reasonably normal in appearance. However, if the individual is using meth on a regular basis, obvious physical indications develop over time. It should be noted that as dramatic as the outside appearance may be, there is also physiological damage occurring on the inside of the body. Three excellent Web sites that have examples of the progression of deterioration from meth use are: 2stopmeth.org; www.co.multnomah.or.us/sheriff/faces_of_meth.htm; and www.mappsd.org. The reader can view some examples of the physical deterioration resulting from prolonged meth use on these sites.

Crank Bugs and Meth Mites

Open sores on the skin are the result of the individual scratching imaginary "crank bugs" or "meth mites." Long-term meth users develop the sensation that insects are crawling on their skin, causing them to scratch themselves. The scratching associated with these imaginary insects (known as formication)

eventually leads to lesions in the skin, topical infections, sore areas, and scabs. These open sores are aggravated by the addict continuing to scratch and thus spreading the infection. The user's lack of proper hygiene contributes to the spread of infection. Those who inject frequently develop abscesses, ulcerations, and scars around the injection sites. These injection sites may become infected and also a target of scratching.

"Meth Mouth"

The long-term or heavy use of meth also leads to severe dental problems, which professionals and addicts refer to as "meth mouth." The American Dental Association (ADA, 2005) concluded that "the oral effects of methamphetamine can be devastating." Long-term drug addicts, especially those using meth, do not take good care of their teeth and do not visit their dentists on a regular basis. Meth addicts do not brush their teeth often, have poor diets, and avoid medical and dental professionals to hide their addiction. Meth acts on the gums and teeth as a corrosive. It softens the teeth, and they basically melt away. When a user smokes meth, the chemicals used to make it, such as sulfuric or muriatic acid, are heated and vaporize and spread around the mouth. The user's mouth is irritated and burned by the chemicals, and sores eventually develop that become infected. This infection spreads throughout the mouth and gums. When coupled with the corrosive action of chemicals on tooth enamel, eventually the teeth rot away to well below the gum line. The gums are also affected because meth use causes blood flow to decrease and thus gums to break down and become diseased. For these and other reasons, it is common for the roots to show and the loss of teeth to occur.

The decay occurring can also be attributed to the effect of meth use on saliva production, or what is medically referred to as "dry mouth" (ADA, 2005). Meth dries out the salivary glands and thus the production of saliva. Normally, the body uses saliva to clean the teeth and neutralize acids, and to control harmful bacteria. With less saliva, the user's mouth cannot perform these functions. Consequently, the acids in anything consumed are free to decay the teeth and gums. The sense of a "dry mouth" or "cottonmouth" (xerostomic) causes some users to drink lots of sugary sodas, which also add to tooth decay (ADA, 2005). Damage to the mouth is not limited to those who smoke the drug; it can also be caused by snorting meth, as caustic chemicals flow through the nasal passage to the back of the mouth.

Another fundamental process that destroys the user's teeth is the grinding that occurs during use. Meth use causes the user to feel anxious or nervous, and the unintentional teeth grinding that consequently occurs leads to cracks

and breaks. Meth users often grind their teeth down hard, causing teeth to break and nerve endings to become exposed. The user may also lose fillings because the teeth grinding causes them to fall out. Just breathing through the mouth and having air pass over the teeth and gums can cause some users to feel pain. Whether from smoking or snorting, the user will eventually develop meth mouth, and many users simply use more meth to alleviate the pain.

The best way to visualize and understand meth mouth is to view some of the images of the mouths of long-term users. The color insert section contains examples of meth mouth and the corresponding tooth and gum decay resulting from prolonged meth use. The consequences of meth mouth go beyond pain and disfiguration. Some long-term recovering users with damaged teeth may be reluctant or unable to find dental help. If they do find it, they may lack the money for dental repairs. With poor teeth, they find it difficult to obtain work because potential employers are turned off by unsightly teeth (mouths), and users face social rejection.

THE COMPARATIVE LOW COST OF METH

In addition to the desired effects of meth on the user, one of the reasons for the drug's popularity is its relative low cost. It is relatively inexpensive to produce, but more important, lasts longer than alternative stimulants, such as cocaine. Prices for the drug vary across the United States:

- It costs $20 to $60 for a quarter gram, which is slightly lower than cocaine but lasts significantly longer (Bonné, 2001).
- The National Council of State Governments estimated a $100 batch of methamphetamine would sell on the street for about $1,000 (Kraman, 2004).
- Meth prices range between $5 and $15 a dose (Leinwand, 2003).
- The Drug Enforcement Administration in 2001 provided a price range from $3,500 to $23,000 per pound, $350 to $2,700 per ounce, and $20 to $300 per gram (Drug Policy Information Clearinghouse, 2003).
- The price of crystal meth runs higher because of its purity and additional manufacturing steps. The National Drug Intelligence Center (2005), using Drug Enforcement Administration data, reported the prices for powdered and crystal meth shown in Table 9.3.

Although prices seem high to the nonuser, they represent a good value to the addict. The long-term high of the drug is a good value compared to cocaine or other substances. Likewise, meth quickly becomes expensive as addicts must use increasing amounts of the drug and do so more frequently over time in pursuit of that lasting high and avoidance of the crash. The consequence is that

Table 9.3
National Price Ranges of Methamphetamine, 2003

Quantity	Powder	Crystal
Pound	$1,600–$45,000	$6,000–$70,000
Ounce	$270–$5,000	$500–$3,100
Gram	$20–$300	$60–$700

Source: National Drug Intelligence Center 2005.

the price of meth becomes very high for children as their needs compete with the meth for family resources.

HOW DOES METH COMPARE TO COCAINE?

Because both are powerful psycho stimulants, meth is often compared with cocaine (National Drug Intelligence Center, 2002). Users who have used both drugs report similar experiences, such as a sense of euphoria. Users of both drugs report experiencing an initial rush, and a longer high and sense of euphoria with meth. If the cocaine is in crack form, the rush and high are much shorter. Users can smoke, inject, snort, or swallow either illicit drug. Both drugs may produce anxiety, increased blood pressure, increased temperature, higher pulse rates, and possible death. Short-term effects of both include increased activity, decreased appetite, and respiration. Prolonged use of either drug can lead to psychotic behaviors, hallucinations, mood disturbances, and/or violence. When users of either drug withdraw, they report craving, paranoia, and depression (London et al., 2004).

Differences between the two drugs exist. Cocaine is derived from the refined leaves of the South American coca plant; consequently, almost all cocaine in the United States is imported. The meth found in the United States is also imported from Mexico, Southeast Asia, and other countries. However, unlike cocaine, it can be domestically manufactured in large or small operations. Large spaces, while often desirable, are not required for production. Meth can be produced in small rooms or spaces. The production of meth is relatively easy compared to importing cocaine. All of the necessary chemicals to produce meth are relatively available, thus making law enforcement control of the illicit drug difficult.

Cocaine and meth abusers have different use patterns. For example, meth users report that they use the drug on a more regular basis than that reported by cocaine users. Meth's effects require less-frequent administration than cocaine, because meth leaves the system more slowly and thus has a longer half-life

than cocaine. Meth has a half-life of between 10 and 12 hours, compared with only about one hour for cocaine (Wermuth, 2000). While cocaine is quickly and almost completely metabolized in the body, meth has a longer duration, and a larger percent of the drug remains unchanged in the body (Center for Substance Abuse Prevention/National Prevention Network, 2006; National Institute on Drug Abuse, 2002). Thus, the brain is affected for more prolonged spans of time. Cocaine is not neurotoxic to dopamine and serotonin neurons, but meth is neurotoxic. "Meth has more long-term, serious effects on the brain than cocaine" (National Institute on Drug Abuse, 2002).

Another difference is cost. Meth is cheaper on the street than cocaine. Meth has a longer duration for the initial rush and high. Crack cocaine offers a high of about 15–20 minutes and meth a high of 8–24 hours. The perceived cost-benefit ratio to the user is much greater for the meth addict. Rawson, Anglin, and Ling (2002, p. 7) wrote, "Methamphetamine effects are long lasting and methamphetamine users typically spend about 25 percent as much money for methamphetamine as that spent by cocaine users for cocaine."

According to research by Dr. Sara Simon sponsored by the National Institute on Drug Abuse (NIDA), abuse patterns differ between meth and cocaine abusers (Zickler, 2005). Meth abusers typically take the drug early in the morning and in two- to four-hour intervals, similar to being on a medication. In contrast, cocaine abusers typically take the drug in the evening and take it over a period of several hours that resembles a recreational-use pattern. They typically continue using until all of the cocaine is gone. In addition, another pattern showed that continuous use was more common among meth abusers than among those abusing cocaine. According to other NIDA-sponsored research by Dr. Simon, the effects of meth and cocaine abuse resulted in similar cognitive deficits, but meth abusers had more problems than cocaine abusers at tasks requiring attention and the ability to organize information (Zickler, 2005).

In the 1980s, cocaine use became epidemic, but in recent years, use has declined among the middle class. Crack cocaine remains a serious blight in some inner cities. Cocaine's use, similar to other drugs, is cyclic, with periodic increases and decreases (Rawson et al., 2002). In contrast, meth has the potential of enduring, similar to marijuana and alcohol.

Cocaine addicts typically experience profound life changes in a relatively short time frame because of higher costs of use and use patterns, which involve binging. Cocaine users typically hit bottom sooner than many meth users. Meth addicts experience the same losses and also hit bottom, but in many cases, do so over a longer period. Some meth addicts use at levels that allow them to maintain jobs, homes, some money, and at least, the appearance of being in control.

NOTE

1. From: *The Methamphetamine Crisis*, Herbert C. Covey; Chapter 1, pp. 3–22, "What Is Methamphetamine and How and Why Is It Used?," by Herbert C. Covey. Copyright © 2006 by Herbert C. Covey. Reproduced with permission of Greenwood Publishing Group, Inc., Westport, CT.

REFERENCES

American Dental Association. (2005, August 9). *Dental topics A to Z: Methamphetamine use*. Retrieved from www.ada.org/prof/resources/topics/methmouth.asp

Anglin, M. D., Burke, C., Perrochet, B., Stamper, E., & Dawud-Noursi, S. (2000). History of the methamphetamine problem. *Journal of Psychoactive Drugs, 32*, 137–141.

Anglin, M. D., Kalechstein, A., Maglione, M., Annon, J., & Fiorentine, R. (1997). *Epidemiology and treatment of methamphetamine abuse in California: A regional report*. Unpublished manuscript, Los Angeles: UCLA, Drug Abuse Research Center.

Bonné, J. (2001). Meth's deadly buzz. *Msnbc.com special report*. Retrieved from msnbc.msncom/id/3071772

Bonné, J. (2004). Hooked in the Haight: Life, death or prison. *Msnbc interactive*. Retrieved from msnbc.com/id/3071769

Center for Substance Abuse Prevention/National Prevention Network. (2006). *Methamphetamine: A resource kit*. Rockville, MD: Substance Abuse and Mental Health Services Administration.

Center for Substance Abuse Treatment. (1999). *Tip 33: Treatment for stimulant use disorders: Treatment improvement protocol* (TIP Series No. 33). Rockville, MD: Substance Abuse and Mental Health Services Administration.

Drug Policy Information Clearinghouse. (2003). *Methamphetamine: Fact sheet*. Rockville, MD: Office of National Drug Control Policy.

Erowid. (2006, March 31). *Meth*. Retrieved from www.erowid.org.

Gibson, D. R., Leamon, M. H., & Flynn, N. (2002). Epidemiology and public health consequences of methamphetamine use in California's central valley. *Journal of Psychoactive Drugs, 34*, 313–319.

Holthouse, D., & Rubin, P. (1997a, December 18). *Methodology—Part I: New Times*. Retrieved from www.phoenixnewtimes.com/issues/1997–12–18/feature2

Holthouse, D., & Rubin, P. (1997b, December 18). *Methodology—Part II: New Times*. Retrieved from www.phoenixnewtimes.com/issues/1997–12–18/feature4

Kraman, P. (2004). *Drug abuse in America—Rural meth*. Lexington, KY: Council of State Governments.

Leinwand, D. (2003). "Meth" moves east. *USA Today*, July 29.

Leshner, A. I. (2000). Addressing the medical consequences of drug abuse. *NIDA Notes, 15*, 3–4.

London, E. D, Simon, S. L., Berman, S. M., Mandelkern, M. A., Lichtman, A. M., Bramen, J., et al. (2004). Mood disturbances and regional cerebral metabolic abnormalities in recently abstinent methamphetamine abusers. *Archives of General Psychiatry, 61*, 73–84.

Marshall, D. R. (2000, July 6). *Congressional testimony to the Senate Judiciary Committee.*

Martin, K. R. (2002). Prenatal exposure to methamphetamine increases vulnerability to the drug's neurotoxic effects in adult male mice. *NIDA Notes, 17*, 1–3.

Mills, K. (1999, December 13). Meth of old has morphed into epidemic proportions. *Seattle Post-Intelligencer.*

Morgan, P., & Beck, J. (1995). The legacy of the paradox: Hidden contexts of methamphetamine use in the United States. In H. Klee (Ed.), *Amphetamine misuse: International perspectives on current trends.* Amsterdam: Harwood Academic Publishers.

Narconon. (2002). *Methamphetamine information: Abuse patterns.* Retrieved from www.narconon.org/druginfo/methamphetamine addiction

National Drug Intelligence Center. (2002, August). Crystal methamphetamine. *Information Bulletin.* (Product Number 2002-L0424–005). Johnstown, PA: U.S. Department of Justice.

National Drug Intelligence Center. (2005). *Methamphetamine drug threat assessment.* Johnstown, PA: U.S. Department of Justice.

National Institute on Drug Abuse. (2002). *Research report: Methamphetamine abuse and addiction.* Bethesda, MD: Author.

National Institutes of Health. (2001, March 1). Methamphetamine abuse leads to long-lasting changes in the human brain that are linked to impaired coordination and memory. *NIH News.* Washington, DC: U.S. Department of Health and Human Services. Retrieved from www.nih.gov/news/pr/mar2001/nida

National Institutes of Health. (2004, January 5). New study suggests methamphetamine withdrawal is associated with brain changes similar to those seen in depression and anxiety. *NIH News.* Washington, DC: U.S. Department of Health and Human Services.

Office of National Drug Control Policy. (2003). *Methamphetamine: Fact sheet.* Rockville, MD: Drug Policy Information Clearinghouse. Retrieved from www.whitehousedrugpolicy.gov/drugfact/methamphetamine

Office of National Drug Control Policy. (2005, November 30). *Fact sheet: Methamphetamine.* Retrieved from www.whitehousedrugpolicy.gov/publications/factsht/methamph/

Rawson, R. A., Anglin, M. D., & Ling, W. (2002). Will the methamphetamine problem go away? *Journal of Addictive Diseases, 21*, 5–19.

Substance Abuse and Mental Health Services Administration. (2004). *Drug abuse warning network, 2003: Interim national estimates of drug-related emergency department visits.* Rockville, MD: Author.

Substance Abuse and Mental Health Services Administration. (2006, March 15). Trends in methamphetamine/amphetamine admissions to treatment: 1993–2003. *The DASIS Report*.

U.S. Drug Enforcement Administration. (2005). *Factsheet: Fast facts about meth*. Washington, DC: Author. Retrieved from www.usdoj.gov/dea/pubs/pressrel/methfact03.html

Volkow, N. D., Chang, L., Wang, G.-J., Fowler, J. S., Franceschi, D., Sedler, M. J., et al. (2001a). Higher cortical and lower subcortical metabolism in detoxified methamphetamine abusers. *American Journal of Psychiatry, 158*(3), 383–389.

Volkow, N. D., Chang, L., Wang, G.-J., Fowler, J. S., Leonido-Yee, M., Franceschi, D., et al. (2001b). Association of dopamine transporter reduction with psychomotor impairment in methamphetamine abusers. *American Journal of Psychiatry, 158*(3), 377–382.

Wermuth, L. (2000). Methamphetamine use: Hazards and social influences. *Journal of Drug Education, 30*, 423–433.

Zickler, P. (2005). Methamphetamine, cocaine abusers have different patterns of drug use, suffer cognitive impairments. *NIDA Notes, 16*, 1–2.

Chapter **10**

The Short- and Long-Term Medical Effects of Methamphetamine on Children and Adults[1]

Kathryn M. Wells, MD

HISTORY OF MEDICAL USE

Methamphetamine is a drug that has been around and known to the medical community for many years. Chemically, it is a synthetic drug that belongs to the amphetamine class of drugs. Medically, its stimulant effects act on both the central and peripheral nervous systems. Throughout history it has had various medical uses, but it has increasingly been used illicitly.

Meth was first synthesized in 1887 by a German chemist. It was not used therapeutically until the 1930s when it began to be promoted by American pharmaceutical companies for various ailments and was thought to be without risk of addiction. At about the same time, Japan began to produce large quantities of meth in the pill form for domestic consumption. After the war, Japanese pharmaceutical companies launched a large campaign to increase the use of over-the-counter meth pills that were in abundance in former military warehouses. This consequently led to the first large-scale epidemic of meth use and abuse (Kato, 1990). In the United States, a prescription was still needed to legally obtain amphetamines, but by the 1950s, the nonmedical use of amphetamines had spread to the civilian population, most commonly being used by individuals who needed to stay awake for long periods of time or to perform well at monotonous tasks. Additionally, meth was being prescribed for the treatment of hyperactivity, obesity, narcolepsy (a disorder causing spontaneous sleep), and depression (Beebe & Walley, 1995).

The "second wave" of the meth epidemic in the United States occurred in the 1960s when intravenous use of the drug became more popular. These users were the first individuals to take the drug solely for its euphoric effects (Wolkoff, 1997). During this period, users had created a way to manufacture meth on the street called the P2P (phenyl-2-propanone) method. This wave was controlled by law enforcement and public efforts to educate potential abusers and treat users. This method of manufacturing meth used lead acetate as a chemical reagent and, because there were often large quantities of lead in the final product, placing the user at risk for lead poisoning, there was the risk of hepatitis, nephritis, and encephalopathy (Allcott, Barnhart & Mooney, 1987).

The "third wave" of meth use in the United States occurred in the 1980s as a result of the advent of another, faster, and easier method for meth manufacturing called the pseudoephedrine reduction method. This method, further discussed elsewhere in this text, contributed to the rapidly rising accessibility and popularity of the drug on the street in the 1990s and 2000s. It produced a more potent and psychoactive form of the drug with a higher percentage of the dextro-isomer of the drug compared to the P2P method, which produced equal proportions of the dextro- and levo-isomers (Burton, 1991; Center for Substance Abuse Treatment, 1997; Cho, 1990). This is important because dextro-methamphetamine is three to four times more potent to the central nervous system (CNS) than levo-methamphetamine (Sowder & Beschner, 1993). The extent of the potential consequences of the impurities of this manufactured form of meth is unclear (unintended by-products and reagent residuals as well as processing errors), but this is of great concern, as many of these laboratories are operated by uneducated "chemists" who get their recipes from unpublished sources or through the Internet and who are frequently using the drug while processing it.

Additionally, on the street a potent, smokable form of meth, known as *crystal*, *glass*, or *ice*, began to gain popularity, and its use grew rapidly because of the more potent and longer "high." For these reasons, many cocaine users began to be attracted to the use of meth (Wolkoff, 1997). By the 1990s, the use of prescription meth was almost completely discontinued due to the understanding of its potential for addiction. Meth is currently classified as a Schedule II stimulant, meaning that it is known to have a high potential for abuse and is available only by prescription. The only accepted medical indications for use of meth are for the treatment of narcolepsy and attention-deficit/hyperactivity disorder, and the dosages prescribed are much smaller than what is used by the abusers.

HOW IS METH USED?

Meth may be snorted, smoked, orally ingested, injected, or absorbed through any mucous membranes such as sublingually, rectally, or vaginally. It is readily absorbed from the gut, nasopharynx (back of the nose and throat), muscle (when injected), mucosa, and placenta. It alters the mood differently depending on the route of ingestion. When the drug is snorted or taken orally, the user describes a feeling of euphoria or a feeling of extreme well-being called a "high." Smoking or injecting the drug results additionally in a more immediate, brief, intense sensation called a "flash" or a "rush." Users describe this as extremely pleasurable, and it has been characterized by some individuals as being equivalent to multiple orgasms. This is then followed by the euphoria. The "high" and the "rush" are both a result of the release of very high levels of the neurotransmitter dopamine into areas of the brain that regulate feelings of pleasure. Further description of the effect of this drug on the brain will be discussed in a later section.

Different methods of use produce different response within the user's body. The length of time until onset of symptoms is dependent upon the method of use. If the drug is snorted, effects occur within 3–5 minutes due to the rapid uptake of the drug through highly vascular nasal passages. If, however, the drug is ingested orally, it must be taken up through the lining of the digestive system, with effects not occurring for 15–20 minutes. Smoking meth usually allows the drug to reach the brain even more rapidly than injecting it (MacKenzie & Heischober, 1997). This is felt to be related to the small particle size of the drug, allowing it to penetrate deep into the lung tissue, where it rapidly crosses into the pulmonary circulation. In fact, meth is available to the body and brain very rapidly after use as it, unlike cocaine, does not have to be converted to a "free base" in order to be smoked effectively (MacKenzie & Heischober, 1997). The stimulant effects of meth have been reported to last up to 24 hours, most commonly 8–12 hours, compared to cocaine's high of only 20–30 minutes (National Institute on Drug Abuse, 1998). Additionally, the route of administration plays a role in the potential for dangerous and unintended consequences or side effects. Intravenous use is frequently associated with additional illnesses related to the administration of the drug (i.e., sharing of needles) such as hepatitis, HIV infection, tuberculosis, pneumonia, cellulitis (tissue infection), bacterial or viral endocarditis (infection of the lining of the heart), wound abscesses, sepsis (blood infection), thrombosis (blood clot in the blood vessels), thrombophlebitis (infection of lining of the blood vessel), and kidney injury (Šlamberová, Charousová, & Pometlová, 2005; Sowder &

Beschner, 1993). Snorting the drug may be associated with sinusitis (infection of the sinuses), loss of the sense of smell, congestion, atrophy (thinning) of the nasal mucosa, nosebleeds, perforation or damage to the nasal septum, hoarseness, and difficulty with swallowing (Sowder & Beschner, 1993; Gold, 1997).

Meth is commonly used in a "binge and crash" pattern—this means that the user will continue to use the drug ("binge") until they completely "crash." Many users go on a "run," during which time they may forgo food and sleep while binging. This period of use can last for several days. After the heavy binge cycle and before the "crash," the user may experience extreme paranoia, hallucinations, aggression, and agitation. This period of use is called "tweaking" and is felt to be the most physiologically dangerous time for the user as he or she may have a tremendous amount of drug in their body. It is also the time period when the user is potentially the most dangerous, due at least in part to their propensity for violence and feelings of paranoia. The "crash" is believed to occur because the chemical messenger (neurotransmitter) dopamine is depleted from the nerve terminals. It will slowly reaccumulate (at least to some level) while the user is crashing, but during this time the user may sleep for days, not even awakening to take care of regular bodily needs such as eating. Unfortunately, this often leads to increased use of the drug following the crash and eventually to difficulty in feeling any pleasure at all as the nerve terminals become injured. This is further discussed below in the section on central nervous system effects.

Tolerance to meth occurs within minutes, and the pleasurable effects disappear even before the blood concentrations fall. This partially explains the lack of direct correlation between blood level and clinical effects seen with meth. Tolerance means that users often need to take repeatedly higher doses and dose more frequently to get the desired effect. In addition, users often change their method of intake to a method that provides the additional "rush" or "flash" but is also more addictive (injection or smoking). However, there is no tolerance for the negative effects on the user's judgment, impulsivity, aggression, and susceptibility to paranoia, delusions, and hallucinations. In fact, it frequently takes an increasingly smaller amount of the drug to produce these symptoms.

When users discontinue their meth use, they will experience at least some symptoms of withdrawal. The more problematic and prominent withdrawal is the psychological withdrawal, which consists of depression, anxiety and agitation, fatigue, paranoia, aggression, and an intense craving for the drug. Physical withdrawal may also occur and is characterized by excessive hunger (polyphagia) and excessive sleepiness (hypersomnolence). Seizures may occur when the user is withdrawing from this drug. Studies have shown that there are brain abnormalities similar to those seen in people with mood disorders such as anxiety and depression (London et al., 2004) in individuals who have

recently discontinued their meth use. This poses additional challenges for individuals in treatment for this addiction.

CLINICAL EFFECTS OF METH USE

There are many factors that may contribute to how a person's body responds to meth use and abuse. The clinical effects from the use of meth are related to the form of the drug used, the dose, the frequency of use, the route of administration, and the length and amount of use. Additionally, the user may have underlying mental health problems for which he or she is trying to self-medicate with the use of the meth. The user may also be using other drugs in conjunction with meth, which may compound and complicate the effects of the drug. Finally, the methods of manufacturing this drug vary greatly, as does the purity of the final product. Therefore, the chemicals used in the manufacturing process as well as unwanted by-products may remain in the final product. This makes it very difficult to differentiate the effects of the drug alone from the effects of the other chemicals and by-products present. These variables also make it very difficult to predict, with any great certainty, the effects of the drug on each individual user.

Short-Term Effects

Pharmacologically, meth is a strong stimulant and is therefore in a class of drugs that includes cocaine, caffeine, and amphetamines. It is structurally similar to amphetamine as well as some of the body's natural neurotransmitters (chemical messengers) such as dopamine, serotonin, epinephrine (adrenaline), and norepinephrine. Meth has a much greater effect on the central nervous system than other amphetamines. Additionally, it seems to exert fewer peripheral nervous system effects (Beebe & Walley, 1995).

Many users report that they began taking meth to try to increase their alertness and to stay awake for longer periods of time, while others begin taking the drug use to lose weight. Still other users begin using to increase their sexual appetite, using it socially to go dancing or "clubbing."

A large portion of the acute physiological symptoms displayed after the use of meth are related to its peripheral effects on the autonomic nervous system and include dilated pupils, dry mouth, suppressed appetite (and consequent weight loss), elevated blood pressure (hypertension), tachycardia (high heart rate), rapid respiratory rate (tachyon), bruxism (involuntary teeth grinding), insomnia (inability to sleep or decreased need for sleep), tremors, and blurry vision. Additionally, meth is a vasoconstrictor of peripheral blood vessels,

which causes decreased oxygen delivery to the extremities, resulting in poor circulation. This may contribute to the multiple skin lesions that users often have, which are further worsened by the frequent picking behavior that the user demonstrates while perseverating on the lesions. Users also often perseverate on other tasks such as taking electronic items apart but then are unable to focus adequately in order to reassemble them (Center for Substance Abuse Treatment, 1999).

The central nervous system effects of meth ingestion are the result of the structural similarity of meth and the neurotransmitters active in the brain (dopamine, serotonin, epinephrine or adrenaline, and norepinephrine). Meth use can initially create feelings of euphoria (well-being), elevated energy, increased sensory perception, improved attention, excitation, intensification of emotions, perception of elevated self-esteem, increased alertness, agitation, aggression, restlessness, irritability, repetitive stereotyped behaviors, and increased physical activity (Jaffe, 1995). Conversely, it can decrease physical appetite with subsequent often marked and rapid weight loss. Users may have pressured speech and flight of ideas with rapid shifts in thinking, poor concentration, exaggerated self-esteem, hypervigilance, enhanced sensory awareness, fearlessness, suspiciousness, impaired judgment, poor impulse control, aggression, and emotional liability (Center for Substance Abuse Treatment, 1999).

Users have described markedly increased feelings of sexual desire, but despite this increased libido (sex drive), they usually begin to have difficulty in sexual performance. It is believed that the result of meth-stimulated serotonin release in the brain gives an initial antidepressant effect and elevates feelings of empathy. However, it is also responsible for bizarre mood changes, psychotic behavior, aggressiveness, and bruxism (involuntary grinding of the teeth). Some users may also experience feelings of nausea and dizziness.

The central nervous system effects of acute ingestion may include psychotic behaviors such as hallucinations and paranoia, which can lead to bizarre, irrational, and even violent, behavior. These effects may persist for days or weeks after the drug was discontinued (Beebe & Walley, 1995). These individuals may therefore have a great potential for violence, are at risk for homicidal and suicidal behavior (Szuster, 1990), and can be very dangerous to approach in any setting. A condition called methamphetamine psychosis has been described in the literature (Murray, 1998). This illness consists of several features, including extreme paranoia, well-formed delusions, hypersensitivity to environmental stimuli including light and sound, stereotyped "tweaking" behavior, panic, extreme fearfulness, and a high potential for violence. In fact, there is a described "hyperviolence syndrome" where the victim is frequently a part of the perpetrator's delusional belief system. A weapon such as a knife or gun is commonly

used in committing a crime and frequently there are multiple wounds inflicted, sometimes even days after the victim's death (MacKenzie & Heischober, 1997). Additionally, agitated delirium has been described in cases of meth-psychotic states and has also been linked to sudden cardiac death in meth users.

Overdose of the drug may be lethal and can even occur in a first-time user who ingests a single large dose (Jaffe, 1990). Acute symptoms of toxic ingestion may include dizziness, tremor, irritability, confusion, hostility, hallucinations, panic, headache, skin flushing, chest pain, palpitations, increased core body temperature (hyperthermia), irregular heart rhythms (arrhythmias), vomiting, cramps, excessive sweating, and severe high blood pressure (hypertension). This can result in brain hemorrhage or stroke, heart attack (myocardial infarction), and acute pulmonary edema (abnormal accumulation of fluid in the lungs) (Furst, Fallon, Reznik, & Shah, 1990; Nestor, Tamamoto, Kam, & Schultz, 1989a, 1989b). Additionally, the hyperthermia may be exacerbated by increased muscular activity due to agitation and can result in massive muscle breakdown (rhabdomyolysis) and potentially, kidney failure (Beebe & Walley, 1995). The development of a very high fever, rapid heart rate, severe hypertension, convulsions, toxic delirium, and cardiovascular collapse may signal a life-threatening situation (Ellinwood, Sudilovsky, & Nelson, 1973; Rowbotham, 1993; Wetli, 1993).

Medical treatment for overdose of meth consists primarily of supportive care. Other potential causes of presentation must be excluded. There are no specific medications or antidotes for the treatment of meth intoxication.

Sedation and rapid cooling may be used to manage the hyperthermia and agitated movements (Ellinwood et al., 1973; Gold, 1997). Ventilation and oxygenation may need to be provided and medications may need to be used to manage hypertension and seizures. Evaluation for cardiac arrhythmias and injury may need to be undertaken as well.

Long-Term Effects

The long-term effects of meth use can be particularly challenging when dealing with a chronic meth abuser. The use and abuse of this drug commonly leads to progressive social and occupational deterioration. Many who work with meth addicts report that the changes caused by the drug lead to a complete rearrangement of the user's priorities. For this reason, meth causes the heavy user to withdraw from anything and everything that is important to them. The reality is that studies now show that these effects are related to brain changes, many of which may be permanent. Individuals who have used meth for long periods of time demonstrate many features of dependence. It is clear that meth

is highly addictive and may lead to a chronic, relapsing disease. Addiction to meth is characterized by compulsive drug-seeking behavior, which is the result of functional and molecular changes in the brain. There is a stronger potential for addiction when utilizing the more rapid-acting routes of administration such as injection or smoking the drug, since there is stronger positive reinforcement for the use with the extremely pleasurable feelings that immediately follow (National Institute on Drug Abuse, 1998).

Chronic users often exhibit concerning behavioral changes, which consist of paranoia, auditory and visual hallucinations, mood disturbances, and delusions. This may result in homicidal or suicidal thoughts. Chronic users may also demonstrate excessive anxiety, confusion, insomnia, weight loss, and extremely violent behavior. It is important to be aware of this when dealing with someone addicted to the drug as they may exhibit very dangerous, unprovoked rages. Additionally, long-term users may display unusual motor movements that appear very similar to a Parkinsonian tremor. When the drug is discontinued, the user may experience depression, anxiety, fatigue, paranoia, aggression, and an intense craving for the drug. Studies have shown that the behavioral changes may persist for months or years after use of the drug is discontinued (National Institute on Drug Abuse, 1998). Chronic meth abuse may lead to the "kindling" phenomenon or "reverse tolerance" where the user can be pushed into frank psychosis by even very small amounts of any stimulant (methamphetamine, amphetamine, caffeine, or nicotine). This is felt to involve alterations that occur in the brain (Jaffe, 1990). Recent studies have suggested that meth psychosis may also spontaneously be brought on by mild stressors (Yui, Goto, Ikemoto, Ishiguro, & Kamata, 2000). Finally, there is another condition called chronic psychosis or "withdrawal" or "abstinence" psychosis, but it is unclear if this may be related to latent schizophrenia that was uncovered by the meth use (Streltzer & Leigh, 1977; Tomiyama, 1990).

Another feature of long-term meth use and abuse relates to the heightened sexuality linked to the use of this drug. After meth use for any length of time, users frequently describe changes in their sexual behaviors. They report that frequently activities that would previously give them sexual gratification no longer do, which leads many users to turn to increasingly bizarre sexual behaviors to meet their sexual needs. This can lead to predatory sexual behavior, increased promiscuity, and the extensive use of pornography. Frequent, often unprotected, sexual activity results in many unplanned pregnancies as well as the transmission of sexually transmitted and blood-borne infections, including HIV/AIDS and multiple forms of viral hepatitis (particularly hepatitis B and C). These risks are increased further when the users are injecting the drug and sharing the injection equipment. In fact, the use of methamphetamine is

so closely linked to sexual behavior that some studies have shown that sexual photos presented to meth users can trigger desire to use, even after long-term abstinence.

Dental decay has become a hallmark of chronic meth use and abuse. This complication has been termed by many as "meth mouth" and is likely multifactorial. First, meth use markedly reduces the production of saliva, causing a very dry mouth (xerostomia). Since saliva normally serves to bathe the teeth and reduce decay-causing bacteria, its reduction may lead to dental decay. Additionally, users often have a high intake of sugary soft drinks, which, coupled with a lack of oral hygiene as well as poor nutrition, may contribute to dental decay (Shaner, 2002). Finally, the significant vasoconstriction caused by meth use may also decrease blood flow to the teeth through the dental pulp, causing further damage. Another hallmark of chronic meth abuse is skin lesions. This is discussed in detail below in the section on the dermatologic system.

MEDICAL COMPLICATIONS OF METH USE

Meth use can affect any major organ system in the body. It has the most profound effect on the brain and central nervous system, but can also affect other organs.

Central Nervous System

The greatest systemic medical concern about meth use is the serious effect that the drug has on the brain of the user. The effects of meth on the central nervous system are numerous. First, this drug affects the brain at the very cellular level, causing nerve damage and loss.

In order to understand the complex effect that meth has on brain cells, it is important to understand the way that brain cells normally work. Nerve cells pass messages from one cell to another through the assistance of chemicals called neurotransmitters. These chemicals are necessary, because the nerve cells do not actually touch and therefore must pass their messages by the release and uptake of a neurotransmitter. These chemical messengers, such as dopamine, epinephrine, norepinephrine, and serotonin, are stored in structures called vesicles that float around in a fluid called cytoplasm within the presynaptic (sending) nerve cell. They remain there until the cell is given the signal to release the chemical. At that time the vesicle moves to the edge of the presynaptic cell, binds to the cell wall, and releases the neurotransmitter into the synapse (the space between the two nerve cells). The message is then picked up by the postsynaptic (receiving) cell when the neurotransmitter attaches to a receptor

on the cell wall. If there is more neurotransmitter in the synapse than is needed, it will either be destroyed or taken back up into the presynaptic cell for storage until it is needed again. This is done through a mechanism in the cell wall of the presynaptic cell called a transporter.

Dopamine is the neurotransmitter most affected by meth, because the two chemicals are very similar in structure. Dopamine is normally released when something pleasurable occurs, since it acts in the regions of the brain that regulate feelings of pleasure. It also elicits effects in the areas of the brain that regulate movement, emotion, judgment, and motivation. Once meth enters the body, regardless of the manner, it makes its way to the brain cells, among other places, where it causes the cells to release dopamine into the nerve synapse. This is what creates the stimulant effects of the drug that the user desires. Early studies showed that meth decreased the transporter function in dopamine neurons (Brown, Hanson, & Fleckenstein, 2000). However, newer studies have shown that the meth actually enters the presynaptic cell and causes massive release of dopamine from the storage vesicles into the cell cytoplasm as well as an additional massive release of dopamine from the presynaptic cell. This causes flooding of the synapse with the dopamine. There are only a limited number of receptors on the postsynaptic cell, and once they are full of the dopamine, they can no longer take up more of the chemical. Therefore, there is an excess of dopamine left in the synapse. Researchers believe that the excess dopamine is broken down by chemicals in the synapse and is ultimately turned into breakdown products that are toxic to the nerve cells. Additionally, the excess dopamine released into the presynaptic cell's cytoplasm is believed to be damaging to the cell.

Studies using a noninvasive brain imaging technique called magnetic resonance spectroscopy (MRS) have shown that the damage done to nerve cells is long-term and is similar to that caused by strokes or Alzheimer's disease (Ernst, Chang, Leonido-Yee, & Speck, 2000). Another study by Dr. Volkow used positron emission tomography (PET) scans to show that dopamine transporter levels in the striatum of the brain were 24 percent lower in meth users than in control subjects. Additionally, the meth users performed more poorly than nonusers on tests that evaluated brain function associated with the striatum, including fine motor skills, gross motor skills, and memory. The reduction in performance was proportional to the deficits in dopamine transporters. She concluded that compared with the normal 6–7 percent reduction in dopamine transporters found in aging, users saw losses roughly equivalent to 40 years of aging (Volkow et al., 2001).

Damage caused by meth to the dopamine system has been compared to that seen in patients with Parkinson's disease, a brain disease characterized by

the progressive loss of dopamine neurons in the regions of the brain involved in movement. Although damage to the brains of Parkinson's patients is more severe than that in meth users, researchers now believe that long-term meth use may lead to symptoms very similar to Parkinson's disease (Volkow et al., 2001). It is unknown if sustained abstinence from meth use will result in recovery of brain changes. Early studies suggested that abstinence was accompanied by dopamine transporter recovery, but a parallel recovery in cognitive function has been more difficult to identify. It is clear that recovery is related to the individual's baseline prior to use, the length of use, and the length of the abstinence (Volkow et al., 2001). A recent study using proton magnetic resonance spectroscopy suggested that following cessation of meth use, adaptive changes occur in the brain, which was felt to potentially contribute to some improvement in function (Nordahl et al., 2005). However, another recent publication suggests that meth use causes persistent hypometabolism in the frontal white matter of the brain with impairment in frontal executive function (Kim et al., 2005). Many studies are ongoing in this important area.

The release of serotonin is also stimulated by meth ingestion. This neurotransmitter has been implicated in states of consciousness, mood, depression, and anxiety. It is believed that meth has a little bit less serotonin effect than dopamine effect. However, damage to cells responsive to this chemical may explain the problems with depression that recovering addicts face.

As the nerve terminals are injured, the user eventually is unable to feel the pleasure that he began to use the drug for. This can contribute to the cycle of addiction and many of the chronic effects that are seen in long-term users.

Meth use can cause seizure activity, strokes, and spontaneous brain bleeds. It can also lead to chronic psychosis as well as movement disorders.

Cardiovascular System

Because meth is a stimulant, it has multiple effects on the cardiovascular system. These effects appear to be manifest at all dose levels and routes of administration even in otherwise healthy young adults. Virtually any kind of heart disease has been linked to meth use and abuse. First, use of this drug causes an increase in heart rate (tachycardia) as well as elevation of blood pressure (hypertension), both of which can be marked and very dangerous. Additionally, meth use can cause sudden cardiac death as well as the sudden rupture of an aneurysm. Meth is a blood vessel constrictor (vasoconstrictor), and that abnormal constriction of blood vessels of the heart can cause heart damage or a heart attack. This may be worsened or exacerbated by the fact that meth use causes increased platelet aggregation, which may clog cardiac vessels. Primarily because

of the catecholamine (epinephrine and other neurotransmitted) excess, meth abuse may result in cardiotoxicity. This may be manifest as inflammation of the heart muscle (myocarditis) or the heart lining (endocarditis) and abnormalities in the heart muscle itself (cardiomyopathy) (Hong, Matsuyama, & Nur, 1991; Gold, 1997). Finally, chronic meth abuse can cause damage to any vessels throughout the body, further damaging the tissues those vessels supply.

Respiratory System

Respiratory system complications from meth abuse include shortness of breath (dyspnea) and severe chest pain. Coughing spasms following inhalation of the drug may result in pulmonary barotrauma and consequent leakiness of air into the pleural cavity, chest cavity (mediastinum), and soft (subcutaneous) tissues of the chest. Pulmonary edema (excess fluid in the lungs) has been noted in meth fatalities and is felt to be the result of deep inhalation of the drug and subsequent aggravation of preexisting conditions (Nestor et al., 1989a, 1989b). Granulomas may form as a result of chronic irritation from adulterants added to the drugs, and constriction of the blood vessels in the lungs may ultimately affect oxygen exchange, potentially leading to chronic lung disease (Center for Substance Abuse Treatment, 1997).

Dermatologic System

The skin of the chronic user is often in poor condition and may be covered in sores. Poor circulation to the skin, poor nutrition, and tactile hallucinations all contribute to this quickly identifiable problem. Users often report that they believe that there are bugs crawling on their skin (a phenomenon called "formication") and will perseverate on trying to pick at the bugs, often with instruments such as knives. These lesions frequently have difficulty healing well and become readily apparent in chronic users. Some parents will even believe that their children have bugs on their skin and cover the children in insect spray or pick at the child's skin. These lesions may be confused with another skin disorder if the drug use is not identified.

There may be evidence of healed burns if the user participated in manufacturing and was burned—often these individuals do not get medical care for the burns when they occur and attempt to treat the injury themselves. Recent data in the literature suggests that if such a burn patient seeks medical care, he needs to be managed differently from routine burn patients, requiring two to three times the usual amount of fluid resuscitation (Warner, Connolly, Gibran, Heimback, & Engrav, 2003).

Immunologic System

Drug abuse has been linked to increased risk of HIV and Hepatitis B, C, and D infection due to high-risk behaviors of users such as unprotected sexual activity and the sharing of injection paraphernalia. Preliminary animal studies have suggested that meth may also affect HIV disease progression by a more rapid and increased brain HIV viral load. Another study suggested that HIV-positive meth users may be at a greater risk of developing acquired immune deficiency syndrome (AIDS) than non–meth-using HIV-positive patients. Finally, additional studies have suggested that interactions between meth and the HIV virus itself may lead to greater neuronal damage and neuropsychological impairment (Volkow, 2005).

Other Organs

Other organs can be affected by meth use as well. Muscle damage can occur as a result of severely elevated body temperature (rhabdomyolysis). This can lead to major organ system damage, including the kidney, liver, and brain. Giant gastrointestinal ulcers can occur as a result of vasoconstriction decreasing blood supply to the intestines. There have also been reports of acute liver failure following intravenous meth use (Kamijo, Soma, Nishida, Namera, & Ohwada, 2002). One recent study showed significantly decreased calcification in the bone of chronic meth users, suggesting possible chronic effects on bone metabolism (Katsuragawa, 1999).

EFFECTS OF METH USE IN PREGNANCY

Meth use during pregnancy is believed to place the unborn fetus at risk, as does the use of other illicit drugs and alcohol. However, the full extent of maternal use of meth on the fetus and newborn infant is not completely known, and the fact that a mother uses a drug while pregnant does not, in and of itself, ensure that the unborn fetus will be affected. In fact, it is very difficult to differentiate the effects of meth exposure from a multitude of other factors such as maternal nutritional status and health, genetics, socioeconomic status, lack of prenatal care, and concomitant exposure to other drugs, including nicotine and alcohol. Additionally, the effect of any prenatal exposure on the fetus is also related to the gestational period during which the drug was used as well as the amount and form of use (Plessinger, 1998). Exposures in the first and second trimester of pregnancy are more likely to result in systemic abnormalities, while exposures later in pregnancy are linked with growth abnormalities.

There is currently a multicenter study underway to further describe this issue but, preliminarily, the investigators report that the effects appear to be similar to those of cocaine-exposed infants. However, although meth and cocaine are both sympathomimetic agents, it is known that meth has a much longer duration of action, potentially complicating its effects further.

There are several components of the potential risk to the fetus in a pregnancy complicated by maternal meth use. First, the effects of the use of the drug on the mother and father, including fertility, must be considered. Studies have shown that chronic, high-dose stimulant use affects reproductive and sexual functioning in both males and females. Male users report loss of sexual interest, impotence, and difficulty in maintaining an ejaculation, while female users may have abnormalities in their menstrual cycles leading to amenorrhea and infertility, as well as difficulty in achieving an orgasm (Gold, 1997). In the pregnant woman, meth may cause hypertension (high blood pressure), tachycardia, and vasoconstriction. Additionally, the mother's poor nutritional habits, high-risk behaviors, and commonly poor prenatal care may contribute to potential risk to the fetus.

Next, the risks to the placenta and the fetus must be considered. Based on its sympathomimetic function, meth is thought to have direct cardiovascular effects on both the fetus and the placenta, potentially causing fetal hypertension (high blood pressure), tachycardia (high heart rate), and vasoconstriction. These effects may result in premature delivery, intrauterine growth retardation, placental hemorrhage, fetal distress, or spontaneous abortion. Additionally, because the placenta provides the source of nutrition for the fetus, constriction of these blood vessels, as caused by meth, may result in reduced blood flow to the fetus and ultimately reduced oxygen and nutrient supply. These findings are consistent with the pharmacologic properties of meth, as it is known that elevated levels of norepinephrine can cause placental vasoconstriction and increased uterine contractility (Lederman, Lederman, Work, & McCann, 1978; Sherman & Gautieri, 1972). One sheep study released in 1993 showed that methamphetamine readily crosses the placenta and produces significant and long-lasting maternal and fetal cardiovascular effects (Stek et al., 1993). It is also known that meth passes through the placenta to the fetus and can cause elevated fetal blood pressure, potentially leading to prenatal strokes and heart or other major organ damage. The drug can cause an increased or extremely variable heart rate in the fetus and slowing or alteration of fetal growth. Additionally, simultaneous with an increase in maternal blood pressure following meth abuse, there is a decreased blood supply as well as oxygen supply to the placenta and fetus. This impaired oxygen supply can retard fetal development (Stewart & Meeker, 1997). Another study in sheep, released in

1994, showed that maternal administration of meth was found to be associated with a short-term increase in circulating fetal catecholamines, which was followed by hyperglycemia, lacticacidemia, and hyperinsulinism. This began to suggest an alteration of fetal sympathoadrenal activity, which may contribute to the perinatal complications seen with meth use (Dickinson, Andres, & Parisi, 1994). Additionally, it has been shown that the norepinephrine transporter and, to a lesser extent, the serotonin transporter are cellular targets in the human placenta for both amphetamine and meth (Ramamoorthy, Ramamoorthy, Leibach, & Ganapathy, 1995).

Another aspect of prenatal meth exposure that must be considered is any possible direct effect on the fetus. Fetal development abnormalities have been described sporadically in the medical literature, but no true syndrome specifically linked with maternal use of meth in the prenatal period has been described. There are limited numbers of studies in this area, particularly studies focusing on human infants. Additionally, most of the research in this area combines all amphetamines (often including cocaine), and only few studies isolate meth exposures. It is known that because of its low molecular weight and lipid solubility, there is considerable transfer of meth from maternal to fetal blood. This, in addition to the immaturity of fetal metabolic activities, may account for the reason that the drug remains in fetal circulation much longer than it does in maternal blood (Inaba & Cohen, 1993; Stek et al., 1993). Multiple studies looking at human exposures to amphetamines have indicated an association between meth or amphetamine use during pregnancy and cleft lip (Little, Snell, & Gilstrap, 1988; Nelson & Forfar, 1971; Saxen, 1975; Thomas, 1995), cardiac defects (Little et al., 1988; Nelson & Forfar, 1971; Nora, McNamara, & Clarke-Fraser, 1967; Nora, Vargo, Nora, Love, & McNamara, 1970), low birth weight (Little et al., 1988; Oro & Dixon, 1987), growth reduction and reduced head circumference (Eriksson, Larsson, & Zetterström, 1981; Little et al., 1988; Oro & Dixon, 1987), biliary atresia (Golbus, 1980; Levin, 1971), prematurity and stillbirth (Ericksson, Larsson, Windbladh, & Zetterström, 1978), hyperbilirubinemia requiring transfusion (Ericksson et al., 1978), cerebral hemorrhage (Dixon & Bejar, 1989), low body fat, and undescended testes (Little et al., 1988). Although these things have been noted to be associated with meth or amphetamine use prenatally, there is little data to suggest any kind of causative relationship, and the direct link between fetal abnormalities and maternal methamphetamine use is not clearly discernable. One study by Oro and Dixon showed that in utero exposure to cocaine or methamphetamine was adversely, negatively associated with gestational age, birth weight, length, and occipitofrontal circumference. They also showed that "the increased rate of prematurity, intrauterine growth retardation, and perinatal complications

associated with prenatal exposure to cocaine or methamphetamine was greater than that predicted by coexisting risk factors and was consistent with the pharmacologic properties of these drugs" (Oro & Dixon, 1987). Another study by Smith et al. showed an association between decreased growth in infants exposed to meth throughout pregnancy relative to those infants only exposed in the first and second trimesters (Smith et al., 2003). Therefore, it is felt that birth outcomes may improve if the mother stops using the drug in the last one to three months of pregnancy. In addition, this study revealed significantly more small-for-gestational-age infants in the methamphetamine-exposed group than those in the nonexposed group (Smith et al., 2003). There also appeared to be a significant decrease in growth in the meth-exposed infants who were born to mothers who additionally smoked cigarettes compared to those who did not smoke (Smith et al., 2003). Finally, these infants may be at increased risk of blood-borne diseases such as HIV, Hepatitis B, and hepatitis C because of the frequent high-risk behaviors of the mother.

Early studies of amphetamine-exposed infants indicated that many of these infants had difficulties with poor feeding and extreme drowsiness throughout the first several weeks to as long as a year of life (Ericksson et al., 1978, 1981; Ramer, 1974). Some of these infants (like infants with exposures to narcotics) may display an array of behavioral disturbances after birth characterized by tremors, irritability, abnormal sleep patterns, and poor feeding, which may represent direct drug effects rather than withdrawal, as the metabolites were found in the infants' urine for up to seven days after birth (Oro & Dixon, 1987). One study that analyzed 294 mother-infant pairs (134 exposed and 160 unexposed) found that 49 percent of exposed infants displayed evidence of withdrawal symptoms and documented the need for pharmacologic intervention for the treatment of withdrawal symptoms in 4 percent of these infants (Smith et al., 2003). Following this initial hyperirritable phase (usually only displayed in the first several days of life), some meth-exposed infants were so extremely drowsy that they required tube feedings, approximating the prolonged sleep, lethargy, and depression ("crash") seen in adult users (Oro & Dixon, 1987). This period of time through the first four weeks of life is felt to be related to the dopamine depletion syndrome and may be characterized by lethargy in the infant with excessive periods of sleep, poor suck and swallow coordination, sleep apnea, and poor habituation (Shah, 2006). Exposed infants may have irregular sleep patterns, poor feeding, tremors, and increased muscle tone. Their poor ability to habituate or self-regulate, especially under stressful situations, will be further intensified if their environment is noisy and chaotic. They will not tolerate this well, which will likely lead to increased irritability and potential for abuse. In the next four months of life, the infant may display symptoms of CNS immaturity, including effects on motor development; sensory integration problems

including tactile, defensive, and texture issues; and neurobehavioral symptoms affecting their interaction and social development. This period is frequently followed by a symptom-free period or the honeymoon phase from 6 to 18 months. However, from 18 months to 5 years, the children may again begin to exhibit difficulties with sensory integration, poorly focused attention, easy distractibility, poor anger management, and aggressive outbursts (Shah, 2006).

Because of the multitude of confounding variables such as other potential drug exposures, genetic predisposition, and environmental factors, it is difficult to identify postnatal features that are directly related to in utero meth exposure. However, scientists have begun to study infants exposed to meth in utero to attempt to identify potential outcomes. One study by Hansen, Struthers, & Gospe (1993) of infants exposed to meth in utero has identified visual cognitive effects (poorer visual recognition memory—a measure correlated with subsequent IQ) and changes in behavior that appear to be permanent in these infants. Several additional studies in rats have demonstrated similar concerns for spatial learning in adult animals exposed to meth in utero (Crawford, Williams, Newman, McDougall, & Vorhees, 2003; Williams, Vorhees, Boon, Saber, & Cain, 2002; Williams et al., 2003).

Several studies have shown that prenatally meth-exposed infants may go on to exhibit further difficulties in childhood. Children exposed to meth in utero may face difficulties with what is called executive-level functioning. This functioning is related to the brain's ability to absorb information, interpret the information, and make decisions based on it. Difficulties in this level of functioning may help explain the problems that many of these children face with impulsivity, judgment, and connecting behavior with consequence. Although there are mounting studies on the effects of meth on adult brains, studies on the brains of infants exposed to meth in utero are limited but beginning to emerge. One study by Smith et al. attempted to study the possible neurotoxic effects of prenatal meth exposure on the developing brain using brain proton magnetic resonance spectroscopy (Smith et al., 2003). In this study, the researchers found the suggestion of an abnormality in the energy metabolism in the brains of children exposed to meth in utero, but acknowledged the need for more studies and the interpretation of this study with caution due to their small sample size and limited behavioral assessments (Smith et al., 2003). The study, however, may have important clinical implications, since the area found to be affected is the frontal-striatal pathway, which is involved in executive-level functioning.

Studies done in Sweden following the short-term legalization of drugs of abuse in the 1960s looked at populations of children exposed to amphetamines prenatally who were then monitored for their progress and performance (Plessinger, 1998). Several reports indicated difficulties with altered growth

and behavior in the exposed children (Plessinger, 1998). Looking at matched groups of exposed and unexposed children at 8 years and then 14 years of age, a larger number of amphetamine-exposed children did more poorly than unexposed controls in mathematics, language, and physical training (Cernerud, Eriksson, Jonsson, Steneroth, & Zetterström, 1996; Ericksson et al., 1978, 1981). These studies also showed that once these exposed children were past puberty, the boys were taller and heavier and the girls were shorter and lighter than the Swedish standards used for comparison (Cernerud et al., 1996). These findings may suggest an effect of in utero exposure of methamphetamines on normal neural development and maturation of the adenohypophysis, which raises concern that the use of amphetamines during pregnancy may cause a wide variety of effects (Plessinger, 1998).

Additional work is being done in an effort to better define any association there may be between prenatal drug exposures and later drug use and medical complications in young adults and adults. One recent study at the University of Chicago indicated that males who were exposed to meth in utero and then went on to take the drug themselves as teens or adults may have hastened onset of brain disorders such as Parkinson's Disease (Heller, Bubula, Lew, Heller, & Won, 2001).

Finally, there have been reports of deaths in fetuses and infants felt to be related to maternal meth use. Although the actual number of these cases reported in the literature is low, there is concern about the risk of death in infant's exposure to this drug either prenatally or in the postnatal period. One study reviewed the deaths of eight fetuses/infants aged from 20 weeks estimated gestational age to a 1-month-old infant (Stewart & Meeker, 1997). In these cases, it was believed that the maternal use of meth played a role in the deaths of the fetuses/infants, and the authors cited the increased vulnerability of the developing nervous system and potential compromise due to fetal acidosis, hypoxernia, decreased uterine blood flow, changes in fetal blood gases, and an increase in fetal glucose levels (Stewart & Meeker, 1997). There have also been some studies suggesting a possible link between cocaine exposure in utero and sudden infant death syndrome (SIDS), but the nature of this connection is unclear. However, because of this potential link as well as the possibility that the infant may have ingested the drug, California law now requires that all SIDS deaths be tested for certain drugs, including meth.

PARENTING ISSUES IN METH USE

A very important factor that must be considered regarding children that are exposed to meth is the environment in which the child is raised. There are

additional and potentially very dangerous consequences that may occur if a child grows up in an environment where there is active use of methamphetamine. These risks include the actual environment, which may include many hazards including the exposure to the drug itself, as well as the actual quality of parenting that the child receives.

The environment of a meth abuser is one that may contain many risks for growing children. These risks include exposure to the actual drug itself (see the next section), weapons, an unkempt and dirty home, inadequate food, inappropriate sleeping conditions, multiple unsavory visitors, and exposure to violence and sexual content and activity. The child may be neglected while the parents sleep for long periods of time during a "crash." They frequently do not receive adequate medical, dental, emotional, and educational care. Additionally, children living in these homes are at an increased risk of sexual abuse, either from witnessing sexually explicit activity or material or from becoming the actual targets of bizarre sexual activity in the home. They may also be physically abused or even killed when the parent becomes easily frustrated or the child becomes the target of the parent's homicidal ideations.

There are few studies in this area, but one study in which meth was administered to premating, gestational, and lactational rat pups demonstrated that the drug had a negative effect on maternal behavior toward the pups (Šlamberová et al., 2005).

EFFECTS ON CHILDREN

When meth is used in a home where children reside, there are several routes of potential exposure and subsequent danger to the children. First, when there is active use in the home, the drug itself is frequently readily accessible to children, often lying on surfaces within easy reach of a curious child. Children display frequent hand-to-mouth behavior, placing them at risk for picking up the drug itself and ingesting it, resulting in a continuum of possible effects ranging from minor physiologic response to significant intoxication, seizures, and death. Although this is not an uncommon event, there are only a few such cases reported in the literature. In 1998 Kolecki reported 18 cases in which children less than 13 years of age were confirmed to be victims of oral methamphetamine poisoning. In these cases, the drugs had been left out with easy access for the children. The children displayed agitation (9), inconsolability (6), increased heart rate (18), abdominal pain, vomiting (6), seizures, muscle breakdown, fever (1), and ataxia (1). Prior to identification of the cause for the children's illness, multiple tests and treatments were undertaken such as head CTs (5), spinal taps (3), and administration of spider (Centruroides sculptu-

ratus) antivenom (3), since the presentation closely resembled spider envenomation. In fact, one child developed an anaphylactic reaction to the antivenom (Kolecki, 1998). Another case reported in 1995 by Gospe profiled the case of an 11-month-old boy who presented to medical care with irritability and transient cortical blindness and involuntary turning of the head and tested positive for meth. Symptoms resolved after 12 hours of supportive care. Mother reported that she had found the infant chewing on a small plastic bag (Gospe, 1995). An additional case was reported by Narogka, who described a 13-month-old girl whose symptoms of restlessness and roving eye movements were initially felt to be from scorpion evenomation, but when she did not respond to antivenom, a urine drug screen was obtained and found to be positive for meth (Nagorka & Bergeson, 1998).

Effects of exposures to meth through breastfeeding must also be considered. Meth, like most drugs, is transferred to the breast milk when a lactating mother uses the drug. Therefore, the American Academy of Pediatrics does not recommend breastfeeding when the mother is using meth, as it is believed that the infant will receive the drug through breast milk, and this has been reported to cause irritability and poor sleeping patterns (American Academy of Pediatrics, Committee on Drugs, 2001).

At this time, there is not enough scientific data in the literature to fully understand the amount of exposure that a child may receive when living in an environment where meth is actively being smoked or used. There is no information on the potential for intake of the drug through transdermal absorption or passive inhalation. Research in this area is clearly needed.

CONCLUSION

Meth use and abuse in this country has far-reaching ramifications for not only the user but for society. While we currently understand a great deal about the medical effects of this drug on both adults and children, there is far more that needs to be studied. Additionally, the nature and extent of the effects on the user of the by-products and other chemicals that may remain in the meth following the manufacturing process are unknown at this time. The studies that are currently available have only addressed the effect of the actual drug. The National Institute of Drug Abuse (NIDA) is aggressively supporting a comprehensive research program to better understand meth's mechanism of action, physical and behavioral effects, risk and protective factors, treatments, and potential predictors of treatment success (Volkow, 2005). Continued collaborative efforts are critical to advancements in understanding the medical effects of meth on the users and children who are exposed.

NOTE

1. From: *The Methamphetamine Crisis,* Herbert C. Covey; Chapter 4, pp. 57–74, "The Short- and Long-Term Medical Effects of Methamphetamine on Children and Adults," by Kathryn Wells. Copyright © 2006 by Herbert C. Covey. Reproduced with permission of Greenwood Publishing Group, Inc., Westport, CT.

REFERENCES

Allcott, J. V., III, Barnhart, R. A., & Mooney, L. A. (1987). Acute lead poisoning in two users of illicit methamphetamine. *Journal of the American Medical Association, 258,* 510–511.

American Academy of Pediatrics, Committee on Drugs. (2001). The transfer of drugs and other chemicals into human milk. *Pediatrics, 108*(3), 776–789.

Beebe, D. K, & Walley, E. (1995). Smokable methamphetamine ("ice"): An old drug in a different form. *American Family Physician, 51*(2), 449–453.

Brown, J. M., Hanson, G. R., & Fleckenstein, A. E. (2000). Methamphetamine rapidly decreases vesicular dopamine uptake. *Journal of Neurochemistry, 74*(5), 2221–2223.

Burton, B. T. (1991). Heavy metal and organic contaminants associated with illicit methamphetamine production. In M. A. Miller & N. J. Kozel (Eds.), *Methamphetamine abuse: Epidemiologic issues and implications* (pp. 47–59). (NIDA Research Monograph Series, No. 115. DHHS Publication No. [ADM] 91–1836). Rockville, MD: National Institute on Drug Abuse.

Center for Substance Abuse Treatment. (1997). Proceedings of the national consensus meeting on the use, abuse, and sequelae of abuse of methamphetamine with implications for prevention, treatment, and research (DHHS Publication No. [SMA] 96–8013). Rockville, MD: Substance Abuse and Mental Health Services Administration.

Center for Substance Abuse Treatment. (1999). *Tip 33: Treatment for stimulant use disorders: Treatment improvement protocol* (TIP Series No. 33). Rockville, MD: Substance Abuse and Mental Health Services Administration.

Cernerud, L., Eriksson, M., Jonsson, B., Steneroth, G., & Zetterström, R. (1996). Amphetamine addiction during pregnancy: 14-year follow-up of growth and school performance. *Acta Pediatrica, 85,* 204–208.

Cho, A. K. (1990). Ice: A new dosage form of an old drug. *Science, 249,* 631–634.

Crawford, C. A., Williams, M. T., Newman, E. R., McDougall, S. A., & Vorhees, C. V. (2003). Methamphetamine exposure during the preweaning period causes prolonged changes in dorsal striatal protein kinase A activity, dopamine Ds-like binding sites, and dopamine content. *Synapse, 48,* 131–137.

Dickinson, J. E., Andres, R. L., & Parisi, V. M. (1994). The ovine fetal sympathoadrenal response to the maternal administration of methamphetamine. *American Journal of Obstetrics and Gynecology, 170,* 1452–1457.

Dixon, S. D., & Bejar, R. (1989). Echoencephalographic findings in neonates associated with cocaine and methamphetamine use: Incidence and clinical correlates. *Journal of Pediatrics, 115,* 770–778.

Ellinwood, E. H., Jr., Sudilovsky, A., & Nelson, L. M. (1973). Evolving behavior in the clinical and experimental amphetamine (model) psychosis. *American Journal of Psychiatry, 130,* 1088–1092.

Ericksson, M., Larsson, C., Windbladh, B., & Zetterström, R. (1978). The influence of amphetamine addiction on pregnancy and the newborn infant. *Acta Paediatrica Scandinavica, 67*(1), 95–99.

Eriksson, M., Larsson, G., & Zetterström, R. (1981). Amphetamine addiction and pregnancy. II: Pregnancy, delivery and the neonatal period: Socio-medical aspects. *Acta Obstetricia et Gynecologica Scandinavica, 60,* 253–259.

Ernst, T., Chang, L., Leonido-Yee, M., & Speck, O. (2000). Evidence for long-term neurotoxicity associated with methamphetamine abuse: A 1H MRS study. *Neurology, 54,* 1344–1349.

Furst, S. R., Fallon, S. P., Reznik, G. N., & Shah, P. K. (1990). Myocardial infarction after inhalation of methamphetamine [Letter to the editor]. *New England Journal of Medicine, 49,* 389–391.

Golbus, M. S. (1980). Teratology for the obstetrician: Current status. *Obstetrics and Gynecology, 55,* 269–277.

Gold, M. S. (1997). Cocaine (and crack): Clinical aspects. In J. H. Lowenson, P. Ruiz, R. B. Millman, & J. G. Langrod (Eds.), *Substance abuse: A comprehensive textbook* (3rd ed.). Baltimore: Williams and Wilkins.

Gospe, S. (1995). Transient cortical blindness in an infant exposed to methamphetamine. *Annals of Emergency Medicine, 26,* 380–382.

Hansen, R. L., Struthers, J. M., & Gospe, S. M., Jr. (1993). Visual evoked potentials and visual processing in stimulant drug-exposed infants. *Developmental Medicine and Child Neurology, 35,* 798–805.

Heller, A., Bubula, N., Lew, R., Heller, B., & Won, L. (2001). Gender-dependent enhanced adult neurotoxic response to methamphetamine following fetal exposure to the drug. *Journal of Pharmacology and Experimental Therapeutics, 298*(2), 769–779.

Hong, R., Matsuyama, E., & Nur, K. (1991). Cardiomyopathy associated with the smoking of crystal methamphetamine. *Journal of the American Medical Association, 265*(9), 1152–1154.

Inaba, D., & Cohen, W. (1993). *Uppers, downers, all arounders* (2nd ed.). Ashland, OR: CNS Productions.

Jaffe, J. H. (1990). Drug addiction and drug abuse. In A. G. Gilman, T. W. Rall, A. S. Nies, & P. Taylor (Eds.), *Goodman and Gilman's the pharmacological basis of therapeutics* (8th ed., pp. 539–545). New York: Pergamon.

Jaffe, J. H. (1995). Amphetamine (or amphetamine-like)-related disorders. In H. I. Kaplan, & B. J. Sadock (Eds.), *Comprehensive textbook of psychiatry* (6th ed., pp. 791–798). Baltimore: Williams and Wilkins.

Kamijo, Y., Soma, K., Nishida, M., Namera, A., & Ohwada, T. (2002). Acute liver failure following intravenous methamphetamine. *Veterinarian and Human Toxicology, 44*(4), 216–217.

Kato, M. (1990). Brief history of control, prevention, and treatment of drug dependence in Japan. *Drug Alcohol Dependency, 25*(2), 213–214.

Katsuragawa, Y. (1999). Effect of methamphetamine abuse on the bone quality of the calcaneus. *Forensic Science International, 101*, 43–48.

Kim, S. J., Lyoo, I. K., Hwang, J., Sung, Y. H., Lee, H. Y., Lee, D. S., et al. (2005). Frontal glucose metabolism in abstinent methamphetamine users. *Neuropsychopharmacology, 30*(7), 1383–1391.

Lederman, R. P., Lederman, E., Work, B. A., Jr., & McCann, D. S. (1978). The relationship of maternal anxiety, plasma catecholamines and plasma cortisol to progress in labor. *American Journal of Obstetrics and Gynecology, 132*, 495–500.

Levin, J. N. (1971). Amphetamine ingestion with biliary atresia. *Pediatrics, 79*, 130–131.

Lewis, D. C., & Millar, D. G. (2005, July 27). Open letter: To whom it may concern.

Little, B. B., Snell, L. M., & Gilstrap, L. C., III. (1988). Methamphetamine use during pregnancy: Outcome and fetal effects. *Obstetrics and Gynecology, 72*, 541–544.

London, E. D, Simon, S. L., Berman, S. M., Mandelkern, M. A., Lichtman, A. M., Bramen, J., et al. (2004). Mood disturbances and regional cerebral metabolic abnormalities in recently abstinent methamphetamine abusers. *Archives of General Psychiatry, 61*, 73–84.

MacKenzie, R. G., & Heischober, B. (1997). Methamphetamine. *Pediatrics in Review, 18*(9), 305–309.

Murray, J. B. (1998). Psychophysiological aspects of amphetamine-methamphetamine abuse. *Journal of Psychology, 132*(2), 227–237.

Nagorka, A. R., & Bergeson, P. S. (1998). Infant methamphetamine toxicity posing as scorpion envenomation. *Pediatric Emergency Care, 14*, 350–351.

National Institute on Drug Abuse. (1998, April). *Methamphetamine abuse and addiction*. (National Institute on Drug Abuse Research Report Series. NIH Publication No. 98–4210). Bethesda, MD: Author.

National Institutes of Health. (2004, January 5). New study suggests methamphetamine withdrawal is associated with brain changes similar to those seen in depression and anxiety. *NIH News,* Washington, DC: U.S. Department of Health and Human Services.

National Jewish Medical and Research Center. (2004). *Toxic brew of chemicals cooked up in methamphetamine labs*. Denver, CO: Author. Retrieved from www.nationaljewish.org/news/meth

Nelson, M. M., & Forfar, O. (1971). Associations between drugs administered during pregnancy and congenital abnormalities of the fetus. *British Medical Journal, 1*, 523–527.

Nestor, T. A., Tamamoto, W. I., Kam, T. H., & Schultz, T. (1989a). Crystal methamphetamine-induced acute pulmonary edema: A case report. *Hawaii Medical Journal, 48,* 457–460.

Nestor, T. A., Tamamoto, W. I., Kam, T. H., & Schultz, T. (1989b). Acute pulmonary edema caused by crystalline methamphetamine. *Lancet, 2*(8674), 1277–1278.

Nora, J. J., McNamara, D. G., & Clarke-Fraser, F. (1967). Dexamphetamine sulphate and human malformations. *Lancet, 2,* 1021–1022.

Nora, J. J., Vargo, T. A., Nora, A. H., Love, K. E., & McNamara, D. G. (1970). Dexamphetamine: A possible environmental trigger in cardiovascular malformations. *Lancet, 1*(7659), 1290–1291.

Nordahl, T. E., Salo, R., Natsuaki, Y., Galloway, G. P., Waters, C., Moore, C. D., et al. (2005). Methamphetamine users in sustained abstinence: A proton magnetic resonance spectroscopy study. *Archives of General Psychiatry, 62,* 444–452.

Oro, A. S., & Dixon, S. D. (1987). Perinatal cocaine and methamphetamine exposure: Maternal and neonatal correlates. *Journal of Pediatrics, 111,* 571–578.

Plessinger, M. A. (1998). Prenatal exposure to amphetamines: Risks and adverse outcomes in pregnancy. *Obstetrics and Gynecology Clinics of North America, 25,* 119–139.

Ramamoorthy, J. D., Ramamoorthy, S., Leibach, F. H., & Ganapathy, V. (1995). Human placental monoamine transporters as targets for amphetamines. *American Journal of Obstetrics and Gynecology, 173,* 1782–1787.

Ramer, C. M. (1974). The case history of an infant born to an amphetamine-addicted mother. *Clinical Pediatrics, 13,* 596–597.

Rowbotham, M. C. (1993). Cocaine levels and elimination in inpatients and outpatients: Implications for emergency treatment of cocaine complications. In H. Sorer (Ed.), *Acute cocaine intoxication: Current methods of treatment* (pp. 147–155). (NIDA Research Monograph Series, No. 123. DHHS Publication No. [ADM] 93–3498). Rockville, MD: National Institute on Drug Abuse.

Saxen, I., (1975). Associations between oral clefts and drugs taken during pregnancy. *International Journal of Epidemiology, 4,* 37–44.

Shah, R. (2006, February 21). *Infants exposed prenatally to methamphetamines: Developmental effects and effective interventions.* Teleconference Series, National Abandoned Infants Assistance Resource Center.

Shaner, J. W. (2002, September). Caries associated with methamphetamine abuse. *Journal of the Michigan Dental Association, 84*(9), 42–47.

Sherman, W. T., & Gautieri, R. F. (1972). Effect of certain drugs on perfused human placenta. X. Norepinephrine release by bradynin. *Journal of Pharmacological Science, 61,* 870–873.

Šlamberová, R., Charousová, P., & Pometlová, M. (2005). Methamphetamine administration during gestation impairs maternal behavior. *Developmental Psychobiology, 46,* 57–65.

Smith, L., Yonekura, M. L., Wallace, T., Berman, N., Kuo, J., & Berkowitz, C. (2003). Effects of prenatal methamphetamine exposure on fetal growth and drug with-

drawal symptoms in infants born at term. *Journal of Developmental Behavior and Pediatrics, 24*(1), 17–23.

Sowder, B., & Beschner, G. (Eds.). (1993). *Methamphetamine: An illicit drug with high abuse potential.* Unpublished report from NIDA Contract No. 271–90–0002. Rockville, MD: T. Head and Company.

Stek, A. M., Fisher, B. K., Baker, R. S., Lang, U., Tseng, C. U., & Clark, K. E. (1993). Maternal and fetal cardiovascular responses to methamphetamine in the pregnant sheep. *American Journal of Obstetrics and Gynecology, 169*(4), 888–897.

Stewart, J. L., & Meeker, J. E. 1997. Fetal and infant deaths associated with maternal methamphetamine abuse. *Journal of Analytical Toxicology, 21,* 515–517.

Streltzer, J., & Leigh, H. (1977, January–February). Amphetamine abstinence psychosis—Does it exist? *Psychiatric Opinion,* 47–50.

Szuster, R. R. (1990). Methamphetamine in psychiatric emergencies. *Hawaii Medical Journal, 49,* 389–391.

Tandy, K. P. (2004, March 24). Statement before the U.S. House of Representatives, Committee on Appropriations, Subcommittee for the Departments of Commerce, Justice, State, the Judiciary and Related Agencies. Washington, DC.

Thomas, D. B. (1995). Cleft palate, mortality and morbidity in infants of substance abusing mothers. *Journal of Paediatric Child Health, 31,* 457–460.

Tomiyama, G. (1990). Chronic schizophrenia-like states in methamphetamine psychosis. *Japanese Journal of Psychiatry and Neurology, 44*(3), 531–539.

Volkow, N. D. (2005, April 21). Methamphetamine abuse—Testimony before the Senate Subcommittee on Labor, Health and Human Services, Education, and Related Agencies—Committee on Appropriations. Washington, DC.

Volkow, N. D., Chang, L., Wang, G.-J., Fowler, J. S., Leonido-Yee, M., Franceschi, D., et al. (2001). Association of dopamine transporter reduction with psychomotor impairment in methamphetamine abusers. *American Journal of Psychiatry, 158*(3), 377–382.

Warner, P., Connolly, J. P., Gibran, N. S., Heimback, D. M., & Engrav, L. H. (2003). The methamphetamine burn patient. *Journal of Burn Care and Rehabilitation, 24*(5), 275–278.

Wetli, C. V. (1993). The pathology of cocaine: Perspectives from the autopsy table. In H. Sorer (Ed.), *Acute cocaine intoxication: Current methods of treatment* (pp. 172–183). (NIDA Research Monograph Series, No. 123. DHHS Publication No. [ADM] 93–3498). Rockville, MD: National Institute on Drug Abuse.

Williams, M. T., Morford, L. L., Wood, S. L., Wallace, T. L., Fukumura, M., Broening, H. W., et al. (2003). Developmental D-methamphetamine treatment selectively induces spatial navigation impairments in reference memory in the Morris water maze while sparing working memory. *Synapse, 48,* 138–148.

Williams, M. T., Vorhees, C. V., Boon, F., Saber, A. J., & Cain, D. P. (2002). Methamphetamine exposure from postnatal day 11 to 20 causes impairments in both behavioral strategies and spatial learning in adult rats. *Brain Research, 958,* 312–321.

Wolkoff, D. (1997). Methamphetamine abuse: An overview for health care professionals. *Hawaii Medical Journal, 56,* 34–36, 44.

Yui, K., Goto, K., Ikemoto, S., Ishiguro, T., & Kamata, Y. (2000). Increased sensitivity to stress in spontaneous recurrence of methamphetamine psychosis: Noradrenergic hyperactivity with contribution from dopaminergic hyperactivity. *Journal of Clinical Psychopharmacology, 20*(2), 165–174.

Chapter **11**

Vapors May Be Dangerous If Inhaled: An Overview of Inhalants and Their Abuse

Jace Waguspack, BS, and Paula K. Lundberg-Love, PhD

The word *inhalant* is a broad term used to describe a heterogeneous group of chemicals that share a common route of administration. In addition to the drugs identified as "inhalants," there are other drugs that are inhaled, but they are excluded from this group as they fall under other drug classifications (i.e., cigarettes, marijuana, or crack cocaine). Inhalants can be effectively subdivided into three separate groups known as volatile substances, nitrous oxide, and nitrites (Balster, 1998).

The first group, volatile substances, is the most commonly abused of the inhalants, and it includes a wide variety of substances that give off vapors at room temperature (Brouette & Anton, 2001). This group typically includes solvents, adhesives, aerosols, cleaning agents, and gasoline (Sharp & Rosenberg, 1997). There are many different types of chemicals that constitute this group, but some of the more common chemicals include toluene, acetone, butane, trichloroethylene, hexane, propane, fluorocarbons, and others (Sharp & Rosenberg, 1997). These chemicals can be found in a variety of easily acquired products, including some glues, spray paint, dry cleaning agents, paint thinner, nail polish remover, and typewriter correction fluid (Sharp & Rosenberg, 1997). The relative ease with which one can acquire these products is one of the proposed explanations for their widespread abuse, especially among young people (McHugh, 1987).

The chemical that constitutes the second group of inhalants is nitrous oxide. Nitrous oxide is commonly known as laughing gas, and can be acquired by those who have access to medical supplies because it is used in dentistry as a

type of anesthetic. It also is often used as a propellant in whipped cream, which makes it available in grocery stores (Brouette & Anton, 2001).

The final group of inhalants comprises chemicals referred to as nitrites. Nitrites include the chemicals butyl nitrite, amyl nitrite, and isobutyl nitrite, and are commonly found in room odorizers (Brouette & Anton, 2001). Due to the fact that they enhance sexual experiences, they are also often sold in sex shops (Brouette & Anton, 2001).

There are several methods by which these chemicals are inhaled, including "sniffing," "huffing," "spraying," and "bagging." "Sniffing" involves inhaling vapors from an open container (Dinwiddie, 1994). "Huffing" is a practice whereby an individual soaks a rag in the chemical of choice and then holds the rag to his or her face to be inhaled (Brouette & Anton, 2001). Inhalant users sometimes directly spray the desired inhalant into the mouth or nose (Brouette & Anton, 2001). "Bagging," on the other hand, involves filling a bag with the chemical and then breathing from the bag (Brouette & Anton, 2001). The practice of bagging can be quite dangerous, as the drowsiness that inhalants induce coupled with the lack of fresh oxygen received when breathing from the bag may cause the individual to lose consciousness. When adhesives are being used in this manner, the bag may seal to the person's face, causing death by asphyxiation if the individual loses consciousness (Brouette & Anton, 2001).

HISTORY AND EPIDEMIOLOGY

The abuse of inhalant drugs is a relatively recent phenomenon. Inhalant use first began to receive public attention in the 1960s, when sniffing glue became popular (Fredlund, Spence, & Maxwell, 1989). During this time, abuse of nitrites began to emerge as well (Haverkos, Kopstein, Wilson, & Drotman, 1994). Nitrites became particularly popular among homosexual men due to the enhancement of sexual experience from vasodilation (widening of blood vessels) resulting in penile engorgement, combined with relaxation of smooth muscle tissue (Goode & Troiden, 1979), as well as prolonged orgasm (Newell, Spitz, & Wilson, 1988). Chronic use of nitrites has several negative hematological effects, including effects on lymphocyte number and function, reduction of monocyte adherence, and the suppression of natural killer cells. Nitrite abuse also compromises T-dependent antibody induction, cytotoxic T cell induction, and the tumoricidal activity of macrophages (Brouette & Anton, 2001; Dax, Lange, & Jaffe, 1989; Soderberg, Chang, & Barnett, 1996). Due to nitrites' negative hematological effects, as well as a high correlation of nitrite use in AIDS patients, it was once postulated that nitrites were in fact the cause of AIDS (Brouette & Anton, 2001). Because of the discovery of the HIV virus, this

theory was disproved. A positive side effect of the association of nitrites and possible susceptibility to AIDS was that the use of nitrites sharply decreased and has never regained its former popularity (Brouette & Anton, 2001).

However, the use of other inhalant substances remains high, particularly among young people. Some sources even report that inhalants have surpassed marijuana in popularity among 12-year-olds (Meyer & Quenzer, 2005). A recent study found that 17 percent of eighth graders in the United States have used inhalants (Johnston, O'Malley, Bachman, & Schulenberg, 2006). It has been proposed that the magnitude of this number is due to the ease of access to inhalants through a variety of common household substances (Ridenour, Bray, & Cottler, 2007). In fact, because they are so readily available, research indicates that inhalants are the gateway drugs for many children (McHugh, 1987).

While inhalant abuse is most common in young people, prevalence rates vary among different ethnic groups. Use among Hispanic Americans was found to be high, but these data are thought to be confounded by the low socioeconomic status and social dysfunction of the particular group of Hispanics studied (Padilla, Padilla, Morales, Olmedo, & Ramirez, 1979). Prevalence of inhalant use by Native American youths on reservations has been reported to be as high as 34 percent of 8th graders and 20 percent of 12th graders (Beauvais, 1992). Conversely, use of inhalants has been found to be particularly low in African Americans as opposed to other ethnic groups (Compton, Cottler, Dinwiddie, Mager, & Asmus, 1994).

Geographically, while inhalant abuse is seen across the United States, it is most pronounced in Alaska, where as many as 22 percent of high school students report having used inhalants (Meyer & Quenzer, 2005). Appropriately, one of the few inhalant abuse treatment centers in the United States, the Tundra Swan Inhalant Treatment Center, is located in Bethel, Alaska (Meyer & Quenzer, 2005).

COMORBIDITY OF PSYCHIATRIC DISORDERS AND INHALANT USE

Inhalant use has a very high rate of comorbidity with psychological disorders. Research indicates that 70 percent of inhalant users met criteria for a mental disorder at some point during their lifetime and 38 percent met criteria for a mental disorder within a year of the survey (Wu & Howard, 2007). Furthermore, female inhalant users exhibit mental disorders more often than do male inhalant users, and female users exhibit multiple mental disorders more often than do their male counterparts (Wu & Howard, 2007). Major depression was the

most common mental disorder found comorbid with inhalant use (41%), and antisocial personality disorder was the second most common mental disorder (32%) (Wu & Howard, 2007). Of the 70 percent of inhalant users that met the criteria for one lifetime mental disorder, 49 percent met the criteria for three or more disorders. The high rate of comorbidity between inhalant use and psychological disorders has important implications for the treatment of inhalant abuse, which will be discussed later in this chapter. Although, at this time, a causal relationship cannot be inferred between inhalant use and psychiatric disorders, it is interesting to note that in the majority of cases, inhalant use precedes the onset of most mood and anxiety disorders identified in inhalant users, and that the onset of these psychiatric disorders tends to occur earlier in inhalant users versus nonusers (Wu & Howard, 2007).

MECHANISMS OF ACTION

Volatile Substances

The physiological mechanisms of action of the volatile substances are the least understood of all of the inhalants. However, recent research has made some progress in identifying and understanding how these agents affect neurons and brain neurochemistry. Many of the mechanisms of action of these substances are similar to those of ethanol. Moreover, these substances exert specific and nonspecific effects on the brain. With respect to their nonspecific mechanisms, volatile substances interact directly with the cell membranes of neurons, thereby fluidizing them (Brouette & Anton, 2001). Such a process reduces the efficiency of neural processing and results in overall central nervous system (CNS) depression. They also interact with and enhance the activity of gamma-aminobutyric acid (GABA)-A receptors and stimulate glycine receptors. Both GABA and glycine are two different neurotransmitter systems whose activation leads to inhibition of the CNS (Beckstead, Weiner, Eger, Gong, & Mihic, 2000). Chemicals in the volatile substance class of inhalants also appear to inhibit the activity of the N-methyl-D-aspartate (NMDA) glutamate receptors, a neurotransmitter system often responsible for central nervous system excitation (Cruz, Balster, & Woodwar, 2000) and the potentiation of learning and memory. This action further induces inhibition of CNS activity. Some volatile substances (specifically toluene) activate dopamine neurons in the ventral tegmental area, a part of the brain involved in the reinforcing effects of many drugs of abuse (Riegel, Zapata, Shippenberg, & French, 2007). Finally, recent research has shown that volatile solvents enhance the activity of serotonin 3A receptors (5-HT 3A), which are also believed to be involved

in the reinforcement of drug-seeking behavior (Lopreato, Phelan, Borghese, Beckstead, & Mihic, 2003). The mechanisms behind this involvement are not well understood. However, some researchers have demonstrated that activation of 5-HT 3A receptors can stimulate dopamine release in the nucleus accumbens, a structure involved in the reinforcing effects of many drugs of abuse (Chen, van Praag, & Gardner, 1991).

Nitrous Oxide

The mechanisms by which nitrous oxide exerts its effects on human behavior are in dispute. Some argue that nitrous oxide works primarily through the opiate receptor system, and that it modulates the release of beta-endorphins and binds to the three types of opiate receptors: mu, kappa, and delta (Brouette & Anton, 2001; Gillman & Lichtigfeld, 1998). This theory would explain the analgesic effects of nitrous oxide, as activation of opiate receptors is known to produce analgesia (Gillman & Lichtigfeld, 1998). Activation of certain opiate receptors is also known to induce euphoria, which is also observed in nitrous oxide intoxication (Beckman, Zacny, & Walker, 2006). However, other research contradicts the opiate receptor theory of nitrous oxide analgesia and intoxication effects. In one such study, naloxone, a known opiate receptor antagonist, was administered following administration of nitrous oxide (Zacny et al., 1999). If the analgesia and euphoria caused by nitrous oxide were the result of opiate receptor activation, administration of naloxone should have reduced both of these effects. Instead, this study found that there was no significant reduction in analgesia or euphoria following naloxone administration, suggesting a separate mechanism of action for both of these effects (Zacny et al., 1999). Further research is necessary to determine the role of the opiate receptors in the analgesic and intoxicating effects of nitrous oxide, though it would appear that there is more current research that supports some degree of opiate receptor involvement. Less disputed is the theory that nitrous oxide blocks NMDA glutamate receptors (Brouette & Anton, 2001; Yamakura & Harris, 2000). This action is likely to underlie the short-term memory deficits often experienced by those under the influence of nitrous oxide (Brouette & Anton, 2001).

Nitrites

The mechanism of action of the third group of inhalants, nitrites, is quite different from the previous two groups. Instead of binding to specific neurotransmitter receptors, nitrites induce vasodilation, which results in the relaxation of blood vessels, and increased blood flow (Brouette & Anton, 2001).

Vasodilation of cerebral arteries is thought to underlie the psychological effects of nitrite intoxication (Brouette & Anton, 2001), while vasodilation of peripheral blood vessels results in lower blood pressure, flushing, syncope (fainting), throbbing sensations, and feelings of warmth (Balster, 1998). Nitrites also cause relaxation of smooth muscle tissue (Brouette & Anton, 2001).

ACUTE EFFECTS

Intoxication

The symptoms observed in inhalant intoxication vary depending upon the class of the inhalant used. Volatile substances cause drowsiness, diplopia (double vision), dysarthria (slurred speech), ataxia (an inability to walk and balance), and general disorientation (Brouette & Anton, 2001). When inhaled in higher concentrations, visual hallucinations can occur (Brouette & Anton, 2001). Because volatile substances have a similar mechanism of action to that of ethanol, it is not surprising that inhalant intoxication closely resembles that of ethanol. Disinhibition is accompanied by perceptual distortions and incoordination (Dinwiddie, 1994). Furthermore, users often experience nausea and vomiting, headaches, coughing, and excessive salivation (Dinwiddie, 1994). Occasionally, one may develop a rash around the nose and mouth (Dinwiddie, 1994). Some abusers even develop delusions, such as the belief that they can fly, which results in injury if these individuals subsequently jump out of windows or trees (Evans & Raistrick, 1987). There is evidence that tolerance develops with prolonged use of volatile substances (Ron, 1986).

In contrast, the symptoms of nitrous oxide intoxication often mimic those of psychedelic drugs as opposed to alcohol (Atkinson & Green, 1983). Nitrous oxide intoxication is often described as a dissociative experience (Brouette & Anton, 2001). While it does not produce the same reaction in all users, those that abuse it report that it induces euphoria, tingling, numbness, dizziness, hallucinations, and sensations of warmth (Brouette & Anton, 2001). Short-term memory loss has also been observed to occur as a result of nitrous oxide intoxication (Brouette & Anton, 2001).

The final group of inhalant drugs, nitrites, induces euphoria, floating sensations, increased tactile sensitivity, disinhibition, heightened sexual arousal, relaxation of the anus, and prolonged orgasm (Newell et al., 1988).

Sudden Sniffing Death Syndrome

Sudden sniffing death syndrome refers to a phenomenon whereby inhalation of chemicals from the volatile substance class of inhalants sensitizes the heart

to the neurotransmitter epinephrine, which is involved in startle and stress responses (Bass, 1970). When the user is then frightened or stressed, as can happen from being caught inhaling by an adult or a startling visual hallucination, the hypersensitivity of the heart to epinephrine can trigger a fatal cardiac arrhythmia (Committee on Substance Abuse, 1996). Such a fatality can occur at any time, even upon an individual's first use of the substance (Committee on Substance Abuse, 1996). There also may be a higher risk of sudden death associated with fuel gases and aerosols than with adhesives (King, Smialek, & Troutman, 1985).

CHRONIC ORGAN EFFECTS

Chronic abuse of the volatile substance class of inhalants can have deleterious effects on multiple organ systems throughout the body. Hepatotoxicity, or toxicity of the liver, is associated with several of the volatile substances (Brouette & Anton, 2001). However, liver toxicity usually decreases shortly after cessation of inhalant use (Fornazzari, 1988). Pulmonary disease, emphysema, and chemical pneumonitis have also been found in individuals chronically exposed to volatile substances (Dinwiddie, 1994). Furthermore, heart complications, such as those found in sudden sniffing death syndrome, are a serious risk (Brouette & Anton, 2001). Some volatile substances, toluene in particular, can cause renal dysfunction (Brouette & Anton, 2001). Other research has even linked volatile substance abuse to bone marrow suppression (Flanagan, Ruprah, Meredith, & Ramsey, 1990). There are no known long-term complications of nitrous oxide abuse, as the side effects of nitrous oxide subside following abstinence (Brouette & Anton, 2001). Meanwhile, the most common complication resulting from chronic abuse of nitrites is a reduction in the functioning and efficacy of the immune system through various mechanisms, as discussed previously (Brouette & Anton, 2001).

CENTRAL NERVOUS SYSTEM EFFECTS

There are multiple brain structures affected by chronic inhalant abuse. Cell death in the cerebellum, a structure vital for balance and gait, often occurs (Fornazzari, Wilkinson, Kapur, & Carlen, 1983). With prolonged use, this damage may become permanent (Lazar, Ho, Melen, & Daghestani, 1983). Furthermore, destruction of white matter via neuronal demyelination is also observed in many chronic volatile substance abusers (Brouette & Anton, 2001). Myelin is a substance that insulates neurons such that the neural transmission of myelinated neurons is much more rapid than that of nonmyelinated neurons.

Therefore, the destruction of myelin in the central nervous system can produce a wide array of neurobehavioral deficits. In severe cases, this may even produce dementia (Meadows & Verghese, 1996). A recent MRI study confirmed neurological abnormalities in subcortical brain structures and white matter in 44 percent of the solvent abusers studied, as opposed to only 25.5 percent of other drug users studied (Rosenberg, Grigsby, Dreisbach, Busenbark, & Grigsby, 2002). These results imply that volatile solvents are neurotoxic, and that they produce neurological abnormalities more often than do other drugs, such as cocaine, marijuana, alcohol, amphetamines, and opiates.

Neuropsychological Deficits and Organic Solvent Neurotoxicity

Neuropsychological testing of chronic inhalant abusers has revealed a variety of neurobehavioral consequences. These include deficits in auditory discrimination, psychomotor speed, visuo-motor functioning, and memory impairment (Tsushima & Towne, 1977). Furthermore, some research suggests that the length of inhalant use correlates with lower neuropsychological test scores in these areas (Tsushima & Towne, 1977). Impairments in executive function (i.e., impulse control, planning, strategy formation, self-monitoring, and problem solving) have also been observed in chronic volatile substance abusers. These types of impairments may interfere with the effective treatment of these individuals due to the individual's lack of insight into his problems or an inability to follow steps to resolve such problems (Rosenberg et al., 2002).

Some individuals exposed to volatile solvents over a prolonged period of time develop a disorder called organic solvent neurotoxicity (OSN). This disorder is characterized by three levels of severity (Ogden, 1993). Symptoms of Type 1 OSN include fatigue, irritability, depression, and anxiety. This is the mildest form of OSN, and no significant types of neuropsychological impairment have been demonstrated in individuals with this type of OSN (Ogden, 1993). With continued abstinence from solvent ingestion, the symptoms diminish and ultimately disappear (Ogden, 1993). Symptoms of Type 2 OSN include personality and mood disturbances, deficiencies in motivation and impulse control, and deficits in memory, learning, concentration, and psychomotor speed (Ogden, 1993). Symptoms of Type 2 OSN generally improve somewhat, although not completely, after abstinence from exposure to solvents (Ogden, 1993). Type 3 OSN is the most severe form of the disorder, characterized by severe dementia accompanied by progressive deterioration of memory, cognitive abilities, and emotional functioning (Ogden, 1993). This condition is irreversible (Ogden, 1993).

FETAL EFFECTS

Administration of inhalants has been shown to affect the fetus in both animals and humans. Due to their high lipid solubility, most volatile substances rapidly and easily cross the placenta (Brouette & Anton, 2001). In animal studies, prenatal exposure to the solvent toluene resulted in lower birth weight and minor malformations (Bowen, Mohammadi, Batis, & Hannigan, 2007). A condition closely mimicking fetal alcohol syndrome has been identified in human infants born to volatile substance abusers (Toutant & Lippmann, 1979). Symptoms include facial deformities, limb abnormalities, growth retardation, and developmental delays. There is also evidence that the infant can be born with a withdrawal syndrome if the mother had used inhalants a few days before the child's birth (Tenenbein, Casiro, & Seshia, 1996).

INHALANT WITHDRAWAL SYNDROME

Despite the fact that a withdrawal syndrome for inhalants is not classified in the *DSM–IV–TR* (American Psychiatric Association, 2000), research indicates that some individuals do have withdrawal symptoms upon abstinence from inhalants. Symptoms include anhedonia, irritability, sleep disturbances, psychomotor slowing, dry mouth, lacrimation (watery eyes), drug craving, headaches, and heart palpitations (Muralidharan, Rajkumar, Mulla, Kayak, & Benegal, 2008; Shah, Vankar, & Upadhyaya, 1999). Pharmacological treatment of inhalant withdrawal symptoms has not been well established, but some research suggests that the administration of benzodiazepines may be effective (Brouette & Anton, 2001). Some new research indicates that baclofen, a GABA-B agonist may be an effective treatment option (Muralidharan et al., 2008).

TREATMENT OF INHALANT ABUSE

The current approach to the treatment of inhalant abuse in the United States is inadequate (Beauvais, Jumper-Thurman, Plested, & Helm, 2002). There are several factors that limit the effectiveness of the treatment of inhalant abuse, including a need for extended detoxification (Reidel, Hebert, & Bird, 1995), common comorbid psychological disorders (Wu & Howard, 2007), the presence of neurological damage (Beauvais et al., 2002), a lack of treatment centers dedicated to inhalant abuse (Beauvais et al., 2002), and an incomplete understanding of the mechanisms of action of many inhalant drugs (Shen, 2007). Furthermore, as a result of these limitations, treatment centers that do accept

inhalant abusers are often pessimistic about the successful rehabilitation of these individuals (Beauvais et al., 2002).

Despite barriers to effective treatment, some recent progress has been made with respect to the primary prevention of inhalant abuse. Primary prevention focuses on stopping potential users from trying the drug. A recent study examined the response of junior high students to anti-inhalant messages (Crano, Siegel, Alvaro, & Patel, 2007). This study exposed inhalant users, vulnerable nonusers, and resolute nonusers to a variety of different anti-inhalant advertisements. The researchers discovered that indirect messages (i.e., messages not overtly targeting the viewer) were more effective in influencing users and vulnerable nonusers. Furthermore, users responded more negatively to messages that threatened physical harm (as opposed to negative social consequences) as a result of inhalant abuse. Finally, vulnerable nonusers were affected more by messages from someone their age, while users were affected more by messages from an adult doctor. The findings of this study can be used to construct more influential and persuasive anti-inhalant messages based upon the target audience. Anti-inhalant messages that are more effective in overcoming users' resistance have greater potential to influence users to quit before they develop patterns of abuse, dependence, and brain impairment.

Despite the incomplete understanding of the mechanisms of action through which inhalants exert their effects, some preliminary pharmacological treatments for inhalant abuse are under investigation. Some research suggests that inhalants act as 5-HT3-A receptor agonists, similar to one of the known mechanisms of action of alcohol (Lopreato et al., 2003). Based on the success of some 5-HT3 receptor antagonists in decreasing alcohol consumption in rodent models and human test subjects, these researchers suggest that antagonism of 5-HT3 receptors may also decrease inhalant abuse (Lopreato et al., 2003). A single subject case study involving administration of lamotrigine (Lamictal) may support this hypothesis. Lamotrigine blocks voltage gated sodium channels, modulates the release of glutamate and aspartate, inhibits dopamine uptake, blocks 5-HT3-A receptors, and may have some effect on calcium channels (Shen, 2007). In this study, administration of lamotrigine to a chronic inhalant abuser decreased craving symptoms and helped the subject remain abstinent throughout the six-month experimental period (Shen, 2007). While these studies show promise for the future of the pharmacological treatment of inhalant abuse, more research must be conducted to investigate the reliability and efficacy of lamotrigine.

There are other research studies that have examined the pharmacological treatment of withdrawal symptoms of inhalant dependence. In a brief three-

subject case series study, Muralidharan et al. (2008) administered baclofen, a GABA-B agonist, in order to reduce or eliminate the withdrawal symptoms of inhalant dependence. Baclofen's agonism of GABA-B receptors appears to modulate the activity of alcohol (and possibly inhalants) on GABA-A receptors. The three subjects in this study all reported a significant decrease in withdrawal symptoms as well as in inhalant cravings within 48 hours of initiation of baclofen treatment. Furthermore, the symptoms and cravings remained low throughout the course of treatment. The researchers also suggest that baclofen may be useful in relapse prevention.

A large number of drug treatment center directors believe that the treatment of inhalant abusers takes longer than recovery from other types of substance abuse, that it is more difficult, and thus is overall less successful (Beauvais et al., 2002). Furthermore, due to the extended length of detoxification, the high comorbidity of mental disorders with inhalant abuse, and the wide range of social dysfunction seen in many inhalant abusers, many treatment center professionals believe that standard treatment programs, which are highly structured with a brief progress schedule, will not be effective for the treatment of inhalant abusers (Beauvais et al., 2002). Many directors of treatment centers also noted that by the time detoxification of chronic users was complete and effective treatment could begin, the patient had to be discharged, often due to third-party payer restrictions on length of treatment (Beauvais et al., 2002). Moreover, many treatment providers believed that chronic inhalant abusers suffered significant neurological damage, which negatively affected their optimism for a positive outcome for these patients (Beauvais et al., 2002). Therefore, the inclusion of professionals trained in neuropsychological assessment and rehabilitation is important in the treatment of chronic inhalant users. These professionals are essential for detecting whether neurological damage has occurred, how extensive the damage may be, and the development of successful techniques for the neuropsychological rehabilitation of these individuals to help them overcome or compensate for their deficits. Furthermore, with as many as 70 percent of inhalant users meeting the criteria for one or more psychological disorders, therapy and/or pharmacological treatment may be necessary to effectively treat the inhalant abuse. The wide range of social dysfunction present in inhalant users must be addressed as well, and young users typically require significant case management (Beauvais et al., 2002). Education is also an important factor in treatment, as more than 27 percent of drug treatment center directors surveyed indicated that a lack of knowledge by the general public and treatment professionals significantly compromised effective treatment (Beauvais et al., 2002).

CONCLUSION

Inhalants are a diverse group of chemicals that share a common route of administration and are not included in other categories of drugs of abuse that are inhaled (i.e., nicotine, marijuana, or crack cocaine). Inhalants can be divided into three groups of chemicals: volatile substances, nitrous oxide, and nitrites. Common methods of administration include "sniffing," "huffing," "spraying," and "bagging." Inhalant use began to receive public attention in the 1960s, and has since become a significant problem, particularly among young people. Use varies among different ethnic groups. While Hispanics and Native Americans show higher-than-average prevalence rates, African Americans show exceptionally low prevalence rates. Inhalant abuse is also particularly high in Alaska. Furthermore, a high percentage of inhalant users have one or more comorbid mental disorders, with major depression and antisocial personality disorder being the most common.

The mechanisms of action of the volatile substances are not completely understood. However, current research indicates that they fluidize cell membranes, enhance the activity of GABA-A and glycine receptors, inhibit the activity of NMDA glutamate receptors, activate dopamine neurons in the ventral tegmental area, and enhance the action of 5-HT-3A receptors. Nitrous oxide is thought by some to be mediated via the actions on a variety of opiate receptors, although others dispute this hypothesis. It also inhibits the action of NMDA glutamate receptors. Nitrites induce central and peripheral vasodilation and the relaxation of smooth muscle tissue.

The acute effects of inhalant use include intoxication and sudden sniffing death syndrome. In volatile substances, intoxication includes drowsiness, diplopia, dysarthria, ataxia, disorientation, and in higher doses, visual hallucinations and delusions. Nitrous oxide intoxication can induce euphoria, tingling, numbness, dizziness, hallucinations, warmth, and short-term memory loss. Nitrite intoxication results in euphoria, floating sensations, increased tactile sensitivity, disinhibition, heightened sexual arousal, relaxation of the anus, and prolonged orgasm. Sudden sniffing death syndrome occurs when a volatile substance sensitizes the heart to the action of epinephrine and the user is then startled. The subsequent release of epinephrine into the sensitized heart can produce a fatal cardiac arrhythmia. This can potentially happen at any time, even during an individual's first use.

The chronic use of inhalants can have potentially damaging effects on multiple organ systems. In the volatile substance group, these can include hepatotoxicity, pulmonary disease, heart complications, renal dysfunction, and bone marrow suppression. Chronic abuse of nitrites can have negative effects on the

immune system. The effects of prolonged use of inhalants upon the central nervous system include cerebellar atrophy, diffuse destruction of white matter, and the possibility of dementia. Chronic inhalant abuse may also result in deficits in auditory discrimination, psychomotor speed, visuo-motor functioning, and memory. Furthermore, a condition called organic solvent neurotoxicity, which is characterized by a variety of cognitive and mood disturbances may develop. Inhalant use by a mother can also affect an unborn child. There are documented impairments in both animal and human models of the disorder, and in human infants, a condition similar to fetal alcohol syndrome can occur. Moreover, the child can also show withdrawal symptoms upon birth if the mother has recently used inhalants.

Withdrawal of inhalants is not included as a diagnostic entity in the *DSM-IV–TR*, but withdrawal effects have been documented. They include anhedonia, irritability, sleep disturbances, psychomotor slowing, dry mouth, lacrimation, craving, headaches, and heart palpitations. This withdrawal syndrome may be treated with some success by using benzodiazepines or baclofen.

Treatment of inhalant abuse is complicated and may be confounded by many factors. However, important areas to address include primary prevention, pharmacological treatment, neuropsychological evaluation and rehabilitation, treatment of comorbid mental disorders, and education about the disorder. Pharmacological intervention is currently rather limited, but there has been preliminary success in treating an inhalant abuser with lamotrigine.

Inhalant abuse continues to be a serious problem, particularly among young people. The incomplete knowledge of the mechanisms of action for some of these substances, combined with insufficient resources for the treatment of the abusers and the complexity of the issues surrounding inhalant abuse, make it difficult for these individuals to receive the help that they need. Further research into these topics is warranted and necessary, and more resources must be devoted to the study and treatment of inhalant abuse to effectively combat this growing problem.

REFERENCES

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Atkinson, R. M., & Green, J. D. (1983). Personality, prior drug use, and introspective experience during nitrous oxide intoxication. *International Journal of Addiction, 18*(5), 717–738.

Balster, R. L. (1998). Neural basis of inhalant abuse. *Drug and Alcohol Dependence, 51*, 207–214.

Bass, M. (1970). Sudden sniffing death. *Journal of the American Medical Association, 212*(12), 2075–2079.

Beauvais, F. (1992). Comparison of drug use rates for reservation Indian, non-reservation Indian, and Anglo youth. *American Indian and Alaska Native Mental Health Research, 5*(1), 13–31.

Beauvais, F., Jumper-Thurman, P., Plested, B., & Helm, H. (2002). A survey of attitudes among drug user treatment providers toward the treatment of inhalant users. *Substance Use and Misuse, 37*(11), 1391–1410.

Beckman, N. J., Zacny, J. P., & Walker, D. J. (2006). Within-subject comparison of the subjective and psychomotor effects of a gaseous anesthetic and two volatile anesthetics in healthy volunteers. *Drug and Alcohol Dependence, 81,* 89–95.

Beckstead, M. J., Weiner, J. L., Eger, E. I., II, Gong, D. H., & Mihic, S. J. (2000). Glycine and gamma-aminobutyric acid-A receptor function is enhanced by inhaled drugs of abuse. *Molecular Pharmacology, 57,* 1199–1205.

Bowen, S. E., Mohammadi, M. H., Batis, J. C., & Hannigan, J. H. (2007). Gestational toluene exposure effects on spontaneous and amphetamine-induced locomotor behavior in rats. *Neurotoxicology and Teratology, 29,* 236–246.

Brouette, T., & Anton, R. (2001). Clinical review of inhalants. *American Journal on Addictions, 10,* 79–94.

Chen, J. P., van Praag, H. M., & Gardner, E. L. (1991). Activation of 5-HT3 receptor by 1-phenylbiguanide increases dopamine release in the rat nucleus accumbens. *Brain Research, 543,* 354–357.

Committee on Substance Abuse and Committee on Native American Child Health. (1996). Inhalant abuse. *Pediatrics, 97*(3), 420–423.

Compton, W. M., Cottler, L. B., Dinwiddie, S. H., Mager, D. E., & Asmus, G. (1994). Inhalant use: Characteristics and predictors. *American Journal on Addiction, 3*(3), 263–272.

Crano, W. D., Siegel, J. T., Alvaro, E. M., & Patel, N. M. (2007). Overcoming adolescents' resistance to anti-inhalant appeals. *Psychology of Addictive Behaviors, 21*(4), 516–524.

Cruz, S. L., Balster, R. L., & Woodwar, J. J. (2000). Effects of volatile solvents on recombinant N-methyl-D-aspartate receptors expressed in xenopus oocytes. *British Journal of Pharmacology, 131,* 1303–1308.

Dax, E. M., Lange, W. R., & Jaffe, J. H. (1989). Allergic reactions to amyl nitrite inhalation. *American Journal of Medicine, 86,* 732.

Dinwiddie, S. H. (1994). Abuse of inhalants: A review. *Addiction, 89,* 925–939.

Evans, A. C., & Raistrick, D. (1987). Phenomenology of intoxication with toluene-based adhesives and butane gas. *British Journal of Psychiatry, 150,* 769–773.

Flanagan, R. J., Ruprah, M., Meredith, T. J., & Ramsey, J. D. (1990). An introduction to the clinical toxicology of volatile substances. *Drug Safety, 5,* 359–383.

Fornazzari, L. (1988). Clinical recognition and management of solvent abusers. *Internal Medicine for the Specialist, 9*(6), 2–7.

Fornazzari, L., Wilkinson, D. A., Kapur, B. M., & Carlen, P. L. (1983). Cerebellar cortical and functional impairment in toluene abusers. *Acta Neurologica Scandinavica, 67,* 319–329.

Fredlund, E. V., Spence, R. T., & Maxwell, J. C. (1989). *Substance use among students in Texas secondary schools, 1988.* Austin: Texas Commission on Alcohol and Drug Abuse.

Gillman, M. A., & Lichtigfeld, F. J. (1998). Clinical role and mechanisms of action of analgesic nitrous oxide. *International Journal of Neuroscience, 93*(1–2), 55–62.

Goode, E., & Troiden, R. R. (1979). Amyl nitrite use among homosexual men. *American Journal of Psychiatry, 136,* 1067–1069.

Haverkos, H. W., Kopstein, A. N., Wilson, H., & Drotman, P. (1994). Nitrite inhalants: History, epidemiology, and possible links to AIDS. *Environmental Health Perspective, 102,* 858–861.

Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2006). *Monitoring the future national results on adolescent drug use: Overview of key findings.* Bethesda, MD: National Institute on Drug Abuse.

King, G. S., Smialek, J. E., & Troutman, W. G. (1985). Sudden death in adolescents resulting from the inhalation of typewriter correction fluid. *Journal of the American Medical Association, 253,* 1604–1606.

Lazar, R. B., Ho, S. U., Melen, O., & Daghestani, A. N. (1983). Multifocal central nervous system damage caused by toluene abuse. *Neurology, 33,* 1337–1340.

Lopreato, G. F., Phelan, R., Borghese, C. M., Beckstead, M. J., & Mihic, S. J. (2003). Inhaled drugs of abuse enhance serotonin-3 receptor function. *Drug and Alcohol Dependence, 70,* 11–15.

McHugh, M. J. (1987). The abuse of volatile substances. *Pediatric Clinics of North America, 34,* 333–340.

Meadows, R., & Verghese, A. (1996). Medical complications of glue sniffing. *Southern Medical Journal, 89*(5), 455–462.

Meyer, J. S., & Quenzer, L. F. (2005). *Psychopharmacology: Drugs, the brain and behavior.* Sunderland, MA: Sinauer Associates.

Muralidharan, K., Rajkumar, R. P., Mulla, U., Kayak, R. B., & Benegal, V. (2008). Baclofen in the management of inhalant withdrawal: A case series. *Primary Care Companion Journal of Clinical Psychiatry, 10*(1), 48–51.

Newell, G. R., Spitz, M. R., & Wilson, M. B. (1988). Nitrite inhalants: Historical perspective. *National Institute on Drug Abuse Research Monogram Series, 83,* 1–14.

Ogden, J. A. (1993). The psychological and neuropsychological assessment of chronic organic solvent neurotoxicity: A case series. *New Zealand Journal of Psychology, 22*(2), 82–93.

Padilla, E. R., Padilla, A. M., Morales, A., Olmedo, E. L., & Ramirez, R. (1979). Inhalant, marijuana, and alcohol abuse among barrio children and adolescents. *International Journal of the Addictions, 14*(7), 945–964.

Reidel, S., Hebert, T., & Bird, P. (1995). *Inhalant abuse: Confronting the growing challenge. Treating Alcohol and Other Drug Abusers in Rural and Frontier Areas* (Technical Assistance Publication Series No. 17). Rockville, MD: Center for Substance Abuse Treatment.

Ridenour, T. A., Bray, B. C., & Cottler, L. B. (2007). Reliability of use, abuse, and dependence of four types of inhalants in adolescents and young adults. *Drug and Alcohol Dependence, 91*, 40–49.

Riegel, A. C., Zapata, A., Shippenberg, T. S., & French, E. D. (2007). The abused inhalant toluene increases dopamine release in the nucleus accumbens by directly stimulating ventral tegmental area neurons. *Neuropsychopharmacology, 32*, 1558–1569.

Ron, M. A. (1986). Volatile substance abuse: A review of possible long-term neurological, intellectual, and psychiatric sequelae. *British Journal of Psychiatry, 148*, 235–246.

Rosenberg, N. L., Grigsby, J., Dreisbach, J., Busenbark, D., & Grigsby, P. (2002). Neuropsychologic impairment and MRI abnormalities associated with chronic solvent abuse. *Clinical Toxicology, 40*(1), 21–34.

Shah, R., Vankar, G. K., & Upadhyaya, H. P. (1999). Phenomenology of gasoline intoxication and withdrawal symptoms among adolescents in India: A case series. *American Journal on Addictions, 8*, 254–257.

Sharp, C. W., & Rosenberg, N. L. (1997). *Substance abuse: A comprehensive textbook*. Baltimore, MD: Williams and Wilkins.

Shen, Y. (2007). Treatment of inhalant dependence with lamotrigine. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 31*, 769–771.

Soderberg, L. S., Chang, L. W., & Barnett, J. B. (1996). Elevated TNF-alpha and inducible nitric oxide production by alveolar macrophages after exposure to a nitrite inhalant. *Journal of Leukocyte Biology, 60*(4), 459–464.

Tenenbein, M., Casiro, O. G., & Seshia, M. M. (1996). Neonatal withdrawal from maternal volatile substance abuse. *Archives of Disease in Childhood—Fetal and Neonatal Edition, 74*(3), F204–F207.

Toutant, C., & Lippmann, S. (1979). Fetal solvents syndrome [Letter to the editor]. *Lancet, 1*(8130), 1356.

Tsushima, W. T., & Towne, W. S. (1977). Effects of paint sniffing on neuropsychological test performance. *Journal of Abnormal Psychology, 86*, 402–407.

Wu, L., & Howard, M. O. (2007). Psychiatric disorders in inhalant users: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Drug and Alcohol Dependence, 88*, 146–155.

Yamakura, T., & Harris, R. A. (2000). Effects of gaseous anesthetics nitrous oxide and xenon on ligand-gated ion channels. *Anesthesiology, 93*, 1095–1101.

Zacny, J. P., Conran, A., Pardo, H., Coalson, D. W., Black, M., Klock, P. A., et al. (1999). Effects of naloxone on nitrous oxide actions in healthy volunteers. *Pain, 83*, 411–418.

Chapter **12**

The Effects and Abuse Potential of GHB: A Pervasive "Club Drug"

Bethany L. Waits, BA, and Paula K. Lundberg-Love, PhD

Gamma-hydroxybutyrate, or GHB, is a widely used drug whose popularity has increased dramatically since its discovery in the late 1960s (Miotto et al., 2001). Once ingested, GHB acts by depressing or downregulating the activity of the central nervous system (CNS), including the brain and the spinal cord, and its effects are similar to other CNS depressants such as alcohol and benzodiazepines (BZD). Although GHB was initially synthesized for use as an intravenous anesthetic for surgical procedures, it has since been marketed therapeutically as a dietary supplement, an anabolic agent, and a drug for the treatment of narcolepsy. Moreover, it has been implicated in preclinical trials for the relief of withdrawal symptoms for several addictive substances, particularly alcohol (Julien, 2005; Levinthal, 2006). In its purest form, GHB is a white powdery substance that is soluble in water (Gonzalez & Nutt, 2005). Although GHB can be obtained as a tablet or a capsule, it is usually distributed, both legally and illegally, as a colorless, odorless, and tasteless solution, and it is typically packaged in small bottles, similar in size to those used for shampoo in hotel rooms (Meyer & Quenzer, 2005). Thus, while oral administration is the most common form of ingestion associated with GHB, the drug is sometimes injected directly into the bloodstream (Jones, 2001).

While GHB was initially developed for medicinal purposes, in recent years it primarily has become a drug of abuse, and is frequently referred to as a "club drug" (Britt & McCance-Katz, 2005). According to Levinthal (2006), a *club drug* is a term that describes a group of drugs utilized primarily by young adults at dance parities, or "raves," which occur at night clubs. Other common "club

drugs" include MDMA (ecstasy), ketamine, Rohypnol, methamphetamine, and LSD (Britt & McCance-Katz, 2005, Levinthal, 2006). The club drugs, including GHB, are readily accessible to purchase at raves and are often combined with other substances to heighten their effects. For instance, one study of GHB users found that 53 percent combined GHB with ecstasy, 50 percent with marijuana, 43 percent with cocaine, 40 percent with amphetamines, and 37 percent with alcohol. The same study reported that 40 percent of the participants used GHB intentionally to increase, or extend, the "high" from other drugs. Overall, 71 percent mentioned using GHB with other substances (Miotto et al., 2001).

In addition to raves and night clubs, GHB can be obtained illegally on the street and on the Internet (Britt & McCance-Katz, 2005; Nicholson & Balster, 2001). Common street names include liquid X, liquid E, grievous bodily harm, G, Georgia home boy, G-rrifick, salty water, scoop, somatomac, gamma 10, gamma g, goop, easy lay, cherry meth, blue nitro, nature's quaalude, GH revitalize, revivarant, renew/trient, and fantasy (Britt & McCance-Katz, 2005; Julien, 2005; Levinthal, 2006). At times, it has also been referred to as "liquid ecstasy" due to its ability to produce a state of euphoria similar to MDMA (Jones, 2001). Recently, GHB has become indirectly available on the Internet in the form of "chemistry kits." These kits contain the biological precursors of GHB, gamma-butyrolactone (GBL) and 1, 4,-butanediol (BD), and instructions that describe how to convert GBL or 1, 4,–BD into GHB (Nicholson & Balster, 2001). Furthermore, according to Jones (2001), several Web sites have GHB "recipes" that illustrate ways to produce GHB in a household kitchen.

The increase in the sale and distribution of GHB has been documented in several studies conducted by the U.S. government. The Drug Abuse Warning Network (DAWN) is a national surveillance agency controlled by the U.S. Substance Abuse Mental Health Service Administration that collects data concerning drug-related visits to hospital emergency rooms throughout the United States. DAWN also documents information concerning drug-related fatalities reported by medical examiners and coroners. According to DAWN, in 1994, 683 emergency departments at various hospitals recorded GHB-related visits. By 2000, this number rose to 4,969 documentations of GHB-related incidents, which is approximately seven times the number reported in 1994 (Meyer & Quenzer, 2005; Rosenthal & Solhkhah, 2005). Due to the increase in information concerning GHB misuse, it is important to discuss the hazardous effects and abuse potential associated with this drug. A review of the current research concerning GHB's history, therapeutic indications, and mechanisms of action in the brain will be discussed. Additionally, the behavioral and physiological effects of GHB as well as its toxicity, withdrawal effects, and approaches to treatment considerations will be addressed in the following sections.

HISTORY

The pharmacological properties of GHB were first reported in 1960 by Dr. Henri Laborit, a French scientist well known for his discovery of Thorazine, the first antipsychotic drug (Meyer & Quenzer, 2005). As a surgeon, Laborit was interested in identifying a compound similar to gamma-aminobutyric acid (GABA), a newly discovered inhibitory neurotransmitter that depressed the activity of the CNS. If such a substance could be identified, Laborit speculated, it would produce sedation and might be utilized as an anesthetic in surgical procedures (Gonzalez & Nutt, 2005; Meyer & Quenzer, 2005). After its synthesis was achieved, it was apparent that GHB was a GABA analog that easily crossed the blood brain barrier (a semipermeable membrane that prevents nonlipid soluble substances from entering the brain). Thus, GHB was capable of directly affecting the brain's biochemistry (Carlson, 2007). Early studies on laboratory animals confirmed Laborit's suspicion that GHB produced sedation and anesthesia. Based on this information, GHB was widely used as an anesthetic in Europe, and is still used for this purpose in some European countries (Meyer & Quenzer, 2005). Recently, however, its use has declined as new studies have revealed that its anesthetic effects are not as therapeutic as other drugs that are currently available, due in part to its lack of analgesic (pain-reducing) properties (Gonzalez & Nutt, 2005).

In the United States, GHB received little attention until the 1980s, when health food stores across the nation began to market the drug as a nutritional supplement and as a sleep aid. Due to manufacturers' claims that GHB had the potential to reduce body fat and increase muscle mass, it became a highly sought after drug (Meyer & Quenzer, 2005). Advertisements stated that GHB had a low risk of toxicity and little danger of dependence, and because of these endorsements, GHB soon became popular among many different groups (Miotto et al., 2001). For instance, its use was particularly prevalent among bodybuilders who used GHB as an allegedly safe alternative to anabolic steroids (Jones, 2001). According to researchers, the most frequent users of GHB, other than bodybuilders, included athletes, gym members, models, and habitual travelers across time zones (Gonzalez & Nutt, 2005). Unfortunately, the distribution and sale of GHB was virtually ignored by the medical community and by public health officials for approximately a decade after it was first released in the United States (Nicholson & Balster, 2001). However, by the 1990s, the Food and Drug Administration (FDA) began to receive numerous reports concerning GHB-related illnesses (Meyer & Quenzer, 2005). For instance, seizures and comas were frequently reported by those using supplements containing GHB (Levinthal, 2006). Additionally, the FDA became

concerned about the dramatic increase in GHB use for sedation and growth enhancement. Many agreed that GHB should be prescribed by a medical doctor to minimize the dangers of over-the-counter misuse (Meyer & Quenzer, 2005).

At the same time, GHB began to be used as a date rape drug, often in combination with alcohol, and such reports began to increase public awareness about its dangerous effects (Julien, 2005; Meyer & Quenzer, 2005; Nicholson & Balster, 2001). Because GHB is a colorless, odorless, and virtually tasteless liquid, it can easily be slipped into alcoholic beverages without it being detected by the drinker (Levinthal, 2006). Like other date-rape drugs (i.e., Rohypnol), GHB's ability to produce unconsciousness, especially in combination with alcohol, has been the primary reason for its use in sexual assault. Moreover, because GHB is rapidly absorbed in the body, unconsciousness persists for several hours after ingestion, making it impossible for a victim to resist the attack (Britt & McCance-Katz, 2005). Although little evidence suggests that GHB enhances sexuality, many believe that it disinhibits sexual inhibitions, making sexual assault easier to accomplish. Furthermore, because GHB is excreted from the body in approximately 12 hours, detection of the drug in blood or urine samples is very difficult.

Thus, the victim's attempts to prosecute are often unsuccessful due to lack of medical evidence (Jones, 2001).

Few published cases exist concerning GHB's alleged role in date rape (Gable, 2004). In one study that investigated cases of drug-related sexual assault, GHB was implicated in only 4.1 percent of the total number evaluated (Nicholson & Balster, 2001). According to Gable (2004), this is not necessarily an indication that GHB is rarely used in date rape. Because few laboratories have the capacity to determine the presence of GHB in the body, it is difficult to establish the true prevalence of GHB involved in cases of sexual assault (Gable, 2004).

These types of events led the FDA to designate the drug as a Schedule I controlled substance in March 2000 (Gable, 2004). As a result, GHB could no longer be obtained over the counter, and it became illegal under FDA regulations to possess or manufacture the drug. However, in 2002, the FDA approved GHB for the treatment of narcolepsy under the trade name Xyrem, and it became a Schedule III controlled substance when utilized for this purpose (Meyer & Quenzer, 2005). According to Nicholson and Balster (2001), GHB's current FDA regulation is similar to tetrahydrocanabinol (THC), or marijuana. For example, THC is classified as a Schedule I controlled substance, but the FDA-approved formulation Marinol, which is used to treat nausea associated with chemotherapy, is designated as a Schedule III drug.

THERAPEUTIC INDICATIONS FOR GHB

As previously mentioned, GHB has been utilized as an anesthetic, a growth promoter, a dietary supplement, a treatment for narcolepsy, and a medication to reduce the adverse effects associated with certain types of drug withdrawal (Hernandez, McDaniel, Costanza, & Hernandez, 1998). Even so, the importance of understanding the biological basis concerning GHB's therapeutic indications cannot be disregarded. For instance, GHB's use as a growth promoter and weight loss supplement was largely supported by research demonstrating its ability to enhance the release of human growth hormone. This effect is a result of GHB's ability to increase stages 3 and 4 sleep, also known as slow-wave sleep (SWS), which is a deeper, more restorative sleep state (Carlson, 2007). Since human growth hormone is primarily released during SWS, GHB indirectly increases its release by amplifying the amount of slow-wave sleep an individual experiences through the night (Gonzalez & Nutt, 2005). Thus, by enhancing the level of growth hormone in the body, it was assumed that GHB ingestion would increase overall muscle mass, similar to anabolic steroids (Rosenthal & Solhkhah, 2005). Although manufacturers of GHB-containing products relied on this research to assert its efficacy (Nicholson & Balster, 2001), there is no scientific evidence supporting GHB's capacity to increase muscle development significantly.

While GHB's efficacy as a growth promoter remains equivocal, it appears to be a useful medication for the treatment of narcolepsy. Narcolepsy is a genetic disorder whose symptoms typically appear during adolescence. It is characterized by excessive daytime sleepiness and a disordered sleep stage architecture with rapid eye movement (REM) sleep occurring at sleep onset (Julien, 2005). One of the most frequent daytime symptoms associated with narcolepsy is cataplexy, a complete loss of muscle tone resulting in paralysis. When a cataplexy attack occurs, a fully awake individual will suddenly fall to the ground unable to move (Carlson, 2007). According to Julien (2005), approximately 60–75 percent of patients with narcolepsy experience cataplexy, and it is usually precipitated by strong emotional reactions such as laughter, anger, surprise, or excitement.

When searching for possible substances to treat narcolepsy, researchers speculated that GHB's ability to promote sleep might eliminate certain symptoms of the disorder, namely, cataplexy attacks. For instance, oral doses of approximately 30–50 mg of GHB produce a state of somnolence, which is readily reversible by external events and is virtually indistinguishable from normal sleep (Nicholson & Balster, 2001). Furthermore, the results of sleep studies evaluating participants' behavior and electroencephalogram (EEG)

patterns suggest that GHB-induced sleep mimics physiological sleep by increasing both SWS and REM sleep (Galloway et al., 1997; Rosenthal & Solhkhah, 2005). GHB's ability to produce a normal sleep state underlies its effectiveness in alleviating the symptoms of narcolepsy. Indeed, when GHB is taken at night, it consolidates sleep, restores sleep patterns to normal levels, and reduces hypnagoic hallucinations (i.e., vivid dreams that occur at sleep onset). Also, GHB improves daytime symptoms by reducing cataplexy attacks and daytime sleepiness (Nicholson & Balster, 2001). To date, the only FDA-approved therapeutic indication of GHB is for the treatment of cataplexy attacks associated with narcolepsy. As mentioned earlier, it is distributed under the trade name, Xyrem, and is manufactured by Jazz Pharmaceuticals (Rosenthal & Solhkhah, 2005).

Currently, researchers are investigating GHB's effectiveness in treating the withdrawal symptoms of several addictive substances, specifically alcohol, opiates, and cocaine (Gonzalez & Nutt, 2005). Support for this hypothesis has been provided by Gallimberti, Schifano, Forza, and Miconi (1994), who found GHB reduced alcohol withdrawal symptoms, including tremors and seizures, in laboratory rats. In clinical studies, GHB has been shown to diminish depression, nausea, and anxiety among alcoholics in detoxification programs (Jones, 2001). Furthermore, the long-term usefulness of GHB among alcoholics in detoxification suggests that the drug significantly increases the number of abstinent days experienced by these individuals (Galloway et al., 1997). GHB also has been found to ease the abstinence syndrome produced by opiate withdrawal. Researchers suspect that GHB may stimulate the release of endogenous opiates, thereby relieving the symptoms associated with opiate abstinence (Nicholson & Balster, 2001). Additionally, one preclinical study conducted by Fattore, Martellotta, Cossu, and Fratta (2000) found data to support the use of GHB for the treatment of cocaine addiction. However, no other studies have replicated these findings. While the use of GHB for the treatment of various withdrawal symptoms is promising, it has not yet been approved by the FDA for the treatment of either alcohol or opiate withdrawal. Therefore, more research must be conducted to determine whether the treatment of certain types of drug withdrawal is, indeed, a therapeutic indication of GHB (Addolorato, Caputo, Capristo, Stefanini, & Gasbarrini, 2000).

MECHANISMS OF ACTION

According to Meyer and Quenzer (2005), two competing hypotheses exist concerning GHB's mechanism of action, or sites of action, in the brain. The first theory states that GHB is an agonist at the GABA-B receptor. An agonist is any drug that binds to a specific receptor and either facilitates or enhances

the naturally occurring effects of that particular receptor. As mentioned previously, GABA, or gamma-aminobutryic acid, is an inhibitory neurotransmitter that depresses, or downregulates, the activity of the central nervous system (Meyer & Quenzer, 2005). Thus, a GABA agonist is any substance that activates GABA receptors causing a CNS-depressant effect. There are two types of GABA receptors in the human brain: GABA-A and GABA-B receptors. Many CNS-depressants (i.e., alcohol, benzodiazepines, and barbiturates) stimulate certain subunits on the GABA-A receptor, and cause behavioral effects such as sedation and relief from anxiety. The GABA-B receptor, when activated, leads to similar behavioral effects, and it is hypothesized that GHB acts on this receptor. Evidence supporting this assumption is based on research showing that the behavioral and physiological effects of GHB in animals can be inhibited by GABA-B receptor antagonists. GHB's anesthetic properties and ability to produce sedation may be the result of its GABA-B receptor agonist properties (Miotto et al., 2001; Meyer & Quenzer, 2005).

The second theory concerning GHB's mechanism of action states that its effects are mediated by specific GHB receptors found in the human brain (Kemmel et al., 1998; Meyer & Quenzer, 2005). This hypothesis was first introduced by Roth and Giarman, who demonstrated that GHB was an endogenous substance and, therefore, might act as a neurotransmitter or neuromodulator in various brain regions (Galloway et al., 1997). Current research indicates that GHB fulfills many of the characteristics associated with neurotransmitters or neuromodulators. For instance, it is released in a calcium-dependent manner. It possesses a distinct reuptake system that transports it into synaptic vesicles. It has specific enzymes that remove it from the synaptic cleft after it is released, and, as previously discussed, it has its own particular receptor sites throughout the brain (Meyer & Quenzer, 2005; Nicholson & Balster, 2001). Furthermore, in rats, monkeys, and humans, GHB-binding receptor sites have been identified in the hippocampus, caudate nucleus, cerebral cortex, and cerebellum. In addition, certain substances have been synthesized in laboratories that specifically bind to GHB receptors. One such substance, NCS-382, is a selective antagonist at GHB receptors, blocking GHB's effects in the brain (Meyer & Quenzer, 2005). This research, along with studies supporting GHB's role as a GABA-B receptor agonist, provide a strong scientific foundation in the understanding of GHB's mechanisms of action.

BEHAVIORAL AND PHYSIOLOGICAL EFFECTS

The behavioral and physiological effects associated with GHB are largely dependent on the amount of the drug an individual ingests. When adminis-

tered for therapeutic purposes, typical doses of GHB range from 15 to 30 mg. Unfortunately, GHB is frequently misused as a recreational drug, and regular users have been known to ingest as much as 57 to 71 mg (Nicholson & Balster, 2001). According to Jones (2001), addiction to GHB is highly probable if an individual consumes between 30 and 50 mg on a daily basis. Therefore, like many other substances, GHB's therapeutic effectiveness and potential for abuse is influenced by the dosage of the drug that a person takes.

Although GHB results in many behavioral and physiological effects, it is typically abused for its ability to induce euphoria, or an "out-of-body" high (Levinthal, 2006). Many researchers believe that this "euphoric" effect underlies its potential for abuse (Julien, 2005). Experiments with laboratory animals, using conditioned place preference, have supported the hypothesis that GHB has reinforcing effects. Conditioned place preference, or CPP, is an experimental procedure in which an animal is given a drug, in this case GHB, and is then placed in a specific compartment to create an association between that environment and the effects of the drug. The animal is also given a placebo and placed in a different compartment. After this place-conditioning is established, researchers allow the animal to have access to both compartments at the same time, and measure the amount of time the animal spends in each environment. Thus, if the animal finds the drug rewarding, it spends the majority of its time in the compartment associated with the drug (Meyer & Quenzer, 2005). In a study by Martellotta, Fattore, Cossu, and Fratta (1997), GHB was shown to induce a CPP, meaning that the laboratory animal preferred the compartment paired with GHB administration to the compartment paired with the placebo. However, it took approximately six drug exposures for a CPP to occur, which is significantly more than other addictive drugs, such as cocaine and the opiates, which produce a CPP after two exposures. This finding suggests that while GHB can be classified as a reinforcer, its effects are much weaker than other highly rewarding drugs. Moreover, those that abuse GHB typically believe that it acts as an aphrodisiac, and that it has the ability to increase or enhance sexual experiences (Julien, 2005; Rosenthal & Solhkhah, 2005). While other substances, like alcohol, have been reported to heighten sexuality, there is no evidence that GHB, or other depressants, aid in sexual performance (Julien, 2005).

When a person consumes GHB, it takes approximately 5 to 15 minutes for it to cross the blood brain barrier, and affect behavior. Theses effects can last between four and seven hours. However, in some cases, users report dizziness for up to two weeks after GHB ingestion (Galloway et al., 1997). Small doses of GHB, 10 to 20 mg or less, typically produce muscle relaxation and relief from anxiety. Additionally, disinhibition, sociability, and light inebriation have been

reported. Those using GHB at low doses often describe an alcohol-like experience, with behavioral effects similar to those experienced after drinking two alcoholic beverages (Gable, 2004). Several clinical studies suggest that GHB at low doses has no effect on attention, vigilance, alertness, short-term memory, or psychomotor skills. The only adverse symptoms mentioned by participants included slight dizziness (Nicholson & Balster, 2001). At moderate doses, between 20 and 40 mg, GHB's behavioral and physiological effects are accentuated. For instance, moderate doses of GHB generally induce sleep and produce euphoria (Rosenthal & Solhkhah, 2005). In addition, other common symptoms include physical disequilibrium, grogginess, tactile sensitivity, increased gag reflex, nausea, and vomiting (Gable, 2004; Gonzalez & Nutt, 2005). According to Nicholson and Balster (2001), at 30 to 40 mg, users may experience mild hypothermia, loss of peripheral vision, confusion, agitation, enuresis, myoclonic jerks, tremors, ataxia, aggression, and hallucinations. Furthermore, as doses increase above 40 or 50 mg, users often become unconscious and may experience severe respiratory depression (Levinthal, 2006). Although high doses of GHB are not generally associated with seizures, some users have reported the occurrence of tonic-clonic (i.e., grand mal) seizures after ingestion of 60 to 70 mg of GHB. Furthermore, as doses increase above 70 mg, individuals often experience a state of unarousable sleep or coma, which can last from approximately one to five hours. Researchers speculate that a lethal dose of GHB is approximately 5–15 times higher than a dose producing unconsciousness (Nicholson & Balster, 2001). Finally, in several rare cases, high doses of GHB, over a period of time, have led to Wernicke-Korsakoff syndrome, which is characterized by a deficiency in thiamine and is often observed in alcoholics (Friedman, Westlake, & Furman, 1996; Gonzalez & Nutt, 2005).

In a noteworthy study conducted by Miotto and colleagues (2001), GHB users were interviewed about the subjective effects of the drug. Of the 42 participants in the study, 75–100 percent reported euphoria, feelings of happiness, increased sexuality, increased well-being, heightened sense of touch, relaxation, talkativeness, tranquility, disinhibition, pleasant drowsiness, and optimism after using GHB. Furthermore, 50–74 percent experienced increased intensity of orgasms, increased energy, giddiness, sensitivity to sound, sweatiness, and loss of consciousness. A smaller percentage, 25–49 percent mentioned symptoms of nausea, auditory and visual hallucinations, headaches, frequent urination, and vomiting. Finally, 1–24 percent of those who participated experienced amnesia or seizures. This research suggests that GHB has the potential to cause serious health effects in individuals who consume large quantities of the substance. More information regarding GHB's toxic effects will be discussed in the following section.

TOXICITY

The term *toxicity* refers to a chemical substance's ability to induce functional or anatomical damage to a living organism. While the toxic effects of many drugs have been documented in the medical literature, GHB's toxicity has been more difficult to determine. This is largely due to the fact that certain amounts of GHB naturally occur in the human brain. Also, GHB is rapidly metabolized after ingestion, giving researchers less time to determine its biological effects (Gable, 2004). Because many physicians have little experience with GHB toxicity, the incidence rates of GHB use are difficult to obtain. Only a few laboratories have the capability to detect the presence of GHB in the body, and clinicians can rarely determine actual blood or tissue levels of the drug (Hernandez et al., 1998; Nicholson & Balster, 2001). Moreover, if a laboratory does have the ability to test for GHB, only a specific type of urinalysis, which tests for the presence of one drug, can determine if GHB was ingested by the individual (Gable, 2004). According to Nicholson and Balster (2001), this makes the evaluation of GHB's toxicity very difficult, because the majority of cases involving hospitalization involve the co-ingestion of other substances, such as alcohol.

While only a limited amount of information in the medical literature exists concerning GHB toxicity, evidence obtained from users, clubs, health food stores, gyms, and the street suggest that the prevalence of GHB overdose is much higher than previously suspected (Hernandez et al., 1998). For example, among one sample of first-time GHB users, approximately 53 percent overdosed on the substance (Degenhardt, Darke, & Dillon, 2003). This percentage increased to 75 percent among users who had ingested GHB on more than 15 occasions (Gonzalez & Nutt, 2005). Typically, an overdose from GHB occurs if an individual takes multiple doses over a short period of time, if there is a higher concentration of GHB in the bottle than expected, or if the drug is taken in combination with alcohol or other sedative drugs (Meyer & Quenzer, 2005).

GHB toxicity, or overdose, can lead to a wide variety of medical conditions, which in some cases, may be life-threatening (Hernandez et al., 1998). Since GHB is a depressant, it is not surprising that the most common manifestations of toxicity involve cardiac, respiratory, and central nervous system depression (Gonzalez & Nutt, 2005). Indeed, a nonfatal overdose of GHB is often characterized by respiratory depression and an unarousable coma. According to Gonzalez and Nutt (2005), unconsciousness may be so unexpected that the patient may be injured as a result of the sudden loss of muscle tone. Although individuals typically regain consciousness after five hours, if other sedatives, such as alcohol, have been taken in combination with GHB, an individual may remain in a comatose state for up to eight days (Nicholson & Balster, 2001).

However, it is not uncommon for patients spontaneously to regain consciousness after several hours. As they become more alert, many report feelings of extreme agitation and combativeness (Rosenthal & Solhkhah, 2005). Clearly, respiratory depression and coma are the most common consequences of GHB overdose; other symptoms have been reported by patients. These include stupor, delirium, nausea, vomiting, vertical nystagmus, hypotension, tachycardia, tremor, anxiety, ataxia, jerking movements, bradycardia, disorientation, and muscle stiffening (Gonzalez & Nutt, 2005; Julien, 2005; Miotto et al., 2001). Additionally, seizures have been reported in rare cases (Meyer & Quenzer, 2005).

As mentioned, the toxic effects of GHB are greatly potentiated when combined with other substances, such as alcohol, opiates, barbiturates, and benzodiazepines (Miotto et al., 2001). Unfortunately, many individuals that abuse GHB do so in order to heighten the effects of other abusive drugs. For instance, the euphoric effects produced by alcohol and some stimulants are enhanced when combined with GHB (Nicholson & Balster, 2001). Additionally, when taken with other club drugs such as ketamine and ecstasy, GHB can reduce several of the adverse effects, such as teeth grinding and jaw-clenching, while simultaneously increasing euphoria and disinhibition (Rosenthal & Solhkhah, 2005). Thus, it is not surprising that many users who overdose on GHB are also under the influence of other substances, often exacerbating GHB's toxicity. Indeed, an increase in the adverse reactions to GHB, when combined with other drugs, has been documented in several case reports of overdose victims. One group of researchers evaluated 88 cases of GHB overdose reported by the emergency room of San Francisco General Hospital. Of these cases, approximately 50 percent involved co-ingestion of one or more additional substances other than GHB. Furthermore, the most common substances taken with GHB included alcohol, amphetamine, and MDMA (Chin, Sporer, Cullison, Dyer, & Wu, 1998). While it is rare that an overdose of GHB will lead to death, the majority of GHB-related fatalities have been associated with other drugs of abuse. One death, reported by Ferrara, Tedeschi, Frison, and Rossi (1995), involved ingestion of GHB in combination with heroin. However, according to Gable (2004), GHB toxicity is the most life-threatening when mixed with alcohol.

WITHDRAWAL EFFECTS

Clinical evidence suggests that chronic use of GHB can produce severe withdrawal symptoms among habitual users (McDonough, Kennedy, Glasper, & Bearn, 2004). Those that consume regular doses of GHB at two- to four-hour intervals throughout the day are thought to be the most susceptible to

experiencing a withdrawal syndrome after discontinuation. Furthermore, many of the symptoms associated with GHB withdrawal are similar to the withdrawal symptoms of other CNS depressants (i.e., alcohol, barbiturates, and benzodiazepine). If an individual is also dependent on a substance other than GHB, the withdrawal syndrome associated with GHB may be more complicated (Rosenthal & Solhkhah, 2005). In general, an individual experiences withdrawal symptoms several hours after the last dose of GHB. However, these symptoms tend to worsen and typically last from 3 to 15 days (Julien, 2005). According to McDonough et al. (2004), the mean duration for GHB withdrawal is approximately 9 days, which is very similar to that of alcohol and other sedative-hypnotics. Several reports have characterized the withdrawal syndrome associated with GHB to occur in two phases. In the first phase (2–12 hours after the last dose), milder symptoms such as insomnia, tremor, anxiety, increased heart rate, confusion, and vomiting have been documented (Britt & McCance-Katz, 2005). According to Meyer and Quenzer (2005), the most common withdrawal symptoms initially seen in patients are insomnia, anxiety, and tremors. Additional withdrawal symptoms commonly seen during the first 12 hours include feelings of doom, sweating, craving for the drug, mood lability, abdominal cramping, heart palpitations, diaphoresis, tachycardia, and miosis (Gonzalez & Nutt, 2005; Jones, 2001; Rosenthal & Solhkhah, 2005). The second phase begins 2 to 3 days after the last dose of GHB, when symptoms become more severe (Britt & McCance-Katz, 2005). Disorientation, fever, horizontal nystagmus, severe agitation, confusion, seizures, paranoia, delusional thinking, and auditory or visual hallucinations have been reported among patients (Rosenthal & Solhkhah, 2005). In some severe cases, full-blown psychosis may develop, and it is not uncommon for many patients to experience delirium during withdrawal (Britt & McCance-Katz, 2005; Meyer & Quenzer, 2005). Indeed, patients who were heavy users prior to cessation are more likely to have withdrawal delirium than those who used the drug less frequently (McDonough et al., 2004). In a meta-analysis conducted by McDonough and colleagues (2004), 38 cases of GHB withdrawal were reviewed, and over half of the patients reported sustained delirium after discontinuation of GHB. In another study, withdrawal delirium and agitation were the most long-lasting symptoms and continued for approximately two weeks after the last dose of GHB (Miotto et al., 2001).

TREATMENT CONSIDERATIONS

In cases of GHB overdose, the most common form of treatment involves supportive medical care, often in the form of cardiovascular and respiratory assistance (Julien, 2005). However, according to Gonzalez and Nutt (2005),

for a patient to receive the best possible care, it is important to discover the amount of the drug that was ingested, as well as any other substances currently in the body. As mentioned, GHB toxicity often leads to unconsciousness and sometimes to unarousable coma. Respiratory support is often mandatory, but the majority of patients typically regain consciousness several hours after GHB overdose, and many are well enough to be discharged after waking. In a study conduced by Chin et al. (1998), similar findings were reported with approximately 13 percent of overdose patients requiring intubation. However, the majority of patients recovered within five hours and were discharged from the emergency department.

Although no specific antidote has been identified for the treatment of GHB overdose, several substances have been used in combination with cardiovascular and respiratory support (Julien, 2005). For instance, naloxone, or Narcan, a drug typically used to counter the effects of opiate overdose, has been utilized to reverse the comatose state associated with GHB toxicity (Jones, 2001). Other drugs such as physostigmine or neostigmine, acetylcholinesterase inhibitors, also have been effective in reviving patients. However, evidence suggests that using these drugs may induce more serious side effects (i.e., seizures). The benzodiazepine antagonist, Romazicon, used for the treatment of benzodiazepine toxicity, also has been administered to patients. Unfortunately, this drug has not proven effective in reversing GHB-related coma (Gonzalez & Nutt, 2005). Clearly, more research is required in order to establish an effective treatment for GHB overdose.

As with overdose, the withdrawal syndrome associated with GHB can be difficult to treat (Gonzalez & Nutt, 2005). According to McDonough et al. (2004), GHB detoxification is usually unplanned, and medical assistance is given only after patients are admitted to emergency departments in a state of crisis. Additionally, users who consume large doses of GHB, at short intervals, are often refractory to treatment during withdrawal, making it difficult for medical professionals to stabilize their symptoms. Even after the first two weeks of detoxification, life-threatening withdrawal symptoms may occur. Therefore, it is crucial for individuals to remain in the hospital for several weeks. If patients are allowed to return home too quickly, there is a high risk that they will relapse and recommence GHB use (Gonzalez & Nutt, 2005).

Several drugs have been implicated in the treatment of GHB withdrawal. These typically include benzodiazepines, mood stabilizers, antipsychotics, and at times, GHB substitutes. Usually, a benzodiazepine is the first drug of choice to suppress withdrawal symptoms (Gonzalez & Nutt, 2005). The most common BZDs prescribed to patients include Valium and Ativan (Glasper, McDonough, & Bearn, 2005). During the first few hours after admission, medical professionals monitor the individual and determine the amount of

BZD required to ease her symptoms. Once this dose has been established, it is given to the patient several times a day for approximately one to two weeks. After the second week, daily doses of BZD are reduced and eventually discontinued (Gonzalez & Nutt, 2005). Although BZDs are typically effective in treating symptoms, in extreme cases, an intravenous anesthetic, such as Propofol, may be administered to those that are unresponsive to high doses of benzodiazepines (Gonzalez & Nutt, 2005). Moreover, if a patient experiences withdrawal delirium, a mood stabilizer, such as Neurontin, is often combined with the BZD to decrease agitation (Miotto et al., 2001).

In addition to mood stabilizers, antipsychotics can be used in conjunction with BZDs to reduce the psychotic symptoms associated with GHB withdrawal (i.e., hallucinations, delusions, and paranoia) (Glasper et al., 2005). For instance, low doses of Haldol, an older antipsychotic, have been successful in alleviating several of these symptoms (Jones, 2001). Additional antipsychotics, such as Zyprexa and Clozaril, are effective in treating the hallucinations and paranoia often seen in patients undergoing withdrawal (Rosenthal & Solhkhah, 2005). Finally, a new alternative in the treatment of GHB withdrawal involves the use of Xyrem, a form of GHB used for the treatment of narcolepsy. During inpatient detoxification, the appropriate dose of Xyrem is given instead of benzodiazepines and then is slowly reduced based on the patient's needs. While this new treatment method is rarely used, it is suspected that its popularity may increase in the coming years (Gonzalez & Nutt, 2005).

CONCLUSIONS

GHB is a white powdery substance that is soluble in water, and is usually distributed, both legally and illegally, as a colorless, odorless, and tasteless solution. Although GHB has been used therapeutically as an anesthetic, a growth promoter, a dietary supplement, a treatment for narcolepsy, and a medication to reduce the adverse effects associated with withdrawal, in recent years it has primarily become a drug of abuse and is commonly referred to as a "club drug." Other common club drugs include MDMA (ecstasy), ketamine, Rohypnol, methamphetamine, and LSD. These drugs, along with GHB, are readily accessible for purchase at "raves" (i.e., dance parties) and are often combined with other substances to heighten their effects. Moreover, GHB can be obtained illegally on the street under the following names: liquid X, liquid E, grievous bodily harm, G, Georgia home boy, G-rrifick, salty water, scoop, gamma 10, gamma g, easy lay, and cherry meth. Recently, GHB has become indirectly available on the Internet in the form of "chemistry kits," which contain the biological precursors of GHB with instructions that describe how to convert the

precursors into the drug. These preceding events, along with GHB's alleged use in sexual assault, led the FDA to designate GHB as a Schedule I controlled substance in March 2000.

According to Meyer and Quenzer (2005), two competing hypotheses exist concerning GHB's mechanism of action in the brain. The first theory states that GHB is a GABA-B agonist, meaning that the drug activates GABA receptors causing a CNS-depressant effect. The second theory states that GHB's effects are mediated by specific GHB receptors found in the human brain. Current research indicates that GHB fulfills many of the characteristics associated with neurotransmitters or neuromodulators. These include its release in a calcium-dependent manner, its distinct reuptake system, and its own endogenous receptor system in the brain.

Furthermore, when an individual consumes GHB, it takes approximately 5 to 15 minutes for it to cross the blood brain barrier and affect behavior. Small doses of GHB typically produce muscle relaxation and relief from anxiety. Moderate doses of GHB induce sleep and euphoria, as well as physical disequilibrium, grogginess, tactile sensitivity, increased gag reflex, nausea, and vomiting. When doses increase above 40 or 50 mg, users often become unconscious and may experience severe respiratory depression. At toxic levels, GHB can produce unarousable coma and seizures. Other symptoms involved in GHB overdose include stupor, delirium, nausea, vomiting, vertical nystagmus, hypotension, tachycardia, tremor, anxiety, ataxia, jerking movements, bradycardia, disorientation, and muscle stiffening. Furthermore, when a patient is admitted to the hospital for GHB overdose, the most common form of treatment involves supportive medical care (i.e., cardiovascular and respiratory assistance). Drugs such as naloxone, physostigmine, neostigmine, and Romazicon have been given to patients in combination with supportive medical care to relieve the toxic effects of GHB.

Individuals that consume regular doses of GHB at two- to four-hour intervals throughout the day are thought to be the most susceptible to experiencing withdrawal after discontinuation. The most common withdrawal symptoms initially seen in patients include insomnia, anxiety, and tremors. As the withdrawal syndrome progresses, feelings of doom, sweating, craving for the drug, mood lability, abdominal cramping, heart palpitations, diaphoresis, tachycardia, and miosis occur. The most severe withdrawal symptoms that patients are likely to experience can involve fever, horizontal nystagmus, severe agitation, confusion, seizures, paranoia, delusional thinking, and auditory or visual hallucinations. Several drugs have been implicated in the treatment of GHB withdrawal. These typically consist of benzodiazepines, mood stabilizers, antipsychotics, and GHB substitutes. For instance, Valium and Ativan are the most common

BZDs prescribed to those in withdrawal. If a patient experiences withdrawal delirium, a mood stabilizer, such as Neurontin, is often combined with the BZD to decrease agitation. Additionally, antipsychotics can be used to reduce the psychotic symptoms associated with GHB withdrawal, and GHB substitutes (i.e., Xyrem) may be given instead of BZDs to reduce symptoms.

REFERENCES

Addolorato, G., Caputo, F., Capristo, E., Stefanini, G., & Gasbarrini. (2000). Gamma-hydroxybutyric acid efficacy, potential abuse, and dependence in the treatment of alcohol addiction. *Alcohol, 20,* 217–222.

Britt, G. C., & McCance-Katz, E. F. (2005). A brief overview of the clinical pharmacology of "club drugs." *Substance Use and Misuse, 40,* 1189–1201.

Carlson, N. R. (2007). *Physiology of behavior.* Boston: Allyn and Bacon.

Chin, R. L., Sporer, K. A., Cullison, B., Dyer, J. E., & Wu, T. D. (1998). Clinical course of gamma-hydroxybutyrate overdose. *Annals of Emergency Medicine, 31,* 716–722.

Degenhardt, L., Darke, S., & Dillon, P. (2003). The prevalence and correlates of gamma-hydroxybutyrate (GHB) overdose among Australian users. *Addiction, 98,* 199–204.

Fattore, L., Martellotta, M. C., Cossu, G., & Fratta, W. (2000). Gamma-hydroxybutyric acid: An evaluation of its rewarding properties in rats and mice. *Alcohol, 20,* 247–256.

Ferrara, S. D., Tedeschi, L., Frison, G., & Rossi, A. (1995). Fatality due to gamma-hydroxybutyric acid (GHB) and heroin intoxication. *Journal of Forensic Science, 40,* 501–504.

Friedman, J., Westlake, R., & Furman, M. (1996). "Grievous bodily harm": Gamma hydroxybutyrate abuse leading to the Wernicke-Korsakoff syndrome. *Neurology, 46,* 469–471.

Gable, R. S. (2004). Acute toxic effects of club drugs. *Journal of Psychoactive Drugs, 36,* 303–313.

Gallimberti, L., Schifano, F., Forza, G., & Miconi, L. (1994). Clinical efficacy of gamma-hydroxybutyric acid in treatment of opiate withdrawal. *European Archives of Psychiatry and Clinical Neuroscience, 244,* 113–114.

Galloway, G. P., Frederick, S. L., Staggers, F. E., Gonzales, M., Stalcup, S. A., & Smith, D. E. (1997). Gamma-hydroxybutyrate: An emerging drug of abuse that causes physical dependence. *Addiction, 92,* 89–96.

Glasper, A., McDonough, M., & Bearn, J. (2005). Within-patient variability in clinical presentation of gamma-hydroxybutyrate withdrawal: A case report. *European Addiction Research, 11,* 152–154.

Gonzalez, A., & Nutt, D. J. (2005). Gamma hydroxyl butyrate abuse and dependency. *Journal of Psychopharmacology, 19,* 195–204.

Hernandez, M., McDaniel, C. H., Costanza, C. D., & Hernandez, O. J. (1998). GHB-induced delirium: A case report and review of the literature on gamma hydroxybutyric acid. *American Journal of Alcohol Abuse, 24,* 179–183.

Jones, C. (2001). Suspicious death related to gamma-hydroxybutyrate (GHB) toxicity. *Journal of Clinical Forensic Medicine, 8,* 74–76.

Julien, R. M. (2005). *A primer of drug action.* New York: Worth Publishing.

Kemmel, V., Taleb, O., Perard, A., Andriamampandry, C., Siffert, J. C., Mark, J., et al. (1998). Neurochemical and electrophysiological evidence for the existence of a functional gamma-hydroxybutyrate system in NCB-20 neurons. *Neuroscience, 86,* 989–1000.

Levinthal, C. F. (2006). *Drugs, behavior, and modern society.* Boston: Allyn and Bacon.

Martellotta, M. C., Fattore, L., Cossu, G., & Fratta, W. (1997). Rewarding properties of gamma-hydroxybutyric acid: An evaluation through place preference paradigm. *Psychopharmacology, 132,* 1–5.

McDonough, M., Kennedy, N., Glasper, A., & Bearn, J. (2004). Clinical features and management of gamma-hydroxybutyrate (GHB) withdrawal: A review. *Drug and Alcohol Dependence, 75,* 3–9.

Meyer, J. S., & Quenzer, L. F. (2005). *Psychopharmacology: Drugs, the brain and behavior.* Sunderland, MA: Sinauer Associates.

Miotto, K., Darakjian, J., Basch, J., Murray, S., Zogg, J., & Rawson, R. (2001). Gamma-hydroxybutyric acid: Patterns of use, effects and withdrawal. *American Journal on Addictions, 10,* 232–241.

Nicholson, K. L., & Balster, R. L. (2001). GHB: A new and novel drug of abuse. *Drug and Alcohol Dependence, 63,* 1–22.

Rosenthal, R. N., & Solhkhah, R. (2005). Club drugs. In D. A. Ciraulo & H. R. Kranzler (Eds.), *Clinical manual of addiction psychopharmacology* (pp. 243–267). Washington, DC: American Psychological Publishing.

This page intentionally left blank

Chapter **13**

HIV and Addiction from an African Perspective: Making the Link

Mary Theresa Webb, PhD with Donald Omonge, BA

Since U.S. president George W. Bush announced the first AIDS awareness day in 2005, billions of U.S. dollars (President's Emergency Plan for AIDS Relief [PEPFAR]) and euro funds have gone into relieving the suffering of millions of Africans infected by the HIV virus and AIDS. African researchers and leaders came together in a conference in Dar Es Salaam, Tanzania, hosted by USG agencies through PEPFAR on August 29–31, 2005, to discuss alcohol/HIV risk behaviors and transmission in Africa. In a media presentation, Senior Technical Advisor and Prevention Technical Team Leader, Office of U.S. Global AIDS Coordinator Caroline A. Ryan, MD, MPH, stated that only 3 percent of all PEPFAR funding in African countries, notably Botswana and Rwanda, has supported programs that dealt with these alcohol-abusive behaviors that effect the transmission of the HIV virus through sexual contact.

All of the funded programs at that time were engaged in community forums and trainings to raise awareness. In Botswana, the community action forums had three components: the formation of a Citizen's Action Committee on Alcohol, the Botswana Alcohol AIDS Project, and the Botswana Movement against Destructive Decision (BMADD). BMADD held trainings for the health care sector on the issues of family dysfunction, co-dependency, alcohol use and abuse, depression, boundaries, grief, loss and bereavement, and self-worth/self-identity. In Namibia, Health Communication, in partner with Johns Hopkins University, held a series of community action forums in 38 sites across the country. Community leaders in all of the six areas surveyed for feedback noted that alcohol abuse combined with unemployment and poverty were

the main problems contributing to the spread of the HIV virus. Rwanda also held community discussions around the same time period.

In 2006, a year after this conference, the Global Outreach in Addiction Leadership and Learning (GOAL) Project, located in Pittsburgh, Pennsylvania, received the first in three rounds of a New Partner's Initiative in PEPFAR funding for supporting the Kenya-based Substance Abuse Rehabilitation and HIV/AIDS (SARAH) Network of grassroots projects to help in the prevention of the spread of the HIV virus by educating communities, teachers, clergy, probation officers, and others in the disease of addiction and its role in the spread of the virus. The Kenya SARAH Network is highlighted here.

MAKING THE LINK

In the World Health report of 2002, cited in the Resolution of the Fifty-eighth World Health Assembly (2006), the resolution pointed out that 4 percent of disease and 3.2 percent of all deaths are attributed to alcohol, and alcohol is the biggest risk factor in developing countries. The resolution called on member states to recognize the threats to public health and to implement programs to reduce the harmful use of alcohol. Presentations a year earlier at the African Regional Conference (sponsored by USG agencies, 2005), had pointed out the consequences of alcohol consumption for HIV transmission and progression: (1) alcohol consumption is a risk factor; (2) misuse of alcohol affects adherence to HIV medication regimens; and (3) there is increased risk because of the interaction between alcohol, noninjection and injection drug use. In Eastern and Southern Africa, estimated alcohol consumption per person aged 15 years or older is 16.6 liters with 43 percent of the entire population within this age bracket being drinkers (WHO, 2004).

Prior to the resolution from the World Health Report of 2002, injecting heroin was widely known to be a major risk factor in the spread of the HIV virus. Since the rise of independent autonomous African countries and the development of these countries in the twentieth century, alcohol consumption had became a greater risk than injecting heroin because alcohol use and misuse had been on the rise among both young and old (Odejide, 2006). Dr. Flisher at the University in Cape Town in South Africa, clearly defined the causes of harmful use: (1) personal factors, such as low self-esteem, depression, sensation seeking and genetics; (2) interpersonal factors, such as peer pressure, relationship problems, negative responses of health care providers, and other environmental influences; and (3) cultural factors and structural factors, such as poverty (Flisher, 2006). Dr. Kasirye, representing the Uganda Youth Development League, Kampala, Uganda, in the same journal (CRISA, 2006, p. 27) stressed that drinking alcohol increases the risk of being infected with

the HIV virus and that those who are under the influence of alcohol are less likely to be concerned about the use of condoms. Even domestic violence is on the increase in areas with high alcohol consumption.

Africa is second only to Eastern Europe in the level of absolute alcohol consumed, and eastern and southern African countries have the highest consumption per drinker in the world. Burundi, Nigeria, South Africa, Swaziland, and Uganda have the highest recorded per capita consumption (Ashley, Levine, & Needle, 2006, p. 193; Obot, 2006, p. 19). These researchers arrived at the consensus that interventions to lessen the spread of the AIDS virus must also target reducing alcohol consumption. They point out that many studies link alcohol use and sexual arousal, acting on that arousal, as well as condom nonuse increases the risk of HIV infection. Alcohol consumption also reduces adherence to antiviral medications and Tuberculosis medications as well as compliance to treatment (Ashley et al., 2006, p. 193). The same authors raised the issue of gender-based violence (GBV) or violence against women in this journal article (p. 195). The UN Population Fund (UNFPA) Gender Theme Group defines GBV as "violence involving men and women, in which the female is usually the victim; and which is derived from unequal power relationships between men and women. It includes, but is not limited to, physical, sexual and psychological harm" (UNFPA, [defined by United Nations Population Fund Gender Theme Group] 1998; Ashley et al., p. 195). In most African countries, GBV is about 40–60 percent. This explains the alarming rates of the rise of the HIV virus among women and children in Sub-Saharan African countries. This inequality of power was made vivid in a skit put on by a Kenyan team training area clergy in Kakamaga, Kenya, one of the towns in western Kenya. In the skit, a husband and wife were discussing what to do about a son's drinking and drug abuse. The husband sat in a chair while the wife sat on the floor at his feet, as it is the custom for the female to sit lower than the male. A peer-based support group, formed for women whose husbands drink too much in the Kisumu District of Kenya, meets in a mud hut. When this author visited with them, she asked the 15 young mothers and wives present how many had been tested for HIV/AIDS. Three women raised their hands. She asked the others what was keeping them from being tested. "We would be beaten," one member said. The rest nodded their heads in agreement. If any of these women do manage to sneak away to be tested and their tests prove positive, they will probably be shunned by their families and fellow villagers.

RECOMMENDATIONS

According to Ashley et al. (2006), the key step to addressing reducing the spread of HIV/AIDS is to reduce alcohol abuse. They recommend: (1) coalition building, (2) community empowerment, (3) professional capacity building

and evidence-based treatment accessibility, (4) national policies and interventions, and (5) changing alcohol-imbibing cultures and behaviors (p. 196). They advocate for changing socially acceptable African male drinking patterns, training public health officials, and school-based interventions. They recommend cross-training in HIV and addiction and voluntary counseling and testing (VCT) (Ashley et al., 2006).

In addition to Botswana, Nambia, and Rwanda, in Uganda churches partnering with the government and NGOS are encouraging alcohol abstinence as a way forward in reducing the spread of the HIV virus. This chapter will highlight a Kenya prevention model, the SARAH Network, funded by the GOAL Project under President Bush's New Partner's Initiative under PEPFAR, and a treatment model in Egypt that is having an impact in that African country.

THE SARAH NETWORK MODEL (KENYA)

In 2005, the Anglican Bishop for Western Maseno Province in Kenya invited a G.O.A.L. Project team to train Kenyans in addictive disease and its relationship to HIV/AIDS. Margaret Namirembe Oketch, a Kenyan leader, and her colleague, invited 52 potential local leaders, community workers, clergy, teachers, and probation officers to the Brackenhurst Conference Center near Limuru for a two-week training. An American all-volunteer professional team worked with Ms. Oketch to put together an addiction/HIV/AIDS training package that included dramatic skits, PowerPoint presentations, and modeling 12 Step recovery and HIV/AIDS support groups (AA and Al-Anon) during the first week of the training. During the second week, the participants grouped themselves by various interests and worked together to prepare their action plans (mini proposals) that they presented to the whole group and made a commitment to follow through with their plans. Meanwhile, the combined GOAL/Kenya team formed a model structure for supervision and follow-up (Figure 13.1) that gave birth to the SARAH Network.

The SARAH Network can be a model for other African countries faced with the escalating rate of HIV/AIDS due to substance abuse addiction because it meets most of the guidelines recommended by Ashley et al. (2006) for successfully reducing the impact of risky behaviors due to the abuse of alcohol. Two recommendations, coalition building and community empowerment, are listed with examples.

A coalition of grassroots projects make up the SARAH Network. The Aniga Women's Initiative is located in western Kenya, in the Kisumu dis-

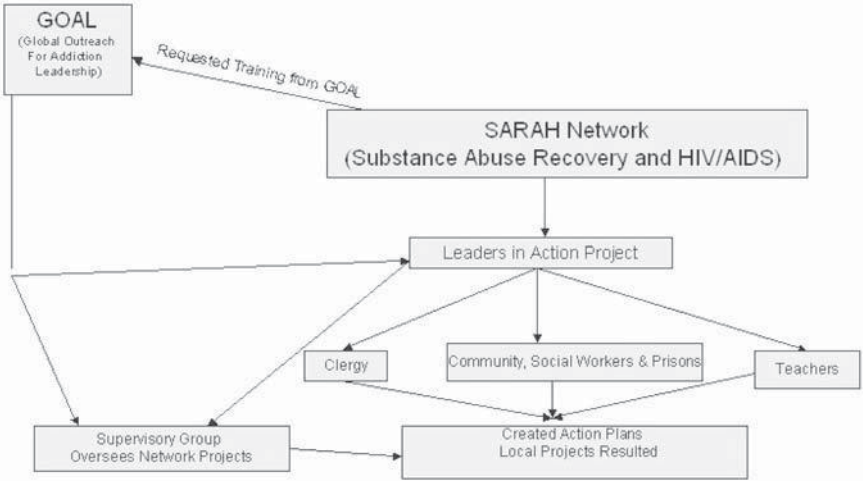

FIGURE 13.1 GOAL/SARAH Network Organizational Chart.

trict, where the statistics for both legal and illegal alcohol abuse and HIV/AIDS among women and children are higher than anywhere else in Kenya. The Kisumu region is also a rural area where village widowed women brew 80 percent proof alcohol called *Chang'aa* and/or are subject to GBV by drunken husbands if they risk going for VCT testing. The women in the Aniga project have been encouraged by their leader to form support groups and to reach out to other village women.

Five new support groups have mushroomed in a two-year period of time. Both men and women in these villages are now planting crops, bee keeping, rearing dairy goats, and raising chickens to bring in sustainable income as alternatives to chang'aa brewing. A mobile unit (van) brings VCT services directly to them.

The Aniga Women Initiative holds community mobilization daylong events in their district. In August, 2007, 2,000 people attended and more than 100 took advantage of VCT located in a tent nearby where the villagers were entertained by skits, poems, and songs that told the HIV and substance abuse prevention message. One reason for the success of this particular project has been the involvement of the local tribal chiefs and community leaders in all of the trainings and that, rather than attempting to change the local customs, such as spousal inheritance, skits are planned around cultural customs to encourage VCT testing by all parties.

Caring Mothers, a group of commercial sex workers in the coastal region of Mombasa, is another rural project. Although commercial sex workers are difficult to work with, the group has grown from 15 to 36 and, with the persistence of their leader; the women are now in peer-based support groups and are learning to protect themselves from acquiring the virus and to stop using alcohol and other drugs. As economic necessity drives many of these women into prostitution, they have begun alternative income-generating activities, such as soap making, growing crops, and hair dressing.

Wholeness Among People Project (WAPIS) is an peri-urban project located in the Banana region, Kiambu District. This project, directed by a member of the Church Army (similar to the Salvation Army), is geared to helping young male drivers of commuting passenger service vehicles called *Matatu*s stop using and dealing in drugs. Drunk-driving traffic accidents have been the major causes of death on Kenyan roads. With the encouragement of their pastor leader and with training in substance abuse prevention and the formation of recovery support groups, about 50 young male drivers now are reaching out to local schools, training peer educators, and doing drug prevention with primary and high school students.

Also, in the Kawangware slums of Nairobi, another pastor has been training peer educators and has started an Alcoholics Anonymous recovery support group at his Upako Center. Under a tin-roofed shack, the young volunteer high school peer educators educate AIDS orphans.

PROFESSIONAL CAPACITY BUILDING

SARAH Network staff conducts weeklong addiction HIV/AIDS training events for clergy, probation officers, and teachers. In a study done in the Kisumu district of Kenya in 1994, the findings indicated that community and church members look up to clergy as trusted community leaders and those impaired seek help (Oketch, 2005). GOAL/SARAH facilitators have trained more than 48 clergy and lay leaders with proven behavior change communication methods and modeling mutual help 12 Step recovery support groups, based on SARAH's motto *Each One Reach One*. These clergy have informed and educated 4,800 persons. SARAH staff collected data and qualitative results using a simple monitoring and evaluation form and followed up with telephone calls, e-mails, and personal visits.

At each workshop, clergy are given a supply of addiction recovery educational books and pamphlets and a list of rehabilitation centers for possible referrals. SARAH also helps affected and infected clergy seek treatment by sponsoring and referring them to a nearby treatment center.

EVIDENCE-BASED TREATMENT ACCESSIBILITY

Although SARAH-trained clergy and teachers refer those persons who request help to existing treatment centers, the majority of Kenyan treatment centers charge a fee and most Kenyans cannot afford to pay those fees. Also, the counselors in these facilities are recovering addicts themselves and have had very little addiction counselor training. Through the SARAH Network, the International Association of Therapeutic Communities, the U.S. Academic Education Development (AED), the Kenya National Campaign Against Drug Abuse Authority (NACADAA), the Support for Addiction Prevention and Treatment in Africa Trust (SAPTA) and the Centre for Addiction Studies and Services in Africa (CASSA), efforts are underway to develop evidence-based interventions, to train addiction counselors, to set up new treatment programs in existing hospitals, and to start a countrywide school and community drug-prevention program.

SARAH Network's motto, *Each One Reach One*, has been successful in spreading the message of hope in healing for both alcohol and other drug abusers and addicts and those infected with HIV/AIDS. Although there are a handful of mutual help 12 Step–based recovery support groups, it will take another 10 years for these to multiply. In Kenya, especially in western Kenya, many more of these peer-based anonymous groups are needed. These recovery support groups have proven successful worldwide for helping addicts stay clean and sober.

CHANGING ALCOHOL-IMBIBING CULTURES AND BEHAVIORS

Alcohol and other drugs have been used and consumed in Kenya as part of the cultural traditions for centuries, but there were certain rules and values of use up until the period just after colonialism and the subsequent breakup of family and tribal cultures. It had been permissible for adults to use alcohol and tobacco, but youth and women were discouraged from using either until after childbearing years. Traditionally, alcohol, including illegal *Bhang'aa* has been used and abused during weddings, birthdays, harvest festivals, funeral ceremonies, and other social events (SINAM/Levi Trust, 1999). However, drunkenness has never been acceptable. In fact, those who exhibit drunkenness as well as those who test positive for the HIV virus become social outcasts. In all their trainings, SARAH staff and project leaders teach the brain disease concept of addiction and the realities of alcohol dependency and the resultant laxity in regard to sexual behaviors.

Khat production and exportation is a booming business among in Kenya. Due to the excessive availability, the abuse of khat is an common activity among long distance truck drivers and others. Among other dangers, excessive use of khat has been blamed for rendering men impotent and hence wives seeking other sexual partners and thus increasing the spread of HIV/AIDS. SARAH staff educates vulnerable individuals on the dangers of the abuse of khat in their HIV prevention activities.

The SARAH staff trainers and project leaders employ dramatic skits, role plays, and dialogues to dispel folklore and myths surrounding alcohol and other drug use. In clergy trainings, clergy also practice methods learned to dispel myths, teach facts, and reduce stigma.

CROSS-TRAINING IN HIV AND ADDICTION AND VCT

Although HIV/AIDS training is now readily available in African countries, thanks to generous funding from the United States, the United Nations, and the European Union, very few programs provide the cross-training in HIV and addiction, as well as modeling both peer-based recovery and HIV+ support groups. SARAH emphasizes a similar mutual help group step for both groups. The basic weeklong training consists of two days of knowledge in drug prevention and treatment, two days in HIV/AIDS prevention and treatment, and one day in implementation planning. SARAH facilitators train people with proven behavior change communication methods. Trainers present topics geared to the specific group to be trained. Participants fill out an action planning sheet provided by the SARAH staff indicating what actions they will undertake and how many persons they anticipate reaching.

The Freedom Center in Egypt, on the continent of Africa and considered a country in the Arab Middle East, an emerging new treatment model that combines the Minnesota Model (i.e., based in peer–based mutual help recovery support group) with a six-week therapeutic community model has been found to be effective. This, combined with a treatment model that includes one-on-one and group counseling as well as attendance at outside recovery support groups, has been accompanied by extensive prevention awareness through television and radio and has taught that treatment for alcohol and other drug addiction is possible.

Three thousand heroin addicts and alcoholics in this Arab African country now wait to get a bed in one of two dozen treatment centers. The Freedom Center, founded by Ehab Elkharrat, MD, former president of the International Substance Abuse and Addiction Coalition (ISAAC) and who now is United Nations Development Programme (UNDP) representative for all Arab coun-

tries, has started other treatment centers in and around Cairo where both heroin and alcohol addiction are huge challenges. Narcotics Anonymous (12 Step) support groups have mushroomed in the past six years. Working on the principle of one addict reaching out to help another and sponsorship, the Egyptian Narcotics Anonymous (NA) support groups have become the lifeline for addicts and are spreading the message of hope in recovery.

CONCLUSIONS

Implementing model programs specifically suited to African cultures and customs to combat the spread of HIV/AIDS must include training components that cross both disciplines, (HIV/AIDS and alcohol and other drugs) that have proven effective for prevention and treatment. They must be geared to those within the culture who are most effective as change agents. The SARAH Network has targeted community opinion-shapers who include clergy, teachers, probation officers, and grassroots community organizers as the most effective as prevention agents.

Difficulties still lie ahead in African countries in meeting the challenges that alcohol and other substance abuse and addiction cause in the spread of HIV/AIDS. Much still needs to be done to assist in the formation of and support for evidence-based treatment facilities, especially for those populations less able to pay. Treatment should embody the establishment and sustaining of mutual help–addiction recovery and HIV+ support groups. Also, unless GBV and rape of vulnerable women and children is curtailed through establishing shelters and a legal protection system, very little hope exists for curbing the spread of the HIV virus in Kenya as well as in other African countries. Limited research has been done to investigate the correlations between HIV and substance abuse issues in Kenya. Despite this fact the SARAH Network's interventions have proven to be effective based on what has worked in other countries. Research-guided interventions would facilitate localized interventions and possibly yield better outcomes. For the existing treatment centers, both public and private, addiction counselor training and certification should be strengthened. More counselor training is needed for family member issues, relapse prevention, therapeutic strategies such as motivational interviewing, and stages of change, as well as a country-wide certification process. These should be put in place in each of the infected African countries.

The Kenya SARAH Network Prevention model and the Egyptian Freedom Center treatment model signal a great new beginning for the African continent in its fight against the spread of the HIV virus.

REFERENCES

African Centre for Research and Information on Substance Abuse (CRISA); R. Kasirye. (2006). Commentary. *African Journal of Drug and Alcohol Studies*. CRISA Publications 5(1), 27–38.

Africa Regional Conference. (2005, August 29–31). (USG agencies: Health and Human Services (HHS), Center for Disease Control (CDC), County Assistance Plans (cAP), National Institute of Alcohol Abuse and Alcoholism (NIAAA), Department of Defense (DoD), and United States Agency for International Development (USAID) President's Emergency Plan for AIDS Relief (PEPFAR) *Alcohol, HIV risk behaviors and transmission in Africa: Developing programs for the United States Emergency Plan for AIDS relief*. Dar Es Salaam, Tanzania 13-32.

Ashley, J. W., Levine, B., & Needle, R. (2006). Summary of the proceedings on alcohol, HIV risk behaviors and transmission in Africa: Developing programmes for the United States President's Emergency Plan for AIDS relief (PEPFAR). *African Journal of Drug and Alcohol Studies,* 5(2), 192–200.

Fifty-eighth World Health Assembly Resolution on Public Health Problems Caused by Harmful Use of Alcohol. (2005, May). In *African Journal of Drug and Alcohol Studies* (2006), CRISA Publications 5(1), 71–72.

Flisher, A. (2006). Public health problems caused by harmful use of alcohol. *African Journal of Drug and Alcohol Studies,* 5(1), CRISA Publications, 73–74.

Obot, I. S. (2006). Alcohol use and related problems in Sub-Saharan Africa. *African Journal of Drug and Alcohol Studies* CRISA Publications 5(1), 17–26.

Odejide, O. A. (2006). Public health problems caused by harmful use of alcohol. *African Journal of Drug and Alcohol Studies*. CRISA Publications 5(1), 17–26.

Oketch, M. (2005). *Substance use and abuse—Kenya.* Presentation at First National Addiction Conference in Kisumu, Kenya.

SINAM/Levi Trust. (1999). Some observations on the misuse of drugs in Mombasa. Unpublished paper.

UNFPA (defined by United Nations Population Fund Gender Theme Group). (1998). Violence against girls and women. From http://www.unfpa.org/intercenter/violence/ntro.htm. Cited also on p. 21, g. Woelk. Alcohol and Gender-Based Violence. Africa Regional Meeting Report, Dar es Salaam, Tanzania. August 29–31, 2005.

World Health Organization (WHO). (2004). Global Status Report on Alcohol, 2004. World Health Organization, Department of Mental Health and Substance Abuse, from http://www.who.int/substance_abuse/publications/alcohol/en/index.html.

Chapter **14**

Drug Abuse–Related HIV/AIDS Epidemic in India: Situation and Responses

Atul Ambekar, MD, and Meera Vaswani, PhD

HIV EPIDEMIC IN INDIA: CONTRIBUTION OF INJECTING DRUG USE

The first case of HIV was detected in India in 1986. In the past two decades since then, the HIV epidemic continues to grow unabated in India. As per the latest estimates, there are about 2.5 million people living with HIV in the country. The prevalence of HIV among the general population is estimated to be about 0.36 percent, with a much higher prevalence among men as compared to women (0.43% vs. 0.29%). Prevalence is also high in the young, productive age group with 88.7 percent of all infections occurring in the age group of 15–49 years (National AIDS Control Organization [NACO], 2007a). In other words, there is still no generalized epidemic (defined as more than 1% HIV prevalence in the general population; Joint United Nations Programme on HIV/AIDS and World Health Organization [UNAIDS/WHO], 2004) of HIV in India at the national level.

The national level data, however, should be interpreted with caution. India, being a vast and heterogeneous country, has a heterogeneous HIV situation. It has often been commented that there is not one, but many, simultaneous HIV epidemics currently spreading in India (Monitoring the AIDS Pandemic [MAP], 2004). In three states of the country, the epidemic is generalized, that is, these states have HIV prevalence of more than 1 percent in the general population. These three states are Andhra Pradesh, Manipur, and Nagaland. It must also be remembered that though these states have a higher *proportion* of

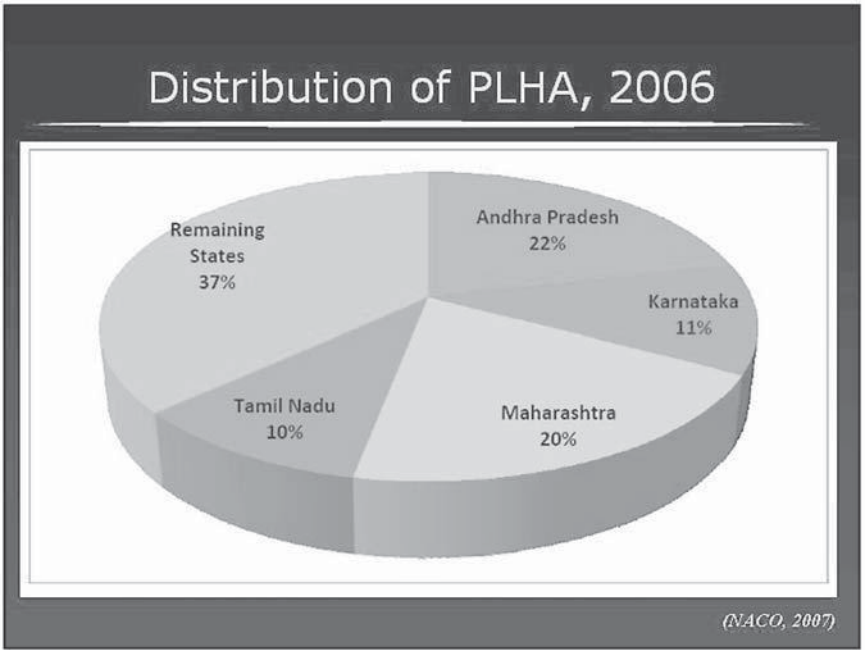

FIGURE 14.1 Distribution of proportion of people living with HIV/AIDS (PLHA) in India, 2006. From *HIV Sentinel Surveillance and HIV Estimation, 2006*, by National AIDS Control Organization, 2007, New Delhi: Ministry of Health and Family Welfare, Government of India.

people living with HIV/AIDS (PLHA), there are other states with far higher *absolute numbers* of people living with HIV (see Figure 14.1).

Besides these three states in the country, there are many other states that are in the "concentrated" stage of the epidemic (UNAIDS/WHO, 2004), in that the prevalence of HIV, though less than 1 percent in the general population, is more than 5 percent in the high-risk groups (HRGs). Certain groups of individuals, based on their enhanced vulnerability to HIV infection, are seen as HRGs. These include female sex workers (FSW), men having sex with men (MSM), and injecting drug users (IDUs). Among these, it is noteworthy that the highest prevalence of HIV at the national level has been found among IDUs (as shown in Figure 14.2).

Since the beginning of the HIV epidemic in India, the major route of transmission has always been heterosexual intercourse, though the proportion of HIV infections attributed to heterosexual intercourse (Correa & Gisselquist,

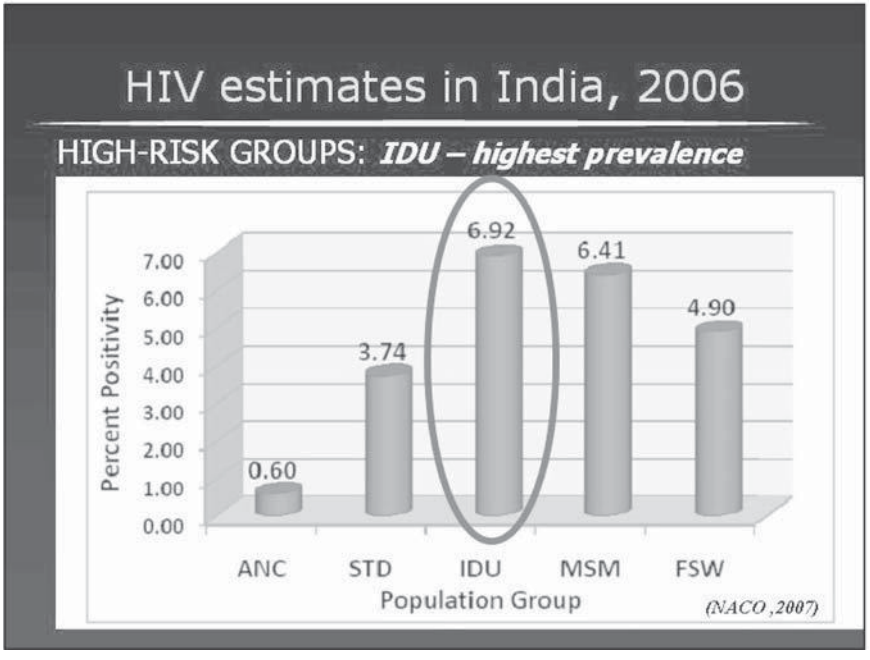

FIGURE 14.2 HIV prevalence among various groups in India, 2006. From *HIV Sentinel Surveillance and HIV Estimation, 2006,* by National AIDS Control Organization, 2007, New Delhi: Ministry of Health and Family Welfare, Government of India.

2006), particularly heterosexual commercial sex (Gisselquist & Correa, 2006), has been debated in the literature. The contribution of injecting drug use (IDU) and consequent sharing of contaminated injection equipment has usually been estimated to be around 2–3 percent of the total HIV infections (Correa & Gisselquist, 2006). Until recently, it has been largely believed that HIV infection among IDUs is concentrated only in the northeastern states (Manipur and Nagaland) and certain large cities of the country (Chennai, Delhi, and Mumbai). Findings from the recent round on sentinel surveillance (conducted annually, at a national level), however, reveal that the HIV infection among IDUs has spread in many states of the country (NACO, 2007a). As seen in Figure 14.3, as many as seven areas of the country have crossed the threshold for a concentrated epidemic among IDUs.

Additionally, many smaller, clinic-based studies have reported high prevalence of HIV infection among injecting drug users in India (Jindal, Arora, &

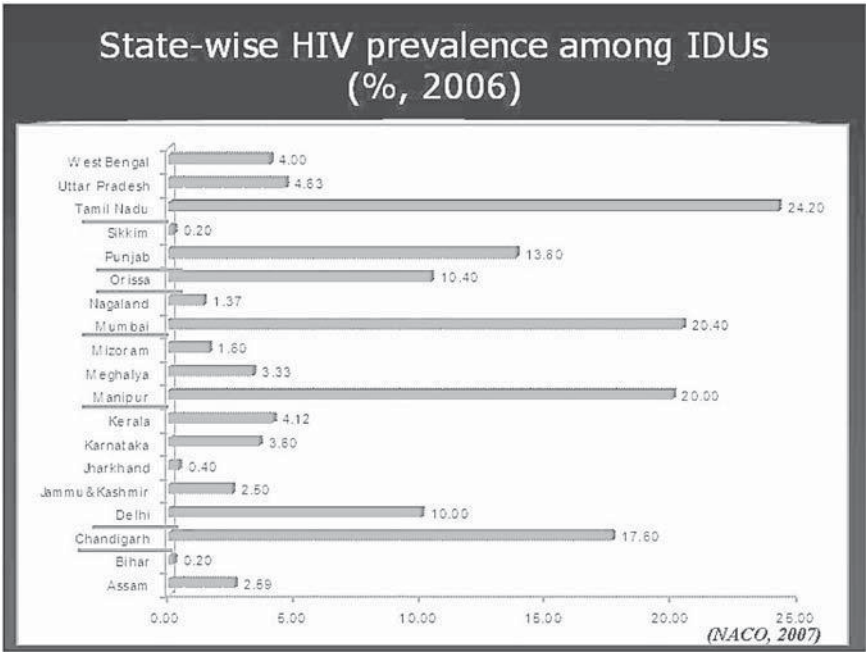

FIGURE 14.3 State-wise HIV prevalence among injecting drug users in India, 2006. From *HIV Sentinel Surveillance and HIV Estimation, 2006*, by National AIDS Control Organization, 2007, New Delhi: Ministry of Health and Family Welfare, Government of India.

Singh, 2008; Saraswathi & Dutta, 2007; Vaswani & Desai, 2004; Vaswani & Rao, 2006). Thus, injecting drug use is a major risk factor for transmission of HIV in India. While, in two of the states (Manipur and Nagaland), it is the primary factor behind a generalized HIV epidemic, the phenomenon may be significantly contributing to the spread of the HIV epidemic elsewhere in the country as well.

INJECTING DRUG USE IN INDIA

Historically, the use of certain intoxicating substances has been prevalent in India for many centuries now. Traditionally, various cannabis derivatives and locally brewed alcohol were the substances of use. There was also a certain degree of social sanction to use of these substances (Dhawan, 1998). Until the early twentieth century, many of the substances of abuse, which are currently illicit, were available through licit channels. Nonmedical or nonprescription

use of psychotropic medications was also observed. Besides opium, cannabis, and alcohol, use of cocaine and even khat was reported in India until 1940. However, many reports up to the late 1960s suggested that most users of these substances used in moderation and only a few did become habitual users or used in excess (Reid & Costigan, 2002).

In the 1980s, however, with the introduction of heroin in India, the situation changed to a great degree. Earlier, opium was available through licensed vendors and a sizable number of registered opium addicts existed in the 1950s (Ray, 1998). Over the years, this practice has been discontinued and new registration of opium addicts has been stopped. In the early 1990s, only a few of the registered opium users were alive and drawing their quota of opium. Consequently, many users of a traditional, plant-based (and relatively less potent) drug like opium were forced to shift to a modern, synthetic (and more potent) drug, that is, heroin (Ambekar, Lewis, Rao, & Sethi, 2005).

Over next few years, many heroin users switched to injecting modes of taking opioid drugs. The phenomenon was first noted in the northeastern states of Manipur and Nagaland, which share their border with Myanmar—the world's second-largest illicit opium-producing country (United Nations Office on Drugs and Crime, Regional Office for South Asia, and Ministry of Social Justice and Empowerment [UNODC/MSJE], 2005). Since taking opioids through an injection route provides a better and quicker high, the injecting route is perceived by the drug users as more cost-effective (UNODC/MSJE, 2004a, 2004b, 2005). The phenomenon was first noted in the northeastern states of Manipur and Nagaland (Sarkar et al., 1991, 1995), which share their border with Myanmar—the world's second-largest illicit opium-producing country (United Nations Office on Drugs and Crime, Regional Office for South Asia, and Ministry of Social Justice and Empowerment [UNODC/MSJE], 2005).

Currently, India figures among the developing and transitional countries with the "largest populations of IDUs," along with Brazil, China, and Russia (Aceijas et al., 2006). However, there are no reliable estimates regarding the number or prevalence of IDUs in the country. The largest of the epidemiological studies in the country—the National Household Survey—found a prevalence of 0.1 percent of IDUs among the adult male population of the country (UNODC/MSJE, 2004a). However, it is widely accepted that household samples are not appropriate to pick up hidden and potentially stigmatizing phenomena such as injecting drug use (Joint United Nations Programme on HIV/AIDS and Family Health International [UNAIDS/FHI], 2003). The same multimodality study (UNODC/MSJE, 2004a) also collected information from patients seeking treatment for substance use problems from more than 200 treatment centers throughout the country through a survey—the

Drug Abuse Monitoring System (DAMS). In this survey, prevalence of injecting drug use ("ever" in lifetime) among treatment seekers was reported to be 14 percent (UNODC/MSJE, 2002a). Yet another component of the same study was the Rapid Assessment Survey (RAS), in which data were collected from drug users contacted at streets in 14 urban locations across the country. Here, the prevalence of injecting drug use was 43 percent (UNODC/MSJE, 2002b). International experts (Aceijas et al., 2004) in a global overview of HIV among IDUs have quoted a rather wide range—from 563,000 to 2,025,000—for the estimated number of IDUs in India.

Like the HIV situation, however, the spread of IDU is also heterogeneous in the country. For a long time now it has been believed that the phenomenon of IDU is a matter of concern only in the northeastern states (Manipur and Nagaland) and the metro cities of the country (Chennai, Mumbai, and Delhi). There have been multiple studies, however, that have produced evidence that IDU is not limited to the aforementioned areas. The 14-city Rapid Assessment Survey (UNODC/MSJE, 2002b) documented the presence of injecting drug use in many other cities of the country other than the northeastern states and metropolitan cities (see Figure 14.4).

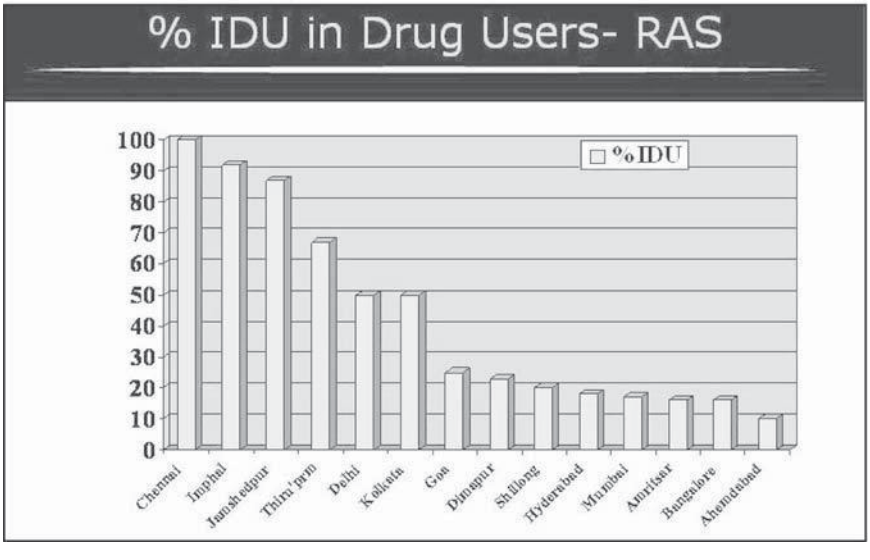

FIGURE 14.4 Proportion of injecting drug users in the total sample of drug users in the Rapid Assessment Survey of Drug Use in India. From *The Extent, Pattern and Trends of Drug Abuse in India: National Survey*, by United Nations Office on Drugs and Crime, Regional Office for South Asia, and Ministry of Social Justice and Empowerment, 2004, New Delhi: UNODC and MSJE.

SHARAN, an NGO working in the area of drugs and HIV, has recently documented the presence of IDU in many parts of the country, including Uttar Pradesh, Maharashtra, Goa, Kerala, and Tamil Nadu (SHARAN, 2007). Similarly, in two of the recent, large-scale studies, presence of IDUs has been documented in many north Indian states, including Punjab, Haryana, Uttar Pradesh, Bihar, Jharkhand, and Orissa (Ambekar & Tripathi, 2006, 2007, 2008).

Thus, there is considerable evidence that IDU is well established in many parts of India. The worrying aspect of this is the presence of IDU in smaller towns and rural areas of the country.

PROFILE OF IDUS IN INDIA

Most of the studies in India have found IDUs to be in their late 20s or early 30s. Notably, most injecting drug users in India are those, who, in the initial phase of their drug-use career, use opioids through a noninjecting route (most commonly, heroin, through smoking or "chasing" routes) and later switch to the injection mode of using opioids. There is usually a time lag—ranging from 2 to 10 years—when people switch from noninjecting to injecting mode of taking drugs (UNODC/MSJE, 2004b). The factors behind initiation of injecting in India have also been studied and are summarized in the accompanying box.

BOX 1

Factors behind Initiation of Injecting Drug Use by Drug Users in India

- Nonavailability of heroin (for smoking or "chasing")
- High cost of heroin (for smoking or "chasing")
- Easy availability of pharmaceutical opiates and other sedatives (injectable) in pharmacies (without prescription)
- Perception that taking drugs through the injecting route produces better and quicker highs and immediate relief from withdrawal symptoms
- Iatrogenic, that is, administration of opioid injections by a health professional
- Curiosity
- Peer pressure

Source: Injecting Drug Use and HIV in India: An Emerging Concern, by United Nations Office on Drugs and Crime, Regional Office for South Asia, and Ministry of Social Justice and Empowerment, 2004, New Delhi: UNODC and MSJE; "My First Time: Initiation into Injecting Drug Use in Manipur and Nagaland, North-East India," by M. Kermode, V. Longleng, B. C. Singh, J. Hocking, B. Langkham, and N. Crofts, 2007, *Harm Reduction Journal, 4*, p. 19.

Most injecting drug users in India prefer to inject either heroin or pharmaceutical preparations. The pharmaceutical preparations include injectable opioids like buprenorphine and pentazocine, which are usually mixed with other sedatives like diazepam, promethazine, and chlorpheniramine. A distinct pattern of using dextropropxyphene capsules for injection has also been observed, particularly from the northeastern states of Manipur, Nagaland, and Mizoram (UNODC/MSJE, 2004b, 2005; Kermode et al., 2007; Ambekar & Tripathi, 2006, 2008).

RISK BEHAVIORS AMONG IDUS: INJECTING RISKS

The risk of transmission of HIV and other blood-borne virus infections due to sharing of contaminated injection equipment is well documented. Injecting serves as an efficient vehicle for transmission of HIV. The injecting route is about three to five times more efficient than the sexual route (0.67% risk for HIV transmission per episode of intravenous needle or syringe exposure as compared to 0.1%–3% risk for HIV transmission per episode of receptive penile-anal sexual exposure and 0.1%–0.2% risk per episode of receptive vaginal exposure; Centers for Disease Control and Prevention [CDC], 1998).

There is ample evidence from the community-based as well as clinic-based studies that a sizable proportion of IDUs in India share their injection equipment and thus are vulnerable to transmission of HIV and other blood-borne virus infections. In the Rapid Assessment Survey of Drug Abuse in India (UNODC/MSJE, 2002b, 2004), injecting drug use was reported to be a group activity. Needle sharing was reported to be "common" among injecting drug users and varied between 52 and 81 percent. Indirect sharing (e.g., sharing of cotton swab, filter, and spoons, etc.) was also common. About half of the IDUs who shared reported that they had shared syringes and needles the last time they injected. Most did not clean the needles and syringes. In the first round of the Behavioral Surveillance Survey (BSS) conducted in India by the National AIDS Control Organization (NACO, 2002a) among 1,355 IDUs, about 44 percent shared injection equipment in the preceding month, while 4 percent reported that they "always" shared injection equipment. In the latest round of BSS also (NACO, 2007b), a large majority of IDUs in Manipur, Delhi, Chennai, and Mumbai reported sharing of injection material. Many of them reported sharing on more than 50 percent of the occasions. In yet another recent study (Ambekar & Tripathi, 2006) conducted at multiple sites nationwide among 5,603 IDUs, 59 percent reported sharing of injections "ever." A large majority of them (77%) shared at the last occasion of injecting. In clinic-based studies, high prevalence of risk behaviors as well as HIV infection has

been reported among IDUs, particularly when compared to non-IDUs (i.e., drug users who take drugs through a noninjecting route). Vaswani and Desai (2004) found a high prevalence of risky injection behavior among IDUs in their sample of 154 consecutive drug users attending a treatment center. Notably, the prevalence of HIV was significantly higher among IDUs (8.2%) as compared to non-IDUs (1.8% in heroin smokers and 0% in alcohol-dependent individuals).

Thus, in this backdrop of widespread injecting drug use and associated practice of sharing contaminated injection equipment, rapid escalation of the prevalence of HIV remains a very likely possibility in India. Some parts of the country have witnessed explosive HIV epidemics among IDUs in the past. For instance, in Manipur in northeast India, the first case of HIV among IDUs was detected in 1989. Six months later, prevalence among IDUs had increased to 50 percent (Sarkar et al., 1993).

RISK BEHAVIORS AMONG DRUG USERS: SEXUAL RISKS

While injecting-related risk for transmission of HIV is well recognized, the contribution of substance use (alcohol as well as illicit drugs like cannabis and opioids) to enhanced transmission of HIV is not recognized so well. There is evidence that substance use (injecting or otherwise) is associated with an enhanced vulnerability to high-risk sexual behavior.

Men who have been drinking alcohol report more contact and noncontact sexual problems as compared to those who do not use alcohol (Verma, Sharma, Singh, Rangaiyan, & Pelto, 2001). In a study on assessment of the risk factors in sexually transmitted diseases (STD), alcohol was one of the risk factors found to be significantly associated with the acquisition of STD (Sharma & Chaubey, 1996). Among studies looking at sexual behavior and its relationship with substance use, in a study from northeast India, among 200 students, sexual behavior was found to be strongly associated with alcohol use (Longkumer, Shrivastava, & Murugesan, 2000). Similarly, in yet another study among adolescents in slums of Mumbai, alcohol use was associated with risky sexual behavior along with the perception that consumption of alcohol before sex heightens sexual pleasure and makes it long-lasting (Singh, Schensul, & Gupta, 2004). Such an association between alcohol and substance use and high-risk sexual behaviors is more evident among groups regarded to be at high risk of HIV such as sex workers and truckers. The number of clients visiting the sex workers decreases during the "dry" (i.e., alcohol-free) periods (Ambwani & Gilada, 1998).

Tripathi, Malhotra, and Sharma (2004) have reported findings of their study carried out in the city of Delhi as a part of a multinational study sponsored by the World Health Organization (WHO). Primary data on alcohol use and sexual risk behavior were obtained from interviews of key informants (KIs), sex workers, truck drivers, and restaurant workers. Among the significant findings, alcohol was found to be a common substance of use among various population subgroups, including sex workers. A clear relationship between use of alcohol and sexual behavior was evident. Though from the general population about 25 percent of respondents reported sexual arousal and enhanced sexual pleasure following consumption of alcohol, among high-risk population groups (sex workers, transport workers, etc.), between 60 and 100 percent reported such feelings. Of more concern was the finding that condom use was uniformly low among all the population subgroups except for sex workers. Consistent condom use was even lower (Tripathi et al., 2004).

Apart from alcohol, other substance use has also been linked to high-risk sexual behaviors. High prevalence of substance use has been found among sex workers and their clients in India. In a nationwide Behavioral Surveillance Survey, among a total of 5,572 female sex workers (FSWs), 22 percent reported that they consumed alcohol every day in the last month (NACO, 2001). Overall, around 15 percent of FSWs reported that they drink regularly before sex. Some (about 6%) had tried other (nonalcohol) addictive drugs, including injections, in the past 12 months. In the same study, among a total of 5,684 clients of FSWs, the proportion of respondents drinking at least once a week was 45 percent and the proportion of those drinking daily was nearly 23 percent. Nearly 13 percent of the respondents regularly consumed alcoholic drinks before having sex with their commercial partners. Around 22 percent reported use of other compounds like cannabis and opium. Some of these respondents (10%) had injected drugs in the last 12 months (NACO, 2001).

In a study from India, comparing the sexual behavior of drug users (excluding alcohol and tobacco) with nondrug users randomly selected from the same community, the average number of sexual partners was found to be significantly higher among drug users (7.4 vs. 4) as compared to nondrug users (Sharma, Aggarwal, & Dubey, 2002). In a recent nationwide study among 12,580 drug users, about 37 percent reported sex with multiple partners. More than a third (35%) reported *never* having used a condom (Ambekar & Tripathi, 2006).

The sexual behaviors of injecting drug users have also been studied. There is evidence that many injecting drug users are sexually active and do visit sex workers (UNODC/MSJE, 2004a, 2004b). In a clinic-based study (Vaswani & Rao, 2006), among 154 consecutive treatment seekers, a significantly higher proportion of IDUs (83.6%) was found to have multiple sex partners as compared to non-IDUs (57.4%). Women IDUs have been found to be engaging in

sex work to sustain their livelihoods (Panda et al., 2001). There is also evidence that HIV infection is being transmitted from injecting drug users to their non-injecting wives and sexual partners (Panda et al., 2000, 2005, 2007).

Thus, in India, a definite association is clearly seen between drug use and high-risk sexual behavior. This association is visible in the general population as well as the known "high-risk" population groups.

RESPONSE TO IDU-RELATED HIV: HARM REDUCTION

While it is well known that drug dependence is a complex, chronic, relapsing condition that is often accompanied by severe health, psychological, economic, legal, and social consequences—or "harms" (McLellan, Lewis, O'Brien, & Kleber, 2000)—with the advent of the HIV epidemic, substance use, in particular, injecting drug use (IDU), is now seen as a major public health issue rather than just a social/legal issue. Injecting drug users are particularly vulnerable to HIV and other blood-borne infections (such as hepatitis C) as a result of sharing contaminated injecting equipment. There is ample evidence from the recent literature that IDUs in India have a high sero-positivity not only for HIV but hepatitis B and C as well (Das, Borkakoty, Mahanta, Medhi, & Chelleng, 2007; Datta et al., 2006; Jindal et al., 2008; Saraswathi & Dutta, 2007; Sarkar et al., 2006). Some of the harms associated with drug use may be attributable to the effects of the drug itself on the body and the mind. More often, drug-related harm is the result of the many social, economic, legal, cultural, and political factors that influence the availability of illicit (illegal) drugs and the conditions under which they are used. Poverty, migration, and social discrimination affect people's vulnerability to the drug-related harm. Stringent laws, unpragmatic policies, and the social stigmatization and discrimination of illicit drug users serve to drive them further toward isolation and away from their social support like family, as well as from various health and social services (Ambekar, Agrawal, & Tripathi, 2008).

Additionally, it should be remembered that "zero tolerance"–based strategies such as legal prohibition of substances and emphasis only on the abstinence-oriented treatment have not been able to eliminate substance use from the society. Despite being illicit and controlled substances, the use of certain drugs like heroin, cocaine, and others is still prevalent in the society and the worldwide illegal trade of these substances is burgeoning (UNODC, 2007). Moreover, making a drug illegal may even increase the harms associated with its use by contributing to the marginalization of a drug user and criminalization of his drug use behavior. There is a growing understanding that the utopian rhetoric, like "a drug-free society" or "a world without drugs," is unrealistic and unachievable. The global community is now realizing that some drug abuse would occur

in any society, despite the best efforts at curbing it. Consequently, a pragmatic response would be to employ strategies that could reduce the harms associated with this drug use. One of the approaches to bring about desirable changes in the behavior of substance-using individuals—and thus reducing the risks of harms—is harm reduction.

While the concept of harm reduction has been used in the context of a variety of risky behaviors, primarily it has been seen as an intervention and management strategy for the individuals using psychoactive substances. Various definitions for the term *harm reduction* (also termed sometimes as *harm minimization*) have been put forward (Lenton & Single, 1998). The International Harm Reduction Association (IHRA) defines harm reduction as "policies and programs, which aim to reduce the health, social and economic harms associated with the use of psychoactive substances" (IHRA, 2006). A defining feature of such policies and programs is their focus on the prevention of drug-related harm rather than the prevention of drug use per se (Hunt, 2003). Instead of viewing drug problems as phenomena caused by individual psychological (or moral) deficiencies, harm reduction views any society's patterns of drug use collectively—holding that many of the most destructive consequences and refractory problems of illicit drug use are not solely attributable to the drugs per se. Rather, many of these problems are more closely linked to the failure of the policies employed to control them (Ambekar & Balhara, 2007; Ambekar et al., 2008).

The adjoining box summarizes some of the key principles of harm reduction (Ritter & Cameron, 2005).

BOX 2

Some Key Features and Principles of Harm Reduction

- The primary goal is reducing harm rather than drug use per se
- Built on evidence-based analysis
- Acceptance that drugs are a part of society and may never be eliminated
- Provides a comprehensive public health framework
- Priority is placed on immediate (and achievable) goals
- Pragmatic—does not seek to pursue policies or strategies that are unachievable or likely to create more harm than good
- Recognizes individual human right—rooted in an acceptance of individual integrity and responsibility

Source: Adapted from *A Systematic Review of Harm Reduction,* by A. Ritter and J. Cameron, 2005, DPMP, Monograph Series No. 06, Fitzroy, Australia: Turning Point Alcohol and Drug Centre.

Though there are many strategies that have been proposed to reduce the harms associated with injecting drug use, in the following section, we limit our discussion to only a few of them. These include (1) needle-syringe exchange programs, (2) agonist maintenance treatment, (3) outreach activities, and (4) legal and policy reforms.

Needle-Syringe Exchange Programs

The programs for needle and syringe exchange are more readily associated with the harm reduction approach than any other type of intervention. At its most basic level, this strategy involves supplying new, clean needles and syringes to IDUs, in exchange for old, used, and potentially contaminated needles and syringes.

As discussed earlier, a sizable number of IDUs in India share their used and potentially contaminated injections with each other, putting themselves and their partners at the risk of acquiring HIV and other blood-borne virus infections. Availability of new, clean injecting equipment, free of cost, would provide these IDUs with an opportunity to protect themselves and their injecting partners from transmission of HIV. Additionally, these programs also provide a point of continuous contact with the drug users that enable these practices and a wide range of other health matters to be discussed. Indeed, most such programs do not limit themselves only to providing sterile injecting equipment but also incorporate a variety of other services such as risk reduction education, condom distribution, bleach distribution, education on needle disinfection, and referrals to substance abuse treatment and other health and social services (Wodak, 2004).

The effectiveness of these programs in preventing the spread of HIV has been a matter of controversy, mainly due to a number of methodological shortcomings in the studies conducted to evaluate their effectiveness. In a review, Gibson, Flynn, & Perales (2001) reported findings from 42 studies, of which 28 found positive effects (reduction in drug-related HIV risk behavior, including self-reported sharing of needles and syringes, unsafe injection and disposal practices, and frequency of injection) and 14 found either no association or a combination of positive and negative effects. Overall, this provides reasonable evidence of the positive impact of needle-syringe exchange programs on HIV risk behavior and HIV infection. Wodak (2004), after his extensive review of 48 studies for the World Health Organization (WHO), has also concluded that not only was there compelling evidence of the effectiveness of needle-syringe exchange programs in reducing HIV infection substantially, there was compelling evidence for their cost-effectiveness as well. Moreover, there was no convincing evidence of any major, unintended negative consequences. However,

and more important, he concluded that these programs on their own are not enough to control HIV infection among IDUs.

Agonist Maintenance Treatment

These programs, known by a variety of names such as "opioid replacement therapy" or "oral substitution treatment" rely on the principle of substituting an illicit, unsafe, short-acting, and more addictive drug with a legal medication of known purity and potency. The origins of opioid replacement therapies can be traced back to the landmark study on the role of methadone maintenance in opioid-dependent subjects (Dole & Nyswander, 1965). Since then, a number of drugs have been used for this purpose such as methadone, LAAM, buprenorphine, slow release oral morphine, and others. However, the search for an ideal agent continues to remain elusive. All of the replacement medications have been shown to be effective in reducing use of illicit drugs, reducing the risk of injecting, reducing the risk of overdose, reducing involvement in illegal activities, and improving socio-occupational functioning (Gowing, Farrell, Bornemann, & Ali, 2004; Mattick, Breen, Kimber, & Davoli, 2003; Mattick, Kimber, Breen, & Davoli, 2003).

For India, these programs are especially relevant because the most favored drugs by the IDUs in India are opioids (Ambekar et al., 2005; Ray & Ambekar, 2005). Though methadone is not available in India as of now, another agent—buprenorphine—is available, and the country has gathered enough experience and expertise in the area of buprenorphine maintenance programs (Dhawan & Sunder, 2008).

Outreach Activities

Several of the drug-using populations may not be accessible through the conventional center-based services. Such populations may remain "hidden" due to a number of factors such as inaccessibility of services, unattractiveness of facilities, distance, stigma, and so forth. Outreach was conceived to overcome these barriers and to reach out to people within their own communities or local milieu, outside of the usual service settings. Community-based outreach involves a number of activities such as establishing contact and rapport with the target populations in their natural environments, providing information about unsafe as well as risk behaviors, and promoting and supporting safe behaviors.

Outreach services have been proved to be effective for reaching hard-to-reach, hidden populations of drug users and provide the means for enabling IDUs to reduce their risk behaviors. According to a WHO review, a significant proportion of IDUs receiving outreach-based interventions reduces their risk

behaviors in drug using, needle, and sexual practices and increases their protective behaviors (Needle et al., 2005).

Outreach services are especially relevant in India, since help seeking among drug users has been found to be low and delayed (UNODC/MSJE, 2004a; Ambekar et al., 2005). Outreach-based activities aimed at identifying and reaching out to hidden IDUs have been employed at many centers in India (Hangzo, Chatterjee, & Sarkar, 1997; Kumar, Mudaliar, & Daniels, 1998). The preliminary evidence from the country suggests that outreach services for IDUs produce significant changes in risk behaviors, especially the injecting-related risk behaviors (Kumar et al., 1998; Kumar, 2008).

Legal and Policy Reforms

The general approach toward drug use in most countries has been "criminalization." This approach may be beneficial in handling drug trafficking but has been shown to be detrimental for an individual drug user, as it promotes marginalization and enhances harm due to increased prices of the prohibited drugs. Such an approach is now under scrutiny and debate. It is now increasingly recognized that by reducing the association of drug use with criminal prosecution, the reform of punitive legal policies can produce clear benefits in the realm of public health and social order.

Since all the harm reduction measures deal basically with illegal drugs and individuals who use these illegal drugs, they are affected by the prevailing legal and policy environment. For instance, there is a possibility that under the existing Indian laws, an agency (or its staff) engaged in providing needle-syringe exchange services in India can be booked under the relevant laws on the grounds of abetment to the crime, that is, facilitating or encouraging drug use by the injecting drug users by providing needle-syringe services to them (Lawyers Collective, 2007).

IDU-RELATED HIV EPIDEMIC IN INDIA: THE INDIAN RESPONSE

The following section will focus upon a critical overview of the response to the IDU-related HIV epidemic in India at the legal, policy, and programmatic level and will attempt to highlight certain existing gaps.

Legal and Policy Issues

In the area of "supply reduction" of drug abuse, India is signatory to all three major international conventions related to drug use, namely, Single Convention

on Narcotic Drugs, 1961, Convention on Psychotropic Substances 1971, and Convention against the Illicit Traffic in Narcotic Drugs and Psychotropic Substances, 1988 (Ambekar et al., 2005). In India, the broad legislative framework on narcotic drugs and psychotropic substances is contained in the three Central Acts: Drugs and Cosmetics Act, 1940, The Narcotics Drugs and Psychotropic Substances Act, 1985 (NDPS Act 1985), and the Prevention of Illicit Traffic in Narcotic Drugs and Psychotropic Substances Act, 1988. The NDPS Act lays down the focus and direction of drug control strategy in the country. Importantly, this Act does attempt to differentiate between a drug user and drug peddler (on the basis of the quantity of the illicit drug found in possession) and also provides for the treatment of drug users (Lawyers Collective, 2007).

India's policy on narcotic drugs and psychotropic substances is based on Article 47 of the Directive Principles of State Policy, Constitution of India, where the "Duty of the State [is] to raise the level of nutrition and the standard of living and to improve public health." However, India does not have a national drug control policy or an apex organization in respect of drug control. The National Health Policy 2002 of India (Ministry of Health and Family Welfare [MOHFW], 2002) does not specifically mention drug use as a component or concern. On HIV/AIDS issues, however, there are explicitly stated and documented policies in the country. The policy document of the National AIDS Control Organization (NACO, 2002b)—National AIDS Prevention and Control Policy (NAPCP)—recognizes various harm reduction measures, as the appropriate strategies to prevent HIV among injecting drug users, although the policy further admits that in India the harm reduction approach is yet to find wider acceptability because of ethical and moral considerations.

As far as the "drug demand reduction" activities are concerned, the nodal agency for drug demand reduction in India is the Ministry of Social Justice and Empowerment (MSJE), and not the Ministry of Health, implying indirectly that drug demand reduction is seen as more of a social justice and welfare issue rather than a health-related issue. The Ministry of Social Justice and Empowerment has developed a three-pronged strategy for drug demand reduction in India. The three components are: (1) building awareness and educating people about ill effects of drug abuse; (2) dealing with addicts through a program of motivation, counseling, treatment, follow-up, and social reintegration; and (3) imparting drug abuse prevention rehabilitation training to service providers. The ministry (MSJE) implements its programs mainly by supporting NGOs all over the country. Currently, the ministry is supporting around 450 drug dependence treatment centers throughout the country, which are being run by NGOs (Ambekar et al., 2005). In addition, the Union Ministry

of Health and Family Welfare and the Health Departments of the States and Union Territories also provide treatment and hospitalization services related to substance use. There are about 122 de-addiction centers, supported at least in part, by the Ministry of Health and Family Welfare (Panda, 2007).

Thus, to summarize, at the policy level, drug use and related issues do not receive a priority status in the health agenda of the government of India. The legal environment in the country, though, is conducive to providing adequate treatment and harm reduction services to injecting drug users.

Programmatic and Service-Delivery Issues

As discussed earlier, treatment services to drug users are delivered through the NGOs (supported by the government, the social welfare sector) as well as the government-run de-addiction centers as part of the health sector. However, it has been repeatedly commented that many of these services remain inadequately utilized (Ambekar et al., 2005; UNODC/MSJE, 2004a). Additionally, the National AIDS Control Organization is also engaged in providing harm reduction services ("Targeted Interventions") to the injecting drug users through NGOs, under the National AIDS Control Programme (NACP), which has just entered its third phase (NACO, 2006).

As of now, there are about 120 targeted interventions in various parts of the country (but concentrated mainly in the northeastern states and the bigger cities), delivering harm reduction (outreach and needle-syringe exchange but *not* agonist maintenance) services to about 90,000 IDUs (Rao & Khumukcham, 2008). This amounts to about 53 percent coverage. As of 2007, there were about 48 intervention sites for buprenorphine substitution also (largely implemented through NGOs, without any direct support from the government), providing services to only a few thousand IDUs (Dhawan & Sunder, 2008). The NACP–III plans, ambitiously, to scale up the existing IDU intervention with various harm reduction services so as to cover about 80 percent of the total number of IDUs in the country (Rao & Khumukcham, 2008). Among the newer initiatives, the NACO proposes to implement oral substitution therapy, in addition to needle-syringe exchange to the IDUs. Clearly, extensive scaling-up of the existing services is required in order to provide adequate coverage to injecting drug users in India to be able to achieve the stated goal of the National AIDS Control Programme—halt and reverse the HIV epidemic (NACO, 2006).

While the effectiveness of the needle-syringe exchange programs in India has not been studied systematically, there is some evidence that these programs may have been successful in bringing IDUs closer to various services, reduction

of risky injection practices, and reducing the prevalence of HIV (Ngully, 2008; Sharma et al., 2003). Similarly, the preliminary evidence regarding the Indian experience with opioid substitution therapy is also encouraging. Dhawan and Sunder (2008), in a brief overview of buprenorphine substitution in India, have concluded that buprenorphine substitution programs have been successful in decreasing the harm associated with drug use, as well as decreasing the drug use per se and improving the quality of life.

To summarize, though various harm-reduction services in India are in place, they remain too few and far in between. There is a visible and obvious gap in terms of the number of services available and the requirements of these services. Without a rapid scale-up in the number and the quality of the services, it would be difficult to provide adequate coverage to the IDU population. This would make it very challenging to realize the goal of halting and reversing the HIV epidemic.

CONCLUSION AND RECOMMENDATIONS

The available evidence indicates that substance use and related HIV is fairly prevalent in almost all parts of India; although, in terms of the HIV prevalence among drug users, different areas within the country are at different stages of the epidemic. However, the presence of risky behaviors—both injecting-related as well as sexual—among drug users would mean continued vulnerability in the country for rapid escalation of the HIV epidemic among drug users and eventually to the general population. Fortunately, among the general population, HIV prevalence is still low in India. The current stage of the HIV epidemic thus should be seen as a window of opportunity for implementing large-scale, evidence-based programs for HIV prevention. Presented below are a few specific measures, which should be urgently adopted by India.

BOX 3

Recommendations for Drug-Related HIV Prevention Programs in India

- Better assessment of epidemiological situation and monitoring/surveillance
- Convergence of abstinence-oriented drug abuse treatment and HIV prevention ("harm reduction") services
- Interventions to address both injecting-related and sexual risk behaviors

Expanding the Knowledge Base

There is an urgent need to expand the knowledge base about drug abuse and vulnerabilities to HIV/AIDS in India. Thus, extensive epidemiological data of adequate quality are urgently required. Additionally, the monitoring and surveillance systems need to be periodically reviewed and, if required, be strengthened and expanded. Behavioral data, wherever possible, should be linked with the biological data. Data collected from clinics by way of "sentinel surveillance" should be supplemented by data collected from the community, that is, the general population. Such an approach has recently been adopted in India for the general population, resulting in a major revision of the HIV estimates in the country (NACO, 2007; UNAIDS, 2007). Finally, a very important research agenda for the country is that of the effectiveness and appropriateness of HIV prevention interventions.

Convergence of "Abstinence" and "Harm-Reduction" Approaches

Services for the treatment for substance use disorders, while available almost everywhere in India, have largely tended to focus upon complete abstinence from drugs. Approaches regarded as "harm reduction" should be urgently scaled up in India. Indeed, both of these—abstinence and harm reduction—should not be seen as two opposite approaches. Rather, emphasis should be on provision of a whole continuum of care, whereby all the services that drug users may require—those aimed at achieving a drug-free life, those aimed at reducing the drug use itself, and those aimed at reducing the consequences of drug use—should be made available to them. Such a comprehensive approach has been recently endorsed by the United Nations as well (UNODC, 2008). Specifically, the country urgently requires oral substitution programs for opioid injectors using agonist agents like methadone and buprenorphine.

Broadening the Scope of the Interventions

Most drug users including IDUs are sexually active and engage in risky sexual behavior. Moreover, a sizable number are married and can potentially transmit the HIV infection to their spouses and regular sexual partners. There is evidence that HIV infection can be transmitted from IDUs to their noninjecting wives leading, ultimately to a generalized epidemic. Thus, it is of utmost importance that interventions aimed at prevention of HIV among IDUs should reach out to their sexual partners as well. Along with the injecting-related risk

behaviors, sexual risk behaviors should also be the targets of various behavior change interventions.

REFERENCES

Aceijas, C., Friedman, S. R., Cooper, H.L.F., Wiessing, L., Stimson, G. V., Hickman, M., on behalf of the Reference Group on HIV/AIDS Prevention and Care among IDU in Developing and Transitional Countries. (2006). Estimates of injecting drug users at the national and local level in developing and transitional countries, and gender and age distribution. *Sexually Transmitted Infections, 82*(Suppl. 3), iii10-iii17.

Aceijas, C., Stimson, G. V., Hickman, M., Rhodes, T., on behalf of the Reference Group on HIV/AIDS Prevention and Care among IDU in Developing and Transitional Countries. (2004, November 19). Global overview of injecting drug use and HIV infection among injecting drug users. *AIDS, 18*(17), 2295–2303.

Ambekar, A., Agrawal, A., & Tripathi, B. M. (2008, March). Harm reduction: An overview. In A. Ambekar & B. M. Tripathi (Eds.), *Drug abuse: News-n-views* (Harm reduction: Prevention of HIV among drug users) (pp. 5–7). New Delhi: National Drug Dependence Treatment Centre, All India Institute of Medical Sciences.

Ambekar, A., & Balhara, Y. P. S. (2007). Harm reduction. In R. Lal & S. Gupta (Eds.), *Substance use disorders: A manual for nursing personnel* (pp. 102–111). New Delhi: National Drug Dependence Treatment Centre, All India Institute of Medical Sciences.

Ambekar, A., Lewis, G., Rao, S., & Sethi, H. S. (2005). *South Asia regional profile: Drugs and crime, 2005*. New Delhi: United Nations Office on Drugs and Crime, Regional Office for South Asia.

Ambekar, A., & Tripathi, B. M. (2006). *Knowledge, attitude, behaviour and practices of drug users in India*. Report submitted to Society for Promotion of Youth and Masses under the peer-led intervention—supported by the Department for International Development (DFID), New Delhi.

Ambekar, A., & Tripathi, B. M. (2007). *Size estimation of IDU at 300 sites in India.* Report submitted to Society for Promotion of Youth and Masses under the peer-led intervention—supported by DFID, New Delhi.

Ambekar, A., & Tripathi, B. M. (2008). *Size estimation of injecting drug use in Punjab and Haryana*. New Delhi: Joint United Nations Programme on HIV/AIDS and Society for Promotion of Youth and Masses.

Ambwani, P. N., & Gilada, I. S. (1998). *Dry alcohol days during festivals to prevent HIV/AIDS*. XII International Conference on AIDS, Geneva. AIDSLINE ICA 12/98410386.

Centers for Disease Control and Prevention. (1998). Management of possible sexual, injecting-drug-use, or other nonoccupational exposure to HIV, including consid-

erations related to antiretroviral therapy. *Morbidity and Mortality Weekly Report* (MMWR), *50*(No. RR-17), 1–14.

Correa, M., & Gisselquist, D. (2006, November). Routes of HIV transmission in India: Assessing the reliability of information from AIDS case surveillance. *International Journal of STD and AIDS, 17*(11), 731–735.

Das, H. K., Borkakoty, B. J., Mahanta, J., Medhi, G. K., & Chelleng, P. K. (2007, September–October). Hepatitis C virus infection and risk behaviors among injection drug users of Nagaland. *Indian Journal of Gastroenterology, 26*(5), 253–254.

Datta, S., Banerjee, A., Chandra, P. K., Mahapatra, P. K., Chakrabarti, S., & Chakravarty, R. (2006, December). Drug trafficking routes and hepatitis B in injection drug users, Manipur, India. *Emerging Infectious Diseases, 12*(12), 1954–1957.

Dhawan, A. (1998). Traditional use. In R. Ray (Ed.), *South Asia drug demand reduction report 1998*(pp. 44–45). New Delhi: United Nations Office on Drugs and Crime, Regional Office for South Asia.

Dhawan, A., & Sunder, S. (2008, March). Opioid substitution therapy: The Indian experience. In A. Ambekar & B. M. Tripathi (Eds.), *Drug abuse: News-n-views* (Harm reduction: Prevention of HIV among drug users) (pp. 8–10). New Delhi: National Drug Dependence Treatment Centre, All India Institute of Medical Sciences.

Dole, V. P., & Nyswander, M. (1965, August). A medical treatment for diacetylmorphine (heroin) addiction: A clinical trial with methadone hydrochloride. *Journal of the American Medical Association, 23*(193), 646–650.

Gibson, D. R., Flynn, N. M., & Perales, D. (2001). Effectiveness of syringe exchange programmes in reducing HIV risk behaviour and HIV seroconversion among injecting drug users. *AIDS, 15*(11), 1329–1341.

Gisselquist, D., & Correa, M. (2006, November). How much does heterosexual commercial sex contribute to India's HIV epidemic? *International Journal of STD and AIDS, 17*(11), 736–742.

Gowing, L., Farrell, M., Bornemann, R., & Ali, R. (2004, October 18). Substitution treatment of injecting opioid users for prevention of HIV infection. *Cochrane Database of Systematic Reviews* (4), CD004145.

Hangzo, C., Chatterjee, A., & Sarkar, S. (1997). Reaching beyond the hills: HIV prevention among injecting drug users in Manipur, India. *Addiction, 92*(7), 813–820.

Hunt, N. (2003). *A review of the evidence-base for harm reduction approaches to drug use.* Forward thinking on drugs: A release initiative. Retrieved December 2, 2008, from http://www.forward-thinking-on-drugs.org/review2-print.html

International Harm Reduction Association. (2006). *What is harm reduction?* Retrieved December 2, 2008, from http://www.ihra.net/Whatisharmreduction

Jindal, N., Arora, U., & Singh, K. (2008). Prevalence of human immunodeficiency virus (HIV), hepatitis B virus, and hepatitis C virus in three groups of populations at high risk of HIV infection in Amritsar (Punjab), Northern India. *Japanese Journal of Infectious Diseases, 61*(1), 79–81.

Joint United Nations Programme on HIV/AIDS. (2007). *Fact-sheet: The process behind India's revised AIDS estimates*. Geneva: Author.

Joint United Nations Programme on HIV/AIDS and Family Health International. (2003). *Estimating the size of populations at risk for HIV: Issues and methods*. Geneva and Washington, DC.

Joint United Nations Programme on HIV/AIDS and World Health Organization. (2004). *Making HIV prevalence and AIDS estimates*. UNAIDS/WHO Working Group on Global HIV/AIDS and STI Surveillance. Retrieved December 2, 2008, from www.who.int/entity/hiv/strategic/en/neff_walker_estimations methods.ppt

Kermode, M., Longleng, V., Singh, B. C., Hocking, J., Langkham, B., & Crofts, N. (2007). My first time: Initiation into injecting drug use in Manipur and Nagaland, north-east India. *Harm Reduction Journal, 4*: 19.

Kumar, M. S. (2008, March). Outreach services: The Indian experience. In A. Ambekar & B. M. Tripathi (Eds.), *Drug abuse: News-n-views* (Harm reduction: Prevention of HIV among drug users) (pp. 11–12). New Delhi: National Drug Dependence Treatment Centre, All India Institute of Medical Sciences.

Kumar, M. S., Mudaliar, S., & Daniels, D. (1998). Community based outreach HIV intervention for street-recruited drug users in Madras, India. *Public Health Reports, 113*(Suppl. 1), 58–66.

Lawyers Collective. (2007). *Legal and policy concerns related to IDU harm reduction in SAARC countries: A review commissioned by UNODC*. New Delhi: Lawyers Collective HIV/AIDS Unit and United Nations Office on Drugs and Crime.

Lenton, S., & Single, E. (1998). The definition of harm reduction. *Drug and Alcohol Review, 17*, 213–220.

Longkumer, M., Shrivastava, H. C., & Murugesan, P. (2000). *Alcohol abuse and risky sexual behavior among the indigenous college students in Shillong, India*. Mumbai: International Institute for Population Sciences.

Mattick, R. P., Breen, C., Kimber, J., & Davoli, M. (2003). Methadone maintenance therapy versus no opioid replacement therapy for opioid dependence. *Cochrane Database of Systematic Reviews* (2), CD002209.

Mattick, R. P., Kimber, J., Breen, C., & Davoli, M. (2003). Buprenorphine maintenance versus placebo or methadone maintenance for opioid dependence. *Cochrane Database of Systematic Reviews* (2), CD002207.

McLellan, T., Lewis, D., O'Brien, C., & Kleber, H. (2000). Drug dependence, a chronic mental illness: Implications for treatment, insurance, and outcomes evaluation. *Journal of the American Medical Association, 284*(13), 1689–1695.

Ministry of Health and Family Welfare, Government of India. (2002). *National health policy 2002*. Retrieved from http://mohfw.nic.in/np2002.htm

Monitoring the AIDS Pandemic. (2004). *AIDS in Asia: Face the facts*. Monitoring the AIDS Pandemic (MAP) Network. Retrieved from http://www.mapnetwork.org/docs/MAP_AIDSinAsia2004.pdf

National AIDS Control Organization. (2001). *National Baseline High Risk and Bridge Population Behavioural Surveillance Survey 2001. PART 1: Female sex workers*

and their clients. Ministry of Health and Family Welfare, Government of India. Retrieved December 2, 2008, from www.nacoonline.org

National AIDS Control Organization. (2002a). *National Baseline High Risk and Bridge Population Behavioural Surveillance Survey 2002. PART 2: Men who have sex with men and injecting drug users*. Ministry of Health and Family Welfare, Government of India. Retrieved December 2, 2008, from www.nacoon line.org

National AIDS Control Organization. (2002b). *National AIDS prevention and control policy*. Ministry of Health and Family Welfare, Government of India. Retrieved December 2, 2008, from http://www.nacoonline.org/program.htm

National AIDS Control Organization. (2006). *National AIDS Control Programme Phase III (2007–2012): Strategy and implementation plan*. Ministry of Health and Family Welfare, Government of India. Retrieved December 2, 2008, from http://www.nacoonline.org/program.htm

National AIDS Control Organization. (2007a). *HIV sentinel surveillance and HIV estimation, 2006*. Ministry of Health and Family Welfare, Government of India. Retrieved December 2, 2008, from www.nacoonline.org

National AIDS Control Organization. (2007b). *Endline Behavioural Surveillance Survey 2006*. Ministry of Health and Family Welfare, Government of India. Retrieved December 2, 2008, from www.nacoonline.org

Needle, R. H., Burrows, D., Friedman, S. R., Dorabjee, J., Touzã, G., Badrieva, L., et al. (2005). Effectiveness of community-based outreach in preventing HIV/AIDS among injecting drug users. *International Journal of Drug Policy, 16*(1), 45–57.

Ngully, P. (2008, March). Needle syringe exchange program (NSEP): Experience from Nagaland. In A. Ambekar & B. M. Tripathi (Eds.), *Drug abuse: News-n-views* (Harm reduction: Prevention of HIV among drug users) (p. 15). New Delhi: National Drug Dependence Treatment Centre, All India Institute of Medical Sciences.

Panda, D. (2007). Drug de-addiction programmes in India. In R. Lal & S. Gupta (Eds.), *Substance use disorders: A manual for nursing personnel* (pp. 10–14). New Delhi: National Drug Dependence Treatment Centre, All India Institute of Medical Sciences.

Panda, S., Bijaya, L., Sadhana, N., Devi, S. N., Foley, E., Chatterjee, A., et al. (2001, July–August). Interface between drug use and sex work in Manipur. *National Medical Journal of India, 14*(4), 209–211.

Panda, S., Chatterjee, A., Bhattacharya, S. K., Manna, B., Singh, P. N., Sarkar, S., et al. (2000, July). Transmission of HIV from injecting drug users to their wives in India. *International Journal of STD and AIDS, 11*(7), 468–473.

Panda, S., Kumar, M. S., Lokabiraman, S., Jayashree, K., Satagopan, M. C., Solomon, S., et al. (2005). Risk factors for HIV infection in injection drug users and evidence for onward transmission of HIV to their sexual partners in Chennai, India. *Journal of Acquired Immune Deficiency Syndrome, 39*, 9–15.

Panda, S., Kumar, M. S., Saravanamurthy, P. S., Mahalingam, P., Vijaylakshmi, A., Balakrishnan, P., et al. (2007). Sexually transmitted infections and sexual prac-

tices in injection drug users and their regular sex partners in Chennai, India. *Sexually Transmitted Diseases, 34*(4), 250–253.

Rao, R., & Khumukcham, S. (2008, March). Prevention of drug related HIV: Role of NACO. In A. Ambekar & B. M. Tripathi (Eds.), *Drug abuse: News-n-views* (Harm reduction: Prevention of HIV among drug users) (pp. 17–18). New Delhi: National Drug Dependence Treatment Centre, All India Institute of Medical Sciences.

Ray, R. (1998). *South Asia drug demand reduction report 1998*. New Delhi: United Nations Drug Control Programme (UNDCP) Regional Office for South Asia.

Ray, R., & Ambekar, A. (2005). *Drug abuse and HIV/AIDS situation in South Asia: Vulnerabilities and responses*. New Delhi: Report submitted to the United Nations Office on Drugs and Crime, Regional Office for South Asia.

Reid, G., & Costigan, G. (2002, January). *Revisiting the hidden epidemic: A situation assessment of drug use in Asia in the context of HIV/AIDS*. Australia: The Centre for Harm Reduction, The Burnet Institute.

Ritter, A., & Cameron, J. (2005). A systematic review of harm reduction. DPMP [Drug Policy Modeling Project], Monograph Series No. 06. Fitzroy, Australia: Turning Point Alcohol and Drug Centre.

Saraswathi, K., & Dutta, A. (2007, April). Study of human immunodeficiency virus and HCV infections in intravenous drug users in Mumbai. *Indian Journal of Microbiology, 25*(2), 174–175.

Sarkar, K., Bal, B., Mukherjee, R., Chakraborty, S., Niyogi, S. K., Saha, M. K., et al. (2006). Epidemic of HIV coupled with hepatitis C virus among injecting drug users of Himalayan West Bengal, Eastern India, Bordering Nepal, Bhutan, and Bangladesh. *Substance Use and Misuse, 41*(3), 341–352.

Sarkar, S., Das, N., Panda, S., Naik, T. N., Sarkar, K., Singh, B. C., et al. (1993). Rapid spread of HIV among injecting drug users in north-eastern states of India. *Bulletin of Narcotics, 45*(1), 91–105.

Sarkar, S., Mookerjee, P., Roy, A., Naik, T. N., Singh, J. K., Sharma, A. R., et al. (1991, September). Descriptive epidemiology of intravenous heroin users—a new risk group for transmission of HIV in India. *Journal of Infections, 23*(2), 201–207.

Sarkar, S., Panda, S., Sarkar, K., Hangzo, C. Z., Bijaya, L., Singh, N. Y., et al. (1995, July–September). A cross-sectional study on factors including HIV testing and counselling determining unsafe injecting practices among injecting drug users of Manipur. *Indian Journal of Public Health, 39*(3), 86–92.

SHARAN. (2007). *A national scale up of IDU interventions across nine states in India: Rapid Assessment*. New Delhi: Author.

Sharma, A. K., Aggarwal, O. P., & Dubey, K. K. (2002, May). Sexual behavior of drug-users: Is it different? *Preventive Medicine, 34*(5), 512–515.

Sharma, A. K., & Chaubey, D. (1996). Risk factors in sexually transmitted diseases. *Indian Journal of Sexually Transmitted Diseases, 17*, 8–10.

Sharma, M., Panda, S., Sharma, U., Singh, H. N., Sharma, C., & Singh, R. R. (2003). Five years of needle syringe exchange in Manipur, India: Programme and contextual issues. *International Journal of Drug Policy, 14*, 407–415.

Singh, S. K., Schensul, J. J., & Gupta, K. (2004, July 11–16). *Alcohol use and risky sexual behavior among adolescents in low income slum communities in Mumbai, India*. Abstract No. C11731. Bangkok, Thailand: International Conference on AIDS.

Tripathi, B. M., Malhotra, S., & Sharma, H. K. (2004). *Alcohol and high-risk sexual behaviour.* New Delhi: National Drug Dependence Treatment Centre, All India Institute of Medical Sciences and World Health Organization.

United Nations Office on Drugs and Crime. (2007). *World drug report 2007*. Vienna, Austria: Author.

United Nations Office on Drugs and Crime. (2008). *Reducing adverse health and social consequences of drug abuse: A comprehensive approach—Discussion Paper.* Vienna, Austria: Author.

United Nations Office on Drugs and Crime, Regional Office for South Asia, and Ministry of Social Justice and Empowerment. (2002a). *Drug Abuse Monitoring System*. New Delhi: Authors.

United Nations Office on Drugs and Crime, Regional Office for South Asia, and Ministry of Social Justice and Empowerment. (2002b). *A Rapid Assessment Survey of Drug Abuse in India*. New Delhi: Authors.

United Nations Office on Drugs and Crime, Regional Office for South Asia, and Ministry of Social Justice and Empowerment. (2004a). *The extent, pattern and trends of drug abuse in India: National Survey*. New Delhi: Authors.

United Nations Office on Drugs and Crime, Regional Office for South Asia, and Ministry of Social Justice and Empowerment. (2004b). *Injecting drug use and HIV in India: An emerging concern*. New Delhi: Authors.

United Nations Office on Drugs and Crime, Regional Office for South Asia, and Ministry of Social Justice and Empowerment. (2005). *Drug use in the north eastern states of India*. New Delhi: Authors.

Vaswani, M., & Desai, N. G. (2004). HIV infection and high-risk behaviors in opioid dependent patients: The Indian context. *Addictive Behaviors, 29*(8), 1699–1705.

Vaswani, M., & Rao, R. (2006). Risk behaviors and HIV infection in alcohol/drug abuse: Indian scenario. *Addictive Disorders and Their Treatment, 5*(1), 35–40.

Verma, R., Sharma, S., Singh, R., Rangaiyan, G., & Pelto, J. (2003, May–June). Beliefs concerning sexual health problems and treatment seeking among men in an Indian slum community. *Culture Health and Sexuality, 5*(3), 265–276.

Wodak, A. (2004). *Effectiveness of sterile needle and syringe programming in reducing HIV/AIDS among injecting drug users*. Evidence for Action Technical Papers. Geneva: World Health Organization.

This page intentionally left blank

Part III

PSYCHOBIOLOGIES

This page intentionally left blank

Chapter **15**

Alcohol Abuse: Impact on Vital Brain Functions and Societal Implications

Mary Theresa Webb, PhD

Recent neuroscientific research provides an important scientific and societal basis for raising an international alarm about the risk of binge or heavy alcohol drinking. Heavy alcohol use damages sections of the brain that modulate emotions, controls intellectual functioning, and thus may contribute to an increase in societal violence, family disruption, and diseases, such as HIV/AIDS.

Heavy drinking before the brain is fully matured arrests the development of the frontal lobe, especially the area of executive function, according to neuroscientific research. For adults, heavy drinking over a long period of time risks the loss of cognitive abilities and increases the risk of having cardiovascular incidents and alcoholic dementias, taxing health care systems.

Statistics from a 2006 European Union Study show that countries who have lowered their legal drinking age to 18 or 16 years old show an increase in binge drinking in these age groups at the same time that 100 American college presidents have suggested lowering the drinking age from 21 to 18 years old (Amethyst Initiative, 2008).

ALCOHOL AND BRAIN RESEARCH

The Centers for Disease Control (CDC) has classified alcohol (ethanol), the addictive drug in all alcoholic beverages, as a central nervous system depressant. Alcohol abuse has plagued humankind since people learned to turn barley and corn into mead. Even today, in many rural regions of Africa, villagers brew maise and turn it into 80-proof alcoholic drinks. In America, until the turn of the twentieth century, a bottle of alcohol was kept in the family medicine

cabinet and used as an anesthetic in surgical procedures. Today, in twenty-first-century America, about 7.7 percent of the population are dependent on or abuse alcohol (Lemonick & Park, 2007, p. 44), one-third of all teenagers binge drink (Kann, et.al, 2000), nearly half of all college students binge drink (Leinwand, 2007), and an uncounted number of young, middle-aged, and older adults abuse alcohol. A *binge* is a pattern of drinking alcohol that brings blood alcohol concentration (BAC) to 0.08 gram-percent or above. For a typical adult, this pattern corresponds to consuming five or more drinks (male), or four or more drinks (female), in about two hours. According to estimates by the U.S. Substance Abuse and Mental Health Services Administration (SAMSHA), 3 to 25 percent of older adults are classified as heavy alcohol users and 2.2 to 9.6 percent abuse alcohol (SAMSHA & National Council on Aging, 1998, p. 4).

Alcohol intake in small and infrequent doses can be flushed out of the body through the action of a catalytic enzyme, alcohol dehydrogenese, that metabolizes alcohol into its constituent chemical components to be eliminated from the body, thus reducing the risk of any toxic residual effects. However, when alcohol rushes into the body through excessive drinking, it is like torrential rain causing a river to rise over its banks, saturating the ground and destroying whatever is in its path. In the case of rapid or excessive intake of alcohol, instead of the alcohol being metabolized and flushed out of the body, the excessive alcohol floods the bloodstream and goes into the brain. Although most mature adults can tolerate consuming 58 liters annually or no more than two drinks a day with little or no effect on cognitive abilities (with the exception of women who have a lower amount of the enzyme alcohol dehydrogenese), alcohol is still toxic to neurons if the drug reaches the brain (Lezak, 1995, pp. 250–251). The more alcohol consumed, depending on age and sex, the greater the risk of permanent brain damage. Areas most affected are the frontal lobe or prefrontal cortex and the cerebellum, as shown in Figure 15.1 (Oscar-Berman and Marinkovic, 2004, p. 4).

The frontal area of the brain contains the neurons that regulate emotions as well as executive functioning, or attention, abstract reasoning, organization, mental flexibility, planning, self-monitoring, and the capacity to use external clues to govern behavior. The prefrontal cortex in a human's brain is 120 percent larger than that of other mammals, giving humans higher intellectual functions than any other mammal. However, alcohol abuse impairs and often destroys those intellectual abilities (Phil, 2003).

Synapses and Neurotransmitter Conductivity

Although it has been known for several decades that drugs such as alcohol replace the neurotransmitter, dopamine, that permits the flow of neurons in

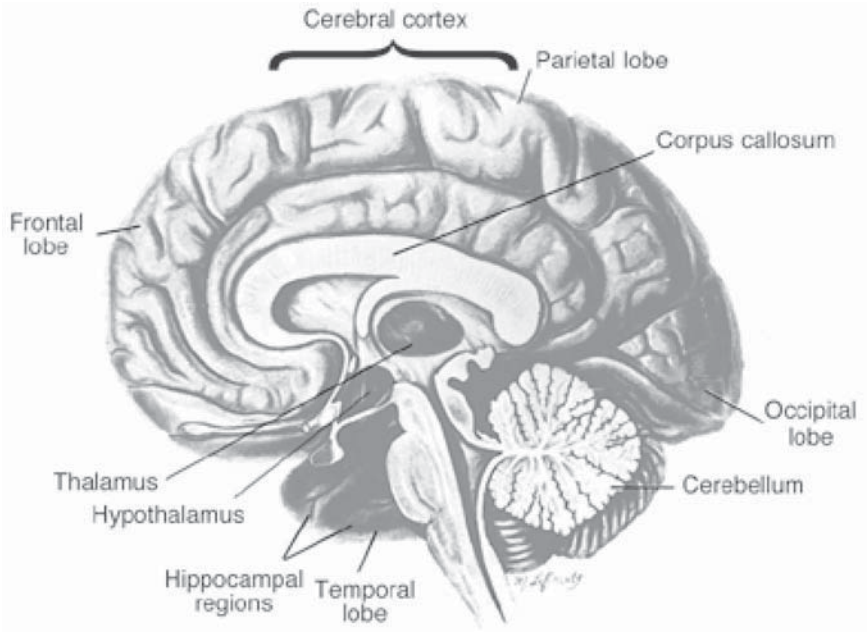

FIGURE 15.1 Alcohol abuse: Impact on the brain. From "Alcoholism and the Brain: An Overview," in *The Neurobiology of Addiction: Introduction to the Brain and the Reward Pathway and Addiction*, by M. Oscar-Berman and K. Marinkovic, 1997, Washington, DC: National Institute on Alcohol Abuse and Alcoholism (NIAAA), p. 4.

the synapse from one nerve ending to another, it has only been in the last 10 years that magnetic resonance imaging (MRI) research studies can show colored pictures of the brain. See Figure 15.2 for how neurotransmitters flow from one synapse to another.

Patterns of brain development and the impact of early onset alcohol use or long-time alcohol abuse and the resultant brain damage have been thus recorded. Through time lapse imaging, researchers at the U.S. National Institute of Mental Health (NIMH) and the University of California showed that the prefrontal cortex doesn't fully develop until young adulthood, for males that is about the age of 25; for females, about the age of 22 (Gogtay, Rapoport, Thompson, Toga, Lusk, and colleagues, 2004). If heavy alcohol use arrests this development, then important executive functions in the prefrontal cortex cannot fully mature.

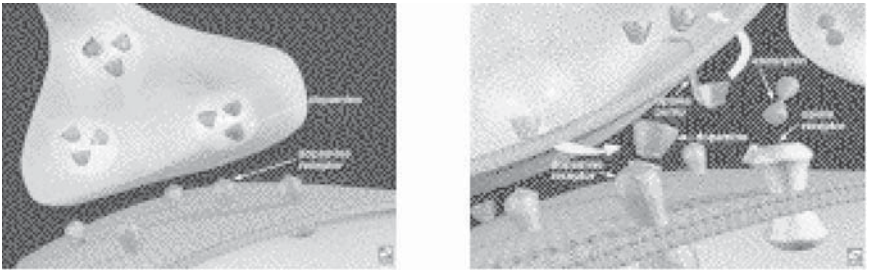

FIGURE 15.2 Synapses and neurotransmitter conductivity. From *Teaching Packets: The Neurobiology of Drug Addiction. Section 1: Introduction to the Brain*, National Institute on Drug Abuse, http://www.nida.nih.gov/pubs/teaching/Teaching2/Teaching2.html

CONCERNS FOR YOUTH

The 2006 European Union study (Anderson & Baumberg, 2006, p. 76) concludes that almost all 15- to 16-year-old students have drunk alcohol, beginning at 12.5 years of age, and start getting drunk at 14 years. Most countries studied show a rise in binge drinking with the prevalence of heavy drinking greatest among young boys (Grube, 2005, p. 3). The highest prevalence of drunkenness and binging among youth being recorded is in Denmark, the Netherlands, Germany, the Isle of Man, Ireland, and the United Kingdom. In countries that have a more positive view of alcohol and have lower legal drinking ages, youth binge drinking increases (p. 108). In this comprehensive report by the Prevention Research Center of the Pacific Insititute for Research and Evaluation prepared for the U.S. Department of Justice (update on ESPAD and MTF surveys, 2003), drinking rates and alcohol-related problems among teenagers (10th graders) in all EU countries and the United States were compared (see Figure 15.3). With the exception of Turkey, the rate of binge drinking was lowest in the United States. Although this comparative study did not prove that the higher legal drinking age in the United States was a deterrent, the fact that binge drinking among youth was lower than in the EU indicates that the U.S. higher legal drinking age (21) should be considered as one of the factors for this finding. In fact, the author concludes, based on his analysis (Grube , 2005), that the perception that American youth drink more and have more problems than their European counterparts is not true. According to this study there is then no support for changing the minimum drinking age in the United States.

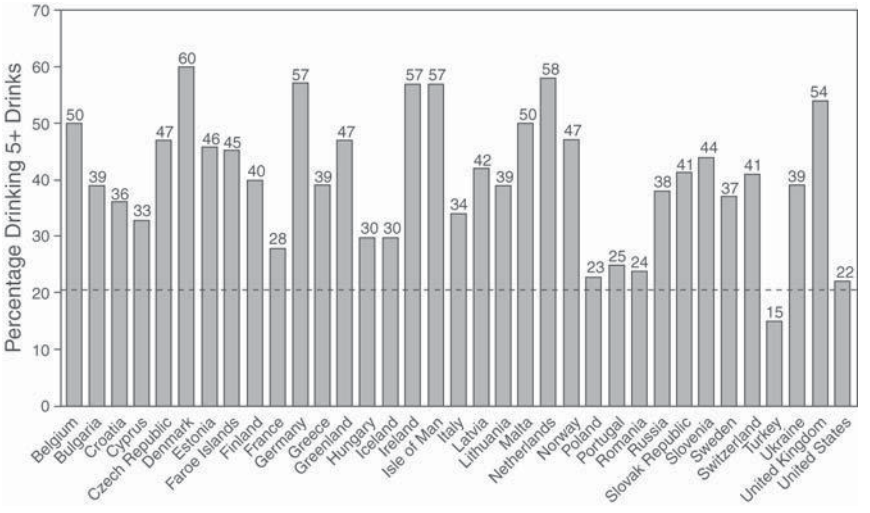

FIGURE 15.3 Prevalence of heavy drinking in the past 30 days: United States and Europe. From Joel Grube of the Prevention Research Center, Pacific Institute for Research and Evaluation, May, 2005 in *Youth Drinking Rates and Problems: A Comparison of European Countries and the United States*, for the U.S. Department of Justice, Office of Juvenile Justice and Delinquency Prevention, Update of 2003 ESPAD and 2003 MTF surveys, Calverton, MD.

The signers of the Amethyst Initiative recognize that binge drinking is a serious problem, with nearly half of America's 5.4 million full-time college students binge drinking once a month, (Leinwand, 2007) many of whom (30% of high school youth) started binge drinking in their high school years (Kann et al., 2000), 2007, p. 1). However, changing the legal age to 18 will not solve this problem. Aggressive measures need to be taken to change the drinking culture on college campuses.

If early alcohol abuse (before the age of 25) damages the brain's intellectual functioning, the ability to think, reason, and make wise decisions, then educating youth to be responsible and intelligent citizens is compromised. Not only might alcohol abuse by adolescents and youth under the age of 25 cause arrested brain development and potential permanent damage, but the resultant brain damage in turn will probably lead to early alcohol and other drug dependency (addiction) and chronic relapse. Improper executive functioning of the prefrontal cortex makes recovery difficult because recovery involves learning to control triggers and cravings. An alcohol-impacted brain also shrinks in size

and has less white matter, leading to cognitive impairment in later life (EU [European Union] Commission, 2006 p. 150).

Occasional or weekend binge drinking, five or six drinks in a row whose purpose is to become intoxicated, popular among teenagers and college students, has long-lasting implications not only for education but for the future of developed and developing countries. As noted in the 2007 U.S. Surgeon General's Report (SAMHSA, 2007), the long-term dangers to America's youth have rung enough alarm bells that the U.S. government has proposed remedial and preventative actions, as has the European Institute of Alcohol Studies in its report to the European Union for implementing public health policies.

CONCERNS FOR OLDER ADULTS

Underdeveloped brains of youth and young adults under the age of 25 are not the only brains affected by chronic heavy alcohol consumption. In a study of male veterans receiving treatment at the Durham Veterans Affairs Medical Center (in the US), researchers Swartzwelder, Zinn, and Stein (2004) administered memory and executive function tests to both alcohol-dependent and age-appropriate primary care outpatients. The alcohol-dependent group scored lower on abstract reasoning, memory, and effectiveness of timed tasks. Although some U.S. National Institute on Alcohol Abuse and Alcoholism (NIAAA) researchers indicate that moderate alcohol use (one drink a day for individuals over 65) reduces the risk of cardiovascular disease and dementia among older adults (Gunzerath, Faden, Zakhari, & Warren, 2004), other researchers (Oscar-Berman & Marinkovic, 2004, p. 5) demonstrated that older adults who had been alcohol users or alcohol abusers as youth had significant brain shrinkage and right hemisphere weakening and were thus more prone to hemorrhagic strokes. Note the shrinkage in cortical grey matter in Figure 15.4.

This condition, known as *alcoholic dementia*, shows cognitive deterioration typically associated with the prefrontal cortex or frontal lobe (Lezak, 1995, p. 255). When years of chronic drinking causes such a stroke in the weakened part of the brain, the frontal lobe in the right brain hemisphere is most often impaired. The affected person can no longer make judgments, use abstract reasoning, or plan and carry out activities without supervision. An estimated 2.3 million older Americans who have or have had problems with alcohol also have this kind of dementia, making it difficult for physicians and caregivers to distinguish between the alcohol-induced dementia and Alzheimer's disease. Two kinds of these dementias, vascular dementia and Wernick-Korsakoff syndrome, are often misdiagnosed as Alzheimer's disease. Korsakoff psychosis (WKS) is a more severe form of alcohol-induced dementia that includes amnesia and

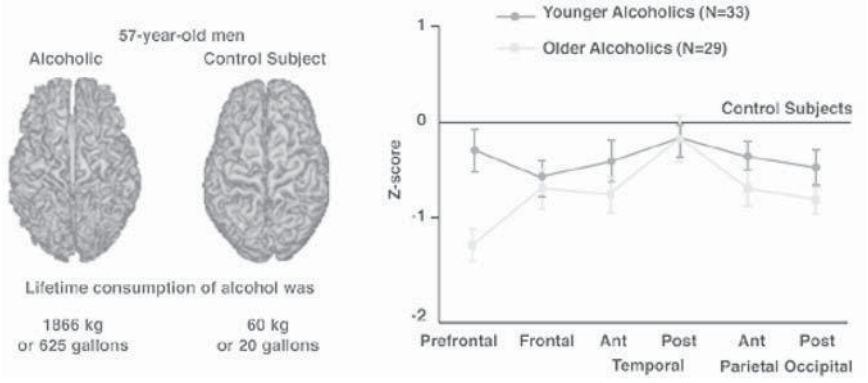

FIGURE 15.4 Cortical gray matter volumes. From "Alcoholism and the Brain: An Overview," in *The Neurobiology of Addiction: Introduction to the Brain and the Reward Pathway and Addiction*, by M. Oscar-Berman and K. Marinkovic, 1997, Washington, DC: National Institute on Alcohol Abuse and Alcoholism (NIAAA). Source: Pfefferbaum et al, 1997.

has an accompanying thiamine deficiency with even more pronounced brain shrinkage and damage. Those older adults so affected have very little ability to change self-destructive behaviors.

CONCERN FOR AN INCREASE IN VIOLENCE

According to the most recent studies in the report from the European Union Commission (Anderson & Baumberg, 2006) concerning alcohol and its societal impact, the authors have some evidence that alcohol abuse is linked with crime, especially violent crime. Heavy alcohol use has been associated with about one-quarter of all assaults and harm done through domestic violence and with about 16 percent of child abuse and neglect. Four out of 10 homicides are attributed to alcohol use (p. 211). These statistics do not take into account unintentional drunk-driving accidents, one of the leading causes of traffic fatalities in all countries.

Because alcohol damages the prefrontal lobe area of the brain where emotions are modulated, it naturally follows that this accounts for the lack of anger control, road rage, rage against women and children, that is, spousal and child abuse. Alcohol is also a central nervous system depressant so that heavy alcohol use is a significant factor in intentional deaths, that is, suicides or rage against oneself.

RECOMMENDATIONS FOR ACTION

The 2007 U.S. Surgeon General's Call for Action recognizes the many developmental and environmental influences on youth drinking and recommends that school officials, parents, and community members come together to determine preventative actions to stop early youth use of alcohol, undertake brief interventions to begin treatment early, and change the way the treatment community treats youth who are already impaired. Some of these are already in place in the United States and may be the reason that the United States has a lower rate of heavy drinking among youth than countries in the European Union. However, as evidenced in the July 2008 Amethyst Initiative, college administrations are still struggling to curb the alarming increase in heavy drinking among college students. Both parents and youth should weigh what potential damage alcohol abuse will do to education and career plans and make choices to avoid those institutions of higher learning whose administrations and alumni continue to permit heavy undergraduate drinking behavior and don't undertake aggressive measures to curb such behavior and treat those so affected. Unfortunately, alcoholic beverage companies contribute to the problem as they target underage youth by sponsoring college sporting events.

Health care professionals, clinicians, and social workers need more training in how to utilize alcohol screening instruments with patients and clients, incorporating them into routine health examinations and patient histories. They also need more training in brief interventions to arrest the alcohol abuse prior to further brain shrinkage and alcohol-induced dementias. Newer treatment options that are based on results of neuropsychological testing to determine the extent of executive function impairment are also needed for both older adults and youth who have abused or are addicted to alcohol and may be in recovery to determine appropriate treatment planning.

There is an absence of robust comparable and comprehensive data on the relation between alcohol and crime. More research is needed in this area (Anderson & Baumberg, 2006, p. 200). Although it has long been estimated that 70–90 percent of those incarcerated have committed a crime while under the influence of alcohol or other drugs, these are only estimates. Interviewing prisoners and asking them whether they would have committed the crime if they had not been drunk was one methodology recommended and used in the EU Commission study. Twenty-four percent of those interviewed in a Canadian study acknowledged they had committed crimes when they were drunk at the time of the crime. However, this low figure is probably due to prisoners still in denial, prisoners high on other drugs, and cultural attitudes toward drinking.

Providing facts and dispelling myths is one of the best ways to change public attitudes and cultural practices surrounding alcohol abuse and addiction. Countries around the world can no longer afford to ignore the public health crisis caused by heavy alcohol use. Smoking cigarettes has become unpopular and now has a negative cultural image in many Western countries due to the efforts of alarmed government public health officials. These enlightened officials have acted upon the knowledge of the dangers of passive smoke and have undertaken aggressive intervention and treatment strategies to help people quit. The same aggressive public health campaign can also change the positive image of heavy alcohol use to a negative one and help those who are abusing or addicted to alcohol to abstain. The long-term biological and societal consequences are too great to ignore. Slogans such as this one, *My brain matters, does yours?*, could change the way the public views alcohol use.

REFERENCES

Amethyst Initiative. http://www.amethystinitiative.org

Anderson, P., & Baumberg, B. (2006). *Alcohol in Europe: A public health perspective*. London: Institute of Alcohol Studies.

Centers for Disease Control (CDC). http://www.cdc.gov/alcohol/faqs.htm#1 Gogtay, N., Rapoport, J., Thompson, P., Toga, A., Lusk, L. Vaituzis, C., et al. (2004, May 17). Imaging study shows brain maturing. *Science News*. Retrieved February 22, 2008, from www.nimh.nih.gov/science-news/2004/pressrelease

EU (European Union) Commission. (2006). Anderson, P., and Blaumberg, B. Alcohol in Europe, a public health perspective. Institute of Alcohol Studies, UK.

Grube, J. (2005). *Youth drinking rates and problems: A comparison of European countries and the United States*. Calverton, MD: Prevention Research Center, Pacific Institute for Research and Evaluation in support of Office of Juvenile Justice and Delinquency Prevention. www.udetc.org

Gunzerath, L., Faden, V., Zakhari, S., & Warren, K. (2004). Alcoholism: Clinical and experimental research. National Institute on Alcohol Abuse and Alcoholism Report on Moderate Drinking. *Alcoholism Clinical and Experimental Research, 28*(6), 829–847.

Kann, L. S., Kinchen, B., Williams, J., Ross, R., Lowry, J., Grunbaum, J., et al. , (2000, June 9). *Youth risk behavior surveillance—United States, 1999*. Retrieved February 22, 2008, from www.marininstitute.org/youth/alcoho0l_youth.htm

Leinwand, D. (2007). College drug use, binge drinking rise. *USA Today*. Retrieved February 22, 2008, from. www.usatoday.com/news/nation/2007-03-15-college-drug-use_N.htm

Lemonick, M. D., & Park, A. (July 16,2007). The science of addiction. *Time*, 42–48.

Lezak, M. D. (1995). Alcohol related disorders. *Neuropsychological Assessment* (3rd ed.), *Theory and practice of neuropsychological assessment* (pp. 246–258). New York: Oxford University Press.

Oscar-Berman, M., & Marinkovic, K. (1997). Alcoholism and the brain: An overview. The neurobiology of addiction: Introduction to the brain and the reward pathway and addiction. *NIAAA. Office of Science Education.* Slides retrieved February 27, 2008.

Oscar-Berman, M., & Marinkovic, K. (2004). Alcoholism and the brain: An overview. *NIAAA*. Retrieved February 22, 2008, from http://pubs.niaaa.nih.gov/publications/arh27-2/125-133.htm

Phil, R. O., & Peterson, J. B. (2003, May 14). Alcohol impairs executive cognitive functioning much longer than expected. *Alcoholism: Clinical and Experimental Research.* Retrieved February 22, 2008, from www.eurkalert.org/pub_releases/2003-05.ace-aie050703

Substance Abuse and Mental Health Services Administration. (2007). *The Surgeon General's call to action to prevent and reduce underage drinking.* Retrieved February 27, 2008, from www.surgeongeneral.gov

Substance Abuse and Mental Health Services Administration & National Council on Aging. (1998). *Substance abuse among older adults*.

Sullivan, E. V. (2003). Alcohol may compound its damaging effects through frontocerebellar circuitry. *Alcoholism: Clinical and Experimental Research.* Retrieved February 22, 2008, from www.eurkalert.org/pub_releases/2003-09/ace-amc090803.php

Swartzwelder, H. S., Zinn, S., & Stein, R. (2004). *Certain components of the brain's executive functions are compromised early in abstinence.* Retrieved December 30, 2007, from medicalnewstoday.com

Chapter **16**

Addiction and Cognitive Control

Vicki W. Chanon, PhD, and Charlotte A. Boettiger, PhD

Substance use disorder, or addiction, is a neurobehavioral disorder characterized by a compulsion to ingest psychoactive substances (such as alcohol, prescription narcotics, nicotine, opiates, stimulants, and marijuana) despite repeated serious negative consequences. Addiction is a widespread problem, occurring among all ethnic groups and socioeconomic classes. Under normal conditions, our base motivations of hunger, thirst, and healthy fear of death or bodily harm are so powerful that they ensure that our basic needs for food, water, and physical safety are met. The need for emotional companionship, financial security, and sexual gratification also normally motivate our actions. However, for those with substance use disorders, some or all of these drives become secondary to obtaining and consuming their abused substance. This misdirection of motivation can lead to bodily harm, loss of vital relationships, loss of employment, loss of shelter, and even death. Observing addiction from the outside can be extremely frightening and puzzling. Why do addicts risk such harm? Evidence suggests that brain circuits that normally guide goal-directed behavior are malfunctioning in addicts. Recent evidence also suggests that perturbations of cognitive control are hallmarks of addiction. These cognitive aspects of addiction and their neural bases are relatively unexplored, but the advent of cognitive neuroscience tools, which allow exploration of brain activity in humans, may allow us to make quantum leaps in our understanding of the brain mechanisms of substance use disorders in the near future.

HOW DOES ADDICTION RELATE TO COGNITIVE CONTROL?

Cognitive control is a broad term describing processes essential to our survival and well-being, encompassing one's ability to act and react in a situation-specific manner (Miller & Cohen, 2001). Appropriate reactions to a specific stimulus can vary greatly from one time or situation to another. For example, a large cobra behind the glass at the zoo may evoke intrigue and cause you to stop at the exhibit to look closer, while the same snake encountered on a wooded hiking trail may evoke fear and cause you to run away. This flexibility in our cognitive control not only allows us to make key decisions through complex and changing contexts, but also allows us to direct our attention to what we find significant and to create important memories of and associations with those things. Disruptions in this cognitive control, or "dysexecutive" behaviors, are an important and defining characteristic in addictive disorder, as people with this disease are often unable to make situation-appropriate responses to stimuli or events.

Moreover, recent evidence suggests that at least two specific cognitive control faculties are functioning abnormally in addicts: attentional control and decision making. Specifically, addicts disproportionately direct their attentional resources toward stimuli associated with their abused substance. For example, the attention of an alcoholic is biased toward items, images, and words that he or she associates with drinking alcohol. This bias, which is directly related to the likelihood of relapse in abstinent individuals, may be the product of profound associative learning in addicts, whereby drug-related cues and the actions of drug seeking and drug taking are tightly linked within the brain (Robinson & Berridge, 2003). These associations are likely to underlie the intense cravings that present a major barrier to maintaining abstinence from drug use. In addition to attentional abnormalities, clinical evidence suggests that decision-making abnormalities exist among people with substance use disorders as well. For example, a number of laboratory studies consistently find that, across a broad spectrum of addictive disorders, addicts show an immediate reward bias; that is, they are more likely than nonaddict participants to choose a smaller immediate reward over a larger reward for which they would need to wait (Reynolds, 2006). This tendency holds true for various types of rewards, including the addict's drug of choice and monetary rewards. Laboratory-controlled gambling tasks have also linked abnormal decision making with addictive disorders (Bechara, 2005; Clark & Robbins, 2002). These cognitive processes (decision making, attentional control, and reinforcement-based associative learning) are all processes that fall under the general umbrella of execu-

tive function and are associated with frontal circuits within the brain. It is still unclear, though, whether these abnormalities always co-occur, or whether they are separate dysfunctions, each of which occurs in some addicts but not others. Some data suggest that the attentional biases may precede addictive disorders, but comparable data are not available in the decision-making domain. Therefore, it is not yet clear whether decision-making abnormalities contribute to the development of addictive disorders, or are instead a byproduct of addiction or prolonged drug abuse.

ADDICTION AND ATTENTIONAL BIAS

Numerous behavioral studies point to an abnormality in attentional allocation to drug cues in addicts. Attention is quickly captured by and/or held on items associated with an addict's substance of choice (Robbins & Ehrman, 2004). A variety of cognitive tasks have been used to explore and measure this bias, one of the most prevalent being the spatial cuing task. In this task, participants are presented with two visual stimuli (or "cues"), one on each side of the screen. Generally one of the cues, which could be words or images, is a drug-related stimulus while the other is neutral. After the cues disappear, a target item requiring some response by the participant appears in the same location that was occupied by one of the two cues. Addicts respond more quickly to targets when they appear in the location where the drug-related cue had appeared than when they appeared in the location of the neutral cue. This finding suggests that, in addicts, attention is preferentially captured or held by drug-related cues. Another task that has been used to measure drug-related bias is a modified Stroop task (MacLeod, 1991; Stroop, 1935). The original Stroop task presents words (including color names) displayed in different ink (or screen) colors, and tests participants on their speed of naming the font color. For example, if the word *red* was printed in blue ink, the correct response would be *blue*. Participants are generally slower at naming the ink color for color words that do not match the ink than they are at naming neutral, noncolor words, and color words written in a congruent font color. This "Stroop Effect" is thought to occur because attention devoted to reading the word interferes with naming the font color. The task has been modified to study drug-related attentional bias, by presenting drug-related words in varying ink colors, rather than color-related words. Such studies find that, in addicts, drug-related words demand more attentional resources than do neutral words.

While the majority of evidence for drug-related attentional bias comes from spatial cuing and modified Stroop studies, such bias has also been detected using other types of cognitive tasks. For example, change blindness tasks, in

which participants detect slight changes between two rapidly alternating visual scenes, demonstrate that addicts more readily detect drug-related changes in the scene. Another task, the attentional blink paradigm, has also recently been used to measure addiction-related attentional abnormalities. In this task, a stream of visual stimuli is rapidly presented, and participants are instructed to detect two specified target stimuli within the stream. When the first target (T1) and the second target (T2) are separated by about 200–400 milleseconds, participants are more likely to miss the second target because attentional resources are already allocated to processing T1. This phenomenon is referred to as the "attentional blink." If T1 is an addiction-related stimulus, addicts demonstrate a larger and more extended attentional blink. In contrast, when T2 is an addiction-related stimulus, an addict's attentional blink is reduced (Liu, Li, Sun, & Ma, 2008). Together, these studies indicate that, in addicts, words and images associated with their drug use have a heightened capacity to capture and hold attention, and that such stimuli interfere with attention to other things in their environment.

This heightened attention toward drug-related stimuli occurs in a wide variety of addictions, including cannabis, cocaine, opiates, tobacco, alcohol, caffeine, and even gambling. Attention abnormalities have been most thoroughly studied for the most commonly abused substances: tobacco and alcohol. Thus, studies exploring attention in alcohol and nicotine addictions have provided a greater understanding of some complex properties of addiction-related attentional bias. For example, data suggest that attentional bias toward tobacco cues requires the stimuli to be consciously perceived; when the smoking cues are presented too quickly or are masked by another stimulus, they do not affect attentional allocation. Data also show that demographic factors, like age and race, and mood factors, like depression, do not impact attentional bias toward alcohol cues in alcoholics. Studies of alcohol attentional bias have found that heavy social drinkers (but not light drinkers) show attentional bias toward alcohol cues, suggesting that such bias may serve as an early warning sign of alcohol addiction; these findings also bring up another possible explanation for the bias: stimulus familiarity. Previous cognitive psychology studies suggest that familiar or overlearned stimuli can affect attentional allocation (Chanon & Hopfinger, 2007). A contribution of familiarity effects to addiction-related attentional bias is suggested by an alcohol-related bias evident among staff members at a treatment center; however, the magnitude of the attentional bias toward alcohol stimuli was predicted by measures of alcohol dependence among the staff members, suggesting that the root cause of the attention bias goes beyond mere stimulus familiarity.

There is abundant evidence that attention is directed disproportionately to drug-related stimuli in addictive disorders, but do such attention abnormalities really matter? Studies that correlate addiction-related attentional bias with treatment outcomes say "Yes!" Mounting evidence suggests that the magnitude of this attention bias is directly proportional to relapse risk (Munafò & Albery, 2006). Moreover, many studies suggest that the size of the attentional bias is also directly proportional to the level of craving during drug abstinence. This clinical relevance underscores the importance of understanding the neural mechanisms of addiction-related attention bias. As noted above, repeated use of an addictive substance may produce changes in the brain that cause the brain to attribute excessive significance or "salience" to people, places, and things associated with drug use; the development of such hypersensitivity is referred to as "incentive salience" learning (Robinson & Berridge, 2003). Understanding which neural processes are affected and contribute to this increased salience of drug cues could lead to new ideas for better treatments for substance use disorders. Thus far, there has been almost no exploration into these neural correlates; however, studies looking at other aspects of attention and addiction provide some hypotheses about the parts of the brain that are involved.

NEURAL CORRELATES OF ATTENTIONAL CONTROL

While empirical studies of the neural mechanisms of attentional bias toward addictive substances remain incomplete, general studies of attentional allocation provide some clues. For example, neuropsychological studies suggest that patients with damage to the parietal lobes have difficulty disengaging and reorienting their attentional focus (Posner, Walker, Friedrich, & Rafal, 1984). The importance of the posterior parietal cortex (PPC) in attentional orienting also comes from neuroimaging studies, which particularly emphasize the intraparietal sulcus (IPS) (Corbetta & Shulman, 2002). Data from single-cell recording in nonhuman primates and fMRI studies in humans also provide evidence that attention leads to increased perceptual processing. For example, enhanced visual attention leads to increased neural activity in the nonprimary visual cortical areas. Study of the childhood development of attention networks has further enhanced our understanding of the neural mechanisms of attentional control. Explorations into the behavior, brain structure, and brain function related to reorienting and executive control of attention in children versus adults implicate a fronto-parietal network in attentional control. As expected, children showed poorer attentional control behaviorally, and they showed reduced brain activity in right temporo-parietal junction (TPJ)

and dorsolateral prefrontal cortex (DLPFC). These behavioral and functional brain differences between children and adults were associated with structural brain differences; poorer performance and reduced brain activity were associated with less gray matter in the TPJ and DLPFC (Konrad et al., 2005). These studies of typically functioning brains give us a place to begin exploring possible sites of brain dysfunction in addiction, but studies specifically exploring these neural mechanisms in addict populations are in great demand.

While addiction-related attentional bias has not been studied at the neural level, other studies of the addicted brain shed some light on possible mechanisms. Attentional bias toward drug-related cues relies greatly on the fact that these cues have become strongly associated with the use of the drug itself. Based on this deep association, the cues that become associated with drug use can produce an intense craving and relapse both in animals and humans (Robinson & Berridge, 2003). Animal studies that have directly examined the effects of cocaine administration on learned associations suggest that drug administration disrupts orbitofrontal pathways to the striatum, causing impairment in the ability to inhibit previously conditioned responses (Everitt et al., 2007). While it is likely that this cognitive inflexibility plays a role in the attentional bias toward drug-related stimuli, the neural underpinnings of the bias remain a mystery for now. Ongoing studies using emerging neuroimaging and pharmacology techniques to explore the underlying neural mechanisms of addiction-related attentional bias aim to fill this gap in our understanding and to suggest new ways in which to treat addictive disorders.

ADDICTION, DECISION MAKING, AND ACTION SELECTION

Two symptoms used as diagnostic criteria for substance dependence according to the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1994) are (1) a persistent desire or unsuccessful efforts to cut down or control substance use, and (2) continued substance use despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance. Research suggests that addiction is related not only to deficits in decision making related to substance use, but also to deficits in more general decision-making processes, and even to deficits in the selection of simple responses to stimuli.

One of the most frequently used tasks to study simple responses selection and inhibition is the "go/no-go" paradigm. This task generally involves presentation of different types of cues that indicate to the participant whether to respond or to refrain from responding on a given trial. Substance-dependent individuals

have difficulty inhibiting planned responses to no-go targets (Lubman, Yucel, & Pantelis, 2004; Verdejo-Garcia, Bechara, Recknor, & Perez-Garcia, 2006). While addicts may demonstrate some generalized inhibitory impairment, one can observe that such impairments appear most evident in the presence of drugs or drug-associated cues. Consistent with this observation, in laboratory go/no-go studies, addicts demonstrate greater inhibitory impairment when drug-related cues are present.

In addition to investigations of simple response selection and inhibition, several neurocognitive studies of addicts have probed higher-level forms of decision making, focusing on two primary forms. The first that we will discuss are known as "gambling tasks," and include the Iowa Gambling Task (IGT) and the Cambridge Gambling Task (CBT) (Bechara, 2005; Clark & Robbins, 2002; Rogers et al., 1999). The IGT requires participants to pick virtual playing cards, one at a time, from one of four decks of cards displayed on a computer screen. Each choice results in either a gain or loss of money. The four decks vary in terms of their riskiness and in their ultimate payoff. Two of the decks have relatively low gains, but even lower losses, while the other two have high gains, and even higher losses; the latter decks also have more frequent losses. Performance on these gambling tasks is assessed by participants' abilities to pick the statistically best decks (i.e., the low-risk decks). One concern with the IGT is that in addition to measuring decision-making behavior, the task is also implicitly measuring learning, as participants learn which decks are best on the basis of their choices. Thus slow learners could be misinterpreted as being poor decision makers. Indeed, some studies have found that performance on the IGT depends on IQ, suggesting an important role for learning in this task. Developed later, the CBT requires participants to guess whether a reward token will randomly appear in a blue box or red box and subsequently place a bet on their decision. There are 10 boxes across the top of the screen and the ratio of red boxes to blue boxes varies from trial to trial (Rogers et al., 1999). Findings with addicts in gambling task studies have been inconsistent; some have found substance abusers to have impairments on the tasks, while others have not. These discrepancies raise the possibility that the type of addiction and/or the duration of abstinence may be important factors affecting performance on the gambling tasks, but greater exploration of these issues is needed before definitive conclusions can be drawn.

The other type of decision-making task most commonly used to study addicts is the so-called delay discounting task. Performance on these tasks has been shown to be IQ independent, and robustly distinguishes between addicts and control subjects. Generally, this type of task requires participants to decide between a reward (e.g., a sum of money) available sooner and a larger reward

available at a later time. The exact implementation of such task varies from lab to lab, but in general, in comparison to control participants, addicts tend to choose the smaller, immediate rewards more often. As with addiction-related attentional bias, delay discounting has been reported for a variety of addictive disorders, including cigarette smokers, alcoholics, amphetamine users, cocaine users, opiate users, and gamblers (Bickel & Marsch, 2001; Reynolds, 2006). While this tendency for addicts to more steeply or more frequently discount delayed monetary rewards is quite consistent, it appears that rewards consisting of the drug of choice are temporally discounted even more severely by addicts (Bickel & Marsch, 2001; Reynolds, 2006). Of clinical and public health significance, heroin-dependent individuals who are willing to participate in risky needle-sharing behavior show greater delay discounting of both heroin and monetary rewards. Together, these findings suggest a generalized mechanism for overvaluing immediate consequences over delayed consequences that may help perpetuate continued drug use in the face of adverse consequences.

NEURAL CORRELATES OF ACTION SELECTION

Numerous studies using various techniques implicate frontal circuits in action selection processes (Duncan & Owen, 2000; Ostlund & Balleine, 2007; Rushworth, Walton, Kennerley, & Bannerman, 2004). For example, studies exploring response selection in nonhuman primates find neuronal activity in prefrontal cortex neurons related to go-no/go responses. Neuropsychological studies of patients with frontal lobe damage have also provided evidence for a role of frontal areas in action selection and response inhibition. In particular, deficits in these functions are frequently observed in frontotemporal dementia (FTD) patients. The term *FTD* is used to describe non-Alzheimer dementias associated with atrophy in the frontal and temporal lobes. Numerous studies indicate that such atrophy, particularly when it involves the orbitofrontal cortex, impairs response selection and the ability to inhibit inappropriate responses (Viskontas, Possin, & Miller, 2007). Similarly, patients with lesions in the right frontal lobe have slower reactions to signals directing them to inhibit their responses than do patients with intact frontal lobes (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003). Finally, a large amount of evidence implicating frontal areas in action selection and complex decision making comes from recent neuroimaging data. The frontal lobes, specifically in the right hemisphere, have been implicated in response inhibition tasks when inhibiting responses to external stimuli (Brass, Derrfuss, Forstmann, & von Cramon, 2005; de Wit, Enggasser, & Richards, 2002; McClure, Ericson, Laibson, Loewenstein, & Cohen, 2007; McClure, Laibson, Loewenstein, & Cohen, 2004). Similarly, an

area of the fronto-median cortex plays a role in voluntary response inhibition when the decision to withhold a response is internally rather than externally cued (Brass & Haggard, 2007). Based on accumulated evidence, Michael Frank and colleagues have recently developed models of natural action selection that posit a central role of the basal ganglia (BG) frontal circuits (Frank, Scheres, & Sherman, 2007). These models point to two separate pathways, with each pathway's contribution regulated by dopamine (DA) levels. These pathways, one of which initiates responses and the other of which inhibits them, derive from two separate populations of cells within the striatum, which in turn target divergent pathways through the BG nuclei, thalamus, and cortex.

While many neuroimaging studies have probed the neural correlates of simple response selection, including several studies in addicted populations, investigations into the neural bases of decision making are less numerous. Fewer studies still have assessed the neural signature of behavioral differences between substance-abusing and non–substance-abusing populations (Bickel et al., 2007). Some fMRI evidence suggests that a dysfunction in the orbitofrontal cortex (OFC) is responsible for response selection differences seen in behavioral studies (Goldstein & Volkow, 2002). For example, cocaine addicts and alcoholics both show greater activation in the OFC correlated with poorer performance on a Stroop task, while controls show the opposite pattern, that is, greater OFC activation is related to better performance in controls. These findings suggest the brains of substance abusers are generally working harder to recruit the usual brain areas needed to perform the task, but that some neural inefficiency is impeding effective response inhibition. In terms of more complex decision making, Bechara (2005) describes a neural framework that would account for our ability to make decisions based on future outcomes. Bechara's theory suggests that two interacting brain systems, an impulsive amygdala-driven system and a more reflective prefrontal-driven system, become unbalanced with drug use, resulting in an overactive amygdala driven system. This imbalance between systems is then proposed to impair a drug user's ability to consider distant future outcomes when choosing whether to continue drug use despite probable negative consequences. Neuroimaging data have also explored complex decision making using delay discounting tasks, with results suggesting that activity differences in frontal and amygdala areas are related to differences in choosing smaller, sooner versus larger, later rewards (Boettiger et al., 2007; McClure et al., 2004, 2007).

While neuroimaging studies are one common means for investigating the neural underpinnings of differences in cognitive functions among addict populations, another valuable method is the use of behavioral pharmacology. This tool has been employed most extensively in studies assessing delay discount-

ing behavior. These studies have determined that acutely elevating dopamine levels with amphetamine *decreases* discounting of delayed rewards (de Wit et al., 2002) and that acutely depressing serotonin levels *increases* discounting of delayed rewards (Schweighofer et al., 2008). Two further studies point to the importance of dopamine in regulating discounting behavior. First, blockade of endogenous opioids with naltrexone (NTX; one of the few FDA-approved treatments for some forms of addiction) changed delay discounting behavior in a personality-dependent fashion (Mitchell, Tavares, Fields, D'Esposito, & Boettiger, 2007). The personality measure, the "Locus of Control" score, measures one's tendency to perceive outcomes as under his or her own control or as controlled by external forces (Rotter, 1966), and is theoretically linked to brain dopamine levels (Declerck, Boone, & De Brabander, 2006). This finding that this cognitive effect of NTX is personality-dependent suggests that its therapeutic effects could be as well. The second finding linked a genetic polymorphism that regulates cortical dopamine levels with delay discounting performance (Boettiger et al., 2007). Further investigation is needed, but these data highlight the importance of the modulatory systems targeted by drugs of abuse (Everitt & Robbins, 2005) in influencing decision-making behavior.

CONCLUSION

Animal studies have been undeniably instrumental in furthering our understanding of the neurobiology of addiction. Such studies have determined which circuits in the brain play a role in addictive behavior, how these areas are connected, and how these circuits are regulated by neuromodulators. Important components of these circuits include a brainstem site called the ventral tegmental area (VTA), the prefrontal cortex, and two areas located deep in the brain, the amygdala and the nucleus accumbens. The VTA neurons produce dopamine, which they release into the other three brain areas listed above. Such dopamine release normally occurs in response to natural rewards like food, water, and sex, but it also occurs in response to all drugs of abuse. These circuits are affected by learning, with the result being that cues associated with rewards also come to trigger dopamine release. Chronic intake of drugs of abuse results in an intensified version of such learning, producing extreme hypersensitivity to drug-related cues. This knowledge gathered in animal studies has enabled scientists to take a more educated approach to empirical studies of addiction in humans.

Despite the importance of these animal studies, it is important to recognize that addiction is a human disorder that does not generally occur in animals outside of a laboratory context. With the advent of cognitive neuroscience tools, we

are beginning to understand the neurobiology of addiction in a new way. These tools now allow the investigation of cognitive aspects of addiction, and thus to ask scientific questions that are only addressable by testing human populations. A growing body of research has begun to specifically focus on addiction as a dysfunction of cognitive control. While it is evident that dysfunctions in cognitive control may be central to the disease of addiction, a vast amount remains to be learned about the underlying neural mechanisms of these dysfunctions. A better understanding of the neural mechanisms underlying cognitive control dysfunctions in addiction will lead to a more complete understanding of how cognitive control systems function normally. In addition, this knowledge will stimulate new avenues for treatment development (both behavioral and pharmacological), ultimately increasing the variety and effectiveness of options for treating substance use disorders.

REFERENCES

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Aron, A. R., Fletcher, P. C., Bullmore, E. T., Sahakian, B. J., & Robbins, T. W. (2003). Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature Neuroscience, 6*(2), 115–116.

Bechara, A. (2005). Decision making, impulse control and loss of willpower to resist drugs: A neurocognitive perspective. *Nature Neuroscience, 8*(11), 1458–1463.

Bickel, W. K., & Marsch, L. A. (2001). Toward a behavioral economic understanding of drug dependence: Delay discounting processes. *Addiction, 96*(1), 73–86.

Bickel, W. K., Miller, M. L., Yi, R., Kowal, B. P., Lindquist, D. M., & Pitcock, J. A. (2007). Behavioral and neuroeconomics of drug addiction: Competing neural systems and temporal discounting processes. *Drug and Alcohol Dependence, 90*(Suppl. 1), S85–S91.

Boettiger, C. A., Mitchell, J. M., Tavares, V. C., Robertson, M., Joslyn, G., D'Esposito, M., et al. (2007). Immediate reward bias in humans: Fronto-parietal networks and a role for the catechol-O-methyltransferase 158(Val/Val) genotype. *Journal of Neuroscience, 27*(52), 14383–14391.

Brass, M., Derrfuss, J., Forstmann, B., & von Cramon, D. Y. (2005). The role of the inferior frontal junction area in cognitive control. *Trends in Cognitive Sciences, 9*(7), 314–316.

Brass, M., & Haggard, P. (2007). To do or not to do: The neural signature of self-control. *Journal of Neuroscience, 27*(34), 9141–9145.

Chanon, V., & Hopfinger, J. (2007). Memory's grip on attention: The influence of item memory on the allocation of attention. *Visual Cognition, 16*(2 & 3), 325–340.

Clark, L., & Robbins, T. (2002). Decision-making deficits in drug addiction. *Trends in Cognitive Sciences, 6*(9), 361–363.

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3*(3), 201–215.

de Wit, H., Enggasser, J. L., & Richards, J. B. (2002). Acute administration of d-amphetamine decreases impulsivity in healthy volunteers. *Neuropsychopharmacology, 27*(5), 813–825.

Declerck, C. H., Boone, C., & De Brabander, B. (2006). On feeling in control: A biological theory for individual differences in control perception. *Brain and Cognition, 62*(2), 143–176.

Duncan, J., & Owen, A. M. (2000). Common regions of the human frontal lobe recruited by diverse cognitive demands. *Trends in Neurosciences, 23*(10), 475–483.

Everitt, B. J., Hutcheson, D. M., Ersche, K. D., Pelloux, Y., Dalley, J. W., & Robbins, T. W. (2007). The orbital prefrontal cortex and drug addiction in laboratory animals and humans. *Annals of the New York Academy of Sciences, 1121*, 576–597.

Everitt, B. J., & Robbins, T. W. (2005). Neural systems of reinforcement for drug addiction: From actions to habits to compulsion. *Nature Neuroscience, 8*(11), 1481–1489.

Frank, M. J., Scheres, A., & Sherman, S. J. (2007). Understanding decision-making deficits in neurological conditions: Insights from models of natural action selection. *Philosophical Transactions of the Royal Society of London B: Biological Sciences, 362*(1485), 1641–1654.

Goldstein, R. Z., & Volkow, N. D. (2002). Drug addiction and its underlying neurobiological basis: Neuroimaging evidence for the involvement of the frontal cortex. *American Journal of Psychiatry, 159*(10), 1642–1652.

Konrad, K., Neufang, S., Thiel, C. M., Specht, K., Hanisch, C., Fan, J., et al. (2005). Development of attentional networks: An fMRI study with children and adults. *Neuroimage, 28*(2), 429–439.

Liu, N., Li, B., Sun, N., & Ma, Y. (2008). Effects of addiction-associated and affective stimuli on the attentional blink in a sample of abstinent opiate dependent patients. *Journal of Psychopharmacology, 22*(1), 64–70.

Lubman, D. I., Yucel, M., & Pantelis, C. (2004). Addiction, a condition of compulsive behaviour? Neuroimaging and neuropsychological evidence of inhibitory dysregulation. *Addiction, 99*(12), 1491–1502.

MacLeod, C. M. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin, 109*(2), 163–203.

McClure, S. M., Ericson, K. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. (2007). Time discounting for primary rewards. *Journal of Neuroscience, 27*(21), 5796–5804.

McClure, S. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. (2004). Separate neural systems value immediate and delayed monetary rewards. *Science, 306*(5695), 503–507.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167–202.

Mitchell, J. M., Tavares, V. C., Fields, H. L., D'Esposito, M., & Boettiger, C. A. (2007). Endogenous opioid blockade and impulsive responding in alcoholics and healthy controls. *Neuropsychopharmacology, 32*(2), 439–449.

Munafò, M., & Albery, I. P. (Eds.). (2006). *Cognition + Addiction*. Oxford: Oxford University Press.

Ostlund, S. B., & Balleine, B. W. (2007). The contribution of orbitofrontal cortex to action selection. *Annals of the New York Academy of Sciences, 1121,* 174–192.

Posner, M. I., Walker, J. A., Friedrich, F. J., & Rafal, R. D. (1984). Effects of parietal injury on covert orienting of attention. *Journal of Neuroscience, 4*(7), 1863–1874.

Reynolds, B. (2006). A review of delay-discounting research with humans: Relations to drug use and gambling. *Behavioral Pharmacology, 17*(8), 651–667.

Robbins, S. J., & Ehrman, R. N. (2004). The role of attentional bias in substance abuse. *Behavioral and Cognitive Neuroscience Reviews, 3*(4), 243–260.

Robinson, T. E., & Berridge, K. C. (2003). Addiction. *Annual Review of Psychology, 54,* 25–53.

Rogers, R. D., Everitt, B. J., Baldacchino, A., Blackshaw, A. J., Swainson, R., Wynne, K., et al. (1999). Dissociable deficits in the decision-making cognition of chronic amphetamine abusers, opiate abusers, patients with focal damage to prefrontal cortex, and tryptophan-depleted normal volunteers: Evidence for monoaminergic mechanisms. *Neuropsychopharmacology, 20*(4), 322–339.

Rotter, J. B. (1966). Generalized expectancies for internal versus external control of reinforcement. *Psychological Monographs, 80*(1), 1–28.

Rushworth, M. F., Walton, M. E., Kennerley, S. W., & Bannerman, D. M. (2004). Action sets and decisions in the medial frontal cortex. *Trends in Cognitive Sciences, 8*(9), 410–417.

Schweighofer, N., Bertin, M., Shishida, K., Okamoto, Y., Tanaka, S. C., Yamawaki, S., et al. (2008). Low-serotonin levels increase delayed reward discounting in humans. *Journal of Neuroscience, 28*(17), 4528–4532.

Stroop, J. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*(6), 643–662.

Verdejo-Garcia, A., Bechara, A., Recknor, E. C., & Perez-Garcia, M. (2006). Executive dysfunction in substance dependent individuals during drug use and abstinence: An examination of the behavioral, cognitive and emotional correlates of addiction. *Journal of the International Neuropsychological Society, 12*(3), 405–415.

Viskontas, I. V., Possin, K. L., & Miller, B. L. (2007). Symptoms of frontotemporal dementia provide insights into orbitofrontal cortex function and social behavior. *Annals of the New York Academy of Sciences, 1121,* 528–545.

This page intentionally left blank

Chapter **17**

Schizophrenia and Substance Misuse

Giuseppe Carrà, MD, MSc, PhD, and
Sonia Johnson, MSc, MRCPsych, DM

Comorbid drug and alcohol problems ("dual diagnosis") are growing concerns among people with schizophrenia and other severe psychoses because of large increases in prevalence and their association with poorer clinical (Margolese, Negrete, Tempier, & Gill, 2006) and psychosocial (Hunt, Bergen, & Bashir, 2002) outcomes. Dual diagnosis clients present more difficulties also from a diagnostic and clinical management perspective than do single diagnosis clients. Substance use has been found to exacerbate positive symptoms (Drake, Osher, & Wallach, 1989; Margolese, Malchy, Negrete, Tempier, & Gill, 2004; Negrete, Knapp, Douglas, & Smith, 1986; Pulver, Wolyniec, Wagner, Moorman, & McGrath, 1989) and to increase aggression and violence (Angermeyer, 2000; Soyka, 2000), as well as medication noncompliance (Bhanji, Chouinard, & Margolese, 2004; Coldham, Addington, & Addington, 2002; Kamali et al., 2001; Margolese et al., 2004; Olfson et al., 2000; Swartz et al., 1998) in people with schizophrenia. Furthermore, time to readmission or community survival is significantly reduced among "dually diagnosed" compared to nonabusing subjects, even when controlling for noncompliance (Hunt et al., 2002).

Improved clinical management of dual diagnosis has been among main concerns of several European National Health Services (Banerjee, Clancy, & Crome, 2002; Carrà & Clerici, 2006; Dervaux et al., 2001; Gouzoulis-Mayfrank, 2004). Options for service development have been widely discussed (Johnson, 1997; Weaver et al., 1999), as models from the United States might perform differently within the context of well-established community mental health services. A variety of treatments exist, but the drive from the United States has been to provide programs integrating treatment of both substance

misuse and severe mental illness. Such programs require additional resources and may require radical redesign of service delivery systems. Actual prevalence rates in European countries and impact on clinical and psychosocial outcomes can inform evidence-based allocation of resources in terms of services and training needed (Johnson, 1997).

THE PREVALENCE OF SUBSTANCE USE IN PEOPLE WITH SCHIZOPHRENIA: STILL AN OPEN QUESTION?

Since the late 1980s, a number of studies have reported that substance use disorders occur at high rates in schizophrenia. U.S. data indicate that approximately 30–50 percent of people with schizophrenia have a lifetime substance use disorder. Nonetheless, whether these rates are generalizable to different countries remains controversial. A number of methodological issues must be considered, which may affect evidence about dual diagnosis prevalence. The first concern is about ascertainment. Most investigations of comorbidity in schizophrenia have been conducted with clinical samples of convenience and this can bias comorbidity rates in an upward direction (Berkson, 1946), and limit generalizability to the general population of individuals with schizophrenia. Studies such as the Epidemiologic Catchment Area (ECA; Regier, Farmer, & Rae, 1990), the National Comorbidity Survey (NCS; Kendler, Gallagher, Abelson, & Kessler, 1996) and the National Psychiatric Morbidity Survey (Jenkins et al., 1997) avoid such sampling problems in that assessments were conducted on both community and institutional populations. Nevertheless, data from these studies are also somewhat limited as the lay interview assessments of psychotic disorders used have been shown to have poor agreement with interviews conducted by clinicians (Kendler et al., 1996). Such poor agreement is indeed common in large-scale surveys and gives rise to expressions of concern among psychiatric epidemiologists (Taub et al., 2005) about validity of evidence. Similarly, the assessment of substance use can also be problematic. Designs often failed to utilize available diagnostic standards of abuse or dependence but instead identify samples based on use, problem use, or combine all levels of severity into one category. Consequently, direct comparisons of rates are limited by the variety of methods of substance use assessment. Reliable multimodal assessment is recommended for dually diagnosed patients (Goldfinger et al., 1996).

The following are the main methods that should be used to assess substance use (Drake, Alterman, & Rosenberg, 1993), with decreasing validity: consensus method (i.e., at least structured diagnosis plus other sources); self-report using formal diagnostic interviews; staff ratings; self-report using screening instrument. A related problem lies in the high prevalence of multiple substance use. This makes it critical to carry out a broad assessment of substance use

and interpretations are limited by the use of more than one substance. Thus, given the frequent occurrence of multiple substance use diagnoses (particularly between alcohol and other drugs), any attempt to attribute observed findings associated with comorbid substance use to a single substance or class of substances is often difficult if not impossible. Finally, a further concern in this research is in relation to the failure to specify time criteria in samples, that is, whether substance diagnoses are based on current, past, or lifetime. Collapsing across current and past use may either obscure or mix up enduring use rates and transient states of intoxication or withdrawal.

WHY AND HOW DO SUBSTANCE-RELATED DISORDERS DEVELOP AMONG PEOPLE WITH SCHIZOPHRENIA?

As mentioned above, comorbidity rates have important implications for nosological issues. The study of comorbidity may enhance the generalizability of research findings. A better understanding both of causal processes underlying comorbidity and of the interplay of individual disorders involved is also needed. Despite the prevalence of substance use disorders in schizophrenia and the worrying effects of dual diagnosis, little is known about its causes. However, attempting to overcome the "chicken and egg" paradox (Meyer, 1986), a number of explanatory models of substance use in schizophrenia have been proposed in the last two decades.

Genetics

There has been accumulating evidence that genetics play a role in schizophrenia (e.g., Sullivan, Kendler, & Neale, 2003), and twin studies have indicated a genetic component to alcohol dependence (Heath et al., 2001; Prescott & Kendler, 1999) and to dependence on other drugs (Kendler, Jacobson, Prescott, & Neale, 2003; Tsuang et al., 1996). Rather more complex is the contribution of genetics to the comorbidity of the two illnesses. Indeed, the available evidence currently suggests little or no genetic relationship between schizophrenia and alcoholism (Kendler & Gardner, 1997), and it may well be that the comorbidity of schizophrenia and substance use disorders reflects the effects of two separate genetic risks in the same individual (Kendler, 1985).

Substance Abuse Leads to Increased Risks of Psychosis

This widely debated association is essentially based on the stress-vulnerability model (Zubin & Spring, 1977), implicating substance use as a stres-

sor precipitating the onset of schizophrenia in vulnerable individuals. Thus, comorbid substance use and schizophrenia may have reciprocal interactions in terms of onset, course, severity, and clinical characteristics (Mueser, Drake, & Wallach, 1998), assuming that the use of substances actually increases the risk of schizophrenia. Indeed, the available evidence supports the hypothesis that cannabis is actually an independent risk factor, both for psychosis and for the development of psychotic symptoms (see Semple, McIntosh, & Lawrie, 2005 for a recent systematic review), with public health concerns (e.g., Fergusson, Poulton, Smith, & Boden, 2006).

Self-Medication Model

An intriguing hypothesis proposes that self-medication by substance abuse is a coping attempt by people with schizophrenia, matching the pharmacological properties of substances with the particular psychiatric symptoms and states experienced (Khantzian, 1985, 1997). However, this should imply diagnostic and symptomatological differences in substance selection (e.g., choice of stimulants and negative symptoms should be associated). Neither prediction is supported by research evidence. Among people suffering from schizophrenia, no replicable patterns of substances chosen and symptoms experienced have been identified (Brunette, Mueser, Xie, & Drake, 1997; Cuffel & Chase, 1994; Hamera, Schneider, & Deviney, 1995), despite preliminary findings supporting the self-medication hypothesis (Schneier & Siris, 1987). As a matter of fact, given the frequent occurrence of multiple substance misuse as discussed above, it is not possible to attribute any observed symptomatological and clinical difference to a single drug class. The only strong evidence is that regarding the association between fewer negative symptoms and dual diagnosis, though it is not still clear whether this may involve lifespan or just current misuse (see Potvin, Sepehry, & Stip, 2006 for a review). Furthermore, the pattern of substance use in schizophrenia is similar to that found in other diagnostic groups (e.g., El-Guebaly & Hodgins, 1992), and reflects a substance use pattern in the general population (Mueser, Yarnold, & Bellack, 1992; Regier et al., 1990) where the sampling took place. However, a less definitive form of the self-medication hypothesis that does have some supporting evidence suggests that substances are used to relieve dysphoria and anxiety and to alleviate tension (Addington & Duchak, 1997; Baigent, Holme, & Hafner, 1995) rather than medicating core psychotic symptoms.

Social and Cross-Cultural Differences

A final framework that may contribute to the model of how substance use and schizophrenia are linked is to assess epidemiological data to determine if

demographic or sociocultural differences in substance use are related to the relative risk for use by people with schizophrenia (Phillips & Johnson, 2001). Availability of substances but also the social context in terms of individual and local or national cultural factors is likely to contribute to the prevalence of dual diagnosis. Internationally, and within a consistent simultaneous sampling framework, the World Mental Health Surveys (WHO World Mental Health Survey Consortium, 2004) reported general population 12-month prevalence for substance disorders ranging between 0.1 and 6.4 percent (IQR, 0.8%–2.6%). This might support the hypothesis about the influence of socioeconomic and cultural factors on dual diagnosis prevalence rates, probably mediated by availability (RachBeisel, Scott, & Dixon, 1999).

THE FIRST WAVE OF EVIDENCE: THE UNITED STATES

Most evidence about the prevalence of this comorbid condition comes from the United States. The largest of these investigations, the Epidemiologic Catchment Area (ECA) study, has shown that the odds of meeting criteria for a substance use disorder are 4.6 times higher for individuals with schizophrenia compared with the rest of the population (Regier et al., 1990). In the National Comorbidity Survey (NCS), roughly half of respondents who met criteria for a substance use disorder at some time in their life also met criteria for one or more lifetime mental disorders (Kessler et al., 1996). Similarly, half of those who met criteria for a mental disorder also met those for a lifetime substance use one. Regarding the general population, the ECA study found that 47 percent of patients with schizophrenia had a comorbid substance abuse disorder, 34 percent related to alcohol, and 28 percent to other drugs (Regier et al., 1990). Further data from the NCS (Kendler et al., 1996) showed that 44.8 percent of individuals with nonaffective psychosis were classified as having a dual diagnosis.

Is Dual Diagnosis Simply Inherent to Psychotic Illness?

Also in the United States, studies in mental health settings suggest variations in dual diagnosis rates, ranging between 20 and 65 percent of persons who have a diagnosis of a severe mental illness (Cuffel, Shumway, Chouljian, & MacDonald, 1994; Drake et al., 1989). For instance, 38 percent of clients of mental health services in a California county reported one or more alcohol dependence symptoms in the previous year, and 21 percent the use of three or more types of illicit drugs (Weisner & Schmidt, 1993), but the prevalence of current substance use in an emergency room among clients with schizophrenia has been reported at 47 percent (Barbee, Clark, Crapanzano, Heintz, & Kehoe,

1989) and among inpatients, it ranges from 12 to 60 percent (Brady, Casto, Lydiard, Malcom, & Arana, 1991; Brady et al., 1990; Dixon, Haas, Weiden, Sweeney, & Frances, 1991; Drake et al., 1989; Havassy & Arns, 1998). On the whole, rates seem higher among patients with psychosis receiving emergency or inpatient treatment (Mueser et al., 1992). The recent Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) study, recruiting at 57 mixed U.S. clinical sites, found 37 percent with current evidence of substance use disorders (Swartz et al., 2006).

However, some U.S. literature also suggests that dual diagnosis rates might vary between rural or urban areas (Mueser, Essock, Drake, Wolfe, & Frisman, 2001), and different backgrounds (Lambert, Griffith, & Hendrickse, 1996; Mowbray, Ribisl, Solomon, Luke, & Kewson, 1997). These variations may reflect factors such as acute versus nonacute illnesses and differences according to mental health care settings sampled (Drake et al., 1989; Havassy & Arns, 1998) and the availability of illicit drugs in the study location (RachBeisel et al., 1999).

Variations in rates reported from the United States prompt questions about the extent to which comorbidity is a product of local social circumstances rather than vulnerability to substance misuse being an inherent clinical feature of psychosis. A number of such social factors could be studied also in order to implement effective preventive and treatment programs (Jeffery, Ley, McLaren, & Siegfried, 2000) as currently research is focused on assessing whether extra resources, required to deliver substance misuse treatment integrated with mental health care for people with severe mental health problems, will lead to benefit. A number of environmental factors may mediate higher incidence of psychotic illnesses in urban areas (e.g., Allardyce et al., 2001), in its turn potentially mediated by social deprivation or migration into urban areas (Freeman, 1994; Van Os, 2004). Analogous social factors—both at individual level and at local/national level—might mediate incidence of substance abuse in people with schizophrenia, explaining part of the variation in dual diagnosis prevalence rates.

SUGGESTIONS FROM ALCOHOL AND DRUG ABUSE PREVALENCE IN THE GENERAL POPULATION

Evidence about the epidemiology of "pure" drug and alcohol abuse in the general population is particularly useful to explore prevalence variations in different cultural and social contexts. First, in reestimating prevalence values by calibration of case definitions (i.e., imposing methodological constraints to unify case definitions), differences were found between the United States and the UK in prevalence of active drug dependence, which was estimated as 1.4

percent in the United States and 0.5 percent in the UK, just somewhat attenuated when the effect of living in an urban setting was controlled (Furr-Holden & Anthony, 2003). The difference was found despite symptom profiles among active cases being very similar. In both countries, being male, unmarried, of a low socioeconomic status (SES), and living in an urban setting were associated with an increased occurrence of drug dependence. Furthermore within the European Union, differences in lifetime and current prevalence of alcohol and drug abuse have long been assessed by the European Monitoring Centre for Drugs and Drug Addiction (EMCDDA) in its annual reports, and despite the general trend toward increasing rates, variations between countries in prevalence rates of both alcohol and drugs abuse remain wide (EMCDDA, 2005). However, accurate comparisons between countries are still problematic because both strengths and weaknesses of different general population surveys on drug use prevalence vary in conjunction with the survey methodology (EMCDDA, 1997). The EMCDDA 2005 annual report attempted to update figures from recently published national surveys, estimating lifetime dependence prevalence rates in the EU countries ranging from 1.1 to 11.9 percent for alcohol, and between 1.0 and 6.9 percent for any other drug (EMCDDA, 2005). Two recent systematic reviews have provided an overview of prevalence of current alcohol (Rehm, Room, van den Brink, & Jacobi, 2005a) and drug (Rehm, Room, van den Brink, & Jacobi, 2005b) use disorders in EU countries and Norway. Including only studies using the *DSM–III–R* or *DSM–IV,* or *ICD–10,* plus validated instruments to assess alcohol use disorders, prevalence rates for dependence ranged from 0.4 to 7.5 percent for males and 0.1 to 2.1 percent for females. Regarding drug dependence, including cannabis, the 12-month prevalence rates varied between 0.3 and 2.9 percent. Overall, these figures suggest wide variations in the extent of alcohol and drug dependence between different Western countries and support the need to explore whether there are similar variations in the dual diagnosed population.

EUROPEAN EVIDENCE ABOUT PREVALENCE OF DUAL DIAGNOSIS

Until fairly recently, little evidence has been available about the epidemiology of dual diagnosis in other countries and whether prevalence and patterns resemble those found in the United States. The literature on dual diagnosis remains very limited in many European countries (EMCDDA, 2004), but a body of evidence of moderate size has been accumulating in the UK since the beginning of the 1990s (Carrà & Johnson, in press) and, though at a different pace, also in other European countries.

Apart from its importance to service planning and policy making in the European Union, this literature is of general interest in two respects. First, it allows assessment of whether American findings regarding the epidemiology of dual diagnosis generalize to other Western countries or whether, as with general population figures, there are substantial differences between the United States and European countries. Second, unlike the United States, EU mental health services are generally catchment area based, serving the whole population of a specific geographical area. This means that findings from EU studies regarding variations between countries are considerably more likely to be representative of the underlying population than those from the United States, where a variety of factors other than place of residence are likely to influence who uses which service (Mueser, Bond, Drake, & Resnick, 1998; Wang et al., 2005). It also allows exploration of the relationship between comorbidity and the characteristics of the local area and its population.

Most recent UK studies report rates between 20 and 37 percent in mental health settings (Menezes et al., 1996; Weaver et al., 2003), but appear to be especially high in inpatient (Phillips & Johnson, 2003) and crisis team (Johnson et al., 2005a, 2005b) and forensic (Wheatley, 1998) settings. In terms of geography, rates appear highest in inner-city areas (McCreadie et al., 2002). Evidence from France is less homogeneous with reported lifetime use rates from a cross-sectional survey (Launay, Petitjean, Perdereau, & Antoine, 1998) of 50 percent for alcohol and 27 percent for cannabis, and current rates for inpatients of 8 percent (Verdoux, Mury, Besancon, & Bourgeois, 1996). However, inner-city inpatients (Dervaux et al., 2003) reported rates of 43 percent for lifetime and 30 percent for current comorbid disorders. In Germany, schizophrenic inpatients consecutively admitted in the Munich (Soyka et al., 1993) area showed a lifetime prevalence for substance use disorders of 43 percent, while a representative first-episode sample reported a lifetime history of abuse due to alcohol (24%) or other drugs (14%) (Hambrecht & Hafner, 1996). In Italy, recent data based on multistage household probability samples yield a 12-month prevalence of World Mental Health Composite International Diagnostic Interview for alcohol abuse and dependence of 0.1 (0.0–0.2), and for a serious mental disorder of 1.0 (0.4–1.7) (WHO, 2004), although cross-national comparisons are hampered by inconsistencies in diagnostic methods. Nevertheless, some Italian data are available from specific clinical populations, namely, on opiate users in residential treatment (Clerici, Carta, & Cazzullo, 1989), with 30 percent having *DSM–III* Axis I and 59 percent Axis II disorders, and on patients on a methadone maintenance treatment program (Pani, Trogu, Contu, Agus, & Gessa, 1997), with 54 percent having *DSM–III–R* Axis I and 43 percent Axis II disorders. Sampling from community addictions services, it was found that dually diagnosed clients represented 36 percent of

the entire caseload (Carrà, Scioli, Monti, & Marinoni, 2006). Furthermore, one multicenter survey in the Lombardy region (Clerici & Carta, 1996), using standardized diagnostic instruments on 606 opiate users in six outpatient or residential treatment programs, showed that, according to *DSM–III*, 108 (17.8%) suffered only from a substance use disorder, 160 (26.4%) had a further comorbid psychiatric diagnosis (except personality disorders), and 338 (55.8%) had a further Axis II diagnosis. A more nationally representative survey (although it was biased by a large rate of refusals for the CIDI's diagnostic assessment) (Pozzi, Bacigalupi, & Tempesta, 1997) on 11 addicts' outpatient clinics located all over Italy showed an overall lifetime psychiatric comorbidity of 32.3 percent, including psychotic (10%), mood (63%), anxiety (19%), and other mental disorders (8%).

THE CLINICAL IMPACT OF ALCOHOL AND DRUG USE ON THE COURSE OF PSYCHOSIS

Numerous studies report that dual diagnosis is strongly associated with youth and male sex (e.g., DeQuardo, Carpenter, & Tandon, 1994; Mueser et al., 1990; Zammit, Allebeck, Andreasson, Lundberg, & Lewis, 2002). More important, comparing with their nonabusing counterparts, those with a comorbid substance abuse present more difficulties from a clinical management perspective. As a matter of fact, higher rates of mental health services use are reported (Bartels et al., 1993). People with this comorbidity spend significantly more days as inpatients (Menezes et al., 1996; Schofield, Quinn, Haddock, & Barrowclough, 2001; Wright, Gournay, Glorney, & Thornicroft, 2000) and in psychiatric intensive care units (Isaac, Isaac, & Holloway, 2005); and use more Accident and Emergency Departments (A&E) out-of-hours (Todd et al., 2005), assertive outreach (Graham et al., 2001), and community (McCrone et al., 2000) psychiatric services. Such evidence about mental health services use suggests the need to explore clinical and psychosocial characteristics dual diagnosis is associated with. These issues should be the main targets of treatment in terms of clinical management. The topic of the effect of substance abuse on the course and outcome of the psychosis has been extensively studied in the last decade. Several reviews have dealt with the topic, providing useful overviews (Crawford, Crome, & Clancy, 2003; Murray, Grech, Phillips, & Johnson, 2003; Ridgely & Johnson, 2001).

Positive Psychotic Symptoms

Substance use has been found to exacerbate psychiatric symptoms in people with schizophrenia, and it has been especially associated with more prominent

positive psychotic symptoms on various measures (e.g., D'Souza et al., 2005; Green, Zimmet, Strous, & Schildkraut, 1999; Lieberman, Kane, & Alvir, 1987; Lysaker, Bell, Beam-Goulet, & Milstein, 1994; Margolese et al., 2004). A recent meta-analysis (Talamo et al., 2006) reviewed eight studies providing Positive and Negative Syndrome Scale (PANSS) (Kay, Fiszbein & Opler, 1987) ratings in both comorbid and noncomorbid clients suffering from schizophrenia. Studies using different definitions of substance abuse and measuring PANSS ratings and substance misuse simultaneously (Addington & Addington, 1998; Bersani, Orlandi, Kotzalidis, & Pancheri, 2002; Lysaker et al., 1994; Margolese et al., 2004; Scheller-Gilkey, Moynes, Cooper, Kant, & Miller, 2004) or at different times (Compton, Furman, & Kaslow, 2004; Gut-Fayand et al., 2001; Sevy, Kay, Opler, & van Praag, 1990) were pooled. Moreover, scores for individual PANSS items, subscale scores for individual subjects, and many other clinical details were not analyzed, nor were other psychopathological and sociodemographic features, that might covary with dual diagnosis, controlled for. On pooling means among 725 subjects, comorbid patients had very significantly higher PANSS-positive, and lower PANSS-negative, scores (Talamo et al., 2006). Also, the recent U.S. CATIE study (Swartz et al., 2006) that recruited 1,460 subjects (37% comorbid) showed higher positive symptoms as well as lower negative symptoms scores on PANSS in comorbid subjects. The clinical relevance of these findings also derives from evidence that the main aspect of mental state associated with violence is the presence of persecutory delusions (Junginger, 1996). Positive symptoms associated with dual diagnosis may thus lead to the increased aggression and violence rates shown by comorbid clients and probably to their use of acute services and inpatient wards. Dual diagnosis patients are as a matter of fact more likely to commit violent offenses (Angermeyer, 2000), and they seem more likely to be convicted of criminal activity (Soyka, 2000) compared to those who do not abuse substances (40.1% vs. 13.7%).

Negative Psychotic Symptoms

Evidence that dual diagnosis patients experience fewer negative symptoms is relevant to the debate on the self-medication hypothesis. People with schizophrenia might attempt to cope with negative symptoms by stimulant pharmacological properties. In fact, studies testing the self-medication hypothesis in dual diagnosis schizophrenia have not been conclusive, with some studies showing that they experience fewer negative symptoms (e.g., Compton et al., 2004; Goswami, Mattoo, Basu, & Singh, 2004; Serper, Chou, Allen, Czobor, & Cancro, 1999), and others not (e.g., Addington & Addington, 1997; Gut-Fayand et al., 2001).

This topic is probably more complex than that in relation to positive symptoms for several reasons. First, negative symptoms need clear conceptualization and description (Andreasen, 1985). This concept—already implicit in Bleuler's distinction between fundamental and accessory symptoms—was developed particularly by Strauss, Carpenter, and Bartko (1974), explicitly classifying the positive and negative symptoms and differentiating the latter from social dysfunction. Indeed, Bleuler's early descriptions are closely aligned with the current nosological description of negative symptoms as restrictions in the range and intensity of emotional expression (affective flattening), in the fluency and productivity of thought and speech (alogia), and in the initiation of goal-directed behavior (avolition). The loss of the ability to feel pleasure (anhedonia) has also been identified as an associated feature, but controversy remains (American Psychiatric Association, 1994). Andreasen described the first operationalized criteria for positive and negative schizophrenia (Andreasen & Olsen, 1982).

Accordingly, the concept of positive versus negative symptoms in schizophrenia led to the development of several rating scales. Clinical investigations in dual diagnosis have used the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962), the PANSS (Kay, Fiszbein & Opler, 1987), and the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1983), along with numerous other scales. The psychometric properties of some of these scales lead to somewhat heterogeneous results in terms of factor-analytic characteristics of positive and negative symptoms. In SANS three dimensions (i.e., psychosis, disorganization, negative) best represent the scale's factor structure. The PANSS shows five factors—positive, negative, depressive, disorganization, and excitement (Lindenmayer, Bernstein-Hyman, & Grochowsky, 1994). In addition: (1) the SANS is explicitly chosen to measure negative symptoms, whereas other scales assess schizophrenia symptoms in broader terms; (2) it covers cognitive symptoms in a less confusing way than the PANSS; and (3) four out of five SANS fall within the negative factor (Buchanan & Carpenter, 1994). However, the most important difference lies in the lack of an anhedonia item in all scales but SANS because of the controversies over including it among negative features. In practice, the self-medication hypothesis assumes that anhedonia is the main target of substance abuse (Khantzian, 1997). In a recent meta-analysis of studies that used just the SANS (Potvin et al., 2006), people with schizophrenia plus a substance use disorder experienced fewer negative symptoms than abstinent ones, and the largest effect size was observed for the anhedonia subscale. Other important findings from this meta-analysis were that the reduction in negative symptoms was associated with the abuse of cocaine and cannabis, but not alcohol.

A number of other studies using the PANSS were recently pooled in a meta-analysis that showed that comorbid patients had significantly lower PANSS-negative scores (Talamo et al., 2006). Nevertheless, even these findings must be considered in the light of several reservations. The results in relation to substance of choice certainly seem consistent with the self-medication hypothesis. However, in terms of neurobiology, the negative symptoms of schizophrenia have been associated with a dopaminergic deficit in the prefrontal cortex (Finlay, 2001), but it remains unclear why people with schizophrenia should select substances that share the common property of acutely increasing dopamine release in the prefrontal cortex (Devous, Trivedi, & Rush, 2001; Volkow et al., 1996). By taking alcohol and other drugs, people with schizophrenia would be self-medicating their neurotransmitter deficits within the prefrontal system, and eventually, their negative symptoms. In addition, the fact that a difference in negative symptoms was observed only for lifetime dependent people with schizophrenia suggests that this does not depend on the *acute* effects of drugs. However, drugs seem unlikely to have long-term effects on negative symptoms. Moreover, further potential hypotheses remain to be tested. Depression, compliance with medication, or factors not yet identified are candidate variables that could explain this relationship. In terms of reverse explanation, patients with fewer negative symptoms might be simply more prone to substance abuse (Kirkpatrick et al., 1996; Kirkpatrick, Messias, & Tek, 2003). Longitudinal studies are required to resolve this issue as well as to explore whether the exposure to current or lifetime substance abuse is related to negative symptoms. More important, several different patient profiles exist among the dual diagnosis population in terms of social skills (e.g., Salyers & Mueser, 2001). Planning and organization skills are required to obtain substances of abuse and maintain substance use. Therefore, these patients would be able to continue their addiction because they have better cognitive function and fewer negative symptoms (Joyal, Hallé, Lapierre, & Hodgins, 2003).

Quality of Life and Social Functioning

In relation to quality of life (QoL) in severely mentally ill substance abusers, the evidence is still somewhat contradictory. In "pure" alcohol dependent subjects, abstinence, controlled, or minimal drinking are associated with improved QoL and psychiatric comorbidity is an important factor in determining a poor QoL (Foster, Powell, Marshall, & Peters, 1999). A few studies examining the effect of substance abuse on outpatients with established severe mental illness (Brunette, Noordsy, Xie, & Drake, 2003; Lam & Rosenheck, 2000) and

with first-episode psychosis (Addington & Addington, 1997, 1998) found significantly lower quality-of-life scores in those with dual diagnosis compared with patients with schizophrenia with no past history of substance abuse, but these findings were not replicated in a similar later study (Van Mastrigt, Addington, & Addington, 2004). Furthermore, Herman (2004) found that dually diagnosed inpatients express higher levels of satisfaction with their quality of life compared to the non–substance-abusing patients with schizophrenia. However, hypotheses emerge from the literature that might explain differences in findings and which deserve further testing. In dually diagnosed people, quality of life also seems related to medication adherence, with findings from a first-episode psychosis cohort showing poorer quality of life in nonadherent patients (Coldham et al., 2002). Also, social skills and cognition and likewise psychotic negative symptoms are features that might explain part of the variance in quality of life, and they should be controlled for when the influence of dual diagnosis is assessed (Joyal et al., 2003; Salyers & Mueser, 2001). Finally, QoL has been the outcome measure in several trials for the dually diagnosed population, often (Bellack, Bennett, Gearon, Brown, & Yang, 2006; Drake et al., 1998; Schaar & Ojehagen, 2003; Shaner, Eckman, Roberts, & Fuller, 2003), but not always (Lehman, Herron, Schwartz, & Myers, 1993), showing improvements.

Medication Compliance

Approximately 40 percent of patients with schizophrenia stop prescribed medication within one year and 75 percent by two years (Owen, Fischer, Booth, & Cuffel, 1996; Perkins, 1999). As mentioned above, it seems possible that increased symptoms expressed by people with comorbid substance abuse and schizophrenia can be accounted for by medication noncompliance as well as by the direct effect of substances on psychiatric symptoms. Findings from numerous studies (Buchanan, 1992; Drake et al., 1989; Kashner et al., 1991; Kovasznay et al., 1997; Owen et al., 1996; Razali & Yahya, 1995; Swartz et al., 1998) reported lower medication compliance in comorbid patients. Furthermore, a recent history of substance abuse or dependence may more accurately predict future medication compliance than a more remote history of a substance use disorder (Olfson et al., 2000). Poor compliance with neuroleptic regimens in comorbid people seem to be associated with more negative subjective or dysphoric responses, but when comorbid substance misuse and other confounding variables are controlled for, level of insight is a significant factor in determining whether a patient is regularly compliant (Kamali et al., 2001). Thus, a better understanding of reasons for noncompliance might be

helpful also in quantifying the amount of noncompliance mediated by increasing positive symptoms and that attributable just to comorbid substance abuse (Kamali et al., 2001). Indeed, when comorbid patients are adequately treated for their psychiatric illness, there is considerable amelioration in their positive psychotic symptoms (Margolese et al., 2006), even when their substance use remains unchanged.

CONCLUSIONS

Dual diagnosis rates among people with schizophrenia in mental health settings are high. Social factors may be very important in explaining dual diagnosis rates in people with schizophrenia.

People with schizophrenia and dual diagnosis—compared with those without a comorbid disorder—have greater positive and fewer negative symptoms, and report poorer adherence to medication compliance and worse global subjective satisfaction with life. However, the self-medication hypothesis needs further research. Conceptualization and measuring of negative features in dual diagnosis are further candidate areas for research.

The negative impact in dual diagnosis on the symptomatological features and on psychosocial functioning is cause of concern. The high rates of dependence (not simply abuse) on alcohol or other drugs, and their impact on different outcomes deserves attention and cautious consideration also in terms of health service research. Standard treatments provided in community mental health centers might not be appropriate in dealing with dual diagnosis. Results on dual diagnosis prevalence cannot assume to be generalizable from one country to another. Furthermore, for social and cultural reasons, people might not want to admit substance misuse, which could also be more stigmatized in some countries than others.

However, social factors such as area of residence are very important in explaining dual diagnosis rates in people with schizophrenia. Periodic, local, surveys are needed to explore the extent of the comorbidity. Mental health and addiction services should take into account results on local prevalence when planning their treatment programs. Staffing, training, and configuration of health services are all areas to be designed and implemented according to relevant evidence. Better identification and treatment of comorbid dependence in adequate settings should be among treatment priorities. Treatment needs of the comorbid population need to be met in order to guarantee a subjective quality of life at least as high as that of their nondependent counterparts. Further research is also needed to explore whether variations in dual diagnosis rates between centers are the result of different distributions of individual

social characteristics (e.g., poverty, unemployment, single marital status, etc.) or of area-level social characteristics (e.g., drug availability, stigma experienced by the mentally ill, benefits regime, etc.).

REFERENCES

Addington, J., & Addington, D. (1997). Substance abuse and cognitive functioning in schizophrenia. *Journal of Psychiatry and Neuroscience, 22,* 99–104.

Addington, J., & Addington, D. (1998). Effects of substance misuse in early psychosis. *British Journal of Psychiatry Supplement, 172*(33), 134–136.

Addington, J., & Duchak, V. (1997). Reasons for substance use in schizophrenia. *Acta Psychiatrica Scandinavica, 96,* 329–333.

Allardyce, J., Boydell, J., Van Os, J., Morrison, G., Castle, D., Murray, R. M., et al. (2001). Comparison of the incidence of schizophrenia in rural Dumfries and Galloway and urban Camberwell. *British Journal of Psychiatry, 179,* 335–339.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Andreasen, N. C. (1983). *The Scale for the Assessment of Negative Symptoms*. Iowa City: University of Iowa.

Andreasen, N. C. (1985). Positive vs. negative schizophrenia: A critical evaluation. *Schizophrenia Bulletin, 11,* 380–389.

Andreasen, N. C., & Olsen, S. (1982). Negative vs. positive schizophrenia: Definition and validation. *Archives of General Psychiatry, 39,* 789–794.

Angermeyer, M. C. (2000). Schizophrenia and violence. *Acta Psychiatrica Scandinavica, Supplement, 407*(102), 63–67.

Baigent, M., Holme, G., & Hafner, R. J. (1995). Self reports of the interaction between substance abuse and schizophrenia. *Australian and New Zealand Journal of Psychiatry, 29,* 69–74.

Banerjee, S., Clancy, C., & Crome, I. (2002). *Coexisting problems of mental disorder and substance misuse (dual diagnosis): An information manual.* London: Royal College of Psychiatrists Research Unit.

Barbee, J. G., Clark, P. D., Crapanzano, M. S., Heintz, G. C., & Kehoe, C. E. (1989). Alcohol and substance abuse among schizophrenic patients presenting to an emergency psychiatric service. *Journal of Nervous and Mental Disease, 177,* 400–407.

Bartels, S. J., Teague, G. B., Drake, R. E., Clark, R. E., Bush, P. W., & Noordsy, D. L. (1993). Substance abuse in schizophrenia: Service utilisation and costs. *Journal of Nervous and Mental Disease, 181,* 227–232.

Bellack, A. S., Bennett, M. E., Gearon, J. S., Brown, C. H., & Yang, Y. (2006). A randomized clinical trial of a new behavioral treatment for drug abuse in people with severe and persistent mental illness. *Archives of General Psychiatry, 63,* 426–432.

Berkson, J. (1946). Limitations of the application of the fourfold table analysis to hospital data. *Biometrics, 2,* 47–53.

Bersani, G., Orlandi, V., Kotzalidis, G. D., & Pancheri, P. (2002). Cannabis and schizophrenia: Impact on onset, course, psychopathology and outcomes. *European Archives of Psychiatry and Clinical Neuroscience, 252,* 86–92.

Bhanji, N. H., Chouinard, G., & Margolese, H. C. (2004). A review of compliance, depot intramuscular antipsychotics and the new long-acting injectable atypical antipsychotic risperidone in schizophrenia. *European Neuropsychopharmacology, 14,* 87–92.

Brady, K., Anton, R., Ballenger, J. C., Lydiard, R. B., Adinoff, B., & Selander, J. (1990). Cocaine abuse among schizophrenic patients. *American Journal of Psychiatry, 147,* 1164–1167.

Brady, K., Casto, S., Lydiard, R. B., Malcom, R., & Arana, G. (1991). Substance abuse in an inpatient psychiatric sample. *American Journal of Drug and Alcohol Abuse, 17,* 389–397.

Brunette, M. F., Mueser, K. T., Xie, H., & Drake, R. E. (1997). Relationships between symptoms of schizophrenia and substance abuse. *Journal of Nervous and Mental Disease, 185,* 13–20.

Brunette, M. F., Noordsy, D. L., Xie, H., & Drake, R. E. (2003). Benzodiazepine use and abuse among patients with severe mental illness and co-occurring substance use disorders. *Psychiatric Services, 54,* 1395–1401.

Buchanan, A. (1992). A two-year prospective study of treatment compliance inpatients with schizophrenia. *Psychological Medicine, 22,* 787–797.

Buchanan, R. W., & Carpenter, Jr., W. T. (1994). Domains of psychopathology and approach to the reduction of heterogeneity in schizophrenia. *Journal of Nervous and Mental Disease, 182,* 193–204.

Carrà, G., & Clerici, M. (2006). Dual diagnosis-policy and practice in Italy. *American Journal on Addictions, 15,* 125–130.

Carrà, G., & Johnson, S. (in press). Variations in rates of comorbid substance use in psychosis between mental health settings and geographical areas in the UK. A systematic review. *Social Psychiatry and Psychiatric Epidemiology*, DOI 10.1007/s00127-008-0458-2.

Carrà, G., Scioli, R., Monti, M. C., & Marinoni, A. (2006). Severity profiles of substance-abusing patients in Italian community addiction facilities: Influence of psychiatric concurrent disorders. *European Addiction Research, 12,* 96–101.

Clerici, M., & Carta, I. (1996). Personality disorders among psychoactive substance users: Diagnostic and psychodynamic issues. *European Addiction Research, 2,* 147–155.

Clerici, M., Carta, I., & Cazzullo, C. L. (1989). Substance abuse and psychopathology: A diagnostic screening of Italian narcotic addicts. *Social Psychiatry and Psychiatric Epidemiology, 24,* 219–226.

Coldham, E. L., Addington, J., & Addington D. (2002), Medication adherence of individuals with a first episode of psychosis. *Acta Psychiatrica Scandinavica, 106,* 286–290.

Compton, M. T., Furman, A. C., & Kaslow, N. J. (2004). Lower negative symptom scores among cannabis-dependent patients with schizophrenia-spectrum dis-

orders: Preliminary evidence from an African American first-episode sample. *Schizophrenia Research, 71*, 61–64.

Crawford, V., Crome, I. B., & Clancy, C. (2003). Co-existing problems of mental health and substance misuse (dual diagnosis): A literature review. *Drugs: Education, Prevention and Policy, 10*, 1–74.

Cuffel, B. J., & Chase, P. (1994). Remission and relapse of substance use disorders in schizophrenia: Results from a one-year prospective study. *Journal of Nervous and Mental Disease, 182*, 342–348.

Cuffel, B. J., Shumway, M., Chouljian, T. L., & MacDonald, T. (1994). A longitudinal study of substance use and community violence in schizophrenia. *Journal of Nervous and Mental Disease, 182*, 704–708.

DeQuardo, J. R., Carpenter, C. F., & Tandon, R. (1994). Patterns of substance abuse in schizophrenia: Nature and significance. *Journal of Psychiatric Research, 28*, 267–275.

Dervaux, A., Bayle, F. J., Laqueille, X., Bourdel, M. C., Le Borgne, M. H., Olié, J. P., et al. (2001). Is substance abuse in schizophrenia related to impulsivity, sensation seeking or anhedonia? *American Journal of Psychiatry, 158*, 492–494.

Dervaux, A., Laqueille, X., Bourdel, M. C., Leborgne, M. H., Olié, J. P., Lôo, H., et al. (2003). Cannabis and schizophrenia: Demographic and clinical correlates. *Encephale, 29*, 11–17.

Devous, M. D., Trivedi, M. H., & Rush, A. J. (2001). Regional cerebral blood flow response to oral amphetamine challenge in healthy volunteers. *Journal of Nuclear Medicine, 156*, 19–26.

Dixon, L., Haas, G., Weiden, P. J., Sweeney, J., & Frances, A. J. (1991). Drug abuse in schizophrenic patients: Clinical correlates and reasons for use. *American Journal of Psychiatry, 148*, 224–230.

Drake, R. E., Alterman, A. I., & Rosenberg, S. R. (1993). Detection of substance use disorders in severely mentally ill patients. *Community Mental Health Journal, 29*, 175–192.

Drake, R. E., McHugo, G. J., Clark, R. E., Teague, G. B., Xie, H., Miles, K., et al. (1998). Assertive community treatment for patients with co-occurring severe mental illness and substance use disorder: A clinical trial. *American Journal of Orthopsychiatry, 68*, 201–215.

Drake, R. E., Osher, F. C., & Wallach, M. A. (1989). Alcohol use and abuse in schizophrenia: A prospective community study. *Journal of Nervous and Mental Disease, 177*, 408–414.

D'Souza, D. C., Abi-Saab, W. M., Madonick, S., Forselius-Bielen, K., Doersch, A., Braley, G., et al. (2005). Delta-9-tetrahydrocannabinol effects in schizophrenia: Implications for cognition, psychosis and addiction. *Biological Psychiatry, 57*, 594–608.

El-Guebaly, N., & Hodgins, D. C. (1992). Schizophrenia and substance abuse: Prevalence issues. *Canadian Journal of Psychiatry, 37*, 704–710.

European Monitoring Centre for Drugs and Drug Addiction. (1997). *Improving the comparability of general population surveys on drug use in the European Union*. Luxembourg: Office for Official Publications of the European Communities.

European Monitoring Centre for Drugs and Drug Addiction. (2004). Comorbidity: Drug use and mental disorders. *Drugs in Focus, 14,* 1–4.

European Monitoring Centre for Drugs and Drug Addiction. (2005). *The state of the drugs problem in the European Union and Norway*. Luxembourg: Office for Official Publications of the European Communities.

Fergusson, D. M., Poulton, R., Smith, P. F., & Boden, J. M. (2006). Cannabis and psychosis. *British Medical Journal, 332,* 172–175.

Finlay, J. M. (2001). Mesoprefontal dopamine neurons and schizophrenia: The role of development abnormalities. *Schizophrenia Bulletin, 27,* 431–442.

Foster, J. H., Powell, J. E., Marshall, E. J., & Peters, T. J. (1999). Quality of life in alcohol-dependent subjects—a review. *Quality of Life Research, 8,* 255–261.

Freeman, H. (1994). Schizophrenia and city residence. *British Journal of Psychiatry Supplement, 164*(23), 39–50.

Furr-Holden, C. D., & Anthony, J. C. (2003). Epidemiologic differences in drug dependence: A US-UK cross-national comparison. *Social Psychiatry and Psychiatric Epidemiology, 38,* 165–172.

Goldfinger, S. M., Schutt, R. K., Seidmanm, L. J., Turnerm, W. M., Penk, W. E., & Tolomiczenko, G. S. (1996). Self-report and observer measures of substance abuse among homeless mentally ill persons in the cross-section and over time. *Journal of Nervous and Mental Disease, 184,* 667–672.

Goswami, S., Mattoo, S. K., Basu, D., & Singh, G. (2004). Substance abusing schizophrenics: Do they self-medicate? *American Journal on Addictions, 13,* 139–150.

Gouzoulis-Mayfrank, E. (2004). Doppeldiagnose Psychose und Sucht. Von den Grundlagen zur Praxis. *Nervenarzt, 75,* 642–650.

Graham, H. L., Maslin, J., Copello, A., Birchwood, M., Mueser, K., McGovern, D., et al. (2001). Drug and alcohol problems amongst individuals with severe mental health problems in an inner city area of the UK. *Social Psychiatry and Psychiatric Epidemiology, 36,* 448–455.

Green, A. I., Zimmet, S. V., Strous, R. D., & Schildkraut, J. J. (1999). Clozapine for comorbid substance use disorder and schizophrenia: Do patients with schizophrenia have a reward-deficiency syndrome that can be ameliorated by clozapine? *Harvard Review of Psychiatry, 6,* 287–296.

Gut-Fayand, A., Dervaux, A., Olié, J. P., Lôo, H., Poirier, M. F., & Krebs, M. O. (2001). Substance abuse and suicidality in schizophrenia: A common risk factor linked to impulsivity. *Psychiatry Research, 102,* 65–72.

Hambrecht, M., & Hafner, H. (1996). Substance abuse and the onset of schizophrenia. *Biological Psychiatry, 40,* 1155–1163.

Hamera, E., Schneider, J. K., & Deviney, S. (1995). Alcohol, cannabis, nicotine, and caffeine use and symptom distress in schizophrenia. *Journal of Nervous and Mental Disease, 183,* 559–565.

Havassy, B. E., & Arns, P. G. (1998). Relationship of cocaine and other substance dependence to well-being of high-risk psychiatric patients. *Psychiatric Services, 49,* 935–940.

Heath, A. C., Whitfield, J. B., Madden, P. A., Bucholz, K. K., Dinwiddie, S. H., Slutske, W. S., et al. (2001). Towards a molecular epidemiology of alcohol dependence: Analysing the interplay of genetic and environmental risk factors. *British Journal of Psychiatry Supplement, 40,* 33–40.

Herman, M. (2004). Neurocognitive functioning and quality of life among dually diagnosed and non-substance abusing schizophrenia inpatients. *International Journal of Mental Health Nursing, 13,* 282–291.

Hunt, G. E., Bergen, J., & Bashir, M. (2002). Medication compliance and comorbid substance abuse in schizophrenia: Impact on community survival 4 years after a relapse. *Schizophrenia Research, 54,* 253–264.

Isaac, M., Isaac, M., & Holloway, F. (2005). Is cannabis an anti-antipsychotic? The experience in psychiatric intensive care. *Human Psychopharmacology, 20,* 207–210.

Jeffery, D. P., Ley, A., McLaren, S., & Siegfried, N. (2000). Psychosocial treatment programmes for people with both severe mental illness and substance misuse. *The Cochrane Database of Systematic Reviews,* 2. Art. No.: CD001088. DOI: 10.1002/14651858.CD001088.

Jenkins, R., Bebbington, P., Brugha, T., Farrell, M., Gill, B., Lewis, G., et al. (1997). The National Psychiatric Morbidity Surveys of Great Britain—Strategy and methods. *Psychological Medicine, 27,* 765–774.

Johnson, S. (1997). Dual diagnosis of severe mental illness and substance misuse: A case for specialist services? *British Journal of Psychiatry, 171,* 205–208.

Johnson, S., Nolan, F., Hoult, J., White, I. R., Bebbington, P., Sandor, A., et al. (2005a). Outcomes of crises before and after introduction of a crisis resolution team. *British Journal of Psychiatry, 187,* 68–75.

Johnson, S., Nolan, F., Pilling, S., Sandor, A., Hoult, J., McKenzie, N., et al. (2005b). Randomised controlled trial of acute mental health care by a crisis resolution team: The North Islington crisis study. *BMJ, 331,* 599–602.

Joyal, C. C., Hallé, P., Lapierre, D., & Hodgins, S. (2003). Drug abuse and/or dependence and better neuropsychological performance in patients with schizophrenia. *Schizophrenia Research, 63,* 297–299.

Junginger, J. (1996). Psychosis and violence: The case for a content analysis of psychotic experience. *Schizophrenia Bulletin, 22,* 91–103.

Kamali, M., Kelly, L., Gervin, M., Browne, S., Larkin, C., & O'Callaghan, E. (2001). Insight and comorbid substance misuse and medication compliance among patients with schizophrenia. *Psychiatric Services, 52,* 161–163.

Kashner, T. M., Rader, L. E., Rodell, D. E., Beck, C. M., Rodell, L. R., & Muller, K. (1991). Family characteristics, substance abuse, and hospitalization patterns of patients with schizophrenia. *Hospital and Community Psychiatry, 42,* 195–197.

Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The Positive and Negative Syndromes Scale (PANSS) for schizophrenia. *Schizophrenia Bulletin, 13,* 261–276.

Kendler, K. S. (1985). A twin study of individuals with both schizophrenia and alcoholism. *British Journal of Psychiatry, 147,* 48–53.

Kendler, K. S., Gallagher, T. J., Abelson, J. M., & Kessler, R. C. (1996). Lifetime prevalence, demographic risk factors, and diagnostic validity of nonaffective psychosis as assessed in a US community sample. *Archives of General Psychiatry, 53,* 1022–1031.

Kendler, K. S., & Gardner, C. O. (1997). The risk for psychiatric disorders in relatives of schizophrenic and control probands: A comparison of three independent studies. *Psychological Medicine, 27,* 411–419.

Kendler, K. S., Jacobson, K. C., Prescott, C. A., & Neale, M. C. (2003). Specificity of genetic and environmental risk factors for use and abuse/dependence of cannabis, cocaine, hallucinogens, sedatives, stimulants, and opiates in male twins. *American Journal of Psychiatry, 160,* 687–695.

Kessler, R. C., Nelson, C. B., McGonagle, K. A., Edlund, M. J., Frank, R. G., & Leaf, P. J. (1996). The epidemiology of co-occurring addictive and mental disorders: Implications for prevention and service utilization. *American Journal of Orthopsychiatry, 66,* 17–31.

Khantzian, E. J. (1985). The self-medication hypothesis of addictive disorders: Focus on heroin and cocaine dependence. *American Journal of Psychiatry, 142,* 1259–1264.

Khantzian, E. J. (1997). The self-medication hypothesis of substance use disorders: A reconsideration and recent applications. *Harvard Review of Psychiatry, 4,* 231–244.

Kirkpatrick, B., Amador, X. F., Flaum, M., Yale, S. A., Gorman, J. M., Carpenter, W. T. Jr., et al. (1996). The deficit syndrome in the *DSM–IV* Field Trial: I. Alcohol and other drug abuse. *Schizophrenia Research, 20,* 69–77.

Kirkpatrick, B., Messias, E. M., & Tek, C. (2003). Substance abuse and the heterogeneity of schizophrenia: A population-based study. *Schizophrenia Research, 62,* 293– 294.

Kovasznay, B., Fleischer, J., Tanenberg-Karant, M., Jandorf, L., Miller, A. D., & Bromet, E. (1997). Substance use disorder and the early course of illness in schizophrenia and affective psychosis. *Schizophrenia Bulletin, 23,* 195–201.

Lam, J. A., & Rosenheck, R. A. (2000). Correlates of improvements in quality of life among homeless persons with serious mental illness. *Psychiatric Services, 51,* 116–118.

Lambert, M. T., Griffith, J. M., & Hendrickse, W. (1996). Characteristics of patients with substance abuse diagnoses on a general psychiatry unit in a VA Medical Center. *Psychiatric Services, 47,* 1104–1107.

Launay, C., Petitjean, F., Perdereau, F., & Antoine, D. (1998). Conduites toxicomaniaques chez les malades mentaux: Une enquête en Ile-de-France. *Annales Médico-psychologiques, 156,* 482–485.

Lehman, A. F., Herron, J. D., Schwartz, R. P., & Myers, C. P. (1993). Rehabilitation for adults with severe mental illness and substance use disorders. *Journal of Nervous and Mental Disease, 181,* 86–90.

Lieberman, J. A., Kane, J. M., & Alvir, J. (1987). Provocative tests with psychostimulant drugs in schizophrenia. *Psychopharmacology, 91,* 415–433.

Lindenmayer, J. P., Bernstein-Hyman, R., & Grochowsky, S. (1994). Five factor model of schizophrenia. *Journal of Nervous and Mental Diseases, 182,* 631–638.

Lysaker, P., Bell, M., Beam-Goulet, J., & Milstein, R. (1994). Relationship of positive and negative symptoms to cocaine abuse in schizophrenia. *Journal of Nervous and Mental Disease, 182,* 109–112.

Margolese, H. C., Malchy, L., Negrete, J. C., Tempier, R., & Gill, K. (2004). Drug and alcohol use among patients with schizophrenia and related psychoses: Levels and consequences. *Schizophrenia Research, 67,* 157–166.

Margolese, H. C., Negrete, J., Tempier, R., & Gill, K. (2006). A 12-month prospective follow-up study of patients with schizophrenia-spectrum disorders and substance abuse: Changes in psychiatric symptoms and substance use. *Schizophrenia Research, 83,* 65–75.

McCreadie, R. G., & Scottish Comorbidity Study Group. (2002). Use of drugs, alcohol and tobacco by people with schizophrenia: Case-control study. *British Journal of Psychiatry, 181,* 321–325.

McCrone, P., Menezes, P. R., Johnson, S., Scott, H., Thornicroft, G., Marshall, J., et al. (2000). Service use and costs of people with dual diagnosis in south London. *Acta Psychiatrica Scandinavica, 101,* 464–472.

Menezes, P. R., Johnson, S., Thornicroft, G., Marshall, J., Prosser, D., Bebbington, P., et al. (1996). Drug and alcohol problems among individuals with severe mental illness in south London. *British Journal of Psychiatry, 168,* 612–619.

Meyer, R. E. (1986). How to understand the relationship between psychopathology and addictive disorders: Another example of the chicken and the egg. In R. E. Meyer (Ed.), *Psychopathology and addictive disorders* (pp. 3–16). New York: Guilford Press.

Mowbray, C. T., Ribisl, K. M., Solomon, M., Luke, D. A., & Kewson, T. P. (1997). Characteristics of dual diagnosis patients admitted to an urban, public psychiatric hospital: An examination of individual, social, and community domains. *American Journal of Drug and Alcohol Abuse, 23,* 309–326.

Mueser, K. T., Bond, G. R., Drake, R. E., & Resnick, S. G. (1998). Models of community care for severe mental illness: A review of research on case management. *Schizophrenia Bulletin, 24,* 37–74.

Mueser, K. T., Drake, R. E., & Wallach, M. A. (1998). Dual diagnosis: A review of etiological theories. *Addictive Behaviors, 23,* 717–734.

Mueser, K. T., Essock, S. M., Drake, R. E., Wolfe, R. S., & Frisman, L. (2001). Rural and urban differences in patients with a dual diagnosis. *Schizophrenia Research, 48,* 93–107.

Mueser, K. T., Yarnold, P. R., & Bellack, A. S. (1992). Diagnostic and demographic correlates of substance abuse in schizophrenia and major affective disorder. *Acta Psychiatrica Scandinavica, 85,* 48–55.

Mueser, K. T., Yarnold, P. R., Levinson, D. F., Singh, H., Bellack, A. S., Kee, K., et al. (1990). Prevalence of substance abuse in schizophrenia: Demographic and clinical correlates. *Schizophrenia Bulletin, 16,* 31–35.

Murray, R. M., Grech, A., Phillips, P., & Johnson, S. (2003). What is the relationship between substance abuse and schizophrenia? In R. M. Murray, M. Cannon, P. Jones, et al. (Eds.), *The epidemiology of schizophrenia* (pp. 317–342). Cambridge: Cambridge University Press.

Negrete, J. C., Knapp, W. P., Douglas, D. E., & Smith, W. B. (1986). Cannabis affects the severity of schizophrenic symptoms: Results of a clinical survey. *Psychological Medicine, 16,* 515–520.

Olfson, M., Mechanic, D., Hansell, S., Boyer, C. A., Walkup, J., & Weiden, P. J. (2000). Predicting medication noncompliance after hospital discharge among patients with schizophrenia. *Psychiatric Services, 51,* 216–222.

Overall, J. E., & Gorham, D. R. (1962). Brief Psychiatric Rating Scale. *Psychology Reports, 10,* 799–812.

Owen, R. R., Fischer, E. P., Booth, B. M., & Cuffel, B. J. (1996). Medication noncompliance and substance abuse among persons with schizophrenia. *Psychiatric Services, 47,* 853–858.

Pani, P. P., Trogu, E., Contu, P., Agus, A., & Gessa, G. L. (1997). Psychiatric severity and treatment response in a comprehensive methadone maintenance treatment program. *Drug and Alcohol Dependence, 48,* 119–126.

Perkins, D. O. (1999). Adherence to antipsychotic medications. *Journal of Clinical Psychiatry Supplement, 60,* 25–30.

Phillips, P., & Johnson, S. (2001). How does drug and alcohol misuse develop among people with psychotic illness? A literature review. *Social Psychiatry and Psychiatric Epidemiology, 36,* 269–276.

Phillips, P., & Johnson, S. (2003). Drug and alcohol misuse among inpatients with psychotic illnesses in three inner-London psychiatric units. *Psychiatric Bulletin, 27,* 217–220.

Potvin, S., Sepehry, A. A., & Stip, E. (2006). A meta-analysis of negative symptoms in dual diagnosis schizophrenia. *Psychological Medicine, 36,* 431–440.

Pozzi, G., Bacigalupi, M., & Tempesta, E. (1997). Comorbidity of drug dependence and other mental disorders: A two-phase study of prevalence at outpatient treatment centres in Italy. *Drug and Alcohol Dependence, 46,* 69–77.

Prescott, C. A., & Kendler, K. S. (1999). Genetic and environmental contributions to alcohol abuse and dependence in a population-based sample of male twins. *American Journal of Psychiatry, 156,* 34–40.

Pulver, A. E., Wolyniec, P. S., Wagner, M. G., Moorman, C. C., & McGrath, J. A. (1989). An epidemiologic investigation of alcohol-dependent schizophrenics. *Acta Psychiatrica Scandinavica, 79,* 603–612.

RachBeisel, J., Scott, J., & Dixon, L. (1999). Co-occurring severe mental illness and substance use disorders: A review of recent research. *Psychiatric Services, 50,* 1427–1434.

Razali, M. S., & Yahya, H. (1995). Compliance with treatment in schizophrenia: A drug intervention program in a developing country. *Acta Psychiatrica Scandinavica, 91,* 331–335.

Regier, D. A., Farmer, M. E., & Rae, D. S. (1990). Co-morbidity of mental disorders with alcohol and other drug abuse: Results from the Epidemiological Catchment Area (ECA) study. *Journal of the American Medical Association, 264,* 2511–2518.

Rehm, J., Room, R., van den Brink, W., & Jacobi, F. (2005a). Alcohol use disorders in EU countries and Norway: An overview of the epidemiology. *European Neuropsychopharmacology, 15,* 377–388.

Rehm, J., Room, R., van den Brink, W., & Jacobi, F. (2005b). Problematic drug use and drug use disorders in EU countries and Norway: An overview of the epidemiology. *European Neuropsychopharmacology, 15,* 389–397.

Ridgely, M. S., & Johnson, S. (2001). Drug and alcohol services. In G. Szmukler & G. Thornicroft (Eds.), *Textbook of community psychiatry* (pp. 347–367). Oxford: Oxford University Press.

Salyers, M. P., & Mueser, K. T. (2001). Social functioning, psychopathology, and medication side effects in relation to substance use and abuse in schizophrenia. *Schizophrenia Research, 48,* 109–123.

Schaar, I., & Ojehagen, A. (2003). Predictors of improvement in quality of life of severely mentally ill substance abusers during 18 months of co-operation between psychiatric and social services. *Social Psychiatry and Psychiatric Epidemiology, 38,* 83–87.

Scheller-Gilkey, G., Moynes, K., Cooper, I., Kant, C., & Miller, A. H. (2004). Early life stress and PTSD symptoms in patients with comorbid schizophrenia and substance abuse. *Schizophrenia Research, 69,* 167–174.

Schneier, F. R., & Siris, S. G. (1987). A review of psychoactive substance use and abuse in schizophrenia: Patterns of drug choice. *Journal of Nervous and Mental Disease, 175,* 641–652.

Schofield, N., Quinn, J., Haddock, G., & Barrowclough, C. (2001). Schizophrenia and substance misuse problems: A comparison between patients with and without significant carer contact. *Social Psychiatry and Psychiatric Epidemiology, 36,* 523–528.

Semple, D. M., McIntosh, A. M., & Lawrie, S. M. (2005). Cannabis as a risk factor for psychosis: Systematic review. *Journal of Psychopharmacology, 19,* 187–194.

Serper, M. R., Chou, J. C., Allen, M. H., Czobor, P., & Cancro, R. (1999). Symptomatic overlap of cocaine intoxication and acute schizophrenia at emergency presentation. *Schizophrenia Bulletin, 25,* 387–394.

Sevy, S., Kay, S. R., Opler, L. A., & van Praag, H. M. (1990). Significance of cocaine history in schizophrenia. *Journal of Nervous and Mental Disease, 178,* 642–648.

Shaner, A., Eckman, T., Roberts, L. J., & Fuller, T. (2003). Feasibility of a skills training approach to reduce substance dependence among individuals with schizophrenia. *Psychiatric Services, 54,* 1287–1289.

Soyka, M. (2000). Substance misuse, psychiatric disorder and violent and disturbed behaviour. *British Journal of Psychiatry, 176,* 345–350.

Soyka, M., Albus, M., Kathmann, N., Finelli A., Hofstetter S., Holzbach, R., et al. (1993). Prevalence of alcohol and drug abuse in schizophrenic inpatients. *European Archives of Psychiatry and Clinical Neuroscience, 242,* 362–372.

Strauss, J. S., Carpenter, W. T., & Bartko, J. (1974). The diagnosis and understanding of schizophrenia. III: Speculations on the processes that underlie schizophrenic symptoms and signs. *Schizophrenia Bulletin, 1,* 61–69.

Sullivan, P. F., Kendler, K. S., & Neale, M. C. (2003). Schizophrenia as a complex trait: Evidence from a meta-analysis of twin studies. *Archives of General Psychiatry, 60,* 1187–1192.

Swartz, M. S., Swanson, J. W., Hiday, V. A., Borum, R., Wagner, H. R., & Burns, B. J. (1998). Violence and severe mental illness: The effects of substance abuse and non-adherence to medication. *American Journal of Psychiatry, 155,* 226–231.

Swartz, M. S., Wagner, H. R., Swanson, J. W., Stroup, T. S., McEvoy, J. P., Canive, J. M., et al. (2006). Substance use in persons with schizophrenia: Baseline prevalence and correlates from the NIMH CATIE study. *Journal of Nervous and Mental Disease, 194,* 164–172.

Talamo, A., Centorrino, F., Tondo, L., Dimitri, A., Hennen, J., & Baldessarini, R. J. (2006). Comorbid substance-use in schizophrenia: Relation to positive and negative symptoms. *Schizophrenia Research, 86,* 251–255.

Taub, N. A., Morgan, Z., Brugha, T. S., Lambert, P. C., Bebbington, P. E., Jenkins, R., et al. (2005). Recalibration methods to enhance information on prevalence rates from large mental health surveys. *International Journal of Methods in Psychiatric Research, 14,* 3–13.

Todd, J., Green, G., Pevalin, D. J, Harrison, M., Ikuesan, B. A., Self, C., et al. (2005). Service uptake in a sample of substance misuse and community mental health service clients: A case control study. *Journal of Mental Health, 14,* 95–107.

Tsuang, M. T., Lyons, M. J., Eisen, S. A., Goldberg, J., True, W., Lin, N., et al. (1996). Genetic influences on *DSM–III–R* drug abuse and dependence: A study of 3,372 twin pairs. *American Journal of Medical Genetics, 67,* 473–477.

Van Mastrigt, S., Addington, J., & Addington, D. (2004). Substance misuse at presentation to an early psychosis program. *Social Psychiatry and Psychiatric Epidemiology, 39,* 69–72.

Van Os, J. (2004). Does the urban environment cause psychosis? *British Journal of Psychiatry, 184,* 287–288.

Verdoux, H., Mury, M., Besancon, G., & Bourgeois, M. (1996). Comparative study of substance dependence comorbidity in bipolar, schizophrenic and schizoaffective disorders. *Encephale, 22,* 95–101.

Volkow, N. D., Gillespie, H., Mullani, N., Tancredi, L., Grant, C., Valentine, A., et al. (1996). Brain glucose metabolism in chronic marijuana users at baseline and during marijuana intoxication. *Psychiatry Research, 67,* 29–38.

Wang, P. S., Lane, M., Olfson, M., Pincus, H. A., Wells, K. B., & Kessler, R. C. (2005). Twelve-month use of mental health services in the United States: Results from the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62,* 629–640.

Weaver, T., Madden, P., Charles, V., Stimson, G., Renton, A., Tyrer, P., et al. (1999). Severe mental illness and substance misuse comorbidity: Research is needed to inform policy and service development. *BMJ, 318,* 37–38.

Weaver, T., Madden, P., Charles, V., Stimson, G., Renton, A., Tyrer, P., et al. (2003). Comorbidity of substance misuse and mental illness in community mental health and substance misuse services. *British Journal of Psychiatry, 183*, 304–313.

Weisner, C., & Schmidt, L. (1993). Alcohol and drug problems among diverse health and social service populations. *American Journal of Public Health, 83*, 824–829.

Wheatley, M. (1998). The prevalence and relevance of substance misuse in detained schizophrenic patients. *Journal of Forensic Psychiatry, 9*, 114–129.

WHO World Mental Health Survey Consortium. (2004). Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization World Mental Health Surveys. *Journal of the American Medical Association, 291*, 2581–2590.

Wright, S., Gournay, K., Glorney, E., & Thornicroft, G. (2000). Dual diagnosis in the suburbs: Prevalence, need, and in-patient service use. *Social Psychiatry and Psychiatric Epidemiology, 35*, 297–304.

Zammit, S., Allebeck, P., Andreasson, S., Lundberg, I., & Lewis, G. (2002). Self reported cannabis use as a risk factor for schizophrenia in Swedish conscripts of 1969: Historical cohort study. *BMJ, 325*, 1199–1204.

Zubin, J., & Spring, B. (1977). Vulnerability—a new view of schizophrenia. *Journal of Abnormal Psychology, 86*, 103–126.

This page intentionally left blank

Chapter **18**

Neural Basis for Methamphetamine Addiction—Rethinking the Definition of Dependence

Mary F. Holley, MD

Addiction to methamphetamine is a serious disabling condition that affects individuals, families, and communities as end-stage addicts destroy their lives, abuse their children, and lose their jobs. The availability of effective treatment is limited by the economic resources available to treat what many consider an incurable moral failure. Taxpayers and donors are often unwilling to devote large amounts of money to rehabilitate people who voluntarily ingest drugs of abuse. The need for prolonged periods of rehabilitation and extended supervision is even greater for methamphetamine addiction than for addiction to other drugs of abuse. A better understanding of the biologic nature of addiction is needed on all fronts, by addicts, their families, health care policy makers and payers, the legal and judicial fields, and the community at large.

Because of the lack of available treatment, an overwhelming burden is being placed on our corrections system by the sheer volume of inmates sentenced primarily for drug offenses. Corrections facilities are designed to punish and deter antisocial behavior in rational persons. They are now being asked to intervene in the disease of addiction although they are neither funded nor staffed to treat addiction. Addicts do not usually make rational choices but rather are driven by a brain disease that must be treated to prevent the recidivism that plagues our current system.

Even in the field of psychiatry, there is tremendous reluctance to admit that methamphetamine is a neurotoxin and that brain tissue is damaged by it. This in spite of the persuasive animal and human data confirming the serious neurotoxicity, particularly to the frontal lobes, related to methamphetamine use. And

this reluctance is seen in card-carrying Darwinists. Can methamphetamine be a neurotoxin in all known animal models and yet fall harmless to the ground when confronted with the mystical, magical human brain? There is nothing special about human neurons that render them impervious to the oxygen free radical compounds released by methamphetamine.

If we wish to convince the general public and health care policy makers that addiction is a brain disease and not merely a moral failure, we must define it in objective terms, not just in behavioral terms. Our current definition of dependence has as much to do with the tolerance of the patient's employer for poor performance as it does the patient's behavior. We should instead define it in terms of neurochemical, behavioral, and personality changes, which appear early in the clinical course, long before "dependence" is diagnosed under the current paradigm.

NEUROTRANSMISSION

Changes in the neurotransmitter levels of the brain underlie many of the personality changes and mental illnesses associated with methamphetamine. The principal chemicals affected by the use of meth are the monoamines dopamine, serotonin, norepinephrine, and cortisol, a stress hormone.

Methamphetamine stimulates a massive release of monoamines, especially norepinephrine, a 2,000 percent increase (Rothman, Bauman, Dersch, Romero, & Rice, 2001), dopamine, a 500–1,000 percent increase (Gough, Imam, Blough, Slikker, & Ali, 2002; Izawa, Yamanashi, Asakura, Misu, & Goshima, 2006), and serotonin, a 300–500 percent increase (Ago, Nakamura, Baba, & Matsuda, 2008) by both presynaptic and postsynaptic mechanisms. Meth causes the accelerated release of vesicles containing these neurotransmitters, and reverses the presynaptic transporter molecule that normally reabsorbs neurotransmitter once released (Zaczek, Culp, & De Souza, 1991). Methamphetamine changes the conformation of the transporter molecule causing the "vacuum cleaner" to go into reverse and spew neurotransmitter out instead of reabsorbing it (Yudko, Hall, & McPherson, 2003, p. 39). This is in contrast to cocaine, which only blocks the reabsorption of neurotransmitter.

Postsynaptically, meth inhibits the enzyme monoamine oxidase (MAO), which is responsible for metabolizing these neurotransmitters once they are released. This increases the length of time the transmitter is active at the receptor site and thus its effects in the brain (Ramsay & Hunter, 2003). When MAO is inhibited, alternate routes of metabolism are used, resulting in delayed metabolism and hydroxy free radical formation. Methamphetamine stimulation also triggers an increase in gluatamine receptor density in nucleus accum-

bens, acutely increasing its responsiveness to repetitive stimulation (Chao, Ariano, Peterson, & Wolf, 2002).

Methamphetamine also disrupts the hypothalamic pituitary axis, causing a release of cortisol, a stress hormone. Higher cortisol levels are associated with greater dopamine release in the striatum and more positive subjective drug effects (Oswald et al., 2005). Glucocorticoid receptors in the midbrain are affected in the striatum and hippocampus, which is thought to underlie stress-related relapse (Wang et al., 2005).

SENSITIZATION STAGE OF ADDICTION

Methamphetamine addiction progresses through two distinct biochemical stages. Only the later stage is characteristically recognized as *addiction* or *dependence* in the terminology of *DSM–IV.* Those patients still in the initial sensitization stage are considered users, but have not yet had negative sequellae from their drug use and so have not tried to quit or cut down. In sensitization, each dose feels even better than before, lasts longer, and is more intense. Having heard about tolerance, the user understands that only when his drug use accelerates would he be considered an addict, so during this early stage, he assures himself he must be ok. He does not believe his methamphetamine use is having any effect on his brain.

During the sensitization period, neural tracts that are hyperstimulated on an occasional basis by low intensity use become more responsive to methamphetamine. These neural tracts have been shown to increase their sensitivity to repeated doses of methamphetamine, both by increasing numbers of receptors for monoamine neurotransmitters, and also by increasing numbers of glutamate receptors in the brain (Xui, Koeltzow, Cooper, Robertson, & White, 2002). We add more lanes to accommodate increased traffic.

And in the sensitization period, the neurons still have a reserve of intracellular neurotransmitter that can be mobilized by axonal transport, to replenish the terminals with fresh supplies of neurotransmitter, thus enabling the cells to function in the face of massive losses in their neurotransmitter supplies (Yudko et al., 2003). This is why, in the early stage of addiction, function is maintained between "highs" and the "crash" after using is just a mild depression, not even as bad as the hangover after an evening of heavy alcohol intake.

This phase may last for years in the case of oral use of small doses of methamphetamine, and there are medical uses for methamphetamine for treatment of Attention Deficit Hyperactivity Disorder (ADHD), narcolepsy, and weight loss in small closely monitored doses. The military has used methamphetamine in small closely regulated doses for many years with a long record of safety.

Unmonitored use, however, readily lends itself to compulsive and repetitive use with resultant personality changes—what any unbiased observer would call *addiction*.

At this early stage the addict is almost never willing to admit she has a problem. She uses frequently, but perhaps not daily. She feels focused and energetic while on methamphetamine. Her family members notice her personality changes, her short temper and impatience, but she thinks everything is fine. She cannot quit using for any prolonged length of time—what the average person would consider addiction—but she has never really tried to quit, nor does she consider herself impaired in any way, and so a psychiatrist would not diagnose her *dependent*. Psychiatry has rejected the word *addiction* as a diagnostic term. It is used here purely as a functional term referring to the habitual and compulsive use of a substance.

Dependence is diagnosed only when strict criteria are met, including three of the following conditions:

Tolerance
Withdrawal symptoms
Escalation of use
Effort to control use
Occupies time, effort
Replaces other activities
Use despite impairment

Only in the later stage of addiction, when drug use impairs functioning and replaces normal healthy activities, resulting in the loss of relationships or employment, does a psychiatrist consider the individual an addict (dependent). This functional definition has more to do with the reaction of his wife and the demands of his employer than it does with the biochemical effects the drug is having in his brain. He won't make an effort to control his use until threatened with an employee drug test. The level of denial in such a person guarantees that the subjective measures used to define dependence will not apply to him until a set of handcuffs has been applied at least once. The changes in his brain are at an advanced stage by now. By the time dependence is diagnosed, serious mental health problems are often evident, and recovery of normal cognition and personality features will require months, if not years, of intense therapy.

Early diagnosis and intervention are impossible with the current definition of dependence (or addiction) used by psychiatry. In fact, people requesting treatment for their addiction are sometimes turned away by mental health providers because they are "not addicted enough," particularly in the public sector.

LATE-STAGE ADDICTION

In later-stage addiction, neurotransmitter reserves have been depleted as continued massive releases of monoamines have exhausted the cell's capacity to manufacture these complex chemicals de-novo (Vacca, Ahn, & Phillips, 2006). Presynaptic function is also impaired by chronic methamphetamine exposure, with corresponding behavioral changes, an effect that persists at least four months into recovery in an animal model (Melega et al., 2008). Striatal dopamine concentration (reserve) is reduced by 20 percent and presynaptic dopamine transporter density is reduced by 35 percent. Chronic presynaptic depression of function is "renormalized" in cortico-striatal pathways by readministration of methamphetamine, restoring the system to apparent normalcy (Bamford et al., 2008).

At the same time, the postsynaptic side has responded to the massive hyperstimulation by phospohorylating, sequestering, and degrading its receptors (Volkow et al., 2001). While D1 receptors in the striatum are preserved, they are delinked from the adenylyl cyclase that serves as its second messenger in the postsynaptic cell (Tong et al., 2003). The dopamine response is suppressed in response to both psycho stimulants and natural rewards in withdrawal from methamphetamine (Vacca et al., 2006). More and more stimulation is required to trigger a postsynaptic response. The crash becomes more symptomatic and longer lasting as neurotransmitters and receptors are depleted. The addict accelerates his drug dose and interval in an effort to reclaim the high and/or avoid the crash.

As dopamine transmission is impaired in the reward circuit, higher doses of methamphetamine are required to maintain function. The recreational user can't wait until Friday to use again, and the functional user needs higher doses to maintain his current level of productivity. As higher doses are used, side effects, including jitteriness and disorganization, are seen, which impair his occupational adjustment. He is generally oblivious to this change and to the personality changes that are also occurring. His boss and his wife, however, usually are not oblivious to these changes.

His irritability has by now progressed to domestic violence—either verbal or physical. His children are afraid of him. His work performance has become erratic and customers and coworkers are complaining. Personality changes are the hallmark sign of the methamphetamine user. They are much more prominent than those seen in heroin or even cocaine users, and they affect every aspect of life. Some of these personality changes are quite reversible since they are mediated by biochemical changes in brain function, not by structural changes.

But they improve very slowly, over the course of many months of abstinence as neurotransmission is reestablished (Wang et al., 2004).

Other changes seen in later-stage methamphetamine addiction are not as readily reversible. The loss of memory, cognitive ability, motivation, and reality testing are related to structural damage to brain tissue caused by cellular damage to the brain (Thompson et al., 2004). These changes also result in the deepening of the addiction as key structures related to self-control are compromised.

CYTOTOXICITY

The massive release of neurotransmitters caused by high dose and chronic methamphetamine use results in high levels of nitrogen and oxygen free radical formation (Acikgov et al., 2000). These free radicals are formed by the metabolism of methamphetamine, and also by the breakdown of the huge amounts of monoamine neurotransmitters that have been released both intra- and extracellularly. These monoamine neurotransmitters must also be broken down, and MAO, the usual enzyme to do that, is inhibited by methamphetamine. Alternative metabolic routes are used, resulting in the generation of large amounts of hydroxyl free radicals nitric oxide and peroxynitrite, which are extremely toxic to brain cells (Jeng, Ramkissoon, Parman, & Wells, 2006).

Free radical compounds denature proteins, damage DNA, and generally wreak havoc in the areas of the brain in which they are concentrated (Cubbells, Rayport, Rajendran, & Sulzer, 1994). Because most of the neurotransmitters are released in the midbrain, nucleus accumbens, and striatum, and in the prefrontal cortex, those areas are disproportionately affected by methamphetamine abuse with progressively worsening cognitive and executive function (Li, Wang, Qiu, & Luo, 2008).

The power of these free radicals to damage the human brain was demonstrated most vividly by Thompson in 2004 when he and his colleagues demonstrated up to 15 percent loss of brain tissue in large areas of the brain, including both cortical and subcortical tissue in methamphetamine users. These findings were correlated with cognitive and memory defects in the subjects studied. Thompson described it as a "forest fire of brain damage" with real-world consequences in occupational failure, disintegration of relationships, and challenges in treatment.

Methamphetamine use causes persistent hypometabolism in the frontal white matter and impairment in frontal executive function on positron emission tomography (PET) scanning (Kim, Lyoo, Hwang, Sung, & Lee, 2005). Abstinent meth users showed impaired performance on the Wisconsin card-

sorting test associated with reduced metabolism in the right superior frontal lobe. Hypo-frontality in methamphetamine addicts has been thought to contribute to the significant cognitive deficits, memory loss, and poor impulse control that cause significant social failure and complicate treatment participation and success (Homer et al., 2008). A realistic assessment of the nature and extent of these deficits is essential to developing effective treatment programs.

THE PLEASURE CIRCUIT

The pleasure circuit has been well described, and consists of the nucleus accumbens, anterior bed nuclei, anterior lateral hypothalamus, stria terminalis, lateral preoptic area, median forebrain bundle, ventral tegmental area, ventral pallidum, and prefrontal cortex. These areas are profoundly affected by methamphetamine administration, with altered sensitivity and receptor changes in animal models (Brady, Glick, & O'Donnell, 2005; Broom & Yamamoto, 2005; Yong & Kauer, 2003).

Direct stimulation of the nucleus accumbens by dopamine results in euphoria. The ventral tegmental area sends numerous dopaminergic neurons to the nucleus accumbens, contributing to reward and motivation in response to natural pleasures. Methamphetamine increases dopamine in the nucleus accumbens by up to 1,000–1,200 percent, provoking a powerful pleasurable sensation and triggering a powerful motivator. As these dopamine receptors are damaged by overstimulation, natural rewards are not appreciated, and motivation is impaired.

The prefrontal cortex is an integral part of the reward circuit. Pleasures are experienced in all their richness in the prefrontal cortex, and cravings originate in these areas as marked by intense neural activity on exposure to triggers (Kalivas, Volkow, & Seamans, 2005). Prefrontal cortex is hyperresponsive to drug cues, driving the nucleus accumbens, while at the same time, executive function is reduced, diminishing cognitive control. Wilson's analysis of these studies showed the orbitofrontal and dorsolateral orbitofrontal cortex more active in addicts anticipating drug usage, while anterior cingulate cortex was more activated in those trying to resist the urge to drug usage—treatment-seeking individuals (Wilson, Sayette, & Fiez, 2004).

Appeals to the pleasure circuit to explain all aspects of addictive behavior are found wanting in that as addiction proceeds, pleasurable sensations decline, and addicts are often motivated to use substances that no longer give them very much pleasure. Motivation shifts from obtaining pleasure and avoiding the pain and anxiety of withdrawal to compulsive use in the face of serious adverse consequences. The ability of the conscious mind to control behavior is

seriously compromised in addiction, particularly methamphetamine addiction, even when competent cognitive behavioral therapy is received and mastered. This suggests a parallel and separate anatomic basis for behavior control apart from hedonic perception.

THE CONTROL CIRCUIT

The control circuit is less understood, and is not included in most textbooks on the neurophysiology of addiction. The original literature, however, supports the existence of a dedicated set of structures that serve to facilitate control of behavior in the face of desire or craving. This control circuit consists of the prefrontal cortex, anterior cingulate gyrus, the lateral habenula, and fasciculus retroflexus, which exerts inhibitory GABA-A control over the midbrain craving centers in the ventral tegmental area (Ji & Shepard, 2007). There are also direct inhibitory connections between prefrontal cortex and VTA that are also GABA-mediated (Carr & Sesack, 2000).

The influence of this circuit in the modulation of addiction has been delineated in several human studies. Volkow and colleagues in 2001 demonstrated a loss of dopamine receptors in the entire orbitofrontal cortex in abstinent methamphetamine users. More specifically, methamphetamine users showed significantly reduced cerebral blood flow in the anterior cingulate gyrus, with a significant persistent reduction even after six months' abstinence. This suggests a structural change, not just a functional neurotransmitter mediated effect, in the anterior cingulate gyrus, an important area for control of impulses and behavior (Hwang et al., 2006).

Multiple studies have demonstrated reduced task-related activation of the anterior cingulated gyrus in methamphetamine users. Paulus, Tapert, and Schuckit in 2005 showed that those addicts who eventually relapsed had markedly reduced activation of the dorsolateral prefrontal cortex and anterior cingulate gyrus compared to addicts who did not subsequently relapse. Subjects were followed for up to three years to observe for relapse, and the predictive power of this functional measure of brain activity in these areas was impressive.

The cingulum bundle conducts impulses from anterior cingulate gyrus and other prefrontal areas posteriorly, primarily to the hippocampus, but also to multiple other midbrain structures, including the lateral habenula. The cells in this transmission line are exquisitely sensitive to methamphetamine, with destruction of more than 90 percent of the cingulum bundle demonstrated after just a single intoxicating dose of methamphetamine in animal models (Zhou & Bledsoe, 1996). This is not just a change in the sensitivity of the

neurons, but the destruction of a key pathway between the cingulate gyrus and midbrain structures, including hippocampus and lateral habenula.

The lateral habenula itself is sensitive to the effects of methamphetamine with specific degeneration of large areas of lateral habenula with continuous exposure to meth as would be seen in a binge pattern of self-administration (Ellison, 1992). In 2000, Carlson and colleagues reported that many drugs of abuse impair function in the habenula and fasciculus retroflexus, dubbing it "the weak link in addiction." This line of research was then almost completely neglected for many years until recent studies have further delineated the significance of Ellison's and Carlson's findings.

The lateral habenula is a significant processing center conveying information from cognitive cortical areas to subcortical areas. Habenular lesions result in learning deficits, and reductions in memory and attention consistent with its central role in cognition (Lecourtier & Kelly, 2007). In humans, the lateral habenula is especially responsive to feedback about errors, exerting inhibitory impulses when errors are detected and response patterns need to be changed (Ullsperger & Cramon, 2003).

Lateral habenula neurons in the primate are activated in a no-reward condition, exerting inhibitory control over dopamine release from the ventral tegmental area (Matsumoto & Hikosaka, 2007). In this study even weak stimulation of lateral habenula elicited strong inhibition of dopamine release. Dopamine levels are thus decreased when predicted rewards do not occur, a biological basis for disappointment (Pagnoni, Zink, Montague, & Berns, 2002).

Specifically, lateral habenula in turn exerts a powerful inhibitory effect on dopamine transmission by the ventral tegmental area via the fasciculus retroflexus, a GABAergic pathway. Ji and Shepard (2007) did the definitive study of this tract, demonstrating that single-pulse stimulation of the lateral habenula effectively shut down the activity of 97 percent of the dopaminergic neurons in the substantia nigra and ventral tegmental area. Stimulation of the lateral habenula resulted in a complete cessation of spontaneous firing in nearly all dopamine neurons in the substantia nigra and ventral tegmental areas. Lesions of the fasciculus retroflexus completely blocked this strongly inhibitory effect on dopamine neurons.

These ventral tegmental dopaminergic signals are responsible for recurrent drug-taking behaviors even in the absence of an external trigger for the hedonic reward pathway (Nakajima et al., 2004). Uncontrolled ventral tegmental stimulation of the nucleus accumbens produces dopamine signals that are experienced in the frontal cortex as cravings and the desire to get high. If sufficient inhibitory control in the frontal cortex is not present to suppress these signals, behavior is likewise uncontrolled.

PROPOSED TREATMENTS

Since a number of interrelated adaptations to drug use occur in multiple areas of the brain, it would be expected that intervention would also have to occur at multiple levels for successful rehabilitation. There are adaptations in the reward areas, motivation and drive, memory and conditioning, and inhibitory control areas that result in long-lasting changes in a person's responsiveness to natural rewards, cognitive ability, and inhibitory control. While acute drug intake increases dopamine release, chronic use impairs it not only in the reward centers but also in the frontal lobes (cognitive ability) and cingulate gyrus (inhibitory control) (Volkow, Fowler, & Wang, 2004).

GABA-mediated inhibitory control is a target for many of the newly proposed treatments for addiction, including baclofen, gabapentin, and vigabatrin. These drugs are GABA A agonists (baclofen), or GABA transaminase inhibitors (gabapentin and vigabatrin), essentially amplifying the inhibitory signals and thus improving impulse control and reducing craving. Animal studies were very promising (Barrett, Negus, Mello, & Caine, 2005; Di Ciano & Everitt, 2004; Filip et al., 2007), as were open-label studies using baclofen and gabapentin (Urschell, Hanselka, Gromov, White, & Baron, 2007) and vigabatrin (Brodie, Fiqueroa, Laska, & Dewey, 2005; Fechtner, Khouri, Figueroa, Ramirez, & Federico, 2006). However, double-blind studies of baclofen and gabapentin showed no effect (Heinzerling et al., 2006; Shoptaw et al., 2003). A randomized controlled trial of vigabatrin is needed. Though vigabatrin has been linked to visual field defects with long-term use, its safety in short-term use is suggested by the open-label studies completed (Brodie et al., 2005).

Reducing the reward value of methamphetamine is another biochemical target area that is open to intervention. As in the case of heroin addiction and its partial agonist buprenorphine, there is a partial agonist for stimulant drugs in the form of modafinil. In animal studies, modafinil substituted partially for both cocaine and amphetamine in rats trained to discriminate these stimulants from saline, but was much less potent (Dopheide, Morgan, Rodvelt, Schachtman, & Miller, 2007). While it is not a dopamine receptor agonist, it has a similar clinical profile to stimulants with alertness and cognitive improvement and does increase dopamine release in the nucleus accumbens (Murillo-Rodríguez, Haro, Palomero-Rivero, Millán-Aldaco, & Drucker-Colín, 2007). A double-blind controlled trial of modafinil showed it is effective in reducing cocaine dependence with few adverse effects (Dackis, Kampman, Lynch, Pettinati, & O'Brien, 2005). It is especially beneficial in improving cognitive performance and thus participation with cognitive behavioral therapy (Makris,

2007; Minzenberg & Carter, 2008). Buproprion, a dopamine and norepinephrine reuptake blocker, has also been shown effective in a subset of men using low doses of methamphetamine, but was not effective for the population at large (Elkashef et al., 2008; Shoptaw et al., 2008).

Alternatively, dopamine blockade has been evaluated for effectiveness in reducing stimulant relapse. Selective D3 receptor antagonists reduced cocaine seeking behavior in rats (Xi et al., 2006; Cervo, Cocco, Petrella, & Heidbreder, 2006). Available D2 blockers including arapiprozole (Beresford et al., 2005) and olanzapine (Smelson et al., 2006) relieve cravings in comorbid schizophrenic stimulant addicts, but with the usual anhedonic side effects characteristic of these medications.

NONPHARMACOLOGIC APPROACHES

But there are nonpharmacologic ways of influencing the self-control tract of the brain and enhancing its function, some of which the rehabilitation industry has used for years without really understanding the neurobiology behind them. One of them is the "boot camp" approach. All rehabilitation programs include at least a component of this approach, and some are almost exclusively a boot-camp experience. Chores and schedules and responsibilities are expected and are recognized as important to recovery from addiction.

The value of these interventions consists of the conditioning, and in some cases, regeneration, of damaged tissue, restoring function in the areas of the brain mediating self-control. Classical rehabilitation techniques are used from the physical therapy paradigms and applied to behavioral rehabilitation with good success. When a neural tract is impaired, for instance, after a stroke, rehabilitation consists of forcing the relevant area of the brain to work, thus facilitating the recruitment of surrounding surviving cells to take over the function of the diseased tissue, a process called activity-dependent plastic change, or neuroplasticity (Ward, 2005).

In the same way, when the self-control pathway is compromised, rehabilitation consists of imposing conditions that force the patient to exert inhibitory control over her behavior, facilitating recruitment of surviving cells to take over the function of the diseased tissue. This is particularly effective at the cortical level in retraining the anterior cingulate cortex to control behavior. Addicts are "forced" to get up at a given time, do chores, and keep a schedule, so that impulse inhibiting areas are stimulated to function on a regular basis. The principles of neuroplasticity ensure that such "exercise" will stimulate recovery of function by increases in brain-derived neurotrophic factor (BDNF), dendritic arborization, and synaptic plasticity.

Contingency management capitalizes on these same principles, as an external motivator is used to improve compliance and modify behavior. Coupled with cognitive behavioral therapy and the social support found in the group therapy setting, success in treatment of methamphetamine addiction is comparable to treatment of other addictions. Cognitive and attentional problems limit the application of cognitive behavioral techniques in early abstinence, but as the brain heals and remodels, retention and insight improve and treatment outcomes are comparable to those obtained with treatment of cocaine and other drug addictions (Rawson et al., 2000).

FUTURE DIRECTIONS

Until now, most neuropharmacology research has been devoted to understanding the reward system of brain physiology with an eye toward blocking the rewards associated with drug abuse with dopamine blockers of various types. Atypical antipsychotic medications have been used and found effective, but anhedonia is a frequent side effect. Clinical usefulness is limited to those patients who are willing to forgo all rewards for an indefinite period of time. Compliance with such therapy is usually confined to the court-ordered population. Substitution therapy with modafinil is more likely to be effective and acceptable to patients, but carries its own risks of exacerbating mental illnesses such as bipolar disorder and psychosis, and also cardiovascular complications (Kampman, 2008).

More attention should be directed toward the self-control system to develop new treatments based on enhancing the addict's control over her own behavior. GABAergic medications offer the potential to amplify normal inhibitory neural tracts to improve impulse control at the midbrain level, while behavioral modification and cognitive behavioral therapy addresses the cortical components of the self-control circuit. This could lead to improvements in the effectiveness of our current contingency management and motivational enhancement techniques, both of which are components of effective cognitive behavioral treatment. A greater understanding of the neurologic components and functional biochemistry of the self-control factor would extend pharmacologic and nonpharmacologic support to empower addicts to control their own lives instead of being controlled by drugs of abuse.

Our enhanced biological understanding of addiction would also permit a more objective definition of drug dependence itself, thus avoiding the denial and deception that complicates the accurate diagnosis of addiction. We are in dire need of an accessible biochemical or radiological marker for drug dependence that does not rely on subjective discomfort, third-party report, or personal desire for change, all of which can be missing in a person who is

shooting up daily and sustaining serious neurologic damage. The level of denial and deception among drug users is legendary. A scan documenting the loss of dopamine activity in the midbrain might be fairly motivating to a patient considering rehabilitation for her drug problem. Earlier intervention would be possible if addiction could be diagnosed at an earlier stage before significant frontal lobe damage has occurred.

Much work needs to be done to identify dopamine, serotonin, or norepinephrine metabolites in peripheral blood, develop scanning techniques to identify brain cell dysfunction, or cognitive testing, or scales of personality parameters that could inform users of the effect drug use is having on their brains before their condition worsens to the point of serious disability. We are close to having the capability of doing just that. Morris, Normandin, and Schiffer (2008) have validated a PET-based technique that accurately measures microdialysis confirmed dopamine levels noninvasively. Our reliance on the recognition of late behavioral changes diagnostic of addiction under *DSM IV* is keeping us behind the curve when assessing the impact methamphetamine is having on individual lives and on society as a whole.

Addiction must be defined and diagnosed in neurologic terms if it is to be recognized as a "brain disease" deserving of comprehensive and compassionate treatment and not a "moral failure" subject to incarceration. A better public understanding of addiction as a brain disease would move it out of the court system and into the therapist's office, with corresponding improvements in public perception, patient self-image, relegation of public resources, and insurance coverage.

REFERENCES

Acikgov, O., Gapnenas, S., Kayatekin, B. M., Pekasetin, C., Uusal, N., Dayi, A., et al. (2000). The effects of a single dose of methamphetamine on lipid peroxidation levels in the rat striatum and prefrontal cortex. *European Neuropsychopharmacology, 10*(5), 415–418.

Ago, Y., Nakamura, S., Baba, A., & Matsuda, T. (2008). Neuropsychotoxicity of abused drugs: Effects of serotonin receptor ligands on methamphetamine and cocaine induced behavioral sensitization in mice. *Journal of Psycopharmacologic Sciences, 106*(1), 15–21.

Bamford, N. S., Zhang, H., Joyce, J. A., Scarlis, C. A., Hanan, W., Wu, N. P., et al. (2008). Repeated exposure to methamphetamine causes long lasting presynaptic cortiostriatal depression that is normalized with drug readministration. *Neuron, 58*(1), 89–103.

Barrett, A. C., Negus, S. S., Mello, N. K., & Caine, S. B. (2005). Effect of GABA agonist and GABA A receptor modulators on cocaine and food maintained respond-

ing and cocaine discrimination in rats. *Journal of Pharmacology and Experimental Therapeutics, 315,* 858–871.

Beresford, T. P., Clapp, L., Martin, B., Wiberg, J. L., Alfers, J., Beresford, H. F. (2005). Aripiprazole in schizophrenia with cocaine dependence: A pilot study. *Journal of Clinical Psychopharmacology, 25,* 363–366.

Brady, A. M., Glick, S. D., & O'Donnell, P. (2005). Selective disruption of nucleus accumbens gating mechanisms in rats behaviorally sensitized to methamphetamine. *Journal of Neuroscience, 25,* 6687–6695.

Brodie, J. D., Fiqueroa, E., Laska, E. M., & Dewey, S. L. (2005). Safety and efficacy of GVG for the treatment of methamphetamine and/or cocaine addiction. *Synapse, 55*(2), 122–125.

Broom, S. L., & Yamamoto, B. K. (2005). Effects of subchronic methamphetamine exposure on basal dopamine and stress induced dopamine release in the nucleus accumbens shell of rats. *Psychopharmacology, 181,* 467–476.

Carlson, J., Armstrong, B., Switzer, R. C., & Ellison, G. (2000). Selective neurotoxic effects of nicotine on axons in fasciculus retroflexus further support evidence that this is a weak link in brain across multiple drugs of abuse. *Neuropharmacology, 39*(13), 2792–2798.

Carr, D. B., & Sesack, S. (2000). Projections from the rat prefrontal cortex to the ventral tegmental area: Target specificity in the synaptic associations with meso accumbens and mesocortical neurons. *Journal of Neuroscience, 20*(10), 3864–3873.

Cervo, L., Cocco, A., Petrella, C., & Heidbreder C. A. (2006). Selective antaqgonism at dopamine D3 receptors attenuates cocaine-seeking behavior in the rat. *International Journal of Neuropsychopharmacology, 23,* 1–15.

Chao, S. Z., Ariano, M. A., Peterson, D. A., & Wolf, M. E. (2002). D1 dopamine receptor stimulation increases GluR1 surface expression in nucleus accumbens neurons. *Journal of Neurochemistry, 83*(3), 704–712.

Cubells, J. F., Rayport, S., Rajendran, G., & Sulzer, D. (1994). Methamphetamine neurotoxicity involves vacuolization of endocytic organelles and dopamine dependent intracellular oxidative stress. *Journal of Neuroscience, 14*(4), 2260–2271.

Dackis, C. A., Kampman, K. M., Lynch, K. G., Pettinati, H. M., & O'Brien, C. P. (2005). A double blind, placebo controlled trial of modafinil for cocaine dependence. *Neuropsychopharmacology, 30*(1), 205–211.

Di Ciano, P., & Everitt, B. J. (2004). Contribution of the ventral tegmental area to cocaine seeking maintained by a paired drug conditioned stimulus in rats. *European Journal of Neuroscience, 19,* 1991–1997.

Dopheide, M. M., Morgan, R. E., Rodvelt, K. R., Schachtman, T. R., & Miller, D. K. (2007). Modafinil evokes striatal [(3)H]dopamine release and alters the subjective properties of stimulants. *European Journal of Pharmacology, 568*(1–3), 112–123.

Elkashef, A. M., Rawson, R. A., Anderson, A. L., Li, S. H., Holmes, T., Smith, E. V., et al. (2008). Buproprion for the treatment of methamphetamine dependence. *Neuropsychopharmacology, 33*(5), 1162–1170.

Ellison, G. (1992). Continuous amphetamine and cocaine have similar neurotoxic effects in lateral habenular nucleus and fasciculus retroflexus. *Brain Research, 598,* 352–356.

Fechtner, R. D., Khouri, A. S., Figueroa, E., Ramirez, M., & Federico, M. (2006). Short term treatment of cocaine and or methamphetamine abuse with vigabatrin: Ocular safety pilot results. *Archives of Ophthalmology, 124,* 1257–1262.

Filip, M., Frankowska, M., Zaniewska, M., Goada, A., Przegaliaski, E., & Vetulani, J. (2007). Diverse effects of GABA mimetic drugs on cocaine evoked self administration and discriminative stimulus effects in rats. *Psychopharmacology, 192,* 17–26.

Gough, B., Imam, S. Z., Blough, B., Slikker, W., Jr., & Ali, S. F. (2002). Comparative effects of substituted amphetamines (PMA, MDMA, and METH) on monoamines in rat coudate: A microdialysis study. *Annals NY Academy of Science, 965,* 410–420.

Heinzerling, K. G., Shoptaw, S., Peck, J. A., Yang, X., Liu, J., Roll, J., et al. (2006). Randomized placebo controlled trial of baclofen and gabapentin for the treatment of methamphetamine dependence. *Drug and Alcohol Dependence, 85,* 177–184.

Homer, B. D., Solomon, T. M., Moeller, R. W., Mascia, A., DeRaleau, L., & Halkitis, P. N. (2008). Methamphetamine abuse and impairment of social functioning: A review of the underlying neurophysiological causes and behavioral implications. *Psychological Bulletin, 134*(2), 301–310.

Hwang, J., Lyoo, I. K., Kim, S. J., Sung, Y. H., Bae, S., Cho, S.N., et al. (2006). Decreased cerebral blood flow of the right anterior cingulate cortex in long term and short term abstinent methamphetamine users. *Drug and Alcohol Dependence, 28,* 177–181.

Izawa, J., Yamanashi, K., Asakura, T., Misu, Y., & Goshima, Y. (2006). Differential effects of methamphetamine and cocaine on behavior and extracellular levels of dopamine and 3,4 dihydroxyphenylalanine in the nucleus accumbens of conscious rats. *European Journal of Pharmacology, 549,* 84–90.

Jeng, W., Ramkissoon, A., Parman, T., & Wells, P. G. (2006). Prostaglandin H synthase catalysed bioactivation of amphetamine to free radical intermediates that cause CNS regional DNA oxidation and nerve terminal degeneration. *The FASEB journal : Official publication of the Federation of American Societies for Experimental Biology, 20,* 638–650.

Ji, H., & Shepard, P. (2007). Lateral habenula stimulation inhibits rat midbrain dopamine neurons through a GABA A receptor mediated mechanism. *Journal of Neuroscience, 27,* 6923–6930.

Kalivas, P. W., Volkow, N., & Seamans, J. (2005). Unmanageable motivation in addiction: A pathology in prefrontal–accumbens glutamate transmission. *Neuron, 45,* 647–650.

Kampman, K. M. (2008). The search of medication to treat stimulant dependence. *Addiction Science and Clinical Practice, 4,* 28–35.

Kim, S. J., Lyoo, I. K., Hwang, J., Sung, Y. H., Lee, H. Y., Lee, D. S., et al. (2005). Frontal glucose hypometabolism in abstinent methamphetamine users. *Neuropsychopharmacology, 30*(7), 1383–1391.

Lecourtier, L., & Kelly, P. H. (2007). A conductor hidden in the orchestra? Role of the habenular complex in monoamine transmission and cognition. *Neuroscience and Behavioral Reviews, 31*, 658–672.

Li, X., Wang, H., Qiu, P., & Luo, H. (2008). Proteomic profiling of proteins associated with methamphetamine-induced neurotoxicity in different regions of rat brain. *Neurochemistry International, 52*, 256–264.

Makris, A. P., Rush, C. R., Frederich, R. C., Taylor, A. C., Kelly, T. H. (2007). Behavioral and subjective effects of d-amphetamine and modafinil in healthy adults. *Experimental and Clinical Psychopharmacology, 15*, 123–133.

Matsumoto, M., & Hikosaka, O. (2007). Lateral habenula as a source of negative reward signals in dopamine neurons. *Nature, 447*, 1111–1115.

Melega, W. P., Jorgensen, M. J., Laatan, G., Way, B. M., Pham, J., Morton, G., et al. (2008). Long term methamphetamine administration in the vervet monkey models aspects of a human exposure: Brain neurotoxicity and behavioral profiles. *Neuropsychopharmacology, 33*(6), 1441–1452.

Minzenberg, M. J. & Carter, C. S. (2008). Modafinil: A review of neurochemical actions and effects on cognition. *Neuropsychopharmacology, 33*, 1477–1502.

Morris, E. D., Normandin, M. D., & Schiffer, W. K. (2008). Initial comparison of ntPET with microdialysis measurements of methamphetamine induced dopamine release in rats: Support for estimation of dopamine curves from PET data. *Molecular Imaging and Biology, 10*(2), 67–73.

Murillo-Rodríguez, E., Haro, R., Palomero-Rivero, M., Millán-Aldaco, D., & Drucker-Colín, R. (2007). Modafinil enhances extracellular levels of dopamine in the nucleus accumbens and increases wakefulness in rats. *Behavioural Brain Research, 176*(2), 353–357.

Nakajima, A., Yamada, K., He, J., Zeng, N., Nitta, A., & Nabeshima, T. (2004). Anatomical substrates for the discriminative stimulus effects of methamphetamine in rats. *Journal of Neurochemistry, 91*(2), 308–317.

Oswald, L. M., Wong, D. F., McCaul, M., Zhou, Y., Kuwabara, H., Choi, L., et al. (2005). Relationships among ventral striatal dopamine release, cortisol secretion, and subjective responses to amphetamine. *Neuropsychopharmacology, 30*(4), 821–832.

Pagnoni, G., Zink, C. F., Montague, P. R., & Berns, G. S. (2002). Activity in human ventral striatum locked to errors of reward prediction. *Nature and Neuroscience, 5*, 97–98.

Paulus, M. P., Tapert, S., & Schuckit, M. A. (2005). Neural activation patterns of methamphetamine dependent subjects during decision making predict relapse. *Archives of General Psychiatry, 62*, 761–768.

Ramsay, R. R., & Hunter, D. J. (2003). Interactions of D-amphetamine with the active site of monoamine oxidase-A. *Inflammopharmacology, 11*(2), 127–133.

Rawson, R., Huber, A., Brethen, P., Obert, J., Gulati, V., Shoptaw, S., et al. (2000). Methamphetamine and cocaine users: Differences in characteristics and treatment retention. *Journal of Psychoactive Drugs, 32*(2), 233–238.

Rothman, R. B., Bauman, M. H., Dersch, C. M., Romero, D. V., & Rice, K. C. (2001). Amphetamine type central nervous system stimulants release norepinephrine more potently than they release dopamine and serotonin. *Synapse, 39*, 32–41.

Shoptaw, S., Heinzerling, K. G., Rotheram-Fuller, E., Steward, T., Wang, J., Swanson, A. N., et al. (2008). Randomized, placebo-controlled trial of bupropion for the treatment of methamphetamine dependence. *Drug and Alcohol Dependence, 96*(3), 222–232.

Shoptaw, S., Yang, X., Rotheram-Fuller, E. J., Hsieh, Y. C., Kintaudi, P. C., Charuvastra, V. C., et al. (2003). Randomized placebo controlled trial of baclofen for cocaine dependence: Preliminary effects for individuals with chronic patterns of cocaine use. *Journal of Clinical Psychiatry, 64*, 1440–1448.

Smelson, D. A., Ziedonis, D., Williams, J., Losonczy, M. F., Williams, J., & Steinberg, M. L. (2006). The efficacy of olanzapine for decreasing cue-elicited craving in individuals with schizophrenia and cocaine dependence: A preliminary report. *Journal of Clinical Psychopharmacology, 26*, 9–12.

Thompson, P. M., Hayashi, K. M., Simon, S. L., Geaga, J. A., Hong, M. S., Sui, Y., et al. (2004). Structural abnormalities in the brains of human subjects who use methamphetamine. *Journal of Neuroscience, 24*, 6028–6036.

Tong, J., Ross, B. M., Schmunk, G. A., Peretti, F. J., Kalsinsky, K. S., Yoshiaki, F., et al. (2003). Decreased striatal dopamine D1 receptor stimulated adenylyl cyclase activity in human methamphetamine users. *American Journal of Psychiatry, 160*, 896–903.

Ullsperger, M., & Cramon, Y. V. (2003). Error monitoring using external feedback: Specific roles of the habenular complex, the reward system, and the cingulate motor area revealed by functional magnetic resonance imaging. *Journal of Neuroscience, 23*, 4308–4314.

Urschel, H. C., Hanselka, L., Gromov, I., White, L., & Baron, M. (2007). Open label study of a proprietary treatment program targeting Type A GABA receptor dysregulation in methamphetamine dependence. *Mayo Clinic Proceedings, 82*, 1170–1178.

Vacca, G., Ahn, S., & Phillips, A. G. (2006). Effects of short term abstinence from escalating doses of D-amphetamine in drug and sucrose evoked dopamine efflux in the rat nucleus accumbens. *Neuropsychopharmacology, 32*, 932–939.

Volkow, N. D., Chang, L., Wang, G. J., Fowler, J. S., Ding, Y. S., Sedler, M., et al. (2001). Low level of brain dopamine D2 receptors in methamphetamine abusers: Association with metabolism in the orbitofrontal cortex. *American Journal of Psychiatry, 158*, 2015–2021.

Volkow, N. D., Fowler, J. S., & Wang, G. J. (2004). The addicted human brain viewed in the light of imaging studies: Brain circuits and treatment strategies. *Neuropharmacology, 47*(Suppl. 1), 3–13.

Wang, B., Shaham, Y., Zitzman, D., Azari, S., Wise, R. A., & You, Z. B. (2005). Cocaine experience establishes control of midbrain glutamate and dopamine by cotricotropin releasing factor: A role in stress induced relapse to drug seeking. *Journal of Neuroscience, 25,* 5289–5296.

Wang, G. J., Volkow, N., Chang, L., Miller, E., Sedler, M., Hitzemann, R., et al. (2004). Partial recovery of brain metabolism in methamphetamine abusers after protracted abstinence. *American Journal of Psychiatry, 161,* 242–248.

Ward, N. (2005). Neural plasticity and recovery of function. *Progress in Brain Research, 150,* 527–535.

Wilson, S. J., Sayette, M. A., & Fiez, J. A. (2004). Prefrontal responses to drug cues: A neurocognitive analysis. *Nature Neuroscience, 7,* 211–214.

Xi, Z. X., Newman, A. H., Gilbert, J. G., Pak, A. C., Peng, X. Q., Ashby, C. R. Jr, et al. (2006). The novel dopamine D3 receptor antagonist NGB 2904 inhibits cocaine's rewarding effects and cocaine-induced reinstatement of drug seeking behavior in rats. *Neuropsychopharmacology, 31,* 1393–1405.

Xui, T. H., Koeltzow, T. E., Cooper, T. E., Robertson, G. S., & White, F. J. (2002). Repeated ventral tegmental area amphetamine administration alters dopamine D1 receptor signaling in the nucleus accumbens. *Synapse, 45,* 159–170.

Yong, L., & Kauer, J. (2003). Repeated exposure to amphetamine disrupts dopaminergic modulation of excitatory synaptic plasticity and neurotransmission in nucleus accumbens. *Synapse, 51,* 1–10.

Yudko, E., Hall, H. V., & McPherson, S. B. (2003). *Methamphetamine use: Clinical and forensic aspects*. Boca Raton, FL: CRC Press.

Zaczek, R., Culp, S., & De Souza, E. B. (1991). Interactions of [3H]amphetamine with rat brain synaptosomes. II: Active transport. *Journal of Pharmacologic and Experimental Therapeutics, 257*(2), 830–835.

Zhou, F. C., & Bledsoe, S. (1996). *Methamphetamine causes rapid varicosis, perforation and definitive degeneration of serotonin fibers: An immunocytochemical study of serotonin transporter.* Retrieved November 15, 2004, from http://www.neuroscience.com/chi0796/htm/main.html

Chapter **19**

Neurobiological Mechanisms and Cognitive Components of Addiction

Jorge Juárez, PhD, and Olga Inozemtseva, PhD

Addictive behavior can be seen as either a public health concern or as a problem of inadequate social adaptation. The boundaries between these two concepts become rather unclear once we consider that such disturbances may be determined by an altered genetic condition or by acquisition in any stage of development due to external agents, including the aforementioned social adaptation factor. Several studies support the position that addictive behavior has a genetic substratum, a condition that may lead individuals to seek contact with drugs and to become dependent on them even after only one or a few exposures. Other hypotheses argue that any healthy subject can become addicted if he or she is exposed to a drug with some frequency. A third point of view holds that repeated exposure to a drug by a subject with a certain genetic charge that confers specific biological or personality characteristics may be more prone to acquiring an addiction than another individual with distinct characteristics. The problem is that we have not yet identified with precision just what those characteristics are that may predispose a person to more easily acquire an addiction. What is more, if such traits do exist, they would not necessarily be the same for all potentially addictive substances. Beyond looking at causes, other studies are focusing on analyses of the mechanisms of action of the different substances that can produce addictive behavior in the organism, in an effort to provide information that may lead to possible forms of treatment. The integrated analysis of causes, mechanisms of action, and treatment is an ideal condition for studying addictions, but analyzing each one of these components apart from the others also constitutes an important task for scientists, one

that requires a great deal of research that will contribute data and information to each objective of study on a daily basis. This chapter focuses primarily on describing the mechanisms of action in the brain of several substances of abuse and analyzing some of the cognitive components that have been associated with addictive behavior by cause and effect.

Generally speaking, addictive behavior is associated with the consumption of one or more of a variety of illicit substances, though it can also be related to the compulsive consumption of completely licit substances, such as certain foods, or to practicing a broad range of social and physical activities that can go from obsessive physical exercise to the behavior of the compulsive gambler. It is clear that addictions can be exemplified by a wide variety of behaviors, but for the purposes of this chapter, discussion is limited to those substances better known as drugs of abuse, the consumption of which is widespread in the population, generally in a form called *recreational* use. The chemical characteristics of such drugs vary greatly and are responsible for each one's particular properties of diffusion and distribution in the body, as well as for the specific mechanisms of action that affect different tissues in the organism. Despite the heterogeneity in the characteristics of the different drugs of abuse, it is clear that they may act on the same sites in the brain and that their activity may produce similar neurophysiologic responses.

THE MESOLIMBIC-CORTICAL SYSTEM

This system is also known as the pleasure circuit. It is intimately associated with reinforcing mechanisms and is of particular importance here because several potentially addictive substances act upon it in one way or another. For this reason, it is necessary to describe in broad terms its composition, both anatomical and neurochemical. From the anatomical perspective, the mesolimbic-cortical system is made up of the ventral tegmental area (VTA), the accumbens nucleus (Acc), the prefrontal cortex (pfC), and the amygdala (Am). Most of these structures have reciprocal connections in which diverse neurotransmitters intervene; the most important of which in terms of its reinforcing action linked to addictive conduct is dopamine (DA). The main dopaminergic projections of this circuit are those that project from VTA to Acc, pfC, and Am (Ford, Mark, & Williams, 2006). The pfC, in turn, sends glutamatergic projections to the VTA and to the Acc, while the basolateral Am sends this same type of projections to pfC. The opioid system also plays an important role in this system's functioning, as the beta-endorphins liberated in the hypothalamus are able to facilitate the liberation of dopamine indirectly. Moreover, the activation of kappa receptors has an inhibiting effect on the dopaminergic neu-

rons of the VTA (Margolis, Hjelmstad, Bonci, & Fields, 2005). Other neurotransmitters and modulators of neuronal activity are involved in the function of the mesolimbic-cortical system, but as it is not the purpose of this chapter to present an exhaustive description of them, they will be mentioned only to the degree in which they are pertinent to the description of the mechanisms of action of the drugs examined.

NICOTINE (C10H14N2)

Nicotine is a natural alkaloid, a nitrogenated compound that acts as a base in relation to acids. It was first isolated from tobacco leaves by Posselt and Reiman in 1828, and is found in concentrations as high as 5 percent. The nicotine content of a cigarette is normally from 1 to 2 percent. Nicotine is a tertiary amine that penetrates membranes easily and is absorbed through the mucosa, skin, and lungs. From there, it spreads through the bloodstream in just a few seconds. Like many other molecules, nicotine requires receptors to produce its biological action. Receptors are proteins that can be found in the membrane, the cytosol, or the cell nucleus. The nicotine receptors, called nicotinic, are found in the cell membrane joined to ionic channels and may be activated by acetylcholine or nicotine. Once the ligand, in this case nicotine, binds to the receptor, it generally produces excitability in the cell. At the peripheral level, it stimulates the autonomous ganglions and the suprarenal medulla, thus increasing blood pressure and cardiac frequency. Its action on the neuromuscular union increases muscle tone. In the central nervous system, it acts on several regions where there are nicotinic receptors, while in terms of its effects related to the acquisition of addiction, it acts upon nicotinic receptors in the mesolimbic-cortical system that, as mentioned above, is related to reinforcing mechanisms.

The neurons in the ventral tegmental area synthesize DA, which is released by specific stimuli. It is precisely in those neurons that we find nicotinic receptors that upon being stimulated by nicotine produce the release of DA by these neurons (Keath, Iacoviello, Barrett, Mansvelder, & McGehee, 2007). This action is associated with the euphoric effects, the increased state of alertness and, probably, nicotine's addictive potential. The activation of other neurotransmission systems also seems to play an important role in the effects of nicotine, as this drug has the ability to inhibit monoaminoxidase (MAO), an enzyme that in addition to degrading DA also degrades norepinephrine (NE) and serotonin (5-HT). In this way, the inhibiting of this enzyme by nicotine provides a greater availability of these three neurotransmitters and thus increases their activity in neuronal communication. Nicotine's action has a specific characteristic

in that during chronic exposure it produces a desensitization of the receptors and their inactivation (Corringer et al., 1998), an effect that may be associated with satiating the desire to smoke; however, in the short term, this inactivation produces an increase in the number of receptors as a compensating phenomenon, a mechanism known as receptor up-regulation that, it has been suggested, may be associated with the abstinence syndrome and the compulsive urge to smoke. Hence, this alternation between the deactivation of the receptors and their up-regulation may generate dependence and, therefore, the cyclical desire to smoke.

ALCOHOL (ETHANOL)

Alcohol is another substance the consumption of which is legal, though we know that it is by no means exempt from abuse and can cause a behavioral state of dependence. Ethanol is an alcohol produced through fermentation, whose molecular formula is CH3CH2OH. Its high degree of solubility in water allows it to penetrate practically all of the organism's tissues, including the brain. Additionally, it has a certain degree of liposolubility that allows it to interact with the lipid bilayer of the cell membrane in such a way that it modifies some of its properties. Alcohol is classified as a substance that depresses the central nervous system, a property intimately related to its affinity for the type-A receptors of gamma aminobutyric acid (GABA), which are widely distributed in the central nervous system. These receptors are inserted in the cell membrane and have different subunits that surround an ionic canal that permits the flow of chlorine (Cl^-). The activation of these GABA receptors makes the chlorine—a negatively charged ion—flow toward the interior of the cell, producing an even higher negative potential there, a phenomenon known as *hyperpolarization* that raises the threshold of cell activation that, in turn, produces an inhibiting action upon it. The activity of these GABA receptors is potentialized by alcohol, thus increasing its inhibiting action. The ansiolitic properties of alcohol, amply described in the literature, seem also to be associated with alcohol's action on the GABA receptors.

In addition, alcohol produces an increase in the liberation of beta-endorphin (an endogenous opioid) by the hypothalamus that, as already described, may be associated with the gratifying and analgesic properties of alcohol. Moreover, some authors have postulated that one of the causes of alcoholism may be associated with a compensatory action on the part of the opioid system that acts through the consumption of alcohol; this, because it has been found that alcoholic subjects present a deficiency of beta-endorphins (Genazzani et al., 1982). Hence, the release of beta-endorphins by alcohol would generate a more

pleasant effect in subjects who have this endogenous opioid deficiency than in normal subjects. Though the direct effect of the release of beta-endorphins by alcohol may be reinforcing per se, there is another mechanism that affects the mesolimbic-cortical system and that seems to potentialize or complement the gratifying properties of alcohol. This mechanism operates as follows: the activity of certain GABAergic type neurons in the VTA exercises an inhibiting effect on the dopaminergic neurons of the VTA and their role is to modulate the dopaminergic discharge toward the accumbens nucleus (Olson & Nestler, 2007). These GABA neurons contain opioid receptors whose activity has an inhibiting effect on the secretion of these same neurons; that is, on the secretion of GABA (Steffensen et al., 2006), such that when beta-endorphins are liberated through alcohol consumption, they bind to the opioid receptors at the GABA terminals and inhibit them (Xiao & Ye, 2008). Given that these receptors then exercise an inhibiting action on the dopaminergic cells of the VTA, the final result is an inhibiting of the inhibition that produces the release of dopamine toward the Acc and the pfC. As mentioned above, this produces a gratifying effect. It is well known that alcohol interacts with other neurotransmission systems; however, as far as its addictive properties are concerned, the action on the opioid and dopaminergic system plays a preponderant role. Alcohol consumption also depends on the functional state of the organism, and that is where the endocrine system seems to play an important role, as studies have described that estrogens can affect alcohol consumption in two ways (Juárez, Vázquez-Cortés & Barrios De Tomasi, 2005); that is, there is evidence that estrogen treatment initially decreases alcohol consumption, but if the subjects are exposed to alcohol after several days of estrogen treatment, consumption increases significantly, a result that seems to depend on the action of the estrogens on the greater or lesser availability of opioid receptors. These data are relevant from the clinical perspective, as estrogens play a very important role in the development of both men and women, and are especially significant in the case of the latter due to their dynamic presence during the reproductive life of women and their subsequent declination during the postmenopausal period.

DRUGS THAT ACT ON THE OPIOID SYSTEM

As is well known, the organism produces its own opioids, molecules derived from amino acids called peptides. These substances are derived from different precursor molecules, and in some cases, are joined to common receptors, though this union presents different degrees of affinity. The principal endogenous opioid peptides are beta-endorphins—enkephalins and dinorphins—that are found widely distributed at both the peripheral and central levels.

Their mu (μ), delta (δ), and kappa (κ) receptors are also widely distributed in the organism, and their activation has been related to multiple functions, some of the most important of which are analgesia, the hedonistic aspects of behavior, and the modulation of nervous activity in the brain (Barrios De Tomasi & Juárez, 2007). It may seem paradoxical that the same type of opioid peptide is secreted in conditions characterized by pain and by pleasure, but it is not really so once we consider that reducing or eliminating pain can be just as pleasant as the presence of an agreeable stimulus. The most common drugs of abuse that act on the opioid system are morphine and heroin, the first of which is often used in clinical medicine because of its powerful analgesic properties, while the second is preponderant in recreational use. Both have a significant addictive potential and produce an intense abstinence syndrome that can be described in two phases: the early phase is generally marked by sweating, rhinorrhea, irritability, dysphoria, trembling, agitation, anorexia, tearing, gooseflesh, and drug craving; the later phase may be characterized by an increase in the signs and symptoms of the early phase, plus nausea, vomiting, diarrhea, increased blood pressure and heart rate, depression, generalized muscle spasms, dehydration, and a low threshold for convulsive crises (Carvey, 1998, chap. 4).

One point of discussion has been whether the dependence on these substances results from the individual seeking the drug intensely in order to eliminate the signs and symptoms of the abstinence syndrome, or if the euphoric effects and reinforcement of such drugs suffice to maintain dependence on them. These hypotheses may, in fact, be complementary rather than mutually exclusive because, as mentioned above, eliminating a harmful stimulus can be just as pleasing as positive reinforcement. Either way, the results would sustain the substance-seeking behavior of the addict. We know that blocking the m opioid receptors in the caudal region of the accumbens nucleus decreases the self-administration of heroin in rats (Martin, Kim, Lyupina, & Smith, 2002), which suggests that in this structure opioid activity on its own produces reinforcing effects that lead the addict to continue consuming the drug. However, the opioids have other mechanisms of action, produced indirectly through the activation or inhibition of other neurotransmission systems that may play an even more important part in the acquisition and continuance of addictions to these drugs. One of the most important of these is precisely the effect on the dopaminergic mesolimbic-cortical system. In this action, the agonistic opioid, which could be morphine or heroin, would act in a way similar to that described for the beta-endorphins liberated by alcohol. In this scenario, these drugs would bind preferentially to the m receptors of the GABA neurons in the ventral tegmental area, and thus inhibit them. This inhibition would, in turn, facilitate the liberation of dopamine. The different opioid receptors may

seem to have different functions in the release of dopamine toward the VTA, as it has been reported that the activation of the kappa receptors inhibits the release of dopamine toward the prefrontal cortex, but not toward the accumbens (Margolis et al., 2006). This finding supports the notion of the different functionality of the DA in the Acc and pfC, and suggests that the release of DA from the VTA toward these structures may take place independently.

PSYCHOSTIMULANTS

Drugs that stimulate the central nervous system (CNS) include substances with diverse structural characteristics that share the common effect of raising a person's state of alertness and level of motor activity. They are also known as sympathomimetic substances, as they increase or imitate the activity of the autonomous sympathetic nervous system. The best-known stimulant drugs are amphetamines, methamphetamines, and cocaine in all of its forms.

Amphetamine and Methamphetamine

Amphetamines are aromatic amines (C9H13N) that exercise a powerful stimulant action on the CNS. Their most common effects are raising the state of alertness, improving concentration, increasing motor activity, producing a sensation of well-being or euphoria, inhibiting sleep, anorexia, and stimulating respiration, among others. In clinical medicine, they have been prescribed to treat obesity, narcolepsy, attention-deficit hyperactivity disorder, and depression, as well as for problems of asthma, as they also have a bronchodilator effect. Because of their euphoric effects and the increased state of alertness they produce, plus their induction of an emotional state of well-being, they are used and abused recreationally. Methamphetamines (C10H15N) are the methylated form of amphetamines; that is, they are differentiated from the latter by the presence of a methyl group in the amine group, a small structural change that reduces their peripheral effects and results, it would appear, in lessening the undesirable effects associated with the activation of the sympathetic nervous system. At the central level, however, the effects of methamphetamines are quite similar to those described for amphetamines, though they tend to be preferred for recreational use because of their lesser undesirable effects. Moreover, methamphetamines are pyrolized more easily and commonly inhaled, which favors their distribution through the lungs, quickly generates higher levels in the brain and, therefore, generates a more intense "high."

Chemical-structural variations between amphetamines and methamphetamines may give them a greater or lesser degree of activity on each neurotrans-

mitter involved; however, given that their mechanisms of action at the cerebral level are practically the same, this activity will be described indistinctly for both substances. Consuming amphetamines or methamphetamines generates a greater availability of dopamine and noradrenaline in the synaptic space and, at least in the olfactory bulb, amphetamines are more efficient in liberating DA than NE (Mesfioui et al., 1998). The action on serotonin seems to be less when compared to that on the other two neurotransmitters, though it has been reported that some amphetamine analogues may present a greater effect on serotonin traffic.

There are basically three mechanisms of action at the cellular level that produce a greater availability of the neurotransmitter: (1) these drugs facilitate the non–calcium-dependent release of the neurotransmitter; there is evidence that amphetamines act on the recently synthesized neurotransmitter and on the contents of the vesicles, given that the previous administration of reserpine, a substance that depletes the neurotransmitter from the vesicles that contain it, attenuates the effect of amphetamines on the release of dopamine (Sabol & Seiden, 1998); (2) they act on the mechanisms that recapture the neurotransmitter and compete with it in its transportation toward the interior of the cell, a competition that makes the neurotransmitter remain available to be utilized by the postsynaptic receptors and thus exercise its effects for a longer period of time (Jayanthi & Ramamoorthy, 2005); and (3) they inhibit the degradation of the DA, NE, or 5-HT by acting competitively on the mononaminoxidase enzyme (MAO), which is in charge, precisely, of deactivating these neurotransmitters. As can be seen, the common result of these mechanisms is a greater availability of the neurotransmitter and, therefore, an increase in its activity. The fact that amphetamines compete with the neurotransmitter in its transportation toward the interior of the cell at the same time as it competes with it to be externalized by that same transporter prevents a dopaminergic overstimulation from occurring with increased doses of amphetamines, because of the greater quantity of amphetamines inside the cell, competition for the transporter favors the amphetamine, while the externalization of dopamine is attenuated. This permits a wider safety margin from harm due to overdoses, compared to that of other drugs whose blocking of the transporter is noncompetitive.

Cocaine (C17H21NO4)

This is an alkaloid obtained from the leaf of the coca plant, which contains approximately 0.5 to 0.7 percent of the active substance. Its most common version is cocaine hydrochloride in powder form that is administered intra-

nasally (snorted) or intravenously (Schuckit, 2000, chap. 5). Another often used form is called *crack*, which is obtained by adding a base—usually ammonia or sodium bicarbonate—to cocaine hydrochloride previously dissolved in water and then filtered. This form of cocaine has a lower point of fusion than cocaine hydrochloride, which allows it to be inhaled (smoked by itself or in combination) at the moment it is pyrolized, and thus take effect more quickly than when it is aspirated. The name *crack* is onomatopoeic of the sound "crack" makes when pyrolized (Carvey, 1998, chap. 12; Schuckit, 2000, chap. 5). Many of cocaine's effects on behavior and the individual's state of mind are similar to those obtained through the consumption of amphetamines or methamphetamines, though there are important differences in the intensity and duration of its effects. Cocaine produces an intense state of euphoria that may be related to the size of the dose taken. It also increases the state of alertness and mental sharpness, creates a sensation of energy, self-confidence, and egocentricity, and apparently augments sensorial sensitivity, while decreasing appetite, the need to sleep, and the signs and symptoms of the activation of the sympathetic nervous system (Gold & Jacobs, 2005, chap. 13). Its mechanism of cellular action consists in blocking the recapture proteins in the presynaptic terminal, where dopamine is primarily released, but it also acts in a similar way in the recapture of noradrenaline and serotonin. However, unlike amphetamines, cocaine does not establish a competitive action with the neurotransmitter through its entry into the cell's cytosol, but simply blocks its entry by producing a greater availability of it in the synaptic cleft, which in turn produces an increase in the dopaminergic, noradrenergic, or serotonergic activity, according to the type of neurotransmitter that is compromised.

As can be appreciated, the common denominator in the action of the drugs described above is their effect on dopaminergic transmission in the mesolimbic-cortical system, which has been related, as discussed above, to the motivational and reinforcing mechanisms in different species of mammals, including humans. Although there can be no question, given the evidence described in the literature, that all of these drugs exercise a common action on the brain, we know that they also affect other systems of neurotransmission that, acting in concert, produce the effects characteristic of each different, potentially addictive substance. The effects of these drugs have varying degrees of severity on the functional state of the organism in general and of the brain in particular. During the acute phase of a drug's action, a series of neuroadaptive changes takes place. In some cases, these changes are reversible, such that once the effect of the drug passes, the organism returns to homeostasis. In other cases, however, the brain may suffer irreversible damage due to the acute or chronic consumption of the substance. In some cases, the magnitude of the damage is so great that it can be

perceived both anatomically and behaviorally, while in others, the harm may be functional and thus more difficult to detect. Nonetheless, it may manifest itself and be detected through testing of the individual's cognitive capacities, and this is precisely the objective of the analysis described in the following section of this chapter.

NEUROPSYCHOLOGICAL COMPONENTS IN ADDICTED SUBJECTS

The main objective of the neuropsychological analysis of addictions is to search for the relationship between the use of substances and the neuropsychological impairments that may become manifest at the behavioral, cognitive, emotional, and personality levels. These neuropsychological impairments have been studied primarily in patients with different degrees of dependency or addiction to one particular substance or among polyabusers. Koob et al. (2004) conceptualize the addictive state as a disorder that progresses from impulsivity to compulsivity through a cycle; that is, in order for the patients to reach the addictive state they must follow a path that leads from the occasional, controlled use of a drug to a point at which they lose behavioral control over the search for, and consumption of, that substance; a situation defined as chronic addiction. The *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV–TR*; American Psychiatric Association, 2002) defines substance dependency as "a cluster of cognitive, behavioural, and physiologic symptoms that indicate that the person has impaired control of psychoactive substance use and continues use of the substance despite adverse consequences (p. 218)." Substance abuse and its chronic use may result in the individual presenting the adverse neuropsychological deficits examined below, based on studies carried out with patients addicted to two of the most widely used and easily accessible kinds of psychostimulants: cocaine and methamphetamines.

As mentioned previously, cocaine is a potent psychostimulant that acts upon the monoaminergic neurotransmission systems, especially the dopaminergic system. The most frequently reported cognitive deficits in cocaine-addicted patients in abstinence indicate problems related to memory, attention, abstract reasoning, and the executive functions. Several researchers have reported deterioration of verbal and visual memory, in the phase of immediate and delayed recall, as well as in verbal learning (Beatty, Katzung, Moreland, & Nixon, 1995; Gillen et al., 1998; Horner, 1997; Rosselli & Ardila, 1996; Serper et al., 2000). The deficit in attention and concentration processes has been evidenced in both visuoperceptual and verbal tasks (Rosselli & Ardila, 1996; Strickland et al., 1993; Toomey et al., 2003). Finally, the decrease in the capacity for abstract rea-

soning has been observed in problem-solving tasks (Beatty et al., 1995; Rosselli & Ardila, 1996). In addition to a deficit in specific cognitive abilities (memory, attention, abstract reasoning), studies have reported a more general decrease in intellectual levels (Rosselli & Ardila, 1996), and in global neuropsychological functioning (Robinson, Heaton, & O'Malley, 1999). Given that cocaine acts primarily on the dopaminergic system, several studies have also revealed some deterioration in motor functions (Robinson et al., 1999; Toomey et al., 2003).

Addiction to cocaine is often associated with addictions to other substances, most often alcohol. Findings in the literature on the comorbid effect of chronic cocaine and alcohol abuse are contradictory, as some studies report no effect of the comorbidity of cocaine and alcohol on cognitive functions, while in others the consumption of "pure" cocaine is associated with more marked deficiencies in certain functions. Thus, Beatty et al. (1995) compared a group of "pure" cocaine addicts and a group of "pure" alcohol addicts to a third group made up of healthy participants. They reported that both groups of addicts presented a significantly poorer execution than the control group in most of the measurements of learning, memory, problem solving, executive functions, and perceptual motor-speed. Furthermore, they considered that the general pattern of neuropsychological impairment was similar in the two addict groups. In contrast, Robinson et al. (1999) compared cocaine addicts, cocaine-alcohol addicts, and healthy persons using the Halstead-Reitan neuropsychological test battery. There, the cocaine addicts presented a significantly lower execution than the cocaine-alcohol co-abusers in both complex and simple motor functioning, as well as on a measure of global neuropsychological functioning. However, the results attained by the cocaine-alcohol co-abusers did not differ significantly from those of the control group in most of the applied tasks. Brown, Seraganian, and Tremblay (1994) found no differences in the execution of a variety of neuropsychological tests between a group of cocaine- and alcohol-dependent patients and individuals addicted only to alcohol.

The complex nature of cocaine addiction includes changes at different levels of the functioning of the organism (including modifications at the level of genetic expression) that may be long-lasting or even permanent (Nestler, 2005). In this vein, Juárez (2004) has suggested that the repeated and prolonged exposure to a variety of drugs, such as cocaine, is capable of producing homeorretic modifications in the organism, which are understood as adaptive changes in different systems within the organism that may be permanent or transitory. Therefore, it is logical to ask: Is the neuropsychological deterioration in addicted patients transitory or permanent? The most natural way of responding to this question would be through studies of patients in long-term abstinence, or through longitudinal studies. In this area, Strickland et al. (1993) reported the

presence of attention and concentration deficit, learning difficulties, and a deterioration of visual and verbal memory in patients addicted to crack cocaine after six months of abstinence. These researchers came to the conclusion that prolonged exposure to cocaine produces a persistent neuropsychological deficit. Di Sclafani, Tolou-Shams, Price, and Fein (2002) examined cognitive functions in abstinent crack-dependent and crack- and alcohol-dependent individuals after six weeks and six months of abstinence. Both groups of addicted patients presented deficits in attention, executive functions, spatial processing, memory (immediate and delayed), and in the global clinical impairment store after six weeks of abstinence. The substance-dependent groups were still significantly impaired at six months of abstinence. Bolla, Funderbuk, and Cadet (2000) detected an association between the dose of both cocaine and alcohol and performance on neuropsychological tasks among patients addicted to cocaine with and without alcohol use at 1–3 days of abstinence. The same effect observed in the first evaluation persisted in the second procedure, carried out with the same patients after four weeks of abstinence.

Cocaine addiction produces a deterioration of the CNS and then proceeds to exercise a negative effect on general neuropsychological functioning and specific cognitive functions, which appears to persist even after the person ceases to consume the drug for a prolonged period of time.

Methamphetamines are another, relatively recent, powerful psychostimulant that have become very popular among the addict population in the past couple of decades. Similar to cocaine, methamphetamines or deoxyephedrine, commonly known as *ice* or *crystal*, act primarily on the dopaminergic system, though their mechanism of action is somewhat different from that of cocaine, as described above. The difference in the mechanism of action at the cellular level between these two psychostimulants may be determinant for the profile of the neuropsychological deficits in persons addicted to one or another of these two substances. We have not found any studies in the literature that report a comparative analysis of the cognitive characteristics of addicts to these two drugs, though numerous works have focused on one or the other of the two. The most frequently reported deficits in methamphetamine addicts are related to learning processes and immediate and delayed verbal memory (Kalechstein, Newton, & Green, 2003; Rippeth et al., 2004; Simon et al., 2000), working memory (Rippeth et al., 2004), attention (Kalechstein et al., 2003; Nordahl, Salo, & Leamon, 2003; Rippeth et al., 2004), psycho-motor speed (Kalechstein et al., 2003), executive functions (Kalechstein et al., 2003; Salo et al., 2005; Simon et al., 2000), motor abilities (Rippeth et al., 2004; Volkow et al., 2001), and abstract reasoning (Simon et al., 2000). No reduction in the premorbid intellectual level, as measured by the Shipley-Hartford Vocabulary test (Simon

et al., 2000), has been reported for methamphetamine addicts. The studies reviewed here in regard to this group of patients point to a profile of neuropsychological impairments similar to that of cocaine addicts. However, additional work is necessary to answer the question whether the neurotoxic effects of cocaine and methamphetamine produce conspicuous differences in the neuropsychological profile of these patients.

The neuropsychological impairments in the area of addictions frequently accompany findings on the functioning of the CNS. The most commonly used techniques in this field are positron emission tomography (PET), functional magnetic resonance imaging (fMRI), and single photon emission computed tomography (SPECT), among others, all of which make it possible to detect changes in patterns of brain blood fluid (CBF) and the metabolism of brain glucose. In relation to cocaine addicts, studies with neuroimaging techniques report a reduction in CBF, both globally and regionally, because, as it turns out, cocaine is a powerful vasoconstrictor. Regional hypoperfusion appears to be more prominent in zones rich in dopamine. Many studies conducted with patients addicted to cocaine report a marked reduction in CBF in the regions of the prefrontal cortex, particularly in the anterior cingulate (Bolla et al., 2004; Volkow et al., 1993) and the orbitofrontal cortex (Adinoff et al., 2003; Volkow et al., 2005). In addition, the activity of the SNC in these patients has been negatively correlated with the consumption dose of the drug (Bolla et al., 2004; Johnson et al., 2005). Moreover, studies of these patients have revealed changes in the pattern of the activation of the limbic system, which is traditionally associated with motivation and the compulsive drug intake (Lu et al., 2005). In contrast, Childress et al. (1999) demonstrated an increased blood flow in the amygdala and anterior cingulate in addicts as compared to healthy individuals when all were shown a video on the consumption of cocaine.

In methamphetamine addicts, neuroimaging studies have been widely used to detect the availability of dopamine transporters (DAT) in the neuronal terminals. It has been shown that methamphetamine addicts present DAT loss in the orbitofrontal and dorsolateral regions of the prefrontal cortex, the amygdala, and other subcortical regions (Ernst, Chang, Leonido-Yee, & Speck, 2000; Sekine et al., 2003; Volkow et al., 2001). Furthermore, in several studies, the density of the DAT correlated negatively with the years of use of the substance (Sekine et al., 2003; Volkow et al., 2001). Sekine et al. related the low density of DAT in the orbitofrontal and dorsolateral cortex to the severity of patients' psychiatric symptoms. Volkow et al. observed DAT loss in the striatum (caudate 27.8% and putamen 21.1%) in methamphetamine addicts, compared to control subjects, associated with a persistent motor deficit. Other alterations of the SNC have also been reported for this type of addict. For

example, Ernst et al., using the proton magnetic resonance spectroscopy (MRS) technique, found a significant reduction in the frontal white matter and basal ganglia. Nordahl et al. (2005) reported a reduction in the metabolism of the anterior cingulate and in the insular cortex (London et al., 2005). Furthermore, Thompson et al. (2004) demonstrated a cortical and hyppocampal structural deficit in patients addicted to methamphetamines.

The prefrontal cortex has been associated with cognitive executive functions (Luria, 1982, p. 185), which Lezak (1995, p. 650) referred to as dimensions of human behavior that are related to the way in which behavior is expressed. They are abilities related to forming objectives, planning, carrying out goal-directed behavior, and the effectiveness of production. Lezak added that these abilities are necessary for appropriate, socially responsible, self-sufficient behavior.

In a review study, Verdejo-García, López-Torrecillas, Giménez, & Pérez-García (2004) referred to the executive functions as those that permit anticipation and the establishment of objectives; the designing of plans and programs; self-regulation and task-monitoring; an appropriate selection, organization, and sequencing of behavior in space and time; monitoring of behavior with respect to affective and motivational states; adaptive decision making; efficiency of execution; and feedback.

In those addicted to several types of substances, including psychostimulants, studies have described impairments in the executive functions that find expression in a lack of cognitive flexibility, verbal fluency (Kalechstein et al., 2003; Simon et al., 2000; Verdejo-García & Pérez-García, 2007), inhibition (Ardila, Rosselli, & Strumwasser, 1991; Kalechstein et al., 2003; Nordahl et al., 2003; Rosselli, Ardila, Lubomski, Murray, & King, 2001), abstract reasoning (Ardila et al., 1991; Rosselli et al., 2001; Simon et al., 2000), and planning and organization (Bechara, 2005). Bechara et al. (2001) and Damasio (1994) consider that substance addicts present a deterioration of the executive functions related mainly to motivational aspects and decision making linked to the orbitofrontal cortex.

At the clinical level, detecting the deficit in executive functions and developing possible treatments have important implications. The problems that addicted patients experience in the area of executive functions are often cited to explain their failure to remain in rehabilitation programs, and their frequent relapse and desertion from treatment. On the other hand, various authors mention that deficiencies in the executive functions prior to addiction may create a predisposition to addiction among these patients through such mechanisms as excessive sensibility to reinforcers, or their inability to control their impulsivity (Rogers & Robbins, 2001; Verheul, 2001). However, to date we have no answer to the question: Is the executive deficit in addicted patients premorbid, or is it

the consequence of the use of substances? It is still difficult to find a response to this question given the methodological complications involved, but advances in scientific knowledge in the area of neuroscience will allow us to posit new research strategies designed to search for answers to questions such as these that may have repercussions for the rehabilitation and treatment of addicts.

REFERENCES

Adinoff, B., Devous, M. D., Cooper, D. B., Best, S. E., Chandler, P., Harris, T., et al. (2003). Resting regional cerebral blood flow and gambling task performance in cocaine-dependent subjects and healthy comparison subjects. *American Journal of Psychiatry, 160,* 1892–1894.

American Psychiatric Association. (2002). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Ardila, A., Rosselli, M., & Strumwasser, S. (1991). Neuropsychological deficits in chronic cocaine abusers. *International Journal of Neuroscience, 57,* 73–79.

Barrios De Tomasi, E., & Juárez, J. (2007). Antagonistas opioides y consumo de alcohol. *Revista de Neurología, 45,* 155–162.

Beatty, W. W., Katzung, V. J., Moreland, V. J., & Nixon, S. J. (1995). Neuropsychological performance of recently abstinent alcoholics and cocaine abusers. *Drug and Alcohol Dependence, 37,* 247–253.

Bechara, A. (2005). Decision making, impulse control and loss of willpower to resist drugs: A neurocognitive perspective. *Nature Neuroscience, 8,* 1458–1463.

Bechara, A., Dolan, S., Denburg, N., Hindes, A., Anderson, S. W., & Nathan, P. E. (2001). Decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. *Neuropsychologia, 39,* 376–389.

Bolla, K., Ernst, M., Kiehl, K., Mouratidis, M., Eldreth, D., Contoreggi, C., et al. (2004). Prefrontal cortical dysfunction in abstinent cocaine abusers. *Journal of Neuropsychiatry and Clinical Neurosciences, 16,* 456–459.

Bolla, K. I., Funderbuk, F. R., & Cadet, J. L. (2000). Differential effects of cocaine and cocaine + alcohol on neurocognitive performance. *Neurology, 54,* 2285–2292.

Brown , T. G., Seraganian, P., & Tremblay, J. (1994). Alcoholics also dependent on cocaine in treatment: Do they differ from "pure" alcoholics? *Addictive Behaviors, 19,* 105–112.

Carvey, P. M. (1998). *Drug action in the central nervous system*. New York: Oxford University Press.

Childress, A. R., Mosley, D., McElgin, W., Fitzgerald, J., Reivich, M., & O'Brien, C. P. (1999). Limbic activation during cue-induced cocaine craving. *American Journal of Psychiatry, 156,* 11–15.

Corringer, P. J., Bertrand, S., Bohler, S., Edelstein, S. J., Changeux, J. P., & Bertrand, D. (1998). Critical elements determining diversity in agonist binding and desen-

sitization of neuronal nicotinic acetylcholine receptors. *Journal of Neurosciences, 18,* 648–657.

Damasio, A. R. (1994). *Descartes' error: Emotion, reason and the human brain.* New York: Grosset/Putnam.

Di Sclafani, V., Tolou-Shams, M., Price, L. J., & Fein, G. (2002). Neuropsychological performance of individuals dependent on crack-cocaine, or crack-cocaine and alcohol, at 6 weeks and 6 months of abstinence. *Drug and Alcohol Dependence, 66,* 161–171.

Ernst, T., Chang, L., Leonido-Yee, M., & Speck, O. (2000). Evidence for long-term neurotoxicity associated with methamphetamine abuse: A 1H MRS study. *Neurology, 54,* 1344–1349.

Ford, C. P., Mark, G. P., & Williams, J. T. (2006).Properties and opioid inhibition of mesolimbic dopamine neurons vary according to target location. *Journal of Neurosciences, 26,* 2788–2797.

Genazzani, A. R., Nappi, G., Facchinetti, F., Mazzella, G. L., Parrini, D., Sinforiani, E., et al. (1982). Central deficiency of ∃-endophin in alcohol addicts. *Journal of Clinical Endocrinology and Metabolism, 55,* 583–586.

Gillen, R. W., Kranzler, H. R., Bauer, L. B., Burleson, J. A., Samarel, D., & Morrison, D. J. (1998). Neuropsychological findings in cocaine-dependent outpatients. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 22,* 1061–1076.

Gold, M. S., & Jacobs, W. S. (2005). Cocaine and crack: Clinical aspects. In J. H. Lowinson, P. Ruiz, R. B. Millman, & J. G. Langrod (Eds.), *Substance abuse: A comprehensive textbook* (pp. 218–251). Philadelphia: Lippincott, Williams & Wilkins.

Horner, M. D. (1997). Cognitive functioning in alcoholic patients with and without cocaine dependence. *Archives of Clinical Neuropsychology, 12,* 667–676.

Jayanthi, L. D., & Ramamoorthy, S. (2005). Regulation of monoamine transporters: Influence of psychostimulants and therapeutic antidepressants. *The American Association of Pharmaceutical Scientists Journal, 27,* 728–738.

Johnson, B. A., Dawes, M. A., Roache, J. D., Wells, L. T., Ait-Daoud, N., Mauldin, J. B., et al. (2005). Acute intravenous low- and high-dose cocaine reduce quantitative global and regional cerebral blood flow in recently abstinent subjects with cocaine use disorder. *Journal of Cerebral Blood Flow and Metabolism, 25,* 928–936.

Juárez, J. (2004). Neurobiología de las adicciones. In J. Velásquez-Moctezuma (Ed.), *Temas selectos de neurociencias III* (pp. 225–235). Distrito Federal, Mexico: Universidad Autónoma Metropolitana (UAM).

Juárez, J., Vázquez-Cortés, C., & Barrios De Tomasi, E. (2005). Different stages in the temporal course of estrogen treatment produce opposite effects on voluntary alcohol consumption in male rats. *Alcohol, 36,* 55–61.

Kalechstein, A. D., Newton, T. F., & Green, M. (2003). Methamphetamine dependence is associated with neurocognitive impairment in the initial phases of abstinence. *Journal of Neuropsychiatry and Clinical Neurosciences, 15,* 215–220.

Keath, J. R., Iacoviello, M. P., Barrett, L. E., Mansvelder, H. D., & McGehee, D. S. (2007). Differential modulation by nicotine of substantia nigra versus ventral tegmental area dopamine neurons. *Journal of Neurophysiology, 98,* 3388–3396.

Koob, G. H., Ahmed, S. H., Boutrel, B., Chen, S. A., Kenny, P. J, Markou, A., et al. (2004). Neurobiological mechanisms in the transition from drug use to drug dependence. *Neuroscience and Biobehavioral Reviews, 27,* 739–749.

Lezak, M. D. (1995). *Neuropsychological assessment.* New York: Oxford University Press.

London, E. D., Berman, S. M., Voytek, B., Simon, S. L., Mandelkern, M. A., Monterosso, P., et al. (2005). Cerebral metabolic dysfunction and impaired vigilance in recently abstinent methamphetamine abusers. *Biological Psychiatry, 58,* 770–778.

Lu, L., Hope, B. T., Dempsey, J., Liu, S. Y., Bossert, J. M., & Shaham, Y. (2005). Central amygdala ERK signaling pathway is critical to incubation of cocaine craving. *Nature Neuroscience, 8,* 212–219.

Luria, A. R. (1982). *El cerebro en acción.* Habana, Cuba: Edición Revolucionaria.

Margolis, E. B., Hjelmstad, G. O., Bonci, A., & Fields, H. L. (2005). Both kappa and mu opioid agonists inhibit glutamatergic input to ventral tegmental area neurons. *Journal of Neurophysiology, 93,* 3086–3093.

Margolis, E. B., Lock, H., Chefer, V. I., Shippenberg, T. S., Hjelmstad, G. O., & Fields, H. L. (2006). Kappa opioids selectively control dopaminergic neurons projecting to the prefrontal cortex. *Proceedings of the National Academy of Sciences of the United States of America, 103,* 2938–2942.

Martin, T. J., Kim, S. A., Lyupina, Y., & Smith, J. E. (2002). Differential involvement of mu-opioid receptors in the rostral versus caudal nucleus accumbens in the reinforcing effects of heroin in rats: Evidence from focal injections of beta-funaltrexamine. *Psychopharmacology, 161,* 152–159.

Mesfioui, A., Math, F., Jmari, K., El Hessni, A., Choulli, M. K., & Davrainville, J. L. (1998). Effects of amphetamine and phenylethylamine on catecholamine release in the glomerular layer of the rat olfactory bulb. *Biological Signals and Receptors, 7,* 235–243.

Nestler, E. J. (2005, December). The neurobiology of cocaine addiction. *Science and Practice Perspectives,* 4–11.

Nordahl, T. E., Salo, R., & Leamon, M. (2003). Neuropsychological effects of chronic methamphetamine use on neurotransmitters and cognition: A review. *Journal of Neuropsychiatry and Clinical Neurosciences, 15,* 317–325.

Nordahl, T. E., Salo, R., Natsuaki, Y., Galloway, G. P., Watres, C., & Moore, C. D. (2005). Methamphetamine users in sustained abstinence: A proton magnetic resonance spectroscopy study. *Archives of General Psychiatry, 62,* 444–452.

Olson, V. G., & Nestler, E. J. (2007). Topographical organization of GABAergic neurons within the ventral tegmental area of the rat. *Synapse, 61,* 87–95.

Rippeth, D. J., Heaton, K. R., Carey, L. C., Marcotte, D. T., Moore, J. D., González, R., et al. (2004). Methamphetamine dependence increases risk of neuropsychological impairment in HIV infected persons. *Journal of the International Neuropsychological Society, 10,* 1–14.

Robinson, J. E., Heaton, R. K., & O'Malley, S. S. (1999). Neuropsychological functioning in cocaine abusers with and without alcohol dependence. *Journal of the International Neuropsychological Society, 5,* 10–19.

Rogers, R. D., & Robbins, T. W. (2001). Investigating the neurocognitive deficits associated with chronic drug misuse. *Current Opinion in Neurobiology, 11,* 250–257.

Rosselli, M., Ardila, A., Lubomski, M., Murray, S., & King, K. (2001). Personality profile and neuropsychological test performance in chronic cocaine-abusers. *International Journal of Neuroscience, 110,* 55–72.

Rosselli, M., & Ardila, A. (1996). Cognitive effects of cocaine and polydrug abuse. *Journal of Clinical and Experimental Neuropsychology, 18,* 122–135.

Sabol, K. E., & Seiden, L. S. (1998). Reserpine attenuates D-amphetamine and MDMA-induced transmitter release in vivo: A consideration of dose, core temperature and dopamine synthesis. *Brain Research, 806,* 69–78.

Salo, R., Nordahl, T. E., Moore, C., Waters, C., Natsuaki, Y., Galloway, G. P., et al. (2005). A dissociation in attentional control: Evidence from methamphetamine dependence. *Biological Psychiatry, 57,* 310–313.

Schuckit, M. A. (2000). Drug alcohol abuse: A clinical guide to diagnosis and treatment. New York: Kluwer Academic/Plenum.

Sekine, Y., Minabe, Y., Ouchi, Y., Takei, N., Iyo, M., Nakamura, K., et al. (2003). Association of dopamine transporter loss in the orbitofrontal and dorsolateral prefrontal cortices with methamphetamine-related psychiatric symptoms. *American Journal of Psychiatry, 160,* 1699–1701.

Serper, M. R., Bergman, A., Copersino, M. L., Chou, J.C.Y., Richarme, D., & Cancro, R. (2000). Learning and memory impairment in cocaine-dependent and comorbid schizophrenic patients. *Psychiatric Research, 93,* 21–32.

Simon, S. L., Domier, C., Carnell, J., Brethen, P., Rawson, R., & Ling, W. (2000). Cognitive impairment in individuals currently using methamphetamine. *American Journal on Addictions, 9,* 222–231.

Steffensen, S. C., Stobbs, S. H., Colago, E. E., Lee, R. S., Koob, G. F., Gallegos, R. A., et al. (2006). Contingent and non-contingent effects of heroin on mu-opioid receptor-containing ventral tegmental area GABA neurons. *Experimental Neurology, 202,* 139–151.

Strickland, T. L., Mena, I., Villanueva-Meyer, J., Miller, B. L., Cummings, J., Mehringer, C. M., et al. (1993). Cerebral perfusion and neuropsychological consequences of chronic cocaine use. *Journal of Neuropsychiatry and Clinical Neurosciences, 5,* 419–427.

Thompson, P. M., Hayashi, K. M., Simon, S. L., Geaga, J. A., Hong, M. S., Sui, Y., et al. (2004). Structural abnormalities in the brains of human subjects who use methamphetamine. *Journal of Neurosciences, 24,* 6028–6036.

Toomey, R., Lyons, M. J., Eisen, S. A., Xian, H., Chantarujipakong, S., Seidman, L. J., et al. (2003). A twin study of the neuropsychological consequences of stimulant abuse. *Archives of General Psychiatry, 3,* 303–310.

Verdejo-García, A., López-Torrecillas, F., Giménez, C. O., & Pérez-García, M. (2004). Clinical implications and methodological challenges in the study of the neuropsychological correlates of cannabis, stimulant, and opioid abuse. *Neuropsychology Review, 14,* 1–41.

Verdejo-García, A., & Pérez-García, M. (2007). Profile of executive deficits in cocaine and heroin polysubstance users: Common and differential effects on separate executive components. *Psychopharmacology, 190,* 517–530.

Verheul, R. (2001). Comorbidity of personality disorders in individuals with substance use disorders. *European Psychiatry, 16,* 274–282.

Volkow, N. D., Chang, L., Wang, G., Fowler, J. S., Franceschi, D., Sedler, M. J., et al. (2001). Higher cortical and lower subcortical metabolism in detoxified methamphetamine abusers. *American Journal of Psychiatry, 158,* 383–389.

Volkow, N. D., Fowler, J. S., Wang, G.-J., Hitzemann, R., Logan, J., Shlyer, D. J., et al. (1993). Decreased dopamine D2 receptor availability is associated with reduced frontal metabolism in cocaine abusers. *Synapse, 14,* 169–177.

Volkow, N. D., Wang, G.-J., Ma, Y., Fowler, J. S., Wong, C., Ding, Y.-S., et al. (2005). Activation of orbitofrontal and medial prefrontal cortex by methylphenidate in cocaine-addicted subjects but not in controls: Relevance to addiction. *Journal of Neurosciences, 25,* 3932–3939.

Xiao, C., & Ye, J. H. (2008). Ethanol dually modulates GABAergic synaptic transmission onto dopaminergic neurons in ventral tegmental area: Role of mu-opioid receptors. *Neuroscience, 153,* 240–248.

This page intentionally left blank

Chapter **20**

Fetal, Neonatal, and Early Childhood Effects of Prenatal Methamphetamine Exposure

Mary F. Holley, MD

Drugs of abuse have a long legacy of causing significant impairment in the development of the human brain. Alcohol abuse during pregnancy results in significant changes that include facial dysmorphisms, neurobehavioral problems such as Attention Deficit Hyperactivity Disorder (ADHD), and mental retardation. The full complex of alcohol-related dysfunction is known as fetal alcohol syndrome. Methamphetamine exposure has not been identified with a syndrome or an identifiable pattern of malformation or dysfunction, but clusters of symptoms have been associated with neurologic deficits identified on sophisticated brain scanning of children exposed to methamphetamine (Chang et al., 2004). These findings require our attention and reasonable changes in our patterns of practice in the prenatal clinic and the nursery.

There are a number of developmental risk factors that are common to all drugs of abuse and that may overlap with the effects of lower socioeconomic status. These include genetic influences, nutrition status of the mother, poverty and associated stressors, mental illness either as a predisposing factor to addiction or as a consequence of drug use, infectious diseases, and lack of prenatal care. Each of these factors contributes to the environmental stressors impacting the development of the child born into a family affected by drug abuse. We will also consider the risk factors specific to methamphetamine use, including placental insufficiency, preterm labor, congenital malformations, and the neurotoxic effects of methamphetamine on the developing brain.

Early identification of drug-exposed infants and children is crucial to our efforts to intervene in the lives of these children. Early childhood development

is strongly affected by the drug use of parents, particularly mothers. Proactive intervention, however, demands that we know at the infancy or preschool stage that a child is at risk for drug- or alcohol-related developmental challenges. In order to effectively intervene in these children's lives, we have to know who they are, and be willing to take action to protect them. The consequences of our failure to do so will be made manifest in the juvenile detention centers of the next decade.

INTRODUCTION

In the United States, methamphetamine use among pregnant women doubled over the six-year span from 1998 to 2004 (Cox, Posner, Kourtis, & Jamieson, 2008), as methamphetamine has gradually replaced cocaine as the drug of choice in many areas of the nation among Americans in general and women of childbearing age in particular. Unlike cocaine, methamphetamine is used by women at rates equal to men (Cohen, Greenberg, Uri, Halpin, & Zweben, 2007), primarily for weight loss and increased energy. Methamphetamine is a much more toxic drug than cocaine, causing more rapid and intense addiction (Gonzalez, Castro, Barrington, Walton, & Rawson, 2000), more symptoms of mental illness (McKetin, McLaren, Lubman, & Hides, 2006), and more rapid personal and family disintegration. For all these reasons, methamphetamine has exacted a higher toll on the family, higher even than cocaine, which has already destroyed countless families across this nation.

Both cocaine and methamphetamine produce sexual arousal and promote promiscuous sexual behavior, but methamphetamine produces this effect for far longer than cocaine, and is commonly used by addicts to enhance their sexual experience. As the people using methamphetamine are predominantly of childbearing age, this drug poses greater risks to their unborn children than other comparable drugs of abuse. Increased risk of disease transmission, including sexually transmitted diseases (STD), hepatitis C, and HIV, higher risk of obstetric complications, prematurity and growth restriction, are of particular concern as methamphetamine use increases in the mothers of our children (Smith, LaGasse, Derauf, Grant, & Shah, 2006).

Of major concern are the numerous anecdotal reports of significant neurologic dysfunction in children exposed to methamphetamine in utero. Caretakers of these children have reported cases of severe ADHD, conduct disorders, learning disabilities, and developmental delays in the children of methamphetamine users. These reports have been of great concern to the family members, adoptive and foster parents who are taking responsibility for these children. Scientifically valid information regarding their prognosis and effective interventions is greatly needed.

Education professionals are concerned about the impact of methamphetamine abuse on the special education demands placed on their schools as large numbers of children with learning disabilities and attention deficits are enrolled in the nation's public school system. And since children with learning disabilities and academic failure have higher rates of delinquency (Aseltine, Gore, & Gordon, 2000), methamphetamine-exposed children are more likely to grow up to be wards of the juvenile justice system, further straining our criminal justice system. Early identification of the drug-affected child would permit intervention and training opportunities before entry into the school system, at a time when such interventions are most likely to be effective. But early identification requires drug testing of neonates at or shortly after birth.

True primary prevention would require identification of the drug-using mother in the first trimester of pregnancy, a time when many mothers are amenable to interventions to help their children. Drug testing in the prenatal clinic, however, is generally not done because of fears such testing would deter women from seeking prenatal care. Lacking reliable prenatal information, we are left with secondary prevention, limiting the effects of a disease by controlling its complications—damage control. This we seek to do by neonatal drug testing to detect exposure early and offer remedial assistance to the child and rehabilitation to the mother after prenatal exposure has already occurred.

There is a great deal of controversy over the utility and validity of drug testing of neonates. There are concerns over the accuracy of the tests, false positive results, interactions with prescribed medications, and possible lawsuits by mothers alleging breaches of their privacy rights. There are also concerns over possible legal prosecution against the mothers of these children, actions taken by child protective services workers, including termination of parental rights, custody battles, and the lack of rehabilitation facilities accepting mothers with their young children.

An accurate understanding of and response to the epidemic of drug use in this nation (and around the world) is essential to prevent a public health disaster of epic proportions as today's children become tomorrow's parents. In view of the significant neurotoxicty of methamphetamine, the anecdotal reports of neurologic dysfunction and learning disability in methamphetamine-affected children cannot be dismissed altogether as unreliable. These reports instead must be investigated and fully understood in order to institute preventative measures and offer meaningful intervention to affected children and their families.

NONSPECIFIC RISK FACTORS

Nutrition is a strong variable associated with poor outcomes in drug-using pregnancies (Knight et al., 1994). Some drugs of abuse, such as alcohol, replace

normal food intake and compete with nutrients for entry into the mother. Other drugs are potent anorectics, among them cocaine and methamphetamine. These drugs can cause profound nutritional deficiencies in chronic users, to the point of starvation. Vitamin deficiencies, anemia, and protein calorie malnutrition are significant risks to a pregnancy already complicated by substance abuse.

Poverty is also strongly associated with drug abuse, as addicts are unable to hold down a job or manage their finances appropriately. Some methamphetamine addicts are employed and hold middle-income jobs, but all of their resources go to procure drugs and alcohol, and adequate housing and nutrition are not priorities for these people. Uninsured mothers living in poverty are less likely to attend prenatal clinics and more likely to have untreated infections and poor nutrition regardless of their drug abuse status.

Chronic stress is often a characteristic of the lifestyle of drug-abusing mothers (Derauf et al., 2007). Homelessness is common, and many addicts move from place to place with no permanent address. Addicts sometimes live in cars or abandoned buildings without utilities or a stable food supply, conditions not conducive to a healthy pregnancy. Legal problems are common with multiple arrests, bonds, and fines. Criminal activity and domestic violence are especially common in methamphetamine-affected households. Methamphetamine addicts are irritable and impatient, and with the onset of psychotic symptoms, sometimes become violent. This violence is often directed at family members.

Mental illness is very common in substance abusers, especially methamphetamine abusers. Addicts sometimes have preexisting major illnesses such as bipolar disorder or schizophrenia, but more often, they develop methamphetamine-induced symptoms of mental illness, which may or may not clear after a period of detoxification (Mahoney, Kalechstein, De La Garza, & Newton, 2008). Addicts may become paranoid, suspicious, and hypervigilant. Real and apparent threats are ubiquitous. Addicts feel threatened by other addicts, aggressive dealers, "snitches," and the authorities and often develop delusions surrounding these and other threats. Their mental problems lead directly to severe distress, and indirectly to occupational failure, economic distress, noncompliance with prenatal care, and poor parental adjustment. Long-term methamphetamine use can cause a form of dementia with memory loss and frontal lobe dysfunction, leading to severe impairment in some users (McCann et al., 2007).

Infectious disease is common in drug-abusing populations regardless of the substance used. Sexually transmitted diseases flourish in the climate of promiscuity, the sex-for-dope economy, and reduced precautions characteristic of methamphetamine abuse. Methamphetamine increases sexual desire and drive much more than other drugs of abuse, and so is associated with extremely

high rates of infection with STDs. Methamphetamine users experience drying of mucous membranes due to the vasoconstrictive effects of the drug. Methamphetamine addicts engage in higher rates of unprotected sex and larger numbers of anonymous partners. They are less likely to present for medical care and less compliant with treatment for STDs.

Increased risks of hepatitis B and C and HIV are well known, particularly in IV drug users and sexually promiscuous users. Methamphetamine appears to act as an adjuvant, increasing the risk of infection when exposed to either hepatitis C (Ye et al., 2008) or HIV (Mahajan et al., 2006; Nair et al., 2006; Talloczy et al., 2008). These infections are transmitted vertically to the neonate at birth. While measures can be taken to protect the developing fetus from HIV, there are currently no prophylactic treatments available to prevent hepatitis C from transmitting to the newborn.

Substance abusers are also less likely to present for prenatal care and so are less likely to have been tested and treated for their infectious diseases. Methamphetamine abusers often first present to the hospital in advanced labor having no prior relationship with any health care provider. Fearing involvement with the legal system, they frequently deny illegal drug exposure. Their drug use can only be detected by drug testing in the presence of high-risk behavioral indicators.

METHAMPHETAMINE SPECIFIC RISK FACTORS

Aside from the general risk factors associated with abuse of any drug, there are also a host of risk factors specific to methamphetamine abuse. These include obstetric complications, poor neonatal growth, increased risk of congenital malformations, and neurologic injury to the neonatal brain related to exposure to a potent neurotoxin. The human fetus and placenta lack enzymes that could metabolize methamphetamine as it crosses the placental barrier (Dixon, 1989). Since methamphetamine crosses the placenta very efficiently, blood and tissue levels in the fetus are comparable to maternal blood and tissue levels (Stek et al., 1993; Won, Bubula, & Heller, 2001). Methamphetamine has a much longer half-life than cocaine, particularly in the human fetus. These factors can result in the accumulation of active drug in the fetus, and higher blood levels of the drug have been seen in the developing fetus than seen in the mother under conditions of chronic administration (Stewart & Meeker, 1997).

The immature organ systems of the fetus are therefore exposed to "big people" doses of a potent stimulant drug with all of its vasoconstrictor and cellular toxic properties. The picture is further complicated by the fact that more than 80 percent of methamphetamine-using mothers also use one or more addi-

tional drugs of abuse, most commonly alcohol (Brecht, Greenwell, & Anglin, 2007). The impact on the developing fetus is thus increased exponentially in its clinical presentation.

Obstetric complications are significantly increased in methamphetamine-abusing pregnant mothers. Because of its prolonged half-life in humans—12 to 20 hours—chronic vascular disruption is seen, leading to long-term complications. Placental insufficiency is often seen, leading to intrauterine growth restriction in the fetus (Smith et al., 2006). Some congenital malformations may also be associated with the intense vasoconstriction associated with methamphetamine use (Hoyme et al., 1990). Maternal hypertension associated with methamphetamine is of much longer duration than similar findings in cocaine abusers and can lead to obstetric catastrophe with abruption and fetal distress or fetal demise (Stewart & Meeker, 1997).

Maternal hypertension is often mistaken for preeclampsia or co-occurs with preeclampsia (Elliott & Rees, 1990). Accurate diagnosis is essential since the treatment differs for methamphetamine abusers. Treatment with beta blockers such as Labetolol can have untoward results in methamphetamine abusers as these drugs do not block the alpha adrenergic effect of methamphetamine and can lead to rapidly progressive heart failure in methamphetamine-abusing gravidas (Samuels, Maze, & Albright, 1979 and author's personal experience). Older antihypertensives are preferred, such as appressoline or nipride, which block alpha receptors and control the hypertension seen in methamphetamine abuse. These drugs must be dosed carefully and slowly to prevent overshoot hypotension and resulting fetal distress.

Preterm labor, premature rupture of membranes, and chorioamnionitis are all increased in methamphetamine-affected pregnancies (Eriksson, Larsson, & Zetterström, 1981). The result is often a low birth weight newborn with signs of drug withdrawal, including tremors, lethargy, poor feeding, excess irritability, and other neurobehavioral abnormalities (Smith et al., 2003). Physical signs of withdrawal in the neonate include tachycardia, hypertension, hyper-reflexia, often mixed with symptoms related to the concurrent use of narcotics such as heroin. The Neonatal Abstinence Scoring system is validated for neonates exposed to all drugs of abuse, although the findings can be widely different for infants withdrawing from heroin versus those exposed to stimulants (Oro & Dixon, 1987). Many of these children are exposed to multiple drugs with conflicting effects, leading to confusing findings and missed detection of exposed children.

The incidence of congenital malformations has been studied in chart review studies (Forrester & Merz, 2006, 2007), or retrospective studies of affected children (Torfs, Velie, Oechsli, Bateson, & Curry, 1994), but these studies did not

include a routine neonatal drug-testing protocol and instead relied on patient report and sporadic drug testing in response to risk factors over the course of normal obstetric care. Both methods of detection are fraught with difficulty as only 25 percent of drug-abusing mothers admit to drug use when questioned (Ostrea, Brady, Gause, Raymundo, & Stevens, 1992), and drug testing is often done based on stereotype, missing many drug-using mothers who do not fit the profile of the "drug addict." As a result, we do not know the true incidence of congenital malformations in the infants of methamphetamine-abusing mothers. Anecdotal reports have linked methamphetamine abuse with gastroschisis, eye and ear malformations, cardiac defects, renal anomalies, and limb reduction defects, all of which are exceedingly rare (Bays, 1991).

Effects of methamphetamine exposure on the developing brain have been extensively studied, particularly in light of the overwhelming evidence that methamphetamine is a neurotoxin in the adult brain (for a review, see Yamamoto & Bankson, 2005). Most of these studies have been done on rodent species. Very few human studies have been done to assess human brain development in methamphetamine-exposed children, but those that have been done suggest a significant effect, and a long-lasting effect, with behavioral changes and increased social problems extending into adolescence.

METHAMPHETAMINE'S EFFECT ON BRAIN DEVELOPMENT

Theoretically, methamphetamine should have a significant impact on brain development. It is a potent vasoconstrictor, reducing blood supply to the placenta and thus delivery of oxygen and nutrients to the developing fetus. It is also a potent neurotoxin, releasing free radical compounds that cause neural cell death (Yamamoto & Bankson, 2005). And indeed stimulants do impact embryogenesis of the developing central nervous system. Cell proliferation and migration are impacted and synaptogenesis is impaired (Weissman & Caldecott-Hazard, 1995). There is reduction in neural growth factor and a marked increase in reactive oxygen species and free radicals (Jeng, Wong, Ting-A-Kee, & Wells, 2005; Wells et al., 2005). These toxic chemicals are known neurotoxins and have been implicated in the progression of cellular damage to the brain in adult methamphetamine users (Acikgoz et al., 2000).

Animal models, rats, mice, and gerbils, have been used to study the fetal effects of many drugs, including methamphetamine. Rodents metabolize methamphetamine differently from humans; the half-life of methamphetamine in rodents is only around 1 hour, compared with 12 hours in humans (Cho, Melega, Kuczenski, & Segal, 2001). Rodents also mature their central nervous

system in the first month postnatally while humans do so in the third trimester of pregnancy (Gomez-DeSilva, Silva, & Tavares, 1998). Rodents are born at an earlier gestational maturity than humans, and so are usually dosed 10 to 20 days after birth to simulate third-trimester human exposure. Neonatal rodents have been found to be relatively resistant to the effects of methamphetamine compared to adults. These factors explain the significant differences in dosing of animals compared to typical human use patterns. These differences often cloud interpretation of animal data by clinicians who accuse animal researchers of vastly overdosing the animals. Because of the markedly reduced half-life of methamphetamine in rodents, these studies more likely underestimate the impact of fetal methamphetamine exposure, rather than overestimating it. Overall, methamphetamine appears to be a much more neurotoxic drug than cocaine, as might be expected based on its metabolic profile.

A summary of the most recent research in the field of methamphetamine effect on brain development reveals some disturbing trends. Acevedo, de Esch, and Raber (2006) studying mice found significant disruption of hippocampus dependent cognitive function in adult animals exposed to methamphetamine in the neonatal stage. Cognitive functions affected included spatial learning, memory, and object recognition. Vorhees, Skelton, and Williams (2007) looked at rats and also found spatial learning and memory deficits that persisted into adulthood, suggesting a permanent structural change in brain development. In an older but well-done study, Hildebrandt, Teuchert-Noodt, and Dawirs (1999) localized the site of methamphetamine-induced injury to the dentate gyrus of the hippocampus, specifically to impaired granule cell proliferation (a 34 percent deficit). Even low-dose methamphetamine exposure caused significant spatial learning deficits in rats, suggesting that there is no safe level of exposure during pregnancy (Williams, Moran, & Vorhees, 2004).

Gomez-DeSilva and colleagues (2004) demonstrated increased catecholamines in neonatal rat brains exposed to methamphetamine, particularly in the substantia nigra, caudate putamen, and nucleus accumbens. Williams, Brown, and Vorhees (2004) demonstrated injury to the hippocampus, nucleus accumbens, and parietal lobes in rats exposed to methamphetamine. These findings have been replicated repeatedly by numerous researchers, demonstrating a significant neurotoxic effect of methamphetamine in brain development with functional loss of memory and coordination.

The mechanism of this damage was elucidated by Jeng et al. (2005), who investigated the metabolic pathways by which methamphetamine damages developing neural tissue. They found that methamphetamine causes oxidative cell injury in the brains of mice exposed to neonatal methamphetamine. Oxygen free radicals are produced by the metabolism of both methamphetamine and

the monoamine neurotransmitters released in response to methamphetamine. These free radicals are potent neurotoxins. The fetal brain lacks key alternate enzymes needed to metabolize monoamines and methamphetamine, leading to the build-up of high levels of oxygen free radicals and consequent long-term impairment of brain development (Wells et al., 2005).

These neurotoxic changes persist into adulthood and sensitize the adult brain to the effects of adult exposure to methamphetamine, particularly in males. Neurotoxicity to the dopaminergic projections in response to methamphetamine challenge in adulthood was significantly increased in animals that had been exposed to methamphetamine prenatally, suggesting a persistent sensitivity (Heller, Bubula, Lew, Heller, & Won, 2001). This finding has significant implications for meth-exposed children growing up in meth-abusing homes who learn early in life how to solve their problems with a pipe.

HUMAN STUDIES OF METHAMPHETAMINE IMPACT

Clearly, methamphetamine should not be given to neonatal rats, mice, or gerbils. It damages their little brains. But what about humans? Do the animal studies apply to the human infant? Most human methamphetamine users are polydrug users and have multiple other risk factors in addition to their drug use, including poverty, stress, nutritional deficiencies, and infections, to name a few. For these reasons, the rodent studies more likely *underestimate* the impact of the total prenatal environment on the human fetus and its development.

The earliest studies on human response to fetal methamphetamine exposure were grim indeed. Tests of visual recognition in human infants exposed to methamphetamine showed significant decreases (Hansen, Struthers, & Gospe, 1993) and corresponding differences in attention, distractibility, and activity level (Struthers & Hansen, 1992). Changes in visual recognition in the infant are strongly associated with cognitive function later in life, and lack of eye contact can also impair maternal–infant bonding in the first days of life. Dixon and Bejar at UCSD (1989) found that 35 percent of neonates exposed to cocaine and/or methamphetamine in utero had abnormalities in brain structure at birth, including intraventricular hemorrhage, necrotic echodensities, and cavitary lesions. These lesions were thought to be due to the severe vasoconstrictive effects of stimulant drugs of abuse. A significant number of stimulant-exposed infants (10%) had ventricular dilation, reflecting diffuse atrophy of cortical tissue.

But the human brain is not the same as a rodent brain. Cortical areas are much more richly developed in humans, and the human cortex continues postnatal development over the first 21 years of life, especially in the first 3 years of life. The developmental window that extends from birth to age three is a time of

explosive growth in cognitive, emotional, verbal, and social development. Given adequate interaction, stimulation, and nutrition, the human cortex increases in size significantly. If the meth-exposed child is sent home with responsible parents, much (though perhaps not all) of the brain injury sustained before birth could be compensated for and its impact diminished. Early intervention in methamphetamine-exposed children could be extremely important to the long-term prognosis for human cognitive and social development.

And indeed that is what the studies demonstrate. When methamphetamine-exposed children are studied at age 8 to 10 years, the cavitary lesions and ventricular dilation are no longer seen (Smith et al., 2001). All of the children in this study had been exposed to methamphetamine with very low levels of exposure to other drugs of abuse, including alcohol. Children with diagnosed developmental delay, impaired growth, seizure disorders, or ADHD were excluded. Only those children who looked perfectly normal were studied. Although these children did have signs of metabolic changes in the basal ganglia, no cavitary lesions were found.

In a follow-up study by the same group, Chang et al. in 2004 investigated further the changes seen in the basal ganglia of methamphetamine-exposed children. Using some of the same subjects, again excluding children with obvious developmental delays or ADHD, significant subcortical changes were seen. Methamphetamine-exposed children had significantly smaller hippocampus, globus palidus, putamen, and caudate (17%–26% smaller), the same areas that are impaired in the animal studies.

A corresponding decrement in neuropsychological testing was also observed in these methamphetamine-exposed children. They scored lower on measures of visual-motor integration, attention, verbal memory, and long-term spatial memory, all essential functions in efficient learning. Again, these were children who had not been identified as having a developmental delay or ADHD by teachers or caregivers. These children looked perfectly normal and were performing normally in school, but they were challenged by the effects of their prenatal methamphetamine exposure.

Long-term follow-up of methamphetamine-exposed children over a period of up to 15 years shows the impact of these challenges. Billings, Eriksson, Jonsson, Steneroth, and Zetterström (1994) demonstrated increased aggressive behavior and poor adjustment in children 8 to 10 years old exposed to methamphetamine prenatally. The same children followed up to 15 years of age (Cernarud, Eriksson, Jonsson, Steneroth, & Zetterström, 1996; Eriksson, Jonsson, Steneroth, & Zetterström, 2000) showed higher rates of academic failure and poor social adjustment. These methamphetamine-exposed children were three times more likely to be behind in school (15% were one year or more behind) and had lower grades than nonexposed peers.

These long-term studies did not attempt to control for alcohol exposure. Indeed, 81 percent of these children were also exposed to alcohol prenatally. Fetal alcohol syndrome has clearly been linked to significant neurobehavioral abnormalities, including ADHD, learning disability, verbal memory deficits, and mental retardation (Pie, Rinaldi, Rasmussen, Massey, & Massey, 2008). Most of these children (80%) were also exposed to nicotine, which has been linked to abnormalities of the auditory association area (Dwyer, Broide, & Leslie, 2008) and dysregulation of emotion and attention (Shea & Steiner, 2008). The majority (78%) of these children did not live with their birth mothers throughout childhood. Many were wards of the state and had moved from foster home to foster home, a condition that is strongly linked to academic failure and delinquency (National Center on Addiction and Substance Abuse at Columbia University, 2004).

We do not know the true incidence or severity of psychomotor or neurocognitive disability in children exposed to methamphetamine because routine drug testing is not being done, and so the majority of affected children are not identified at birth or assessed as they mature. Human studies will always be complicated by concurrent alcohol and other drug abuse, since most meth addicts use other drugs as well. We do not yet have the necessary data to assess the true incidence and risk of methamphetamine exposure to the development of the human brain.

EARLY DETECTION AND INTERVENTION

Primary prevention of drug-related disability in the infant requires that the drug-abusing mother be identified and counseled early in the prenatal course so that disease and impairment is prevented. Screening of obstetric patients for drug and alcohol abuse is required by the laws of most states, yet is inconsistently done by most providers and even more seldom reported or acted upon during the pregnancy. This is in spite of the fact that a poor obstetric outcome is likely in the event of drug abuse during pregnancy, and that the poor outcome is likely to be attributed to substandard obstetric care if the existence of drug abuse has not been documented.

Prenatal screening is fraught with difficulty because of the fear that the drug-abusing gravida will simply avoid prenatal care if serious attention is directed to her drug or alcohol abuse, especially in the form of drug testing. Screening is thus limited to verbal questioning with its attendant underestimation of the extent of the problem at hand.

For verbal screening to be effective, questioning should be incorporated into the routine intake interview so that a rapport is established with the health care provider and suspicion as to the intentions of the provider is minimized.

Questioning should begin with an inquiry into family history of substance abuse, ideally in the context of a general family history review. This line of questioning is less threatening to the patient and less likely to raise resistance than a direct assault on her fitness to be a mother. If the family history of addiction is then recorded as an illness, not a moral failure, the patient may be more likely to admit to her own "addictive illness" than she would be to admit to her "illegal drug use" with its criminal intonations.

Early prenatal identification of a drug or alcohol problem permits primary prevention of the neonatal complications—the ideal situation—rather than just the damage containment obtainable with neonatal drug testing. Prenatal identification requires an informed, concerned, and honest patient, who has been apprised of the risks drug abuse poses to her and her baby's health, a nonjudgmental attitude on the part of the medical provider, and the availability of appropriate inpatient or outpatient treatment facilities.

Since primary prevention is often not accomplished, we are left with damage containment measures that focus on identification of the affected newborns and rehabilitation of their mothers after delivery. But even postdelivery "damage control" interventions are often not carried out because of poor detection of drug and alcohol abuse in the neonatal stage. The period of hospitalization presents our best opportunity to detect and intervene in the addiction process because of the prolonged period of intense observation by skilled professionals in the labor and delivery suite. Professionals trained in recognition of the signs of addiction can identify mothers and infants with risk factors for addiction, and in most states, can order a neonatal drug test at the slightest hint of a problem. The most seriously affected children are likely to be identified in this way; however, not all drug addicts look, act, and smell like drug addicts. Not all labor and delivery professionals are alert to the signs and symptoms of addiction, and not all doctors and hospitals permit drug testing on their favored (insured) patients. Some doctors and hospitals, wishing to avoid all the hassle associated with drug testing, do testing on only the most egregious cases, missing many affected children. The extremely short hospitalizations for delivery that are now common also limit the ability of trained professionals to observe mothers over an extended time and recognize abnormal patterns of behavior and failures in bonding.

Most children exposed to drugs of abuse are not identified at birth, and most of them go home with their drug-abusing parents without any intervention. These children, who have already been exposed to a potent neurotoxin, are then exposed to poor nutrition, domestic violence and child abuse, infectious diseases, and a host of environmental problems associated with poverty and addiction. They are at significant risk for academic and social failure if their

problems are not addressed in a proactive manner. Proactive intervention, however, demands that we know at the infancy or preschool stage that a child is at risk for drug- or alcohol-related developmental challenges. Early identification offers us the opportunity to intervene in the development of a child and reduce the likelihood of a poor outcome.

Despite a long history of neonatal testing for medical problems and inborn errors of metabolism in our newborns (e.g., PKU [phenylketonuria] and hypothyroid), the United States has no systematic program for testing all neonates for exposure to neurotoxic drugs of abuse. This is in spite of the large numbers of children thought to suffer from these exposures and the ease of testing by meconium or hair analysis (Garcia-Bournissen, Rokach, Karaskov, & Koren, 2007). Testing is instead based on stereotypes and behavioral indications that may unfairly stigmatize some segments of the population, and at the same time, fail to detect the majority of cases. Basic public health science tells us that this is a totally inadequate system of detection of a major public health risk.

In a large study of meconium drug testing, 44 percent of all neonates in an inner-city hospital tested positive for illicit drug exposure. The majority of children with prenatal exposure to illegal drugs appeared normal at birth, and so would not have been tested under most protocols. Only one in four drug-abusing mothers admitted to her drug use on questioning in the perinatal setting (Ostrea et al., 1992). Of those who admitted to drug use at any time during pregnancy, around 90 percent of their infants had a positive meconium drug screen at birth.

Current neonatal indications for drug testing include obvious withdrawal symptoms, low birth weight, neurobehavioral abnormalities, seizures, stroke, cardiac problems, and necrotizing enterocolitis in a term baby. Only severely affected infants will display symptoms this severe—around 4 percent of methamphetamine-positive neonates (Smith et al., 2003). Most methamphetamine-affected children (> 50%) do not have any significant withdrawal symptoms, yet are still at risk for developmental delay or learning disability. Identification of these children is essential for effective intervention to prevent social and academic failure.

Current maternal indications for neonatal drug testing include a known history of drug abuse, lack of prenatal care, home delivery or precipitous delivery, preterm labor, poor weight gain in pregnancy, hypertension without proteinuria, abruption, fetal distress, sexually transmitted disease, and obvious intoxication. Poor, single, or homeless mothers are much more likely to be tested for drug abuse than mothers who are employed and have stable family relationships.

The currently practiced methods for drug testing of neonates rely heavily on stereotypes of how drug-abusing mothers typically behave. This approach

is dangerous and misleading for two reasons. First, women from disadvantaged backgrounds may fit the "profile" of the drug-abusing mother and may be selected for testing at a higher rate. This risk factor–based testing leads to a stigmatized feeling of reproach, being held under suspicion merely for being poor or unmarried or homeless.

Second, stereotype-based drug testing misses many infants whose mothers do not fit the profile but who are abusing illegal drugs, especially methamphetamine. Methamphetamine differs from other major drugs of abuse in that many low-dose users do not consider themselves "drug users." Methamphetamine abusers are often well educated, highly functioning individuals, working two jobs, highly driven and successful. They use methamphetamine to work harder and longer, get more focus and enhance attention, or to lose weight and/or prevent weight gain. They consider methamphetamine a "medication," not a "drug," and so will deny "drug use." Methamphetamine users frequently do not fit the stereotype of the "addict." Drug testing based on stereotypes misses this sizable population of mothers, and so their methamphetamine-exposed newborns are not detected.

Universal drug testing offers a major improvement to our current protocols based on perceived risk factors. The currently high cost of meconium drug testing would be greatly reduced if all newborns were tested with resulting economies of scale, just as the cost of the PKU test was reduced when it became part of standard care. State laws to provide for testing of all newborns for exposure to drugs of abuse, just as we currently test all neonates for PKU and thyroid dysfunction, would permit detection of the vast majority of drug-affected newborns. The basis for testing for PKU and thyroid dysfunction is that early intervention is life saving and prevents severe disability in affected children. This rationale also applies to the drug-exposed newborn. Identified mothers could be offered treatment and counseling, and a major cause of child abuse would be prevented. Children could be screened for learning disabilities and receive the special attention they need to develop normally.

We currently screen children for learning disabilities in early elementary school, long after the best window of opportunity for intervention—birth to age three—has passed. Universal neonatal testing for drug exposure would permit earlier intervention in the child's cognitive and social development, and would also permit earlier intervention for the mothers of drug-exposed children, allowing more of them to obtain the treatment they need to preserve the family. None of these interventions can happen if we do not know a newborn has been exposed to substance abuse.

If all newborns were tested, no mother would feel singled out or stigmatized, and no drug-affected children would be missed. Counseling and follow-up

could be offered to mothers and their children, and early detection of learning difficulties could be facilitated. Early detection could lead to better outcomes for children and their families as addictions are identified in earlier stages when treatment outcomes are more favorable. Earlier detection of the subtle learning disabilities associated with methamphetamine exposure permits educational intervention to mitigate the long-term impact of these challenges.

The major limitation of universal neonatal meconium drug testing is that it would only detect late second- and third-trimester use, and would miss those children whose mothers didn't like the hyperemesis treatment offered by their obstetrician, and elected to use their college friend's hangover treatment instead. These women are extremely unlikely to admit to drug use, and if their children exhibit learning difficulties or neurologic injury, they will find someone else to blame.

Another limitation is the incidence of false positive results even with the use of gas chromatography or mass spectrometry confirmation testing. Meconium screens are not reliable for PCP (Phencyclidine) exposure—the use of dextromethorphine (Robitussin) cough medicine is enough to make the PCP screen positive. The meconium screen would also be positive for amphetamines in the case of prescription use of stimulants such as Adderall and Desoxyn. These drugs are not recommended for use in pregnancy (Category C) as alternative treatments are readily available for their indications.

But the most common reason for failure to obtain a drug test is denial, usually based on stereotyping of the mother and fear of offending the favored (insured) patient. Patients are in denial, their families are in denial, and often health care workers join them in the assurance that everything must be just fine. A good girl like her couldn't possibly be using illegal drugs. Denial is generally discouraged in the medical field, since lives are at risk if a serious diagnosis is missed.

DENIAL DOESN'T WORK

Denial on a societal scale is just as dangerous as denial on an individual level. Our reluctance to "label" drug-affected children results in a complete inability to assist them since we do not know who they are. Universal drug testing of neonates would break down barriers to treatment and intervention for children and their families. Family drug courts could assure compliance with treatment on the part of parents and assemble the wide range of services and programs that could prevent the disintegration of these fragile families.

When affected children are missed, opportunities for intervention are missed.

We lose opportunities to impact the child and family with drug-treatment options, prevent child abuse and neglect, improve cognitive performance, and prevent academic failure. We miss the opportunity to prevent second-generation drug abuse in these children as they grow up with poor self-esteem and fall into delinquency. Universal drug testing offers us an inroad to identify and assist families and children at risk. We would be remiss if we fail to take action.

REFERENCES

Acikgoz, O., Gonenc, S., Kayatekin, B. M., Pekcetin, C., Uysal, N., Daye, A., et al. (2000). The effects of a single dose of methamphetamine on lipid peroxidation levels in the rat striatum and prefrontal cortex. *European Journal of Neuropsychopharmacology. 10*(5), 415–418.

Acevedo, S. F., de Esch, I. J., & Raber, J. (2007). Sex and histamine dependent long term cognitive effects of methamphetamine exposure. *Neuropsychopharmacology, 32*(3), 665–672.

Aseltine, R. H., Gore, S., & Gordon, J. (2000). Life stress, anger, and anxiety, and delinquency: An empirical test of general strain theory. *Journal of Health and Social Behavior, 41*, 256–275.

Bays, J. (1991). Fetal vascular disruption with prenatal exposure to cocaine or methamphetamine. [Letter to the Editor]. *Pediatrics, 87*, 416–417.

Billings, L., Eriksson, M., Jonsson, B., Steneroth, G., & Zetterström, R. (1994). The influence of environmental factors on behavioral problems in eight year old children exposed to amphetamine during fetal life. *Child Abuse and Neglect, 15*, 3–9.

Brecht, M. L., Greenwell, L., & Anglin, M. D. (2007). Substance use pathways to methamphetamine use among treated users. *Addictive Behaviors, 32*(1), 24–38.

Cernarud, L., Eriksson, M., Jonsson, B., Steneroth, G., & Zetterström, R. (1996). Amphetamine addiction during pregnancy: 14 year follow-up of growth and school performance. *Acta Pediatrics, 85*, 204–208.

Chang, L., Smith, L. M., LoPresti, C., Yonekura, L. M., Kuo, J., Walot, I., et al. (2004). Smaller subcortical volumes and cognitive deficits in children with prenatal methamphetamine exposure. *Psychiatry Research and Neuroimaging, 132*, 95–106.

Cho, A. K., Melega, W. P., Kuczenski, R., & Segal, D. S. (2001). Relevance of pharmacokinetic parameters in animal models of methamphetamine abuse. *Synapse, 39*, 161–166.

Cohen, J. B., Greenberg, R., Uri, J., Halpin, M., & Zweben, J. E. (2007). Women with methamphetamine dependence: Research on etiology and treatment. *Psychoactive Drugs, 4*, 347–351.

Cox, S., Posner, S. F., Kourtis, A. P., & Jamieson, D. J. (2008). Hospitalizations with amphetamine abuse among pregnant women. *Obstetrics and Gynecology, 111*, 341–347.

Derauf, C., LaGasse, L. L., Smith, L. M., Grant, P., Shah R., Arria, A., et al. (2007). Demographic and psychosocial characteristics of mothers using methamphet-

amine during pregnancy: Preliminary results of the infant development, environment, and lifestyle study (IDEAL). *American Journal of Drug and Alcohol Abuse, 33*, 281–289.

Dixon, S. D. (1989). Effects of transplacental exposure to cocaine and methamphetamine on the neonate. *Western Journal of Medicine, 150*(4), 436–442.

Dixon, S. D., & Bejar, R. (1989). Echoencephalographic findings in neonates associated with maternal cocaine and methamphetamine use: Incidence and clinical correlates. *Journal of Pediatrics, 115*, 770–778.

Dwyer, J. B., Broide, R. S., & Leslie, F. M. (2008). Nicotine and brain development. *Birth Defects Research Part C: Embryo Today, 84*(1), 30–44.

Elliott, R. H., & Rees, G. B. (1990). Amphetamine ingestion presenting as eclampsia. *Canadian Journal of Anaesthesiology, 37*, 130–133.

Eriksson, M., Jonsson, B., Steneroth, G., & Zetterström, R. (2000). Amphetamine abuse during pregnancy: Environmental factors and outcome after 14–15 years. *Scandinavian Journal of Public Health, 28*(2), 154–157.

Eriksson, M., Larsson, G., & Zetterström, R. (1981). Amphetamine addiction and pregnancy. II: Pregnancy, delivery and the neonatal period: Socio-medical aspects. *Acta Obstetrics and Gynecology Scandinavia, 60*(3), 253–259.

Forrester, M. B., & Merz, R. D. (2006). Comparison of trends in gastroschisis and prenatal illicit drug use rates. *Journal of Toxicology and Environmental Health,* A *69*, 1253–1259.

Forrester, M. B., & Merz, R. D. (2007). Risk of selected birth defects with prenatal drug use, Hawaii 1986–2002. *Journal of Toxicology and Environmental Health,* A *70*, 7–18.

Garcia-Bournissen, F., Rokach, B., Karaskov, T., & Koren, G. (2007). Methamphetamine detection in maternal and neonatal hair: Implications for fetal safety. *Archives of Disease in Childhood: Fetal Neonatal Ed., 92*, F332–F333.

Gomez-DeSilva, J., de Miguel, R., Fernandez-Ruiz, J., Summavielle, T., & Tavares, M. A. (2004). Effects of neonatal exposure to methamphetamine: Catecholamine levels in brain areas of the developing rat. *Annals of New York Academy of Science, 1025*, 602–611.

Gomez-DeSilva, J., Silva, M. C., & Tavares, T. (1998). Developmental exposure to methamphetamine: A neonatal model in the rat. *Annals of New York Academy of Science, 844*, 310–313.

Gonzalez Castro, F., Barrington, E. H., Walton, M. A., & Rawson, R. A. (2000). Cocaine and methamphetamine: Differential addiction rates. *Psychology of Addictive Behaviors, 14*, 390–396.

Hansen, R. L., Struthers, J. M., & Gospe, S. M., Jr. (1993). Visual evoked potentials and visual processing in stimulant drug exposed infants. *Developmental and Medical Child Neurology, 35*, 798–805.

Heller, A., Bubula, N., Lew, R., Heller, B., & Won, L. (2001). Gender dependent enhanced adult neurotoxic response to methamphetamine following fetal exposure to the drug. *The Journal of Pharmacology and Experimental Therapeutics, 298*, 769–779.

Hildebrandt, K., Teuchert-Noodt, G., & Dawirs, R. R. (1999). A single neonatal dose of methamphetamine suppresses dentate granule cell proliferation in adult gerbils which is restored to control values by acute doses of haloperidol. *Journal of Neural Transmission, 106,* 549–558.

Hoyme, H. E., Jones, K. L., Dixon, S. D., Jewett, T., Hanson, J. W., Robinson, L. K., et al. (1990). Prenatal cocaine exposure and fetal vascular disruption. *Pediatrics, 85*(5), 743–747.

Jeng, W., Wong, A. W., Ting-A-Kee, R., & Wells, P. G. (2005). Methamphetamine enhanced embryonic oxidative DNA damage and neurodevelopmental deficits. *Free Radical Biology and Medicine, 39,* 317–326.

Knight, E. M., James, H., Edwards, C. H., Spurlock, B. G., Oyemade, U. J., Johnson, A. A., et al. (1994). Relationships of serum illicit drug concentrations during pregnancy to maternal nutritional status. *Journal of Nutrition, 124,* 973S–980S.

Mahajan, S. D., Hu, Z., Reynolds, J. L., Aalinkeel, R., Schwartz, S. A., & Nair, M. P. (2006). Methamphetamine modulates gene expression patterns in monocyte derived mature dendritic cells: Implications for HIV-1 pathogenesis. *Molecular Diagnostics and Therapeutics, 10,* 257–269.

Mahoney, J. J., III, Kalechstein, A. D., De La Garza, R., II, & Newton, T. F. (2008). Presence and persistence of psychotic symptoms in cocaine- versus methamphetamine- dependent participants. *American Journal of Addiction, 17*(2), 83–98.

McCann, U. D., Kuwabara, H., Kumar, A., Palermo, M., Abbey, R. Brasic, J., et al. (2007). Persistent cognitive and dopamine transporter deficits in abstinent methamphetamine users. *Synapse, 62*(2), 91–100.

McKetin, R., McLaren, J., Lubman, D. I., & Hides, L. (2006). The prevalence of psychotic symptoms among methamphetamine users. *Addiction, 101*(10), 1473–1478.

Nair, M. P., Mahajan, S., Sykes, D., Bapardekar, M. V., & Reynolds, J., (2006). Methamphetamine modulates DC SIGN expression by mature dendritic cells. *Journal of Neuroimmune Pharmacology,* (3), 296–304.

National Center on Addiction and Substance Abuse at Columbia University. (2004). *Criminal neglect: Substance abuse, juvenile justice, and the children left behind.* New York: Author.

Oro, A. S., & Dixon, S. D. (1987). Perinatal cocaine and methamphetamine exposure: Maternal and neonatal correlates. *Pediatrics, 111*(4), 571–578.

Ostrea, E. M., Brady, M., Gause, S., Raymundo, A. L., & Stevens, M. (1992). Drug screening of newborns by meconium analysis: A large-scale, prospective, epidemiologic study. *Pediatrics, 89,* 107–113.

Pei, J. R., Rinaldi, C. M., Rasmussen, C., Massey, V., & Massey, D. (2008). Memory patterns of acquisition and retention of verbal and nonverbal information in children with fetal alcohol spectrum disorders. *Canadian Journal of Clinical Pharmacology, 15*(1), e44–56.

Samuels, S. I., Maze, A., & Albright, G. (1979). Cardiac arrest during cesarean section in a chronic amphetamine abuser. *Anesthesia and Analgesia, 58,* 528–530.

Shea, A. K., & Steiner, M. (2008). Cigarette smoking during pregnancy. *Nicotine and Tobacco Research, 10*(2), 267–278.

Smith, L. M., Chang, L., Yonekura, M. L., Grob, C., Osborne, D., & Ernst, T. (2001). Brain proton magnetic resonance spectroscopy in children exposed to methamphetamine in utero. *Neurology, 57,* 255–260.

Smith, L. M., LaGasse, L. L., Derauf, C., Grant, P., & Shah, R. (2006). The infant development, environmental, and lifestyle study: Effects of prenatal methamphetamine exposure, polydrug exposure, and poverty on intrauterine growth. *Pediatrics, 118,* 1149–1156.

Smith, L., Yonekura, M. L., Wallace, T., Berman, N., Kuo, J., & Berkowitz, C. (2003). Effects of prenatal methamphetamine exposure on fetal growth and drug withdrawal symptoms in infants born at term. *Journal of Developmental and Behavioral Pediatrics, 24,* 17–23.

Stek, A. M., Fisher, B. K., Baker, R. S., Lang, U., Tseng, C. Y & Clark, K. E. (1993). Maternal and fetal cardiovascular responses to methamphetamine in the pregnant sheep. *American Journal of Obstetrics and Gynecology, 169*(4), 888–897.

Stewart, J. L., & Meeker, J. E. (1997). Fetal and infant deaths associated with maternal methamphetamine abuse. *Journal of Analytic Toxicology, 21,* 515–517.

Struthers, J. M., & Hansen, R. L. (1992). Visual recognition memory in drug-exposed infants. *Behavioral Pediatrics, 13,* 108–111.

Talloczy, Z., Martinez, J., Joset, D., Ray, Y., Gacser, A., Toussi, S., et al. (2008). Methamphetamine inhibits antigen processing, presentation, and phagocytosis. *Plos Pathogens, 4,* e28.

Torfs, C. P., Velie, E. M., Oechsli, F. W., Bateson, T. F., & Curry, C. J. (1994). A population based study of gastroschisis: Demographic, pregnancy, and lifestyle risk factors. *Teratology, 50*(1), 44–53.

Vorhees, C. V., Skelton, M. R., & Williams, M. T. (2007). Age dependent effects of neonatal methamphetamine exposure on spatial learning. *Behavioral Pharmacology, 18,* 549–552.

Weissman, A. D., & Caldecott-Hazard, S. (1995). Developmental neurotoxicity to methamphetamines. *Clinical and Experimental Pharmacology and Physiology, 22,* 372–374.

Wells, P. G., Bhuller, Y., Chen, C. S., Jeng, W., Kasapinovic, S., Kennedy, J. C., et al. (2005). Molecular and biochemical mechanisms in teratogenesis involving reactive oxygen species. *Toxicology and Applied Pharmacology, 207*(2 Suppl.), 354–366.

Williams, M. T., Brown, R. W., & Vorhees, C. V. (2004). Neonatal methamphetamine administration induces region specific long term neuronal morphologic changes in the rat hippocampus, nucleus accumbens, and parietal cortex. *European Journal of Neuroscience 19,* 3165–3170.

Williams, M. T., Moran, M. S., & Vorhees, C. V. (2004). Behavioral and growth effects induced by low dose methamphetamine administration during the neonatal period in rats. *International Journal of Developmental Neuroscience, 22,* 273–283.

Won, L., Bubula, N., & Heller, A. (2001). Methamphetamine concentrations in fetal and maternal brain following prenatal exposure. *Neurotoxicology and Teratology, 23*(4), 349–354.

Yamamoto, B. K., & Bankson, M. G. (2005). Amphetamine neurotoxicity: Cause and consequence of oxidative stress. *Critical Reviews of Neurobiology, 17,* 87–117.

Ye, L., Peng, J. S., Wang, X., Wang, Y. J., Luo, G. X., & Ho, W. Z. (2008). Methamphetamine enhances hepatitis C virus replication in human hepatocytes. *Journal of Viral Hepatitis, 15,* 261–270.

Chapter **21**

Endocannabinoid Hypothesis of Drug Addiction

Emmanuel S. Onaivi, MSc, PhD

In reviewing the history of human drug addictions, one finds previous misconceptions that people addicted to drugs lacked willpower and were morally weak. But we now know that drug addiction is a chronic relapsing brain disease characterized by the compulsive use of addictive substances despite adverse consequences to the individual and society. For more than 50 years, it has been assumed that all drugs of abuse release dopamine in the brain's reward system to produce pleasure and euphoria and, consequently, lead to addiction in vulnerable individuals (Salamone, Correa, Mingote, & Weber, 2005; Spanagel & Weiss, 1999). However, many agents, such as inhalants, barbiturates, or benzodiazepines, do not activate midbrain dopamine-mediated transmission consistently, despite the fact that these drugs have rewarding properties and are heavily abused (Spanagel & Weiss, 1999). Therefore, dopamine is not a simple marker of reward or hedonia, and it might no longer be tenable to suggest that drugs of abuse are simply activating the brain's "natural reward system" (Salamone et al., 2005). In this chapter, I propose that the dopamine hypothesis of drug abuse and reward is another misconception. The dopamine projections in the brain do not convey a specific "reward" signal because dopamine release occurs not only to all drugs of abuse but also to stress, foot shock, aversive and salient stimuli (Horvitz, 2000; Roll, 2005). Mice that cannot make dopamine (DD mice) have been used to test the hypothesis that dopamine is necessary for reward. The results show that dopamine is not required for natural reward (Cannon & Palmiter, 2003) and morphine-induced reward (Hnasko, Sotak, & Palmiter, 2005). Thus, there are numerous problems associated with

Table 21.1
Problems Associated with Dopamine Hypothesis of Reward

Beyond the nucleus accumbens and dopamine hypothesis of reward
• Not all studies point to a unitary role for dopamine in one brain circuitry as the most relevant system in drug abuse.
• Dopamine may not be involved in brain reward mechanisms as previously thought.
• Reward centers in the brain consist of multiple systems and neuroanatomical sites other than the mesoaccumbens dopamine circuitry.
• Dopamine-independent mechanisms involving other neurotransmitters like glutamate, GABA, serotonin, endocannabinoids, stress hormones, and dynorphin are potential substrates for the rewarding effects of abused substances.
• In schizophrenics, dopamine excess in mesoaccumbens causes heightened state of arousal and not pleasure.
• Smokers and cocaine addicts continue to take hits long after the cigarettes become distasteful or after the effects of cocaine have worn off.
• Addictions arise from a complex pattern of pathogenetic and environmental situations.
• Differential effects of abused substances on the complex network of genes, hormones, neurotransmitters, and modulators do not support the concept of a single reward transmitter.
• Manipulation of dopamine circuitry as a pharmacology target should provide medication for substance abuse.
• There is no causal relationship for dopamine as a pleasure or reward transmitter triggered by abused substances.
• Activation of dopamine pathways is not involved in brain-stimulation reward of all brain sites relevant to addiction.
• Electrolytic lesions and 6-OH dopamine lesion studies of dopamine cell bodies in the ventral tegmental area and other brain sites did not attenuate brain-stimulation reward.

dopamine hypothesis of reward (Table 21.1). For example, self-administration of opiates and alcohol occurs even when the mesolimbic dopamine system is lesioned (Koob & Le Moal, 1997) and interference with accumbens dopamine transmission does not substantially blunt the primary motivation for natural rewards (Salamone et al., 2005). Although we cannot underestimate the role of dopamine in the central nervous system, recent studies in schizophrenia, where dopamine hypothesis had dominated the treatment approaches, now show that glutamate receptor offers promise for a new class of antipsychotic agents (Patil et al., 2007).

It appears that the strong and long-held belief of dopamine hypothesis as a basis for drug reward and addiction will not change overnight. However, the recent evidence indicating that the endocannabinoid system plays a pivotal role in reward processes (Solinas, Goldberg, & Piomelli, 2008) suggests that the endocannabinoid system may be a target for the treatment of addictive behaviors. So the activation of the "natural reward system," supposedly mediated by the accumbens dopamine, cannot reasonably be used as a general explanation for drug abuse and addiction (Salamone et al., 2005). Furthermore, the discov-

ery that dopamine neurons fail to respond when animals receive anticipated reward contradicts the neurobiological model of acute drug reward and reinforcement associated with the mesolimbic dopamine system (Sullivan et al., 2008). Most of the addictive substances are plant-derived, including cocaine, caffeine, morphine, ephedrine, nicotine, and so on, and from an evolutionary perspective, the natural function of these plant alkaloids evolved to deter consumption, which is incompatible with the mesolimbic dopamine hypothesis of drug reward and reinforcement—an incompatibility that has been termed "the paradox of drug reward" (Sullivan et al., 2008).

A number of observations have led to renewed questions of the dopamine hypothesis. For example, first-time users of cannabis, or tobacco and other recreational drugs, usually have unpleasant responses with nausea and vomiting. It is also now recognized that these abused substances selectively activate specific pathways, so that while nicotine binds with cholinergic receptors, cannabinoids and opiates bind to cannabinoid and opioid receptors, respectively, and not dopaminergic receptors. It is therefore timely that there is accumulating evidence indicating a central role for the endocannabinoid system (ECS) in the regulation of the rewarding effects of abused substances. Such recent studies have shown that this endocannabinoid system is involved in the common neurobiological mechanism underlying drug addiction (De Vries & Schoffelmeer, 2005; Fattore et al., 2007; Maldonado, Valverde, & Berrendero, 2006; Parolaro, Vigano, & Rubino, 2005), and see Table 21.2. Thus, an endocannabinoid hypothesis of drug reward is postulated from data from our studies and those of others. Endocannabinoids mediate retrograde signaling in neuronal tissues and are involved in the regulation of synaptic transmission, to suppress neurotransmitter release by the presynaptic cannabinoid receptors (CB-Rs). This powerful modulatory action on synaptic transmission has significant functional implications and interactions with the effects of abused substances. Additional support for the endocannabinoid hypothesis of drug reward is derived from the action of cannabinoids or marijuana use on brain reward pathways that is similar to other abused substances. Furthermore, administration of cannabinoids or the use of marijuana exerts numerous pharmacological effects through their interactions with various neurotransmitters and neuromodulators (Table 21.2).

In our preliminary studies to test the endocannabinoid hypothesis of drug reward, we investigated the interaction between vanilloid and cannabinoid agonists and antagonists in the mouse model of aversion using the elevated plus-maze test. The vanilloid agonist used is a natural product, capsaicin, the active ingredient in hot chili peppers that is known to be habit forming. In a follow-up study, we determined the effect of the CB1 antagonist, rimonabant, on withdrawal aversions from chronic treatment with abused drugs. Our

Table 21.2
Framework for an Endocannabinoid Hypothesis of Drug Reward

- The existence of an endocannabinoid physiological control system (EPCS) with a central role in the regulation of the rewarding effects of abused substances.
- The EPCS is intricately involved in almost all the biological processes of the human body and brain.
- The EPCS appears to exert a powerful modulatory action on retrograde signaling associated with cannabinoid inhibition of synaptic transmission.
- The retrograde signaling appears to be involved in the modulation of neurotransmitter release by cannabinoids and endocannabinoids.
- The abundant distribution of the cannabinoid receptors in the brain provides the EPCS limitless signaling capabilities of cross-talk within, and possibly between, receptor families.
- A missense mutation in human fatty acid amide hydrolase may be associated with problem drug use in vulnerable individuals.
- Cannabinoids induce alterations in brain disposition and pharmacological actions of drugs of abuse.
- Changes in endocannabinoid contents in the brain of rats chronically exposed to nicotine, ethanol, or cocaine.
- "Runners high," the sense of euphoric well-being that comes with vigorous exercise such as running, stimulates the release and elevated levels of endocannabinoids.
- The mechanisms of dependence to different substances appear to be different in terms of their impact on the EPCS.
- The endocannabinoid transmission is a component of the brain reward system and appears to play a role in dependence/withdrawal to abused substances.
- Reduced sensitivity to reward in CB1 knockout mice. But mice that cannot make dopamine (mice lacking tyrosine hydroxylase) respond to rewarding stimuli, indicating reward without dopamine.
- Overeating, alcohol and sucrose consumption is decreased in CB1 receptor deleted mice.
- Involvement of endocannabinoid system in the neural circuitry regulating alcohol consumption and motivation to consume alcohol.
- Evidence for the existence of a functional link between the cannabinoid and opioid receptor systems in the control of alcohol intake and motivation to consume alcohol.
- Decreased alcohol self-administration and increased alcohol sensitivity and withdrawal in CB1 receptor knockout mice.
- Endocannabinoid system modulates opioid rewarding and addictive effects by cross-talk between endogenous opioid and endocannabinoid systems in drug reward.
- Involvement of endocannabinoid and glutamate neurotransmission in brain circuits linked to reward and mnemonic processes. Abolition of LTP in mice lacking mGluR5 receptors and enhanced LTP and memory in mice lacking cannabinoid CB1 receptors.
- Endocannabinoid system in memory-related plasticity may be a common mechanism in the control of conditioned drug seeking by cannabinoids.

results suggest that the endocannabinoid physiological control system may be important natural regulatory mechanisms for reward.

ENDOCANNABINOID PHYSIOLOGICAL CONTROL SYSTEM (EPCS) AND REWARD, DRUG ABUSE, AND ADDICTIONS

Cannabinoids are the constituents of the cannabis plant, and endocannabinoids are the marijuana-like substances produced on demand by the human body (Onaivi, Sugiura, & Di Marzo, 2006). Currently, there are two well-characterized CB-Rs with known splice variants, CB1-Rs and CB2-Rs (Onaivi et al., 2006), that are known to mediate the effects of marijuana use and those of the endocannabinoids. 2-Arachidonyl glycerol has emerged as one of the endocannabinoids that acts as a full agonist at CB-Rs. Anandamide, the first endocannabinoid isolated, is a partial agonist at CB-Rs and a full agonist at the vanilloid receptor. CB1-Rs are known to be expressed in the brain and in peripheral tissues. Although CB2-Rs was once believed to be restricted to immune cells, we and others have demonstrated that it is also functionally expressed in neurons, albeit at lower levels than CB1-Rs (Onaivi, Ishiguro, et al., 2006; Van Sickle et al., 2005). These cannabinoid receptors are G-protein coupled receptors with seven transmembrane domains.

Many studies now show that the endocannabinoid system is involved as the major player, and most likely, the common neurobiological mechanism underlying drug reward. There is substantial evidence supporting a role for the endocannabinoid system as a modulator of dopaminergic activity in the basal ganglia (Giuffrida & Piomelli, 2000). The endocannabinoid system therefore participates in the primary rewarding effects of alcohol, opioids, nicotine, cocaine, amphetamine, cannabinoids, and benzodiazepines through the release of endocannabinoids that act as retrograde messengers to inhibit classical transmitters, including dopamine, serotonin, GABA, glutamate, acetylcholine, and norepinephrine (Onaivi, Sugiura, et al., 2006). Furthermore, the endocannabinoid system is intricately involved in the common mechanisms underlying relapse to drug-seeking behavior by mediating the motivational effects of drug-related environmental stimuli and drug reexposure (Maldonado et al., 2006). Therefore, substantial data now point to a role of the endocannabinoid system in triggering and/or preventing reinstatement of drug-seeking behavior (Fattore et al., 2007). It appears that the effects of perturbation of the endocannabinoid system by drugs of abuse can be ameliorated by restoring the perturbed system using cannabinoid ligands.

It is not surprising that preliminary studies with cannabinoid antagonists are showing promise in the reduction of drug use, in smoking cessation,

and reduction in alcohol consumption and, of course, rimonabant has been approved in Europe for treating obesity. It is hoped that these encouraging positive results will lead to new therapeutic agents in the treatment of drug dependency. The promiscuous action and distribution of cannabinoid receptors in most relevant biological systems provides the EPCS limitless signaling capabilities of cross-talk within, and possible between, receptor families that may explain the myriad behavioral effects associated with smoking marijuana. The EPCS, therefore, appears to play a central role in regulating the neural substrate underlying many aspects of drug addiction, including craving and relapse (Yamamoto, Anggadiredja, & Hiranita, 2004). The findings that the EPCS is involved in the reinstatement model provided evidence of the EPCS in the neural machinery underlying relapse. Relapse, the resumption of drug taking following a period of drug abstinence, is considered the main hurdle in treating drug addiction, and pharmacological modulation of the endocannabinoid tone with rimonabant gave positive results in human trials. A summary of data from recent studies of the efficacies of cannabinoid antagonist and mutant mice have recently been reviewed (Parolaro et al., 2005). As the usefulness of the pharmacotherapy of substance abuse has been limited, there is sufficient preclinical evidence for clinical trials to evaluate the efficacy of cannabinoid-based drugs in the treatment of drug dependency.

INTERACTION BETWEEN CB1 AND CB2 RECEPTORS IN DRUG ABUSE AND ADDICTION

The expression of CB1 cannabinoid receptors (CB1-Rs) in the brain and periphery has been well studied, but the brain neuronal expression of CB2-Rs had been ambiguous and controversial and its role in substance abuse is unknown. There is now ample evidence for the functional presence of CB2-Rs in mammalian brain neurons (Gong et al., 2006; Onaivi, 2006; Van Sickle et al., 2005). We have investigated the involvement of CB2-Rs in alcohol preference in mice and alcoholism in humans (Ishiguro et al., 2007). So we tested if CB2-Rs in the brain play a role in alcohol abuse/dependence in animal model and then examined an association between the CB2 gene polymorphism and alcoholism in a Japanese population. There is an association between the Q63R polymorphism of the CB2 gene and alcoholism in the Japanese population. Our data therefore revealed that CB2-Rs are functionally expressed in brain neurons and play a role in substance abuse and dependency (Gong et al., 2006; Ishiguro et al., 2007; Onaivi, 2006). The next question is now that we know that CB2-Rs are present in the brain, what is the nature of and contribution to the known effects of CB1-Rs in the rewarding effects of drug abuse? With

the recent definitive demonstration of neuronal CB2-Rs in the brain, one possible explanation may be that CB2-Rs and CB1-Rs work independently and/or cooperatively in different neuronal populations to regulate a number of physiological activities influenced by drugs of abuse, cannabinoids, endocannabinoids, and marijuana use. Nevertheless, our studies and those of others demonstrate the functional expression of CB2-Rs in the brain that may provide novel targets for the effects of cannabinoids in substance abuse disorders beyond neuro-immunocannabinoid activity.

NEW PERSPECTIVES IN STUDYING THE INVOLVEMENT OF THE ENDOCANNABINOID SYSTEM IN REWARD PROCESSES

We used the elevated plus-maze test—a validated method to measure the performance of rodents that are nocturnal in nature and to obtain an index of anxiety when control and experimental animals are exposed to the maze. The plus-maze consists of two open arms and two enclosed arms linked by a central platform and arranged in a plus sign (+). The elevated plus-maze system was used to study withdrawal anxiogenesis from selected drugs with abuse potential, including cocaine, diazepam, and ethanol. The synthetic methanandamide was included in the study. Adult C57Bl/6 mice were evaluated in the elevated plus-maze test of anxiety following abrupt cessation from chronic treatment with selected doses of cocaine, diazepam, ethanol, 8%w/v, and methanandamide. In a separate group of these mice, the ability of the CB1 cannabinoid antagonist, rimonabant, to block the withdrawal aversions of mice from alcohol and selected drugs with abuse potential was determined. The in-vivo pharmacological interaction was evaluated by the co-administration of cannabinoids and vanilloid ligands. Capsaicin known to activate CB-Rs and vanilloid receptors (VR1-Rs) in the central nervous system (CNS) was used to study the involvement of the ECS in the habit-forming properties of capsaicin using mice. The interaction between the vanilloid and cannabinoid system was performed using selected agonists and antagonists. The ability of the antagonist drug to block agonist drug effect was also evaluated in this paradigm.

INTERACTION OF CANNABINOIDS AND VANILLOIDS: A BIOLOGICAL BASIS OF WHY SOME LIKE IT HOT AND OTHERS DO NOT

We tested the hypothesis that the endocannabinoid physiological control system (ECS) may play a role in the habit-forming properties of capsaicin and

may also be involved in the rewarding effects of abused substances. In the first set of studies, the cross-talk between cannabinoids and vanilloids was determined by evaluating the interactions of capsaicin, a vanilloid receptor 1 agonist, and WIN5512–2, a cannabinoid agonist. The effect of the vanilloid receptor antagonist, capsazepine, and the cannabinoid antagonist, rimonabant, in the actions of capsaicin and the WIN55212–2 were determined. The aversive behavior induced in mice following treatment with capsaicin, which was dependent on gender and strain of mice used, was enhanced by pretreatment with WIN55212–2. While the 30 minutes with WIN55212–2 enhanced aversions induced by capsaicin, capsazepine or rimonabant blocked the aversions induced by capsaicin and WIN55212–2. The effect of capsaicin on the performance of mice in the plus-maze test was dependent on the gender and mouse strain, with C57BL/J male mice being more susceptible to the behavioral aversions induced by capsaicin.

ANTAGONISM OF WITHDRAWAL REACTION FROM ALCOHOL AND DRUG ABUSE BY CB1 RECEPTOR ANTAGONISM IN THE PLUS-MAZE TEST

The effects of rimonabant on the withdrawal aversions from cessation with chronic treatment of mice with the selected addictive drugs were evaluated. Cannabinoid CB1 receptor antagonism reduced behavioral aversions following withdrawal from alcohol, cocaine, and diazepam. It is important to note that rimonabant, the CB1-R antagonist alone, produced variable effects characterized by reduced aversion at low dose and increased aversions to the open arms of the maze at higher doses.

THE ENDOCANNABINOID SYSTEM IN REWARD PROCESSES

The major goal of the study is to understand the role of endocannabinoids (the marijuana-like substances produced by the human body) in drug reward and addiction. Cannabinoids, the constituents in marijuana, mediate their effects by acting on the endocannabinoids and their receptors (CB-Rs), while capsaicin, the pungent chemical in hot chili peppers, a habit-forming food substance, activates the vanilloid type 1 (VR1) receptors to produce its effects. Marijuana and capsaicin enhance appetite, taste, and perhaps addiction and habit-forming properties through CB-Rs and VR1 receptors. The data obtained reveal differential sensitivities of capsaicin in mice that were gender- and strain-dependent that may indicate why some like hot chili peppers and

others do not. Mice naturally do not like capsaicin, and these data are supported by the finding that genetically modified mice lacking the VR1 receptor no longer avoid spicy peppers (Caterina et al., 2000). This may be due to the interaction between the cannabinoid and vanilloid systems. This is because both capsazepine and rimonabant (antagonists at vanilloid and cannabinoid receptors) blocked the aversions induced by WIN55212–2 and capsaicin, indicating a cross-talk between cannabinoid and vanilloid systems. Although the interaction between the EPCS and the vanilloid system is not well established and studied, the results from the investigation on whether the interaction between endocannabinoid and endovanilloid systems induced by the natural ligands could be a basis why some like hot chili peppers and others do not is intriguing. Therefore, it is tempting to speculate and extend the known regulation of the neural substrates altered by abused substances to an interaction or a cross-talk between the EPCS and the endovanilloid (VR1) system. The existence and involvement of the EPCS in this in-vivo model is presented as additional evidence that manipulating the EPCS could be exploited in reducing the behavioral consequences of withdrawal from drug dependency.

Finally, we tested the hypothesis that the EPCS is an integral component of the reward circuit using in-vivo tests. We determined the influence of CB-R antagonism on withdrawal anxiogenesis from chronic alcohol, cocaine, and diazepam treatment in vivo using the mouse plus-maze test. The CB1-R antagonist rimonabant blocked the behavioral aversions to the open arms of the plus-maze, which was precipitated from withdrawal from cocaine, diazepam, and alcohol. Similar to the withdrawal aversions in the plus-maze test, conditioned place preference (CPP) paradigm has been used to investigate the rewarding effects of cannabinoids. Rimonabant has been shown to counteract the CPP supported by classical reinforcers, including food, cocaine, and morphine (Chaperon, Soubrie, Puech, & Thiebot, 1998). This is in agreement with data that demonstrate the antagonistic activity of rimonabant against disruption of cognition or reward-enhancing properties of morphine, amphetamine, and cocaine (Poncelet, Barnouin, Breliere, Le Fur, & Soubrie, 1999), which we have extended to ethanol and diazepam. The blockade of the behavioral aversions by cannabinoid antagonist following chronic administration with alcohol, cocaine, and diazepam is in agreement with data obtained during cannabinoid-induced alterations in brain dispositions of drugs of abuse that correlated with behavioral alterations in mice (Reid & Bornheim, 2001). These results taken together suggest that in the CNS, the EPCS may be a directly important natural regulatory mechanism for reward in the brain and also contribute to reduction in aversive consequences of abused substances. Thus, the data presented, along with those of others, not only support the existence of the EPCS but also

an endocannabinoid hypothesis of drug reward and addiction that may last beyond the dopamine hypothesis of drug reward.

CONCLUSIONS

The preliminary data on the association between activation of cannabinoid and vanilloid receptors show that the interaction of abused substances with the endocannabinoid system is pivotal in habit forming and a neural basis of drug abuse and dependency. Cannabinoids therefore appear to be involved in adding to the rewarding effects of addictive substances, including cocaine, alcohol, and benzodiazepines. Again, these results taken together suggest that in the CNS the EPCS may be a directly important natural regulatory mechanism for reward in the brain and also contribute to reduction in aversive consequences of abused substances. The existence and involvement of an endocannabinoid physiological control system in this in-vivo model is presented as additional evidence that manipulating the EPCS could be exploited in reducing the behavioral consequences of withdrawal from alcohol and drug dependency. It is a good thing that controversy is one of the fuels of science. Thus, there is a lot more research to be done to better understand the nature and the neurobiology of the endocannabinoid physiological control system in health and disease. In the end, the eternal bliss may not be dopamine but endocannabinoids—the brain and body's marijuana and beyond (Onaivi, Sugiura, et al., 2006).

ACKNOWLEDGMENTS

This work would not have been possible without the support, in part, by William Paterson University, Center for Research, the Dean College of Science and Health, Dr. DeYoung Student Worker Fund for maintenance of research animals, and the Provost Office for release time. I thank my colleagues in the Biology Department, and especially the Chairperson, Dr. Gardner, and Norman Schanz in the mouse laboratory. I acknowledge the assistance and support of Dr. Patricia Tagliaferro and my collaborators, including Drs. Hiroki Ishiguro, B. Emmanuel Akinshola, and Sanika Chirwa. I wish to thank past and present students for technical assistance in working with me, especially, Zoila Mora, Alex Perchuk, Danielle Colas, and Chiara Brandoni for direct involvement with some part of these studies. I would also like to thank Drs. John McPartland, Richard Rothman, Mike Baumann, and William Freed for constructive comments. I am indebted to Dr. George Uhl, the Chief of Molecular Neurobiology Branch at NIDA-NIH, where I am a Guest Scientist.

REFERENCES

Cannon, M. C., & Palmiter, R. D. (2003). Reward without dopamine. *J. Neuroscience, 23,* 10827–10831.

Caterina, M. J., Leffler, A., Malmberg, A. B., Martin, W. J., Traffon, J., & Petersen-Zeitz, K. R. (2000). Impaired nociception and pain sensation in mice lacking the capsaicin receptor. *Science, 288,* 306–313.

Chaperon, F., Soubrie, P., Puech, A. J., & Thiebot, M. H. (1998). Involvement of central cannabinoid (CB1) in the establishment of place conditioning in rats. *Psychopharmacology, 135,* 324–332.

De Vries, T. J., & Schoffelmeer, A. N. M. (2005). Cannabinoid CB1 receptors control conditioned drug seeking. *Trends Pharmacol Sci., 26,* 420–426.

Fattore, L., Spano, M. S., Deiana, S., Melis, V., Cossu, G., Fadda, P., et al. (2007). An endocannabinoid mechanism in relapse to drug seeking: A review of animal studies and clinical perspectives. *Brain Res Rev, 53,* 1–16.

Giuffrida, A. & Piomelli, D. (2000). The endocannabinoid system: A physiological perspective on its role in psychomotor control. *Chem Phys Lipids,* 108, 151–158.

Gong, J. P., Onaivi, E. S., Ishiguro, H., Liu, Q.-R., Tagliaferro, P. A., & Brusco, A. (2006). Cannabinoid CB2 receptors: Immunohistochemical localization in rat brain. *Brain Res, 1071,* 10–23.

Ishiguro, H., I., Iwasaki, S., Teasenfitz, L., Higuchi, S., Horiuchi, T., Saito, T., et al. (2007). Involvement of cannabinoid CB2 receptor in alcohol preference in mice and alcoholism in humans. *Pharmacogenomics J., 7,* 380–385.

Hnasko, T. S., Sotak, B. N., & Palmiter, R. D. (2005). Morphine reward in dopamine-deficient mice. *Nature, 438,* 854–857.

Horvitz, J. C. (2000). Mesolimbocortical and nigrostriatal dopamine responses to salient non-reward events. *Neuroscience*, 96, 651–656.

Koob, G. F., & Le Moal, M. (1997). Drug abuse: Hedonic homeostatic dysregulation. *Science, 278,* 52–58.

Maldonado, R., Valverde, O., & Berrendero, F. (2006). Involvement of the endocannabinoid system in drug addiction. *Trends Neurosci., 29,* 225–232.

Onaivi, E. S. (2006). Neuropsychobiological evidence for the functional presence and expression of cannabinoid CB2 receptors in the brain. *Neuropsychobiology, 54,* 231–246.

Onaivi, E. S., Ishiguro, H., Gong, J. P., Patel, S., Perchuk, A., Meozzi, P. A., et al. (2006). Discovery of the presence and functional expression of cannabinoid CB2 receptors in brain. *Ann N Y Acad Sci, 1074,* 514–536.

Onaivi, E. S., Sugiura, T., & Di Marzo, V. (Eds.). (2006). *The brain and body's marijuana and beyond.* Boca Raton, FL: CRC Taylor and Francis.

Parolaro, D., Vigano, D., & Rubino, T. (2005). Endocannabinoid and drug dependence. *Curr Drug Target—CNS Neurol Disorders, 4,* 643–655.

Patil, S. T., Zhang, L. U., Martenyi, F., Lowe, S. I., Jackson, K. A., Andrew, B. V., et al. (2007). Activation of MGlu2/3 receptor as a new approach to treat schizophrenia: A randomized phase 2 clinical trial. *Nature Medicine, 13,* 1102–1107.

Poncelet, M., Barnouin, M. C., Breliere, J. C., Le Fur, G., & Soubrie, P. (1999). Blockade of CB1 receptors by SR141716 selectively antagonizes drug-induced reinstatement of exploratory behavior in gerbils. *Psychopharmacology, 144,* 144–150.

Reid, M. J., & Bornheim, L. M. (2001). Cannabinoid-induced alterations in brain disposition of drugs of abuse. *Biochem. Pharmacol., 61,* 1357–1367.

Roll, E. T. (2005). *Emotion explained.* New York: Oxford University Press.

Salamone, J. D., Correa, M., Mingote, S. M., & Weber, S. M. (2005). Beyond the reward hypothesis: Alternative functions of nucleus accumbens dopamine. (2005). *Curr Opin Pharmacol. 5,* 34–41.

Solinas, M., Goldberg, S. R., & Piomelli, D. (2008). The endocannabinoid system in brain reward processes. *Br. J. Pharmacol, 154,* 369–383

Spanagel, R., & Weiss, F. (1999). The dopamine hypothesis of reward: Past and current status. *Trends Neurosci., 22,* 521–527.

Sullivan, R. J., Hagen, E. H., & Hammerstein, P. (2008). Revealing the paradox of drug reward in human evolution. *Proc. R. Soc. B, 275,* 1231–1241.

Van Sickle, M. D., Duncan, M., Kingsley, P. J., Mouihate, A., Urbani, P., Mackie, K., et al. (2005). Identification and functional characterization of brainstem cannabinoid CB2 receptors. *Science, 310,* 329–332.

Yamamoto, T., Anggadiredja, K., & Hiranita, T. (2004). New perspectives in the studies on endocannabinoid and cannabis: A role for the endocannabinoid-arachidonic acid pathway in drug reward and long-lasting relapse to drug taking. *J Pharmacol Sci., 96,* 382–388.

Chapter **22**

Regulation of μ-Opioid Receptor Desensitization in Sensory Neurons

Cui-Wei Xie, MD, PhD

A common property of G protein-coupled-receptors (GPCRs) is desensitization, loss of signaling after repeated or continued stimulation of the receptor. Desensitization of the μ-opioid receptor, which is the primary target of clinical analgesia and drugs of abuse, has been a subject of intense investigation because of its potential role in the development of opiate tolerance and dependence. Recent studies examining μ-opioid desensitization in dorsal root ganglion (DRG) sensory neurons have revealed multiple mechanisms that regulate signaling and trafficking of the μ receptor, its interactions with other GPCRs, and G protein-effector interactions during acute and chronic opioid exposure. Several protein kinase systems, including phosphoinositide 3-kinase (PI3K), Akt, extracellular signal-regulated kinases (ErK) and p38 mitogen-activated protein kinase (p38 MAPK), have been shown to play important roles in these mechanisms. Adding another layer of complexity, emerging evidence indicates that formation of hetero-oligomers between the μ receptor and other GPCRs permits cross regulation of receptor signaling and trafficking, which in some cases leads to the development of cross desensitization. Further understanding of these dynamic processes could provide insight into the adaptive changes at cellular levels that initiate the complex behavioral changes in opiate tolerance and dependence.

DIFFERENT FORMS OF μ-OPIOID DESENSITIZATION IN DRG NEURONS

Primary cultures of rodent DRG neurons have been extensively used to study opioid regulation of voltage-gated Ca^{2+} channels (VGCCs). It is well documented that opioid receptors couple with Ca^{2+} channels through Gi and Go proteins. Activation of μ-opioid receptors significantly reduces multiple components of VGCC currents, predominantly N- and P/Q-type currents, through a mechanism involving direct interactions between G-protein βγ subunits (Gβγ) and the α_1 subunit of Ca^{2+} channels (Ikeda, 1996). The μ receptor-mediated VGCC inhibition contributes directly to the analgesic action of opiate drugs and has been demonstrated at multiple levels of the pain-processing pathways, including thalamic and periaqueductal grey neurons (Connor & Christie, 1998; Formenti et al., 1995) and peripheral sensory neurons (Nomura, Reuveny, & Narahashi, 1994; Taddese, Nah, & McCleskey, 1995). DRG sensory neurons in cultures express all major types of opioid receptors and VGCCs, providing a convenient model system to study opioid signaling and desensitization with regard to ion channel regulation. Notably, DRG cultures consist of a mixed population of neurons of various sizes. Expression of μ receptors in these neurons appears to be developmentally regulated with the highest level expressed in cells of all sizes during the first postnatal week. The expression then gradually declines in larger cells and is confined to small and medium cells by postnatal day 21 (Beland & Fitzgerald, 2001). Consistent with this expression pattern, only a subset of DRG neurons are sensitive to the effect of a selective μ agonist [D-Ala^2, N-$MePhe^4$, Gly-ol^5]-enkephalin (DAMGO) in cultures derived from adult animals (Schroeder & McCleskey, 1993), whereas the majority of DRG neurons (~90%) respond to DAMGO in cultures derived from early postnatal mice (Tan et al., 2003). The latter can therefore be readily used for the study of μ opioid desensitization.

Desensitization, defined as a loss of cell responsiveness to the agonist, can be mediated by a number of cellular regulatory events. At the receptor level, opioid desensitization has been associated with agonist-induced rapid receptor uncoupling with G proteins, reduction in cell surface receptors, or downregulation of total receptors (Connor, Osborne, & Christie, 2004; Johnson, Christie, & Connor, 2005; Marie, Aguila, & Allouche, 2006). Postreceptor changes that affect Gi/Go proteins, downstream signaling pathways, and the final effector such as ion channels can also contribute to desensitization of opioid signaling (Tan et al., 2003). Two relevant terms often mentioned in this context are the *homologous* and *heterologous* desensitization. Homologous desensitization

refers to the process in which only the activated receptor reduces its responsiveness. Heterologous desensitization, however, reduces signaling of other receptors not activated by the agonist. Another way to define opioid desensitization is based on its time course. With regard to VGCC modulation, acute or rapid desensitization can occur within minutes of agonist exposure, whereas chronic desensitization often develops gradually over several hours. In DRG cultures, both acute and chronic μ opioid desensitization have been observed following DAMGO treatment (Nomura et al., 1994; Samoriski & Gross, 2000; Tan et al., 2003). The acute desensitization has rapid onset within one to five minutes, causes incomplete loss of opioid responses, and is often insensitive to the modulators of major protein kinases. In contrast, chronic desensitization has a much slower time course, leads to complete loss of opioid responses in 24 hours, and is heavily regulated by several protein kinase systems (Nomura et al., 1994; Tan et al., 2003). Acute and chronic μ opioid desensitization also differentially affect VGCC modulation by other Gi/Go-coupled receptors in DRG neurons. For example, γ-aminobutyric acid $(GABA)_B$ receptor-mediated VGCC inhibition can be heterologously reduced following acute but not chronic DAMGO desensitization. On the other hand, chronic DAMGO treatment induces heterologous desensitization to α2-adrenergic receptor-mediated VGCC inhibition (Samoriski & Gross, 2000; Tan et al., 2003). These findings suggest that acute and chronic desensitization are mechanistically different and may affect distinct components in the G protein-receptor-Ca^{2+} channel pathway.

How these different forms of desensitization are related to opioid tolerance in vivo remains an open question. Tolerance is defined as a decreased physiological response to the same dose of drug during repeated exposure. Compared to *in-vitro* desensitization, tolerance develops over a more extended period (hours to days) and involves complex changes not only at the cellular level but also in various neural networks. Many believe that cellular mechanisms of desensitization serve to initiate the cascade of events ultimately leading to opioid tolerance and dependence (Bailey & Connor, 2005; Borgland, 2001; Marie et al., 2006). However, the exact role of desensitization-related processes in the development of tolerance remains to be clarified. Studies have shown that such roles may vary in different experimental settings, depending upon the agonist applied, cell types, and the form of desensitization examined (Connor et al., 2004). As discussed below, prolonged desensitization coupled with poor receptor recycling is likely to promote tolerance. On the other hand, desensitization associated with rapid receptor internalization and recycling may in fact be a protective mechanism that prevents extended receptor signaling leading to tolerance (Martini & Whistler, 2007).

Protein Kinase Modulation of Desensitization

Two major groups of protein kinases have been implicated in GPCR desensitization observed in nonneuronal cells: members of G-protein receptor kinase (GRK) family and second messenger-activated protein kinases. Agonist-induced receptor phosphorylation by GRKs, followed by binding of β-arrestin and receptor uncoupling from G proteins, is considered a common mechanism for acute homologous desensitization. This process is often complemented by feedback phosphorylation via second messenger-activated kinases, such as protein kinase A (PKA), protein kinase C (PKC), and MAPK. These kinases can target both homologous and heterologous receptors (Chuang, Iacovelli, Sallese, & De Blasi, 1996) and affect postreceptor components, including G proteins (Fields & Casey, 1995) and effector ion channels (Hamid et al., 1999). Thus, GPCR desensitization could be mediated by multiple protein kinases with changes occurring at the receptor, G_i/G_o proteins, effectors, or their regulators.

Numerous studies have examined whether the above general mechanisms can apply to desensitization of μ opioid signaling. The μ-opioid receptor has various phosphorylation sites on Ser, Thr, and Tyr residues located on its three cytoplasmic loops and intracellular carboxyl terminal domain. Phosphorylation of these sites regulates signaling efficacy (McLaughlin & Chavkin, 2001) and receptor trafficking (El Kouhen et al., 2001). In cells expressing modified μ receptors with C-terminal truncation or point mutation at the putative phosphorylation sites, DAMGO- or morphine-induced desensitization is significantly attenuated or completely abolished (Deng et al., 2000; Pak, O'Dowd, & George, 1997; Schulz et al., 2004). These findings support the view that μ receptor phosphorylation may be a major contributor to opioid desensitization. It is particularly tempting to link acute desensitization with agonist-induced, rapid μ receptor phosphorylation because of their similar time courses. However, several issues remain to be addressed with this assumption. First, despite extensive investigation, the kinases responsible for μ receptor phosphorylation have not been firmly established. Overexpression of GRK2 in heterologous cells reportedly increases agonist-induced μ receptor phosphorylation (Schulz et al., 2004; Zhang et al., 1998), but evidence exists that activation of μ receptors fails to trigger accumulation of GRKs at the plasma membrane, indicating a weak interaction between the two (Schulz, Wehmeyer, & Schulz, 2002). Second, many relevant studies are conducted using heterologous expression systems, and so far, direct evidence is still lacking to confirm phosphorylation of the μ receptor by GRKs or other kinases in native neurons. Finally, as shown in DRG neurons (Nomura et al., 1994; Tan et al., 2003) and

nonneuronal cells (Kaneko et al., 1997; Morikawa et al., 1998), acute desensitization of μ opioid regulation of VGCCs is not affected by altering the function of major protein kinases, including PKA, PKC, PI3K, Akt, and ErK, nor is it affected by nonspecific inhibitors of protein kinases or phosphatases such as H-8 and okadaic acid. These observations do not support a significant role of protein phosphorylation in this form of desensitization.

In contrast to the acute desensitization, our recent studies show that chronic desensitization in DRG neurons is regulated by a number of second messenger-activated protein kinases in the PI3K-Akt pathway and MAPK cascades (Tan et al., 2003). PI3K comprises a family of dual specificity enzymes with both lipid kinase and protein kinase activity, which can be directly activated by Gβγ dimmers upon activation of μ receptors. One of the key downstream targets of PI3K is Akt, a serine-threonine kinase. The activity of Akt is enhanced by the presence of phosphatidylinositol-3,4,5-P_3 (PIP_3), a lipid product of PI3K, but reduced by phosphates and tensin homolog (PTEN) that reduces the cellular level of PIP_3. In mouse DRG neurons, pharmacological blockade of PI3K activity during a four-hour DAMGO treatment significantly reduces the extent of chronic desensitization, whereas upregulation of cellular Akt levels in neurons lacking PTEN accelerates the desensitization in a PI3K-dependent manner. These findings clearly indicate a facilitating effect of the PI3K-Akt pathway on the chronic desensitization. Similarly, inhibition of the MAPK kinase (MEK)-Erk pathway with PD98059 attenuates DAMGO desensitization. Concurrent blockade of PI3K and Erk activity with selective inhibitors produces no additive relieve of the desensitization, suggesting that the two kinases act in a serial manner. This is consistent with the finding in other cell types that μ agonists activate Erk through Gβγ- and PI3K-dependent mechanisms (Ai, Gong, & Yu, 1999). Further analyses demonstrate that chronic DAMGO exposure leads to loss of prepulse facilitation (PPF), a measure of voltage-dependent Gβγ-Ca^{2+} channel interactions, and that such a loss can be partially reversed by inhibition of PI3K or Erk activity. Thus, PI3K and Erk cascades may facilitate chronic desensitization through postreceptor modifications that weaken Gβγ-Ca^{2+} channel interactions. Interestingly, another component of the MAPK cascade, p38 MAPK, is also crucially involved in DAMGO desensitization but through different mechanisms. In HEK293 cells, p38 MAPK facilitates DAMGO-induced μ receptor internalization by enhancing the function of endocytic machinery regulated by the small GTPase Rab5 (Cavalli et al., 2001; Mace, Miaczynska, Zerial, & Nebreda, 2005). Blocking p38 MAPK activity attenuates both desensitization and internalization of the μ receptor in chronic DAMGO-treated DRG neurons. On the other hand, inhibition of p38 MAPK does not affect desensitization induced by morphine, a μ agonist

triggering little receptor internalization. It is thus likely that involvement of p38 MAPK in the desensitization is closely related to its ability to regulate μ receptor internalization (Tan, Evans, & Xie, 2007).

μ-Opioid Receptor Trafficking and Desensitization

The μ-opioid receptor undergoes agonist-induced endocytosis via clathrin coated-pits, a process regulated by receptor phosphorylation and association with nonvisual β-arrestins. The exact role of this process in regulating opioid signaling has been a subject of intense investigation (Connor et al., 2004; Marie et al., 2006; Martini & Whistler, 2007). Studies in primary neurons and AtT20 cells have indicated that receptor internalization does not contribute to acute desensitization of μ receptors coupled to VGCCs (Borgland, Connor, Osborne, Furness, & Christie, 2003; Walwyn et al., 2006) or inward rectifying potassium channels (Arttamangkul, Torrecilla, Kobayashi, Okano, & Williams, 2006). The relationship between chronic desensitization and μ receptor internalization, however, is more complex. The internalization induced by prolonged agonist treatment not only attenuates opioid responsiveness via physical removal of the receptor from the cell surface but also promotes receptor dephosphorylation and recycling (Qiu, Law, & Loh, 2003). The internalized GPCRs can return to the cell surface through at least two recycling pathways, a fast/early sorting pathway occurring within minutes and a slow/late sorting pathway taking several hours (Sheff, Daro, Hull, & Mellman, 1999). When rapid recycling occurs, the internalization serves as an important means for receptor resensitization, effectively reducing the extent of apparent desensitization (Koch et al., 1998; Qiu et al., 2003). Alternatively, if the internalized receptors are trapped in late endosomes, desensitization can be enhanced because of significant loss of surface receptors (Law et al., 2000). Extended or repeated opioid exposure can also target internalized receptor to lysosomes for degradation, causing receptor downregulation and more persistent signaling reduction. Thus the functional consequence of μ receptor internalization may vary in different model systems, depending upon the rate and extent of endocytosis as well as its coupling with distinct intracellular sorting pathways that determine the postendocytic fate of the receptor.

In mouse DRG neurons, DAMGO treatment induces substantial μ receptor endocytosis within 20 minutes and results in an approximately 40 percent loss in surface μ receptors after 4- to 24-hour treatments. The internalized receptors are found to colocalize with Rab4 and Rab11, the respective marker of the early and late endosomes, suggesting that both the fast and slow sorting pathways participate in μ receptor recycling in these neurons (Walwyn et

al., 2006). Chronic DAMGO desensitization can be prevented or significantly reduced when receptor endocytosis is abolished with p38 MAPK inhibitors (Tan et al., 2007) or when the number of surface μ receptors increases dramatically through adenovirus-mediated overexpression (Walwyn et al., 2004). It is hence conceivable that chronic DAMGO-induced μ receptor internalization may be coupled with delayed recycling through the late sorting pathway, resulting in sustained loss of surface receptors and desensitization. Such a link between internalization and chronic desensitization appears to be agonist selective. After a four-hour exposure to morphine, VGCC inhibition by morphine is significantly reduced but there is no evident μ receptor internalization (Tan et al., 2007). Interestingly, the effect of DAMGO remains relatively intact in chronic morphine-treated neurons (Walwyn et al., 2006). These findings indicate that morphine can induce desensitization through internalization-independent mechanisms, but such desensitization may be incomplete as revealed by testing with DAMGO, a more efficacious full agonist of the μ receptor. A possible interpretation here is that inability of morphine to stimulate endocytosis-related cellular processes, such as activation of p38 MAPK (Mace et al., 2005), may have prevented development of a full-fledged μ opioid desensitization in DRG neurons.

The differences in internalization efficacy of various opiate drugs have been linked with their distinct propensity in promoting tolerance and dependence. Full μ receptor agonists that stimulate internalization, such as etorphine and fentanyl, tend to produce less pronounced tolerance compared to morphine that is least effective in triggering internalization (Duttaroy & Yoburn, 1995). Expression in HEK293 cells of a chimeric receptor (μ/δ receptor), in which the C-terminus of the μ receptor is replaced with that of the δ opioid receptor, facilitates morphine-induced internalization and reduces cellular tolerance and cAMP superactivation, a cellular hallmark of opiate withdrawal (Finn & Whistler, 2001). More recent studies have provided *in-vivo* evidence that morphine tolerance and withdrawal are significantly reduced in a knockin mouse expressing a mutant μ receptor that internalizes in response to morphine (Kim et al., 2008). Thus, internalization could reduce the liability of opiate drugs for promoting tolerance and dependence, likely by limiting receptor signaling and downstream adaptive changes such as superactivation of the cAMP pathway.

Receptor Oligomerization and Desensitization

GPCRs interact with each other through formation of homo- or heteromeric receptor complexes. Such events have divergent impact on receptor binding, signaling or trafficking, and allow cross-regulation between different

receptor systems (Bouvier, 2001; Devi, 2001). An increasing number of studies have shown that heterodimerization between the μ-opioid receptor and other GPCRs often reduces signaling of both receptors, promoting heterologous or cross-desensitization. In HEK293 cells expressing the heterodimer of μ and sst_{2A} somatostatin receptors, exposure to the selective agonist of either receptor induces phosphorylation and desensitization of both receptors (Pfeiffer et al., 2002). Similarly, oligomerization of the μ receptor with the substance P receptor (NK1) facilitates agonist-induced cross-phosphorylation and cointernalization of μ and NK1 receptors in HEK293 cells (Pfeiffer et al., 2003). Another example is hetero-oligomerization of μ opioid and α_{2A} adrenergic receptors. When both receptors are expressed in cell lines or transfected neurons, the heterocomplex formation enhances μ receptor signaling in response to morphine, but significantly reduces opioid responses following simultaneous application of morphine and the α_{2A} agonist clonidine (Jordan, Gomes, Rios, Filipovska, & Devi, 2003). In DRG neurons, functional interactions between naturally existing μ and α_{2A} receptors promote chronic cross-desensitization and cointernalization of the two receptors (Tan, 2005). An interesting common finding in these studies is modification of trafficking profiles of μ receptors in the presence of receptor oligomers. The observed cointernalization of the two interactive receptors in response to a single selective agonist may be explained by the "dragging" phenomenon in which the ligand-activated receptor can "drag" another receptor in the same complex to the endocytic pathway (He, Fong, von Zastrow, & Whistler, 2002). Formation of receptor complexes can also alter receptor interactions with β-arrestin, a key adaptor protein regulating receptor endocytosis and sorting, causing delayed receptor recycling and resensitization (Pfeiffer et al., 2003). As it often occurs between functionally related GPCRs, this type of heterodimeric interaction may serve as a negative feedback mechanism to suppress the activity of shared downstream signaling pathways during prolonged receptor stimulation.

While receptor cotrafficking may contribute significantly to the chronic cross-desensitization, direct inter-receptor communication within hetero-oligomers by conformational changes has been proposed for more rapid signaling regulation (Franco et al., 2005; George, O'Dowd, & Lee, 2002). Using fluorescence resonance energy transfer microscopy, a recent report demonstrates that μ and α_{2A} receptors communicate with each other in the heterodimer through a cross-conformational switch that permits direct inhibition of one receptor by the other with subsecond kinetics (Vilardaga et al., 2008). It should also be noted that not all events of heteromerization lead to cross inactivation or desensitization. Formation of μ and δ opioid receptor heterodimers, for example, results in synergistic enhancement of receptor binding and signaling

by μ and δ ligands (Gomes et al., 2000). Taken together, receptor oligomerization is a dynamic process regulated by different cellular mechanisms; further investigation is required to fully understand its molecular basis and functional consequence. Finally, most studies addressing this issue to date have been conducted in heterologous cells or in systems where receptors are overexpressed. This may lead to interactions nonexistent with endogenously expressed receptors. It is therefore necessary for future study to identify and characterize interactions between endogenous receptors in native neurons. Such knowledge could shed light on the control of opioid receptor signaling and treatment of opiate tolerance and dependence.

REFERENCES

Ai, W., Gong, J., & Yu, L. (1999). MAP kinase activation by mu opioid receptor involves phosphatidylinositol 3-kinase but not the cAMP/PKA pathway. *FEBS Lett., 456,* 196–200.

Arttamangkul, S., Torrecilla, M., Kobayashi, K., Okano, H., & Williams, J. T. (2006). Separation of mu-opioid receptor desensitization and internalization: Endogenous receptors in primary neuronal cultures. *J Neurosci, 26,* 4118–4125.

Bailey, C. P., & Connor, M. (2005). Opioids: Cellular mechanisms of tolerance and physical dependence. *Curr Opin Pharmacol, 5,* 60–68.

Beland, B., & Fitzgerald, M. (2001). Mu- and delta-opioid receptors are downregulated in the largest diameter primary sensory neurons during postnatal development in rats. *Pain, 90,* 143–150.

Borgland, S. L. (2001). Acute opioid receptor desensitization and tolerance: Is there a link? *Clin Exp Pharmacol Physiol, 28,* 147–154.

Borgland, S. L., Connor, M., Osborne, P. B., Furness, J. B., & Christie, M. J. (2003). Opioid agonists have different efficacy profiles for G protein activation, rapid desensitization, and endocytosis of mu-opioid receptors. *J Biol Chem, 278,* 18776–18784.

Bouvier, M. (2001). Oligomerization of G-protein-coupled transmitter receptors. *Nat Rev Neurosci, 2,* 274–286.

Cavalli, V., Vilbois, F., Corti, M., Marcote, M. J., Tamura, K., Karin, M., et al. (2001). The stress-induced MAP kinase p38 regulates endocytic trafficking via the GDI:Rab5 complex. *Mol Cell, 7,* 421–432.

Chuang, T. T., Iacovelli, L., Sallese, M., & De Blasi, A. (1996). G protein-coupled receptors: Heterologous regulation of homologous desensitization and its implications. *Trends Pharmacol Sci, 17,* 416–421.

Connor, M., & Christie, M. J. (1998). Modulation of Ca2+ channel currents of acutely dissociated rat periaqueductal grey neurons. *J Physiol, 509,* 47–58.

Connor, M., Osborne, P. B., & Christie, M. J. (2004). Mu-opioid receptor desensitization: Is morphine different? *Br J Pharmacol, 143,* 685–696.

Deng, H. B., Yu, Y., Pak, Y., O'Dowd, B. F., George, S. R., Surratt, C. K., et al. (2000). Role for the C-terminus in agonist-induced mu opioid receptor phosphorylation and desensitization. *Biochemistry, 39,* 5492–5499.

Devi, L. A. (2001). Heterodimerization of G-protein-coupled receptors: Pharmacology, signaling and trafficking. *Trends Pharmacol Sci, 22,* 532–537.

Duttaroy, A., & Yoburn, B. C. (1995). The effect of intrinsic efficacy on opioid tolerance. *Anesthesiology, 82,* 1226–1236.

El Kouhen, R., Burd, A. L., Erickson-Herbrandson, L. J., Chang, C. Y., Law, P. Y., & Loh, H. H. (2001). Phosphorylation of Ser363, Thr370, and Ser375 residues within the carboxyl tail differentially regulates mu-opioid receptor internalization. *J Biol Chem, 276,* 12774–12780.

Fields, T. A., & Casey, P. J. (1995). Phosphorylation of Gz alpha by protein kinase C blocks interaction with the beta gamma complex. *J Biol Chem, 270,* 23119–22325.

Finn, A. K., & Whistler, J. L. (2001). Endocytosis of the mu opioid receptor reduces tolerance and a cellular hallmark of opiate withdrawal. *Neuron, 32,* 829–839.

Formenti, A., Arrigoni, E., Martina, M., Taverna, S., Avanzini, G., & Mancia, M. (1995). Calcium influx in rat thalamic relay neurons through voltage-dependent calcium channels is inhibited by enkephalin. *Neurosci Lett, 201,* 21–24.

Franco, R., Casado, V., Mallol, J., Ferre, S., Fuxe, K., Cortes, A., et al. (2005). Dimer-based model for heptaspanning membrane receptors. *Trends Biochem Sci, 30,* 360–366.

George, S. R., O'Dowd, B. F., & Lee, S. P. (2002). G-protein-coupled receptor oligomerization and its potential for drug discovery. *Nat Rev Drug Discov, 1,* 808–820.

Gomes, I., Jordan, B. A., Gupta, A., Trapaidze, N., Nagy, V., & Devi, L. A. (2000). Heterodimerization of mu and delta opioid receptors: A role in opiate synergy. *J Neurosci, 20,* RC110.

Hamid, J., Nelson, D., Spaetgens, R., Dubel, S. J., Snutch, T. P., & Zamponi, G. W. (1999). Identification of an integration center for cross-talk between protein kinase C and G protein modulation of N-type calcium channels. *J Biol Chem, 274,* 6195–6202.

He, L., Fong, J., von Zastrow, M., & Whistler, J. L. (2002). Regulation of opioid receptor trafficking and morphine tolerance by receptor oligomerization. *Cell, 108,* 271–282.

Ikeda, S. R. (1996). Voltage-dependent modulation of N-type calcium channels by G-protein beta gamma subunits. *Nature, 380,* 255–258.

Johnson, E. E., Christie, M. J., & Connor, M. (2005). The role of opioid receptor phosphorylation and trafficking in adaptations to persistent opioid treatment. *Neurosignals, 14,* 290–302.

Jordan, B. A., Gomes, I., Rios, C., Filipovska, J., & Devi, L. A. (2003). Functional interactions between mu opioid and alpha 2A-adrenergic receptors. *Mol Pharmacol, 64,* 1317–1324.

Kaneko, S., Yada, N., Fukuda, K., Kikuwaka, M., Akaike, A., & Satoh, M. (1997). Inhibition of Ca2+ channel current by mu- and kappa-opioid receptors coexpressed

in Xenopus oocytes: Desensitization dependence on Ca2+ channel alpha 1 subunits. *Br J Pharmacol, 121,* 806–812.

Kim, J. A., Bartlett, S., He, L., Nielsen, C. K., Chang, A. M., Kharazia, V., et al. (2008). Morphine-induced receptor endocytosis in a novel knockin mouse reduces tolerance and dependence. *Curr Biol, 18,* 129–135.

Koch, T., Schulz, S., Schroder, H., Wolf, R., Raulf, E., & Hollt, V. (1998). Carboxyl-terminal splicing of the rat mu opioid receptor modulates agonist-mediated internalization and receptor resensitization. *J Biol Chem, 273,* 13652–13657.

Law, P. Y., Erickson, L. J., El-Kouhen, R., Dicker, L., Solberg, J., Wang, W., et al. (2000). Receptor density and recycling affect the rate of agonist-induced desensitization of mu-opioid receptor. *Mol Pharmacol, 58,* 388–398.

Mace, G., Miaczynska, M., Zerial, M., & Nebreda, A. R. (2005). Phosphorylation of EEA1 by p38 MAP kinase regulates mu opioid receptor endocytosis. *Embo J, 24,* 3235–3246.

Marie, N., Aguila, B., & Allouche, S. (2006). Tracking the opioid receptors on the way of desensitization. *Cell Signal, 18,* 1815–1833.

Martini, L., & Whistler, J. L. (2007). The role of mu opioid receptor desensitization and endocytosis in morphine tolerance and dependence. *Curr Opin Neurobiol, 17,* 556–564.

McLaughlin, J. P., & Chavkin, C. (2001). Tyrosine phosphorylation of the mu-opioid receptor regulates agonist intrinsic efficacy. *Mol Pharmacol, 59,* 1360–1368.

Morikawa, H., Fukuda, K., Mima, H., Shoda, T., Kato, S., & Mori, K. (1998). Desensitization and resensitization of delta-opioid receptor-mediated Ca2+ channel inhibition in NG108–15 cells. *Br J Pharmacol, 123,* 1111–1118.

Nomura, K., Reuveny, E., & Narahashi, T. (1994). Opioid inhibition and desensitization of calcium channel currents in rat dorsal root ganglion neurons. *J Pharmacol Exp Ther, 270,* 466–474.

Pak, Y., O'Dowd, B. F., & George, S. R. (1997). Agonist-induced desensitization of the mu opioid receptor is determined by threonine 394 preceded by acidic amino acids in the COOH-terminal tail. *J Biol Chem, 272,* 24961–24965.

Pfeiffer, M., Kirscht, S., Stumm, R., Koch, T., Wu, D., Laugsch, M., et al. (2003). Heterodimerization of substance P and mu-opioid receptors regulates receptor trafficking and resensitization. *J Biol Chem, 278,* 51630–51637.

Pfeiffer, M., Koch, T., Schroder, H., Laugsch, M., Hollt, V., & Schulz, S. (2002). Heterodimerization of somatostatin and opioid receptors cross-modulates phosphorylation, internalization, and desensitization. *J Biol Chem, 277,* 19762–19772.

Qiu, Y., Law, P. Y., & Loh, H. H. (2003). Mu-opioid receptor desensitization: Role of receptor phosphorylation, internalization, and representation. *J Biol Chem, 278,* 36733–36739.

Samoriski, G. M., & Gross, R. A. (2000). Functional compartmentalization of opioid desensitization in primary sensory neurons. *J Pharmacol Exp Ther, 294,* 500–509.

Schroeder, J. E., & McCleskey, E. W. (1993). Inhibition of Ca2+ currents by a mu-opioid in a defined subset of rat sensory neurons. *J Neurosci, 13,* 867–873.

Schulz, R., Wehmeyer, A., & Schulz, K. (2002). Opioid receptor types selectively cointernalize with G protein-coupled receptor kinases 2 and 3. *J Pharmacol Exp Ther, 300,* 376–384.

Schulz, S., Mayer, D., Pfeiffer, M., Stumm, R., Koch, T., & Hollt, V. (2004). Morphine induces terminal micro-opioid receptor desensitization by sustained phosphorylation of serine-375. *Embo J, 23,* 3282–3289.

Sheff, D. R., Daro, E. A., Hull, M., & Mellman, I. (1999). The receptor recycling pathway contains two distinct populations of early endosomes with different sorting functions. *J Cell Biol, 145,* 123–139.

Taddese, A., Nah, S. Y., & McCleskey, E. W. (1995). Selective opioid inhibition of small nociceptive neurons. *Science, 270,* 1366–1369.

Tan, M., Walwyn, W. M., Evans, C. J., Xie, C. W. (2009). p38 map kinase and beta-arrestin 2 mediate functional interactions between endogenous mu-opioid and alpha 2A-adrenergic receptors in neurons. *J Biol Chem*. Epub ahead of print, Jan 6, 2009, http://www.jbc.org/cgi/doi/10.1074/jbc.M806742200.

Tan, M., Groszer, M., Tan, A. M., Pandya, A., Liu, X., & Xie, C. W. (2003). Phosphoinositide 3-kinase cascade facilitates mu-opioid desensitization in sensory neurons by altering G-protein-effector interactions. *J Neurosci, 23,* 10292–10301.

Tan, M., & Xie, C. W. (2005). Interactions between mu-opioid and alpha 2A adrenergic receptors promote receptor internalization and desensitization. *Internat. Narc. Res. Conf. Abs, M42.*

Vilardaga, J. P., Nikolaev, V. O., Lorenz, K., Ferrandon, S., Zhuang, Z., & Lohse, M. J. (2008). Conformational cross-talk between alpha2A-adrenergic and mu-opioid receptors controls cell signaling. *Nat Chem Biol, 4,* 126–131.

Walwyn, W. M., Keith, D. E., Jr., Wei, W., Tan, A. M., Xie, C. W., Evans, C. J., et al. (2004). Functional coupling, desensitization and internalization of virally expressed mu opioid receptors in cultured dorsal root ganglion neurons from mu opioid receptor knockout mice. *Neuroscience, 123,* 111–121.

Walwyn, W. M., Wei, W., Xie, C. W., Chiu, K., Kieffer, B. L., Evans, C. J., et al. (2006). Mu opioid receptor-effector coupling and trafficking in dorsal root ganglia neurons. *Neuroscience, 142,* 493–503.

Zhang, J., Ferguson, S. S., Barak, L. S., Bodduluri, S. R., Laporte, S. A., Law, P. Y., et al. (1998). Role for G protein-coupled receptor kinase in agonist-specific regulation of mu-opioid receptor responsiveness. *Proc Natl Acad Sci U S A, 95,* 7157–7162.

Index

About the Editor and Contributors

EDITOR

Angela Browne-Miller, PhD, DSW, MPH, is the Set Editor of the *Praeger International Collection on Addictions* (2009), Director of Metaxis Institute for Personal, Social, and Systems Change, based in northern California, United States; Director of Browne and Associates Violence, Substance Abuse, and Trauma Treatment and Prevention Program, also in northern California; has been a keynote speaker at conferences around the world on addiction, violence, and behavior change; and is the author of numerous books, including *To Have and to Hurt: Seeing, Changing or Escaping Patterns of Abuse in Relationships* (2007) and *Rewiring Your Brain to Change Your Behavior* (2009). Dr. Browne-Miller earned two doctorates and two master's degrees at the University of California, Berkeley, where she lectured in three departments for 14 years, and has served as a National Institute of Mental Health Postdoctoral Fellow, a U.S. Department of Public Health Fellow, the public relations director for Californians for Drug Free Youth, the Research Education and Treatment Director for the Cokenders Alcohol and Drug Program, a member of the board of directors of the Employee Assistance Society of North America, an advisor to addiction treatment programs in the United States and several other countries, and project director on three California Department of Health violence prevention projects.

CONTRIBUTORS

Atul Ambekar, MD, is Assistant Professor at the National Drug Dependence Treatment Centre and Department of Psychiatry, All India Institute of Medical Sciences in New Delhi, Delhi, India. He earned his MD degree from All India Institute of Medical Sciences, a prestigious and internationally recognized institute in India. He worked as Senior Resident and later joined the United Nations Office on Drugs and Crime (UNODC), Regional Office for South Asia, New Delhi, as Research Officer. Subsequently, Dr. Ambekar joined the All India Institute of Medical Sciences as Assistant Professor. He is a resource person for trainings and research for various national and international organizations, such as UNODC, Joint United Nations Programme on HIV/AIDS (UNAIDS), and the government of India. He has several publications to his credit.

Charlotte A. Boettiger, PhD, is Assistant Professor of Psychology and Biomedical Research Imaging at the University of North Carolina Chapel Hill, in Chapel Hill, North Carolina, United States. She was formerly an Associate Investigator at the Ernest Gallo Clinic and Research Center and an Adjunct Assistant Professor in the University of California, San Francisco, Department of Neurology. She studied Biology as an undergraduate at the University of California Berkeley, and received her PhD in Neuroscience at UCSF, where she studied the cellular and synaptic mechanisms of vocal learning. She completed a postdoctoral fellowship with Dr. Mark D. Esposito at the Henry H. Wheeler, Jr., Brain Imaging Center in the Helen Wills Neuroscience Institute at UC Berkeley. Her research focuses on the cognitive neurobiology of addiction. Her experimental approach employs neuroimaging, behavioral techniques, genetics, and pharmacology studies in humans. Dr. Boettiger was awarded the Hugh O'Connor Memorial Fellowship by the Wheeler Center for the Neurobiology of Addiction in 2002.

Kathleen Bradbury-Golas, DNP, RN, APN, is Assistant Professor of Nursing at Richard Stockton College of New Jersey in Pomona, New Jersey, United States. She currently teaches across the nursing curriculum in both undergraduate and graduate nursing programs. She also practices part-time as a family nurse practitioner in southern New Jersey. Dr. Bradbury-Golas holds a Doctorate in Nursing Practice from Case Western Reserve University of Cleveland, Ohio, United States. She is certified as both a Family Nurse Practitioner, by the American Association of Nurse Practitioners, and an Adult Health Clinical Nurse Specialist, by the American Nurses' Credentialing Center. Dr. Bradbury-Golas completed her dissertation research studying health promotion practices and opiate-dependent users.

Giuseppe Carrà, MD, MSc, PhD, Consultant Psychiatrist and Research Fellow at University College London, England, studied Medicine at Milan University in Italy, and did his psychiatric training at Pavia University in Pavia, Italy. He has a PhD in Public Mental Health from the University of Pavia. Dr. Carrà did an MSc in Psychiatric Research at University College London. Since 1995, he has been a Consultant Psychiatrist at S. Paolo University Hospital Trust, Milan, though he worked for two years (2004–2006) in the Camden and Islington CMHTs. Since 2004, Dr. Carrà has been a Research Fellow at UCL. He is a Board Member of the International Society of Addiction. He claims the following governmental and institutional appointments: Member of the Guideline Development Group for Schizophrenia at the Italian National Institute of Health; Member of the Addiction Panel at the Italian Department of Health; Member of the panel of independent experts assisting the European Commission at Directorate General of Justice, Freedom and Security in the priority field "Prevent Drug Consumption and Inform the Public," July 2007–June 2010. Research interests include "dual diagnosis" of psychosis and substance misuse, psychoeducational interventions in schizophrenia, and adaptation and implementation of local guidelines.

Vicki W. Chanon, PhD, completed her PhD in the Cognitive Psychology Program at the University of North Carolina at Chapel Hill in 2007. Her dissertation focused on the neural correlates of varying types of attentional allocation (voluntary, reflexive, and social) in healthy adults using event-related potentials. As an undergraduate, Vicki earned her BA in Psychology and Philosophy at the University of Delaware. She is currently a postdoctoral researcher working with Dr. Charlotte Boettiger in the Behavioral Neuroscience Program at the University of North Carolina at Chapel Hill in Chapel Hill, North Carolina, United States. Her current research focuses on dysfunctional attentional processes in addictive disorders. Specifically, she is using behavioral, neuroimaging, and pharmacological methods to gain an understanding of the neurobiological underpinnings of attentional biases toward drug-related words and images in those suffering with addictive disorders.

Kyle M. Clayton, MS, is a Clinical Psychology graduate student at the University of Texas Tyler, in Tyler, Texas, United States. While at UT Tyler, he has been involved in research concerning neuropsychological testing for children with learning disabilities, correlations between olfaction and Alzheimer's disease, the impact of Lorazepam on neuropsychological measures in suspected dementia patients, the effect of exercise on cognition in women receiving adjuvant chemotherapy for breast cancer, and family-focused treatment of juvenile sex offenders. Mr. Clayton provides intervention for children and families as a counselor at a residential treatment center, a teacher/mentor for a youth center,

and a caseworker for a foster care/adoption agency. Following the completion of his master's degree in May 2008, Mr. Clayton entered the doctoral program in clinical psychology at the University of Texas Southwestern Medical Center in Dallas.

Herbert C. Covey, PhD, of the Adams County Social Services Department, and the College of Continuing Education, University of Colorado Boulder in Boulder, Colorado, United States, and has taught juvenile delinquency for more than two decades. Dr. Covey received his PhD in Sociology from the University of Colorado at Boulder. He has published several articles on a wide array of subjects, including community corrections, disabilities, religion, street gangs, crime measurement, boomtown crime, research methods, determinant sentencing, violent mentally ill offenders, and social gerontology, among other topics. His current research involves substance abuse treatment and dietary practices of enslaved African Americans. He has also served as an academic journal reviewer for *Criminal Justice Review*, *Journal of Contemporary Criminal Justice*, *The Gerontologist*, *Journal of Applied Sociology*, and *Journal of Crime and Justice*. He is also a part-time instructor at the University of Colorado at Boulder. He has encountered many families and youth who have been involved with meth use and manufacture. He has authored or coauthored nine books and numerous academic articles. He is the editor of *The Meth Crisis* (2006), to which he contributed three chapters. He was a coauthor of the text *Youth Gangs* (2006) and sole author of *African American Slave Medicine: Herb and Non-Herbal Treatments* (2007). He coordinated four major symposiums on methamphetamine for judges, caseworkers, public health officers, law enforcement officers, child protection caseworkers, public administrators, ex-users, and other stakeholders.

Ann N. Dapice, PhD, is Director, Education and Research, of T. K. Wolf, Inc., a nonprofit Native American organization in Tulsa, Oklahoma, United States. With a PhD in Psychology, Sociology, and Philosophy from the University of Pennsylvania, she has served as professor and administrator at a number of universities, including the University of Pennsylvania, Widener University, Penn State University, and Goddard College, teaching Social Sciences and Native American Studies. Her cross-cultural research has been reported in professional journals, books, and academic presentations regionally, nationally, and internationally. Speaking engagements include topics related to addiction, stalking, and Native American issues. Dr. Dapice has served on the Elders Council for the American Indian Chamber of Commerce, is a Member of the Board of Directors of the Mental Health Association in Tulsa and the Tulsa Metropolitan Ministry/Domestic Violence Intervention Services Committee.

She consults with the University of Pennsylvania on Native American issues, where she is founder and Chair of the Association of Native Alumni.

Ted Goldberg, PhD, is Professor of Sociology in Stockholm, Sweden. He was awarded an undergraduate degree in Anthropology and Sociology from Uppsala University and a PhD in Sociology from the same university. Combining anthropological methodology with sociological theory, he has done extensive participant observation research on the drug scene in Stockholm. His main field of study is drug consumption and drug policy, and he has written numerous books and articles on these subjects. He has also written about modern Swedish society, the relevance of sociological theory for practical social work, migration, and alternative methods of teaching on the university level. For several decades, Dr. Goldberg has been one of the most outspoken critics of Swedish drug policy. He is currently teaching at the Department of Social Work at Stockholm University, and the Department of Caring Sciences and Sociology at the University of Gävle.

Dirk Hanson, MA, is a freelance science writer and novelist based in Ely, Minnesota, United States. Mr. Hanson has written extensively on technology, science, and medicine. He is the author of the books *The New Alchemists* and *The Incursion*, as well as a third, *Addiction: The Search for a Cure*. He maintains a blog, "Addiction Inbox," devoted to news and issues related to the science of substance abuse. He has also written for *California Magazine*, *Omni*, *CoEvolution Quarterly*, *Willamette Week*, *Whole Mind Newsletter*, and other magazines. He has worked as a business and technology reporter for the *Des Moines Register* and for numerous trade publications.

Mary F. Holley, MD, trained at Baylor College of Medicine in Houston, Texas. She is a licensed physician, formerly an obstetrician-gynecologist in northern Alabama, United States. She is founder and Director of Mothers Against Methamphetamine, a national nonprofit drug education organization. Dr. Holley is author of *Crystal Meth: They Call It Ice*, and has produced several educational videos for drug education and prevention. She serves on the Alabama Attorney General's Methamphetamine Task Force as Chairperson of the Education Committee. Her Web site can be seen at www.mamasite.net.

Olga Inozemtseva, PhD, obtained her PhD in the Behavioral Sciences (area of Neurosciences) at the Neuropsychology and Neurolinguistics Laboratory of the Institute of Neurosciences at the University of Guadalajara in Guadalajara, Jalisco, Mexico. Currently, Dr. Inozemtseva works in the same laboratory and gives courses concerning the structure and development of cognitive processes and executive functions. She directs theses and participates in tutoring committees at undergraduate, master's, and doctoral levels. She is the author of

several publications (articles, book chapters, articles for dissemination). She has participated in projects in the area of infant neuropsychology that focus on development of children with genetic and hormonal alterations. At present, her professional interests are leading to neuropsychology of drug addiction, particularly, the neuropsychological consequences of substance abuse among addicted patients in a phase of abstinence. Dr. Inozemtseva is a member of the National System of Researchers, CONACyT, México.

Sonia Johnson, MSc, MRCPsych, DM, Professor in Social and Community Psychiatry, has studied Social and Political Sciences and Medicine at Cambridge and Oxford Universities, and then did an MSc in Social Psychology at the London School of Economics. She has a Doctorate in Medicine from the University of Oxford. Dr. Johnson did her psychiatric training in South London and was a Clinical Lecturer in Community Psychiatry at the Institute of Psychiatry. Since 1997, she has been a Senior Lecturer and Reader in Social and Community Psychiatry at University College London, in London, England. Since 2003, her clinical work has been in the Camden and Islington Early Intervention Service for young people with psychosis. Since 2001, Dr. Johnson has been course director for the MSc in Psychiatric Research, and has experience as a PhD supervisor. Her research interests include the evaluation of innovative service models, such as crisis resolution teams, crisis houses, and assertive outreach teams; epidemiology and treatment outcomes in early psychosis; women's mental health needs; "dual diagnosis" of psychosis and substance misuse; the development of the European Service Mapping Schedule (ESMS); and staff morale.

Jorge Juárez, PhD, obtained his master's degree in Psychobiology and his PhD in Biomedical Sciences at the Universidad Nacional Autónoma de México. Currently, he is Researcher and Professor at the Institute of Neurosciences at the University of Guadalajara in Guadalajara, Jalisco, Mexico. He has published numerous scientific articles and several chapters in scientific books and coordinated the edition of books. Dr. Juárez has been reviewer of scientific articles in journals of recognized prestige. Since 1988, his main area of research has focused on the neurophysiologic substratum of motivational aspects of behavior, primarily in relation to addictions. Dr. Juárez has imparted courses at the master's and doctorate levels and has directed undergraduate, master's and doctoral theses. He has been Coordinator of the Doctoral Program, Research Coordinator, and Member of the Ethics Committee, and the Editorial Council of the Institute of Neurosciences at the University of Guadalajara. Since 1990, has he belonged to the National System of Researchers, CONACyT, México.

Paula K. Lundberg-Love, PhD, is a Professor of Psychology at the University of Texas Tyler (UTT) in Tyler, Texas, United States, and the Ben R. Fisch

Endowed Professor in Humanitarian Affairs for 2001–2004. Her undergraduate degree was in Chemistry, and she worked as a chemist at a pharmaceutical company for five years prior to earning her Doctorate in Physiological Psychology with an emphasis in Psychopharmacology. After a three-year postdoctoral Fellowship in Nutrition and Behavior in the Department of Preventive Medicine at Washington University School of Medicine in St. Louis, she assumed her academic position at UTT where she teaches classes in psychopharmacology, behavioral neuroscience, physiological psychology, sexual victimization, and family violence. Subsequent to her academic appointment, Dr. Lundberg-Love pursued postgraduate training and is a licensed professional counselor. She is a member of Tyler Counseling and Assessment Center, where she provides therapeutic services for victims of sexual assault, child sexual abuse, and domestic violence. She has conducted a long-term research study on women who were victims of childhood incestuous abuse, constructed a therapeutic program for their recovery, and documented its effectiveness upon their recovery. She is the author of nearly 100 publications and presentations and is co-editor of *Violence and Sexual Abuse at Home: Current Issues in Spousal Battering and Child Maltreatment*. As a result of her training in psychopharmacology and child maltreatment, her expertise has been sought as a consultant on various death penalty appellate cases in the state of Texas.

Mark K. Mathews, LADC, BCCR, is currently pursuing a master's degree in Holistic Health Studies. Mr. Mathews works as a bio-counselor at Health Recovery Center in Minneapolis, Minnesota, United States. He is also the president of Bio-Recovery, a nutritional supplement company specializing in formulas to address addictions and mental illnesses.

Joan Mathews-Larson, PhD, LADC, holds a Doctorate in Nutrition and is the founder and Executive Director of the pioneering Health Recovery Center in Minneapolis, Minnesota, United States, an orthomolecular (psycho-biological) model for treating addiction and emotional disorders. This unique focus combines therapy with physiological intervention to repair biochemical damage that often manifests as impaired mental functioning and behavioral problems. Her work has received national recognition because of the high recovery rates this model produces. The loss of her 17-year-old son to suicide directly after he completed a treatment program fueled Dr. Mathews-Larson's ongoing search for more effective solutions. Her clinic has now successfully treated many thousands of people over three decades. Her published research in the *International Journal of Biosocial and Medical Research* in 1987 and 1991 describes this work. Her approach is outlined in the best-selling book *Seven Weeks to Sobriety* in 1992; followed by *Depression Free, Naturally*, in 2001. Her books have been reprinted in Finnish, Japanese, Icelandic, and Russian. Articles about her work

have appeared in *Psychology Today, Let's Live, Alternative Medicine, Chicago Daily Herald, Harper's Bazaar, Prevention Magazine, Natural Health, Mpls, St. Paul Magazine, Minnesota Physician, Journal of Orthomolecular Medicine, San Francisco Chronicle, LA Times, New Orleans Times, Norway Times, Minneapolis Star Tribune,* and *Townsend Letter for Doctors*.

Scott E. McClure, PhD, is currently employed as a Senior Learning Skills Counselor with the University of California San Diego, Department of Psychiatry, Center for Criminality and Addiction Research, Training, and Application (CCARTA) in San Diego, California, United States. At CCARTA, he provides technology transfer of the most current evidence-based treatment procedures for substance use disorders to substance use treatment staff throughout the state of California. Dr. McClure has more than seven years of experience working with individuals who have co-occurring mental health and substance use issues across a broad scope of populations ranging from the severally mentally ill to university students. He has facilitated groups on post-traumatic stress disorder (seeking safety), adult attention deficit/hyperactivity disorder, dialectical behavior therapy, as well as implemented motivational interviewing and life skills groups while employed at the UCSD Co-occurring Disorder Outpatient Clinic. His clinical and research interests include criminality and addiction, trauma, ADHD, and multicultural issues.

Donald Omonge, BA, is pursuing his post-graduate degree (MA) in Disaster Management from the University of Nairobi in Nairobi, Kenya, and holds a Bachelor of Arts degree in sociology from Egerton University. He has six years of experience working with community-based organizations engaged in community development interventions, research, capacity building, and training. He is currently a training officer with SARAH (Substance Abuse Recovery And HIV/AIDS) Network, a Kenyan faith-based organization sponsored by the U.S. government for community-based HIV/AIDS and substance abuse prevention through behavior change interventions.

Emmanuel S. Onaivi, MSc, PhD, is Associate Professor in the Department of Biology at William Paterson University in Wayne, New Jersey, United States, and a Guest Scientist at U.S. National Institutes of Health. He received his MSc and PhD degrees from the University of Bradford, England. He conducted cannabinoid research as a Postdoctoral Fellow at MCV-VCU. He was an Instructor at LSU-Pennington Biomedical Research and a Visiting Research Scientist at Stanford University, before he was appointed to the faculty of Meharry Medical College, where his laboratory was the first to clone and sequence the mouse CB1 gene. At Vanderbilt University, his team reported racial and gender differences in the expression of CB1 cannabinoid receptors.

His current research is on cannabinoid genomics and proteomics on behavior and changes in anxiety and depression as a basis for addiction. Dr. Onaivi has published several papers, edited four books, including *The Biology of Marijuana from Gene to Behavior* (2002), supervised doctoral and postdoctoral fellows, and his student research was selected and presented on Capitol Hill in Washington, DC. He is a member of the Society for Neuroscience, IACM, and the newsletter editor for the International Drug Abuse Research Society. He received the William Paterson University Award for Excellence in Scholarship.

Meera Vaswani, PhD, earned her PhD degree from Delhi University, a leading university in India. She is a member of the National Drug Dependence Treatment Centre, Department of Psychiatry, All India Institute of Medical Sciences in New Delhi, Delhi, India, a prestigious and internationally recognized institute in India, where she was first a postdoctoral fellow, subsequently joined as a lecturer, and rose to be a professor. During this time, she was elected as Fellow of the Royal Society, London, and Member of the National Academy of Medical Sciences, India. She was selected for a United Nations fellowship, for which she worked in the University of Glasgow in the UK. She was one of the three scientists in the world selected for the Distinguished International Scientist Collaborative Award (DISCA) from the National Institute on Drug Abuse, National Institutes of Health, in 2007. Dr. Vaswani has completed several projects on addiction (alcohol and heroin addiction) and published many papers in both national and international journals. She has been a guest speaker in several national and international forums. She was the only delegate who was invited by Japan to represent India at the Asia Pacific Society of Biological Research in Alcoholism. Subsequently, she was elected to the Board of Directors for the Asia Pacific Society for Alcohol and Addiction Research (APSAAR).

Jace Waguspack, BS, received his BS in Psychology from Texas Christian University in May 2007. He is currently a master's level graduate student at the University of Texas Tyler in Tyler, Texas, United States. He is training in Clinical Psychology with an emphasis on Clinical Neuropsychology, and he intends to follow a PhD program in Neuropsychology upon attaining his master's degree. He hopes thereafter to become a licensed psychologist and assist in the assessment and rehabilitation of individuals that have received traumatic brain injury in a hospital setting. He has performed research in a neuroscience laboratory on the effects of neuro-immunological activation on learning in a mouse model. His current research interests include the study and use of neuropsychological assessment tools in populations with TBI and dementia.

Bethany L. Waits, BA, is a graduate student in Clinical Psychology at the University of Texas Tyler in Tyler, Texas, United States. She anticipates graduation in May 2010 and plans to pursue the licensed professional counselor designation upon completion of her degree. Ultimately, she would like to work as a therapist for a nonprofit community organization, such as a crisis center. In May 2007, she obtained her BA in Psychology from UT Tyler, graduating summa cum laude. Ms. Waits was accepted as a lifetime member of Psi Chi, the national honor society in psychology, and Alpha Chi, a national college honor society. While an undergraduate, she participated in a student panel addressing issues related to sexual assault on campus. Additionally, she has worked with several professors on various research projects.

Mary Theresa Webb, PhD, is founder and past Director of the Global Outreach for Addiction Leadership and Learning (GOAL) Project based in Aliquippa, Pennsylvania, United States. She was one of the founders of the International Substance Abuse and Addiction Coalition (ISAAC), based in the United Kingdom, and the OPORA Training Center in Moscow, Russia. (*Opora* is the Russian word for *support*. Moscow-based nongovernmental OPORA, which works with the Russian government to fight addiction, has trained more than 3,000 persons in addiction and Twelve-Step programs, and implemented 60 recovery groups in 31 cities in Russia.) As a global educator, she has personally and with others facilitated addiction trainings in the United States, Russia, Romania, Tajikistan, Bulgaria, Kenya, Tanzania, Honduras, and Egypt. Currently, Dr. Webb is the Project Director of the Kenya-based, USAIDS-funded Substance Abuse Rehabilitation and HIV/AIDS (SARAH) Network in Kenya. Dr. Webb holds a master's in Education from the University of Pittsburgh and a Doctor of Philosophy Degree in Family Systems/Addiction Intervention from the University of America. She is the author of *Tree of Renewed Life*, *Church Training Manual*, and coauthor of *Codependence in Family Systems*.

Kathryn M. Wells, MD, is Assistant Professor of Pediatrics and Pediatrician at the Kempe Center for the Prevention and Treatment of Child Abuse and Neglect at the University of Colorado Denver in Denver, Colorado, United States. She received her BA in Biology and Psychology with magna cum laude honors from Carroll College in Helena, Montana, and then attended medical school at Creighton University School of Medicine in Omaha, Nebraska. She received her MD in 1993. She received postgraduate training as a Pediatric Resident at Creighton University-Nebraska Universities Health Foundation Joint Pediatric Residency Program in Omaha. Dr. Wells has had fellowship training in Denver, Colorado, at the University of Colorado Health Sciences

Center, the Kempe Children's Center, and the Children's Hospital in the pediatric subspecialty field of Child Abuse and Neglect. She is often called on as an expert witness in child abuse and neglect cases as well as those involving substance abuse. She travels across the country as a medical expert on the effects of substance abuse on users and the children of users, especially methamphetamine. She is also actively involved in the Colorado Alliance for Drug Endangered Children and has made many presentations on the medical effects of methamphetamine use.

Cui-Wei Xie, MD, PhD, is a Professor in the Department of Psychiatry and Biobehavioral Sciences, David Geffen School of Medicine, and in the Semel Institute for Neuroscience and Human Behavior, at the University of California Los Angeles (UCLA), in Los Angeles, California, United States. She earned her doctoral degrees (MD, PhD) in 1984 from Peking University Health Science Center in Beijing, China. Her doctoral thesis work addressed opioid regulation of pain and cardiovascular function and received several awards from the National Ministry of Public Health in China. After receiving her degrees, Dr. Xie taught at Peking University and continued her research there as Associate Director of the Laboratory of Cardiopulmonary Endocrinology. She was named one of the 10 Outstanding Young Scientists by the Beijing Association of Science and Technology in 1986. Dr. Xie came to United States in 1987 as a visiting scholar at National Institutes of Health (NIH). She moved to Duke University two years later where she studied opioid regulation of hippocampal synaptic plasticity. Since joining UCLA faculty in 1995, Dr. Xie has continued her study on opioid receptor signaling and the cellular mechanisms underlying opioid desensitization and tolerance. She is the recipient of multiple research grants from NIH and has served on many review panels for the research grant programs of federal agencies and private foundations.

This page intentionally left blank

The Praeger International Collection on Addictions

Recent Titles in
Abnormal Psychology

Mental Disorders of the New Millennium, Volumes 1–3
Thomas G. Plante, editor

Bleeding to Ease the Pain: Cutting, Self-Injury, and the Adolescent
Lori G. Plante

Understanding and Treating Depression: Ways to Find Hope and Help
Rudy Nydegger

THE PRAEGER INTERNATIONAL COLLECTION ON ADDICTIONS

Volume 3
Characteristics and Treatment Perspectives

Edited by Angela Browne-Miller

Praeger Perspectives
Abnormal Psychology
Thomas G. Plante, Series Editor

PRAEGER

Westport, Connecticut
London

Library of Congress Cataloging-in-Publication Data

The Praeger international collection on addictions / edited by Angela Browne-Miller.
v. cm. — (Abnormal psychology, ISSN 1554–2238)
Includes index.
Contents: v. 1. Faces of addiction, then and now—v. 2. Psychobiological profiles—v. 3. Characteristics and treatment perspectives—v. 4. Behavioral addictions from concept to compulsion.
ISBN 978–0–275–99605–5 (set : alk. paper)—ISBN 978–0–275–99607–9 (v.1 : alk. paper)—ISBN 978–0–275–99609–3 (v.2 : alk. paper)—ISBN 978–0–275–99611–6 (v.3 : alk. paper)—ISBN 978–0–275–99613–0 (v.4 : alk. paper) 1. Substance abuse. 2. Alcoholism. 3. Compulsive behavior. 4. Addicts. I. Browne Miller, Angela, 1952–
HV4998.P72 2009
616.86—dc22 2008051468

British Library Cataloguing in Publication Data is available.

Library of Congress Catalog Card Number: 2008051468
ISBN: 978–0–275–99605–5 (set)
978–0–275–99607–9 (Vol. 1)
978–0–275–99609–3 (Vol. 2)
978–0–275–99611–6 (Vol. 3)
978–0–275–99613–0 (Vol. 4)
ISSN: 1554–2238

First published in 2009

Praeger Publishers, 88 Post Road West, Westport, CT 06881
An imprint of Greenwood Publishing Group, Inc.
www.praeger.com

Printed in the United States of America

The paper used in this book complies with the Permanent Paper Standard issued by the National Information Standards Organization (Z39.48–1984).

10 9 8 7 6 5 4 3 2 1

Contents

PART III: PRACTICES

Series Foreword

Tragically, most people across the globe have either struggled with a health- and relationship-damaging addiction or know someone who has. Addictions, broadly defined, have touched the lives of the majority of people in multiple cultures and locations. For centuries, numerous people have suffered with their addictions to alcohol and drugs as well as with other addictions, with often devastating outcomes. Sadly, important relationships, jobs and careers, and many lives have been lost due to the destructive power of addiction. These tragedies not only occur for those who suffer from addiction, but for their loved ones, coworkers, and community members, and for innocent victims who are perhaps in the wrong place at the wrong time when an addiction-related accident, crime, or violence occurs. The enormous cost of addiction in health care, traffic accidents, crime, violence, loss of workplace productivity, and broken families is too large to quantify. The global spread and success of organizations such as Alcoholics Anonymous (as well as related organizations such as Narcotics Anonymous, Sexoholics Anonymous, and Overeaters Anonymous) is a testament to the numerous people trying to recover from their addictions. Sadly, for every person seeking help for his addiction problem, there are likely to be many more people who never do. Clearly we need help to better understand, evaluate, treat, and cope with those who suffer from addictions.

In this remarkable four-volume set, *The Praeger International Collection on Addictions,* Angela Browne-Miller, PhD, DSW, MPH, has assembled an all-star and diverse team of leading experts from across the globe to provide a state-of-the-art understanding of the various facets of addiction. Each chapter

is written in a manner that is suitable for professionals working in the field as well as educated lay readers and those who either struggle with addictions or live or work with someone who does. What is especially remarkable about the four-volume set is its emphasis on addiction from around the globe, examining multicultural and international issues in addiction, as well as its coverage of so many multifaceted aspects of diverse addictions. For example, it certainly makes sense to cover fully addiction topics such as alcohol abuse and illegal drug use of, say, cocaine and heroin, yet chapters are also offered that examine addictions to caffeine, Internet pornography, work, television, intimate relationship abuse, and shopping. The chapters highlight biological, psychological, social, spiritual, and public health perspectives, with chapter authors who are psychologists, psychiatrists, other physicians, nurses, social workers, counselors, clergy, and other professionals. Dr. Browne-Miller is uniquely qualified to assemble this project as she is someone who has worked in the field of addiction for many years and has training in a unique blend of both the policy and the clinical sides of psychology, social work, education and public health.

The set is complete, state of the art, and highly informative and engaging. There is something for everyone interested in the field of addiction for professional or personal reasons. It is hoped that professionals and lay readers will greatly benefit from this important work and, in doing so, will find a way to improve the lives of those touched by addiction. It is my hope that both research and practice in the field of addiction will be greatly improved thanks to this set. The lives of those who either struggle with addiction or live with those who do will ultimately be improved thanks in part to this critical series. I am grateful to Dr. Browne-Miller and her assembled contributors for providing us all with such important and high-quality volumes that are now available to the public and professional communities. If only one life is saved or improved thanks to this set, it will be a great success in my view; yet I expect that many lives will ultimately be saved or greatly improved thanks to *The Praeger International Collection on Addictions*.

Thomas G. Plante, PhD, ABPP
Santa Clara University and Stanford University School of Medicine,
Praeger Series Editor, Abnormal Psychology

Preface

Angela Browne-Miller, PhD, DSW, MPH

Welcome to *The Praeger International Collection on Addictions,* addressing the insidious, pervasive, worldwide problem of human addiction. Addiction is clearly a global issue, touching every population, every nation, and every age group, people from all walks of life everywhere, directly or indirectly. Indeed, we are talking about an affliction of epic and epidemic proportions. We cannot look away. This is the health of the human species we are talking about.

The World Health Organization (WHO, 2008, p. 1) reports that "psychoactive substance use poses a significant threat to the health, social and economic fabric of families, communities and nations. The extent of worldwide psychoactive substance use is estimated at 2 billion alcohol users, 1.3 billion smokers and 185 million drug users" (p. 1). The WHO has estimated there to be at least 76.3 million persons with alcohol use disorders worldwide, and at least 15.3 million persons with drug use disorders worldwide. Alcohol use and abuse as well as the use and abuse of other psychoactive substances contributes to substantial individual and public health costs. Alcohol is but one substance playing a major role in this global addiction epidemic, but clearly there are many others, despite efforts to prevent new addictions and addicts, and to contain world drug markets (UN, 2008, p. 7).

For example, cocaine shares the stage with other abused drugs. Its prevalence is estimated to be up to 3 percent of the population in developing countries, with severe medical, psychological, social, and economic consequences including, but not limited to, the spread of infectious diseases (e.g., AIDS, hepatitis, and tuberculosis), plus crime, violence, and neonatal drug exposure.

Amphetamine-type stimulant (ATP) abuse is more widespread than cocaine abuse in at least 20 countries. Methamphetamine is presumed to lead in ATP addiction rates, with massive meth epidemics affecting several whole countries and entire regions of others. Social and public health costs of methamphetamine production and use via smoking, sniffing, inhaling, and injecting are staggering and growing in many regions. Additionally, there has been a global increase in the production, transportation, and use of opioids, especially heroin, with worldwide heroin production doubling or even tripling since the mid-1980s. Global estimates are that 13.5 million persons consume opioids, with 9.5 million of these being heroin users who face health risks including hepatitis, HIV, and death. And cocaine, meth, and heroin are just one piece of the picture.

The hotly debated drug cannabis—or the *Cannabis* family of drugs with the euphoric tetrahydrocannabinols, or THCs, including marijuana and hashish preparations—is said to be the most widely abused drug. Research is now suggesting the risk for acute health effects of long-term, chronic cannabis use, including potential impairment of cognitive development, learning, memory, recall, attention, and coordination. (Certainly the presence and extent of long-term effects of casual, of regular and of chronic use are as yet not entirely ascertained.) Both casual use of marijuana and medical use of forms of what is termed medical marijuana (e.g., dronabinol sold as Marinol, the cannabidiols, or CBDs) are subsets of all forms of cannabis use. There are legitimate therapeutic uses of this substance, and these uses make it all the more difficult to regulate marijuana drugs fairly and effectively.

We have here, and in the use of any psychoactive medication for therapeutic purposes, a gray area in which illicit and licit use overlap and can confuse many adult and youth consumers, researchers, and policy makers, among others. In the emergence (or reemergence in history, some will argue) of cannabis as medicine, we have a model for asking which, if any, abused substances may, and perhaps should, be repurposed for medicinal or treatment purposes, and how this is best done against the backdrop of the global addiction epidemic.

Regarding marijuana, we are confronted with the age-at-first-use issue, which suggests that early onset of regular cannabis use may affect not only the academic and social performance of children and teens, but also their future susceptibility to addictions. It was in the 1960s that the hotly debated label "gateway drug" was applied to marijuana, perhaps to scare off its use, and only in the decades since have we understood better what this might actually mean to us. It may not be that marijuana provides the training wheels for drug addiction, but rather that it may serve as an indicator of future use of the same or other drugs. Of course, today, with so many young people having access, and taking advantage of their access, to the whole range of psychoactive substances,

the question of which drug might be a gateway to which other drug dissolves into the fury of the countless addiction conundrums of our constantly changing times.

There is always a new, or rediscovery of an old, addiction on the horizon. There is also always a new (or rediscovered) psychoactive substance for exploratory, research, and perhaps even treatment purposes emerging (or re-emerging). Labeling all of these substances as addictive right out the gate may or may not serve science or even humanity itself. How can we be certain the approach we take will be a constructive one? With new legal (where licensed for development and experimentation) and illegal (where not being utilized under protection of law) so-called designer drugs emerging at a staggering rate, we must admit that we cannot know what is coming, nor whether the new compound will be addictive, or popular, or of medicinal value, or even accessible. We can only imagine what the brave new world of chemistry will continue to bring and whether any benefits can be made available without accompanying risks and detriments.

Moreover, the desire to explore and achieve various altered states of consciousness in religious, spiritual, ritual, and perhaps even treatment settings, is unfolding into debates about rights (Browne-Miller, 1989, pp. 258–260). When there is no demonstrated risk to self or others, we have to ask ourselves whether this right should be protected, especially in circumstances of traditional uses for religious purposes. Again, this dilemma arises against the backdrop of the global and runaway epidemic of substance addiction. How do we balance pressures from opposite directions (freedom protecting the right to use versus control to stop injury and costs of using), when these pressures are not balancing themselves?

Also against the backdrop of global addiction levels, is the massive level of addiction to legal drugs, many of which are heavily marketed to consumers. The legal drug tobacco is said to be the substance causing the most damage globally, with at least one-third of the global population smoking. While smoking rates may be dropping in some countries, the reverse is true globally. As just one of its effects, smoking accounts for some 90 percent of all lung cancer in men and 70 percent of all lung cancer in women. And yet tobacco use is overwhelmingly viewed as being "the single most avoidable cause of disease, disability and death" in the United States (CDC, 2008, p. 2).

And perhaps nothing here has touched so many lives as the regularly consumed, legal drug caffeine, perhaps because coffee drinking is considered so very normal and acceptable, even necessary, in everyday life. However, we must ask whether there is a level of caffeine use that is abuse—or perhaps self-abuse. Surely we do not want to throw caffeine use onto this list of substance abuses

and addictions. Still, a collection on addiction would not be complete without at least touching on this matter, and therefore we do address caffeine herein.

And then there are also the addictions to prescription drugs (such as Vicodin, Percocet, OxyContin, and Darvon), which we find increasing rapidly and already a worldwide phenomenon, with the most commonly abused prescription drugs being opiates. The U.S. National Institute of Mental Health characterizes prescription drug addiction as the second most common illegal use of drugs in the United States, second only to marijuana.

We must also note that unusual, virtually invisible psychoactive substances are working their way into our everyday lives. Household and workplace products contain many volatile substances, exposure to which can be not only damaging, but also intoxicating, and perhaps addicting. Although this domain of substance use and abuse is not specifically addressed herein, we must acknowledge the severe and perhaps largely unmeasured effects of this domain of even routine, legal substance use as well as unintentional and intentional abuse.

So as not to exclude nonsubstance addictions in this overview of addiction today, the fourth volume in this collection on addiction reminds us that work, television, shopping, food (with its particularly difficult-to-call-addiction nature), intimate partner relationship, gambling, Internet, and even pornography addictions make their marks in our lives, either indirectly or directly. These behavioral, nondrug addictions, which occur alone and co-occur with each other, also do co-occur with substance uses, abuses, and addictions. Every human being is in some way affected by the prevalence of behavioral addictions, either directly or indirectly. The study of behavioral addictions teaches us a great deal about addiction itself.

All this suggests the picture of an addiction-prone and largely chemically dependent human species. And this is just the tip of the iceberg. With this truly incomplete laundry list of human fallibilities—or better stated, perhaps, human *vulnerabilities*—this four-volume collection on addiction is truly that: a collection of perspectives, approaches, and findings. Each chapter is a snapshot of the work and thinking taking place in many fields of addiction. Contributors to this collection work with addiction on the various social, philosophical, psychological, spiritual, policy, political, economic, biological, and even cellular levels, all places where this thing we call "addiction" lives. Certainly this collection would have to comprise hundreds of volumes, rather than the four that it does, to address addiction in all its iterations.

Here we give voice to a diverse cross section of perspectives on addiction. This is in no way an exhaustive cross section (of either perspectives or addictions); rather, this collection suggests the diversity of perspectives, theories, practices, and types of addiction in the field—or better stated, *fields*—of addiction. The four volumes of this collection represent the voices of those who have

graciously and even bravely stepped forward from their numerous countries and arenas of work to contribute their ideas, research, and experiences. Certainly there are many others out there, many other aspects of addiction, and many other drugs and objects of addiction not addressed in these volumes.

This work is divided into four volumes, with the first three addressing addictions to substances and the fourth addressing behaviors that show characteristics of addiction. Volume 1, *Faces of Addiction, Then and Now,* offers a sampling of the depth and breadth of addiction today and in the past; volume 2, *Psychobiological Profiles,* surveys some of the interlinked psychological and biological aspects of addiction; volume 3, *Characteristics and Treatment Perspectives,* samples the range of addiction treatment perspectives and approaches; and volume 4, *Behavioral Addictions from Concept to Compulsion,* gives the reader a glimpse of behavioral addictions other than substance addictions.

Readers will observe that the content of these volumes is indeed diverse and in no way represents any one view or theory of addiction. There are many other voices out there who must also be heard, and only in the interest of time and space are we stopping here, at these volumes. The content of these volumes in no way expresses the opinion of this editor, nor of this publisher, regarding what is right, best proven, or even most en vogue in the addiction world; rather, this *International Collection on Addictions* seeks a display of, a sampling of, the diversity of effort to quell the detrimental effects of addiction on individuals, families, communities, societies, economies, and international relations; on ecologies; and in fact, on the human population of planet Earth.

REFERENCES

Browne-Miller, Angela. (2008, reprint of 1989). Synaptic rights. In *Working dazed: Why drugs pervade the workplace and what can be done about it* (pp. 258–260). Houston, Texas: Questia Media. Also available at http://www.questia.com/library/book/working-dazed-why-drugs-pervade-the-workplace-and-what-can-be-done-about-it-by-angela-browne-miller.jsp

Centers for Disease Control (CDC). (2008). *Targeting tobacco use: The nation's leading cause of preventable death* (March 2008 revision). Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

United Nations (UN). (2008). 2008 world drug report. UNODC (United Nations Office on Drug Control), Vienna, Austria: Vienna International Centre, Full report in English available at http://www.unodc.org/documents/wdr/WDR_2008/WDR_2008_eng_web.pdf

World Health Organization, Management of Substance Abuse, Facts and Figures. (2008). "The Global Burden," 2008, (Accessed 9-01-08), p. 1 http://www.who.int/substance_abuse/facts/global_burden/en/index.html. **See Also:** http://www.who.int/substance_abuse/facts/en/index.html

This page intentionally left blank

Introduction to Volume 3: Characteristics and Treatment Perspectives

Angela Browne-Miller, PhD, DSW, MPH

This is volume 3 of *The Praeger International Collection on Addictions: Characteristics and Treatment Perspectives*. How addiction is treated has a great deal to do with how addiction is perceived, explained, and theorized. Addiction treatment philosophies range from spiritual to educational, social, psychological, medical, and to combinations of these. We might also include legal and correctional approaches although these are generally not considered treatment in themselves, and are too frequently anything but treatment. Both the designs and the outcomes of addiction treatment are measured and reported based on underlying philosophies and theories, implicit as well as explicit.

In this volume, we examine samples from the fascinating and diverse range of treatment as per theorized etiology, life course and other perspectives. The intention is not to favor one approach, philosophy, or theoretical stance over another, but rather to see at least some of the many characteristics of the field. Certainly there are viewpoints and approaches not addressed herein, and this is merely a time and space limitation, not one of interest or respect.

From the various vantage points presented herein, the very characteristics of addiction itself can appear different. The material in this volume builds on the psychobiological discussions in volume 2, where addictions were described in various ways, including as public health problems, as self-destructive behaviors, as adaptation gone awry, as frequently co-occurring with trauma and substance abuse, as related to or similar to allergies, as cognitive control conditions, and as neurobiological processes including the desensitization of sensory neurons.

"Part I: Paradigms" opens with a look at "The Social Construction of Drug Policy and Its Impact on Substance Abuse Treatment Philosophies in the

United States," contributed by Sean R. Hogan, PhD, MSW, at the Department of Social Work, California State University Fullerton, California, United States. The social construction of drug users and addicts as deviants and criminals is a relatively new (from a longer historical standpoint) conceptualization of this condition. Hogan calls for policies that can integrate the criminal justice system with the health professions (e.g., medical, psychological, social work, counseling) to begin shifting away from the present-day and quite dominant societal perception of drug users and addicts as criminals. Drug policy reformers call for far more than just this shift; however, this is at least a start. Hogan contends that reforming the current "prohibition-criminalization paradigm" and replacing it with a public health model will be a large job. However, we can work at this incrementally, beginning perhaps with the decriminalization—"depenalization"—of particular drug-related activities such as possession of a small amount of marijuana. This step can reduce law enforcement and prosecution costs, Hogan says, without raising the cost to society of marijuana use.

Next we hear from Lorraine T. Midanik, PhD, at the School of Social Welfare, University of California at Berkeley, Berkeley, California, United States, in chapter 2, "The Philosophy of Denial in Alcohol Studies: Implications for Research." Midanik discusses the machinations of denial in alcohol addiction, directing us to the distinction between denial and other "untruths." Denial is frequently misunderstood to be something that necessitates volition as per standard dictionary definitions. Denial and its definitions have a long history. Psychiatric definitions have referred to various processes that characterize denial, such as defense mechanisms. As early as 1911, T. D. Crothers wrote of "a progressive anesthesia of certain areas of the brain with unconsciousness of the real condition." And E. Morton Jellinek (who wrote the famous book, *Alcohol Addiction and Chronic Alcoholism*, in 1942) described the "rationalization of drinking behavior" and accompanying "well-known alcoholic 'alibis'." Midanik takes us on a journey through the social construction of denial—or at least of definitions of denial, and also of the politics of denial, reporting that, "we do live in a world where *denial* is an important mainstay in the alcohol field. It has provided a central ideology for the disease model." Midanik questions "the assumption that *denial* needs to be the central core of alcohol treatment."

In chapter 3, we have "The Spirituality of Recovery and the Twelve Steps of Alcoholics Anonymous," by Rev. Robert Davis Hughes, III, PhD, of the School of Theology, University of the South (Sewanee), in Sewanee, Tennessee, United States. The *Twelve Steps of Alcoholics Anonymous* was first written by "Bill W." (Wilson) in 1938 and was the next year published in what has come to be called "The Big Book." The Twelve Steps have become what Hughes describes as "the dominant gold standard in defining the spirituality of recovery." Hughes

notes that while the "recovery community" has accepted much of the new neuropsychological knowledge of addiction, it remains to be seen whether this community can expand to fully include "the broad range of other faiths and communities of spiritual practice, including new religions and persons of little or no faith, as the founders intended." This would "define a program of recovery that is 'spiritual but not religious'" and that would serve a broader population.

Consideration of denial and of spirituality in recovery from addiction takes us to the quite central if not always visible question of free will, in chapter 4, "Addictions and Free Will: A Structured Exploration of the Boundaries of Personal and Societal Responsibility," by Julian G. Martins, MA, MB, BS, of the Academy of Nutritional Medicine, Cambridge, England, and Basant K. Puri, MA, PhD, MB, BChir, MRCPsych, MMath, of the Medical Research Council (MRC) Clinical Sciences Centre (CSC), Hammersmith Hospital and Imperial College, London, England. When addiction is described as a "moral condition," the addicted person is said to be exercising "free choice" when choosing to behave in a certain way. Martins and Puri explain, "Although this position has the advantage of promoting a strong expectation of personal responsibility for behavior, it carries with it the risk of an excessive retributive response to addictive problems. . . . The potential advantage of addiction being seen as a chronic, relapsing brain disorder is that it is able to reduce the stigma associated with these problems, allow earlier access to more effective treatment programs, and avoid the overuse of ineffective and costly prison sentences. . . . In fact, not only is prison ineffective at tackling drug problems, it is also a high-risk environment for drug initiation." Martins and Puri note something taking place in some prison settings, "the neurosurgical ablation of the nucleus accumbens to treat addiction on subjects in penal settings in Russia and China where informed consent is doubtful." (Note that the nucleus accumbens is, in essence, a collection of neurons within the forebrain said to play a central role in the reward circuit, operating on dopamine and serotonin, promoting desire, and satiety and inhibition, respectively.)

Looking past, or perhaps deeper into, free will, we see the potential of religiosity to ward off addictions. Patrick McNamara, PhD, of the Boston Veterans Administration (VA) Healthcare System and the Boston University School of Medicine, Department of Neurology, Boston, Massachusetts, United States; and James Burns, PhD, of The Danielsen Institute at Boston University, Boston, Massachusetts, United States, share their perspectives on "Religiosity as Protective against Addictions in Adolescents" in chapter 5. McNamara and Burns underscore the "protective effects of religion against addictions in adolescents," discussing the potential mechanisms for this "protective effect," noting that among these "are the [very important] interactions between religious

beliefs/practices and the developing brain." McNamara and Burns suggest that, "religiosity and executive control processes such as impulse control and the planning and generation of implementation intentions mutually interact during development in such a way as to yield effective protection against addiction and risky behaviors in children and adolescents." While this chapter speaks largely from a prevention standpoint, per the "protective effort" described herein, we must ask, What might such an interaction between religiosity and the mind's own executive control processes play in addiction treatment itself? If this interaction works before an addiction is fully formed, can it also be important once the addiction is in place?

Part I of this volume is concluded in chapter 6 with an "Analysis of Intervention, Treatment, Policy, and Law That Contradict Ethical Criteria," contributed by Ann N. Dapice, PhD, of T. K. Wolf, Inc., a Native American–focused addiction treatment program in Tulsa, Oklahoma, United States. Dapice reviews the characteristics of truly moral decision making. Among these characteristics are: have no personal gain or interest; obtain as much information as possible about the problem; try to understand the consequences; react in the same manner to the same actions; and do not be sick or overly tired or stressed when making moral decisions. Dapice tells us that the tenets of moral decision making should inform and guide addiction intervention, treatment, law, and policy. But do they? Not entirely, as Dapice reminds us: "Nicotine [for example] remains in first place as a cause of preventable death . . . yet use of nicotine has often been condoned and encouraged through reward systems in mental health and drug rehabilitation facilities, other addiction settings, and prisons. Recently, U.S. federal funding has required that smoking not be allowed in federally funded mental health, addiction facilities, or prisons. Yet well-intentioned counselors can still be heard to say, 'We can't take their last drug away.'"

We then turn to Part II of this volume, "Models," which opens with consideration of "Family Issues in the Field of Addiction Treatment" in chapter 7, written by Giuseppe Carrà, MD, MSc, PhD, of the Department of Mental Health Sciences, Royal Free and University College Medical School, London, England; Massimo Clerici, MD, PhD, of the Department of Neurosciences and Biomedical Technologies, University of Milan-Bicocca, Milan, Italy; Hamid Ghodse, MD, PhD, DSc, of the International Centre for Drug Policy, St. George's Hospital Medical School, University of London, England; Flavio Poldrugo, MD, PhD, of the Office for Research and Innovative Projects on Alcohol, Other Addictions and Mental Health, Departments of Psychiatry and Addiction, University of Trieste, Italy; and, Nady el-Guebaly, MD, of the Department of Psychiatry, University of Calgary, Calgary, Canada. Carrà,

Clerici, Ghodse, Poldrugo, and el-Guebaly have graced this collection with their insights into the great complexity of family member roles in substance abuse treatment against the backdrop of essential cultural sensitivity. Among the findings are: confrontational approaches may or may not work within particular cultural contexts; families may split into rescuing and rejecting factions; and family systems approaches can be cost-effective when conducted with cultural sensitivity. The authors of this chapter note that "People with schizophrenia from families that express high levels of criticism, hostility and emotional over-involvement, according to the Expressed Emotion (EE) Index, have more frequent relapses than people with similar problems from families that tend to be less expressive."

In chapter 8, Mary Theresa Webb, PhD, of the Global Outreach for Addiction Leadership and Learning (GOAL) Project based in Aliquippa, Pennsylvania, United States, and of the International Substance Abuse and Addiction Coalition (ISAAC) based in the United Kingdom, also of the OPORA Training Center in Moscow, Russia; and, Ann Fabean, PhD, MA, of Greenburg, Pennsylvania, United States, retired from private practice, write on "Codependence from a Family Systems Perspective." Webb and Fabean note that, 30 years ago, the addiction field and the family systems field were just beginning to explore the effect of addiction on family systems. Delving into complex matters such as codependence, counterdependence, family emotional affect, and family roles can illuminate this aspect of co- and counter-addiction. Webb and Fabean emphasize, "Codependent family members frequently exhibit symptoms of the family disease for a longer period of time than the addict does. The addict can become abstinent and receive individual and group support and treatment and be *in recovery*. Family members who don't understand that their reactive patterns or maladaptive behaviors need to be healed are not *in recovery*. They need as much support and treatment—if not more in many cases—than the addict." Unfortunately, as the authors find, "in today's Western controlled-care climate, which allows only short outpatient treatment, family member treatment often is ignored. Even in Eastern and/or developing countries where therapeutic modalities of treatment exist, little is known or practiced for family recovery. In African countries, women who live with alcoholic or HIV-infected males need special culturally sensitive care and support. Many family members who live or have lived with active addicts and who still suffer from depression or posttraumatic stress deserve the same attention and care as the addict."

Next, in chapter 9, we study "Best Practices for Dual Diagnosis Treatment and Program Development: Co-occurring Mental Illness and Substance Disorders in Various Combinations," contributed by Kathleen Sciacca, MA,

of Sciacca Comprehensive Service Development for Mental Illness, Drug Addiction and Alcoholism, MIDAA, New York City, New York, United States. Sciacca directs our attention to dual diagnosis concerns and asks what the best practices are for addressing these issues. Detailing best practices, client readiness to change, and other factors, Sciacca presents the wisdom of her many years as a consultant to the design and implementation of dual diagnosis treatment and integrated services. Although the field of dual diagnosis has expanded markedly in recent decades, there remains a great need for expansion of the knowledge—and utilization of the knowledge—in this area. Given that some "40 to 60 percent of individuals presenting in mental health settings have a co-occurring substance abuse diagnosis, and 60 to 80 percent of individuals presenting in a substance abuse facility have a co-occurring mental health disorder . . . [and] at least 50 percent of the 1.5 to 2 million Americans with severe mental illness abuse illicit drugs or alcohol," it is not only humane to attend further to dual diagnosis issues, but it is also cost-effective to do so.

Then, in chapter 10, Ray B. Smith, PhD, private consultant based in Forestville, Maryland, United States, at the time of this writing with a CES research project in a field army hospital in Iraq, contributes "Cranial Electrotherapy Stimulation in the Treatment of Addictions." Here Smith reports on what evolved out of his earlier studies funded by a U.S. National Institute of Mental Health (NIMH) grant to work at the District of Columbia's (Washington, DC, United States) 600-bed Rehabilitation Center for Alcoholics. Smith concludes that cranial electrotherapy stimulation (CES, also known as "electrosleep") has been shown to, more effectively than pharmaceutical interventions, address "drug abstinence syndrome" in the treatment of addiction. CES is basically "a small electrical stimulation pulsed across the head for up to one hour a day for three weeks or less for FDA-accepted treatment claims of depression, anxiety, and insomnia. All these are symptoms of persons recovering from addictions, the drug abstinence syndrome." Smith points out that CES has been used outside the United States longer and more widely, and that perhaps the confusion of CES with electric shock therapy has dampened interest in the United States.

From here, Maria Dinis, PhD, DSW, at the Division of Social Work, California State University Sacramento, Sacramento, California, United States, shares "An Analysis of Service Providers' Treatment Model Choices for Cocaine-Using Clients: A Case Study," in chapter 11. Dinis inquires as to how treatment providers select their treatment approaches. She finds that educational background, professional and experiential training, work setting, personal belief system, age, and gender all affect the professional's selection of treatment to be applied. Dinis concludes that "It is, therefore, important to understand the relation between the background characteristics of treatment providers and

their treatment choices." Focusing on the choice of cocaine treatment models to exemplify this, Dinis finds additionally that providers' selection of treatment models is also complicated in light of their interaction with clients' beliefs about treatment models and other client characteristics that cause a model to work for one client and not for the next.

Van L. King, MD, Michael Kidorf, PhD, Kenneth B. Stoller, MD, Karin J. Neufeld, MD, MPH, and Jessica Peirce, PhD, all at Johns Hopkins University School of Medicine, Baltimore, Maryland, United States; and Robert K. Brooner, PhD, at the Johns Hopkins University Department of Psychiatry and Behavioral Sciences, also in Baltimore, Maryland, United States, contribute chapter 12, "Translating Efficacy to Effectiveness in Opioid Dependence Treatment." Here the focus is on opioid dependence in the forms of both heroin and the increasingly popular opioid of abuse, prescription opioid (oxycodone). The authors report that "Opioid dependence is a severe, persistent disorder often requiring long-term and at times intensive psychiatric treatment." The Motivated Stepped Care (MSC) treatment model is discussed, as it integrates medication, psychotherapy, and behavioral reinforcement into a comprehensive model of treatment. MSC is advantageous both clinically and as per its cost-effectiveness via its "matching the intensity of service to the severity of the problem in a dynamic fashion over time using behavioral reinforcement to motivate counseling adherence and treatment plan progress. In this way, only as much treatment as needed to improve and stabilize functioning is prescribed, and then intensity of care is reduced to minimize intrusiveness and cost." A "wide spectrum of pro-rehabilitation behavior change" is provided by this MSC treatment model.

Here, we pause to consider alternate aspects of rehabilitation and harm reduction, in chapter 13, "Shamanistic Harm-Reduction Practices," contributed by Michael Winkelman, MPH, PhD, at Arizona State University, in Tempe, Arizona, United States. Winkelman applies an evolutionary psychology approach to understanding similarities in drug and non-drug methods for altering consciousness, which is termed the "shamanic paradigm." Identifying the biological similarity between altered states of consciousness (ASCs) and drug-induced states suggests the potential for what may be called "shamanic practices" (based to some extent on traditional indigenous shamanic practices) for treating addiction. Winkelman contends that, "The shamanic approach supports the harm-reduction principles of alternatives to abstinence with natural and nonaddictive ways of meeting human needs to experience transcendent states of consciousness." Winkelman's approach is one of harm reduction, plus self-realization of goals and alternate futures, and then behaviors to reach these futures. Winkelman suggests that assisting the client to recognize the "spiritual dimension" can help the client to access the personal resources to make sig-

nificant behavior changes, including in drugging and drinking behaviors and addictions.

From here, we move into Part III of this volume, "Practices," turning first to chapter 14, "Therapeutic Approaches, Beliefs, and Perceptions of Providers When Treating Alcohol- or Drug-Using Clients: Selecting Best Practices," submitted by Maria Dinis, PhD, MSW, at Division of Social Work, California State University, Sacramento, California, United States; Joseph R. Merighi, PhD, MSW, at Boston University School of Social Work, Boston, Massachusetts, United States; and Kelly Anders, MAL, at the University of California Davis in Davis, California, United States. Dinis, Merighi and Anders warn that without programs to provide best practice approaches, these new models are virtually irrelevant. "The treatment-client community as a whole may not adequately benefit from the evidence-based knowledge professionals are currently developing or the gains made in substance abuse treatment strategies, including medications, until the treatment infrastructure is supported and the public demands quality of treatment care in the same way as they recognized that alcohol/drug abuse was associated with public health and safety problems." Again, we see how critical understanding of the public health perspective is to treating addiction. Funding must support public health approaches in programs that require "vital resources to maintain and utilize evidence-based practices in the treatment of addictions."

Chapter 15, "Brief Telephone-Based Interventions for Clients with Alcohol and Other Drug Abuse Problems," is written by Thomas Beers, PhD, of the California Screening, Brief Intervention, Referral and Treatment program (CASBIRT) in San Diego, California, United States; Melinda Hohman, PhD, of School of Social Work, San Diego State University, San Diego, California, United States; Katharine Slack, MSW, at CASBIRT and the California State University School of Social Work, Center for Alcohol and Drug Studies and Services in San Diego, California, United States; Daniela Sisneros, MSW, also at the Center for Alcohol and Drug Studies and Services in San Diego, California; and Susan I. Woodruff, PhD, at the San Diego State University School of Social Work, in San Diego, California. The authors explain that "Alcohol and other drug (AOD) abuse and dependency continue to be major problems . . . as they contribute to elevated health risks related to cardiovascular disease, stroke, pancreatitis, cirrhosis, and various types of cancer . . . [and associated problems such as] violence, family dysfunction, poverty, poor work performance, and criminal behavior." The authors suggest and report on the "CASBIRT Brief Treatment Program," which offers "short-term, harm-reduction treatment services for high-risk AOD abusers who typically would not be treated by community-based services." This innovative and cost-effective

treatment model utilizes motivational interviewing principles and cognitive behavioral approaches, with the advantage that this model minimizes barriers to program access with "no-cost services, bilingual counselors, flexible scheduling, and phone or in-person sessions." The findings of the authors' study of this treatment model indicate that telephone counseling can be "a viable treatment alternative to traditional face-to-face counseling" and that telephone service delivery is having "a number of positive effects and effects similar to in-person counseling," with "positive changes in AOD behavior" being suggested.

Ramadugu Shashikumar, MD, a consulting psychiatrist in Jallandhar, Punjab, India, shares his insights in chapter 16, "Screening and Brief Intervention for Alcoholics." "Screening is the procedure that identifies a disease or disorder in its early or presymptomatic stages and provides efficient identification of those users of alcohol who need a more comprehensive diagnostic evaluation and treatment planning." The author also notes that "in view of its easy availability, low cost, and greater sensitivity (which is an essential characteristic in screening tests and also a tool to monitor abstinence states), GGT [gamma glutamyl transferase] is an ideal screening biological marker."

Chapter 17, "Service Profiles: Provider Choices for Treatment of Alcohol- and Cocaine-Using Clients," is contributed by Maria Dinis, PhD, MSW, at California State University, Sacramento, Division of Social Work, and the Alcohol Research Group, Berkeley, California, United States; Lyndsay Ammon, MPH, at the University of California Berkeley, School of Public Health, and the Alcohol Research Group, Public Health Institute, Berkeley, California, United States; and Kelly Anders, MAL, at the University of California Davis, in Davis, California, United States. The authors conduct an important study regarding treatment provider choices to apply either one or a combination of services. "For instance, does pregnancy status play a role in treatment selection; and does the choice vary depending on the provider's own work setting and demographic characteristics? If so, what are the actual treatment choices being made? For example, professionals working in a hospital setting may tend to recommend only inpatient hospital treatment for their clients rather than referring them either to halfway houses, which cost a fraction (13%–17%) of inpatient care and about 50% of outpatient hospital-based treatment, or to no-cost self-help groups. . . . Conversely, those working in alcohol/drug treatment programs may recommend a broader range of services that they believe would result in better treatment outcomes for some clients, such as pregnant women."

Next, Jana Cook, MD, PhD, Scott E. McClure, PhD, Igor Koutsenok, MD, MS, and Scot Lord, CATC, FACT, all at the School of Medicine, Department of Psychiatry, Center for Criminality and Addiction Research, Training and Application (CCARTA) the University of California, San Diego (UCSD),

San Diego, United States, contribute chapter 18, "An Innovative Approach to Prison-Based Substance Use Treatment: The Implementation of Inmate Peer Mentor Programs." The authors note that, "The United States currently maintains the largest prison population in the world. Over the past 30 years there has been a continuous increase in the number of inmates in U.S. state and federal prisons. . . . The relationship between substance use and criminal incarceration is an undeniable factor in the nation's prison crisis." Other approaches to the criminalization of substance use—and to inmates' substance addictions—are essential and have emerged. The authors share the story of an important approach, that of the Therapeutic Community (TC). Cook, McClure, Koutsenok, and Lord explain: "In October 2006, the California Men's Colony (CMC) prison in San Luis Obispo, faced with staff recruitment and retention difficulties, decided to utilize long-term sentenced inmates as peer mentors to lead their prison-based TC program. Prior to this decision, several attempts to utilize contracted treatment providers from within the state as well as one out-of-state treatment provider had failed. This protracted struggle inspired the correctional counselor . . . to use the human resources available, inmates with long prison terms." There was a large pool of participants in need of treatment services and a valuable resource in the peer-mentor inmates with long-term sentences who had had previous experience in TC treatment. In fact, "Some of these participants had a history of active involvement in prison-based TC treatment that stretched back over 15 years, and their experience with the TC model was unquestionable." It became clear that peer mentors can "provide substance abuse treatment under close correctional and clinical supervision." In fact, the CMC Substance Abuse Treatment Program "Our House" became the first custody-based TC program within CDCR entirely run by peer mentors, independent from any outside treatment providers.

Chapter 19, "Advances in Corrections-Based Treatment: Building the Addiction Treatment Workforce," submitted by Igor Koutsenok, MD, MS, and Scott E. McClure, PhD, (who both are, as noted previously, at CCARTA and the University of California San Diego, California, United States). Koutsenok and McClure report that, "Cross-trainings are unique to correctional addictions treatment environments and have emerged to help foster productive relationships between treatment and correctional staff. Cross-trainings aim to help both staffs develop an enhanced understanding of their role in offender treatment." They go on to write that "trainings need to follow scientifically driven principles, be facilitated by well-trained and educated professional training staff, and be reinforced through clinical supervision, organizational acceptance, follow-up trainings, and implementation analysis. The ability to successfully improve the addiction treatment workforce is highly dependent on these fac-

tors." And the authors tell us how very important it is that we "bring science to front-line practice." We can learn valuable lessons regarding what makes treatment effective from this area of front line practice.

From here we shift gears to see another face of addiction treatment, in chapter 20, "Sacred Medicines for Harm Reduction and Substance Abuse Rehabilitation," by Michael Winkelman, MPH, PhD, who is, as noted earlier, of Arizona State University in Tempe, Arizona, United States. Winkelman reports that "the concept of sacred medicines refers to substances also known as psychedelics, hallucinogens, entheogens, and psychointegrators" and that "humans have used plants as sacred medicines for thousands of years for their vision-inducing properties, their ability to shift awareness and consciousness to a spiritual domain, and their ability to induce healing." Winkelman makes the case for including these substances in research and treatment, although they have been prohibited since at least the 1970s. Winkelman reports that "there has emerged significant evidence that these substances have important applications in the treatment of a variety of conditions, including addictions." Winkelman contends that given the "limited efficacy of current treatments" for drug addiction, the use of such "medicinal" substances for treatment of drug dependence is an "ethical responsibility of the medical field."

And last is chapter 21, "Entheogen-Enhanced Transpersonal Psychotherapy of Addictions: Focus on Clinical Applications of Ketamine for Treating Alcoholism." The authors are Eli Kolp, MD, in private practice, Tampa Bay, Florida, USA; Evgeny Krupitsky, MD, PhD, DMedSci, of the St. Petersburg State Pavlov Medical University, also of the Bekhterev Research Psychoneurological Institute, and also of the Health Care Committee of the Government of the Leningrad Region, Russia; Harris Friedman, PhD, Department of Psychology, University of Florida, Gainesville, Florida, United States; and M. Scott Young, PhD, at the University of South Florida, in Tampa, Florida, United States. Kolp, Krupitsky, Friedman, and Young present the results of studies researching the treatment of alcoholism, heroin addiction, and other problems such as treatment-resistant depression with ketamine. "Undeniably, alcoholism is an extremely destructive problem that resists most conventional treatment approaches. . . . Ketamine hydrochloride (ketamine) is a prescription medication widely used for general anesthesia. It was first synthesized in 1962 and later patented by Parke-Davis in 1966 as a human anesthetic. During the Vietnam War, ketamine was the most widely used battlefield anesthetic. The U.S. Food and Drug Administration (FDA) approved ketamine anesthesia for use with all ages (children, adults, and the elderly) in 1970. Since then, ketamine has been adopted by many hospitals and medical offices due to its rapid onset, proven safety, and short-duration of action." More than 7,000 publications describe its

"high effectiveness and safety" and "numerous clinical studies have detected no long-term psychological impairment following ketamine use."

This then is volume 3, *Characteristics and Treatment Perspectives,* of *The Praeger International Collection on Addictions*. This collection of essays reveals a shifting field, a brave new field of addiction treatment, expanding in several directions, building on an extensive knowledge base, years of research, and years of debates. The range of possibilities on the addiction treatment horizon is vast, exciting and perhaps unlimited. We look to the future of addiction treatment based on the work of the authors of this volume and their colleagues around the world.

Part I

PARADIGMS

This page intentionally left blank

Chapter 1

The Social Construction of Drug Policy and Its Impact on Substance Abuse Treatment Philosophies in the United States

Sean R. Hogan, PhD, MSW

> *Our grievous error was in allowing the narcotics addict to be pushed out of society and relegated to the criminal community.*
>
> —Rufus King (1953)

Historically, American society and its political representatives have interpreted illegal drug use from a moral perspective requiring some type of punitive response. From this perspective, drug use and addiction are seen as the self-inflicted consequence of characterologically weak, spiritually bankrupt, and socially deviant individuals whose behavior warrants stringent antidrug legislation and criminal justice intervention. This prohibition-criminalization paradigm (Rouse & Johnson, 1991) represents a consensus among the American people that illicit drug use is an immoral behavior and that drug users should be subject to arrest, prosecution, and incarceration. The rehabilitation and treatment of drug users and addicts are secondary goals to the elimination of an illegal drug-using presence in society (Jensen & Gerber, 1998).

Most of the discourse associated with the criminalization of addiction in U.S. society has focused on the negative social, legal, and economic consequences of illegal drug use. Outcomes, such as the creation of organized criminal networks and markets, the violence associated with drug manufacturing and trade, and the subsequent legal, health, and economic costs to U.S. society have become anathemas among advocates for drug policy reform (MacCoun & Reuter, 2001; Mosher & Atkins, 2007; Nadelmann, 1991). The role that criminalizing addiction has played in the subsequent substance abuse treatment philosophies

for drug addicts has not attracted as much attention. In this chapter, the early social and political foundations of America's response to drug use as a social problem are presented. Then, the implications of this response in the substance abuse treatment of persons who are addicted to drugs are examined. Last, some policy alternatives to America's current reaction toward illicit drug use are suggested. The intention of this chapter is to provide a clearer understanding as to why the United States embraces prohibitionist and punitive drug policy ideals and how these ideals directly affect the clinical treatment of drug addicts. In this chapter, all mentions of drug use refer to illicit drug use.

EARLY SOCIAL CONTEXTS

Interestingly, in the United States, drug policies are often constructed based on the characteristics of the people that are using the drugs rather than the harmful properties of the drugs themselves (Robinson & Scherlen, 2007). Prior to 1875, the United States had no antidrug policies or practices (Courtwright, 1982). The use of drugs was not considered particularly detrimental to society or its members. Individuals who became addicted to drugs were generally placed in the care of a physician and labeled as "unfortunate" (Booth, 1996). These individuals were regarded with compassion rather than with some type of social stigma or disapproval. Drugs and drug use had yet to fall subject to any serious moral distinction. As a result, substance use during the early history of the United States was not regulated or controlled. The true consequences of substance abuse had yet to be fully appreciated or comprehended by early Americans. While alcohol had been a part of U.S. society since the country's inception, drug use was just beginning to flourish during the nineteenth century (Courtwright, 1982).

The first drug to attain social prominence in the United States, other than alcohol, was morphine. Morphine sulfate, an opium alkaloid, had been discovered in 1804 by a German chemist named Friedrich Serturner (Lindesmith, 1957). The effectiveness of morphine as an analgesic and narcotic was quickly recognized by the medical and patent medicine establishments of the time. Without any antidrug regulation or legislation, patent medicines were readily and legally available to any individual. Patent medicines, or proprietary medicines as they were sometimes called, were popular elixirs sold by businesses and entrepreneurs as tonics or cure-alls (Courtwright, 1982). Opiate preparations, such as Mrs. Winslow's Soothing Syrup and McMunn's Elixir of Opium, as well as laudanum and paregoric, could be purchased at any local drug store or in any mail-order catalog (Lindesmith, 1957). These preparations were also very inexpensive. The combination of low price and high availability made

morphine and other opiate use during the last half of the nineteenth century fairly prevalent. Many early Americans were opiate consumers (Musto, 1999).

According to Courtwright (1982), by far, the greatest use of morphine in nineteenth-century America was by white, middle- and upper-class women. Women of this period used morphine as a panacea for many of their physical and emotional problems. Physicians prescribed morphine for everything from a toothache to menstrual cramps to melancholia (depression) (Aldrich, 1988). If a woman went to see a doctor in the late 1800s, chances are she would leave with a prescription for morphine. Another population that contributed to the high prevalence of morphine use during this period was Civil War veterans. Many soldiers, having incurred chronic or extensive injuries during the war, required prolonged morphine treatment. Following the end of the war, these soldiers were often maintained on morphine by their local physicians. The administration of morphine by physicians is regarded by some historians as the single greatest cause of opiate addiction in the United States during the nineteenth century (Courtwright, 1982; Durrant & Thakker, 2003).

So-called infant doping was another widespread practice of this period (Aldrich, 1988). Sedating children and babies with morphine mixtures was a common method for controlling loud, crying, and unruly children (Musto, 1999). Screaming babies were often given laudanum medicines as a calming agent (Aldrich, 1988). This practice led to early infant mortality studies in England, which resulted in that country's first opiate prohibition law, the Pharmacy Act of 1868. This law banned laudanum use unless it was prescribed by a physician (Berridge & Edwards, 1987). In the United States, the first legislation addressing the use of morphine and patent medicines came in 1906 with the passage of the Pure Food and Drug Act. This act was intended to curtail the use of adulterants in foods and, particularly, patent medicines, by requiring manufacturers to list a product's ingredients on its label. It did not, however, make patent medicines, morphine, or any other drug illegal.

One reason for the naïveté associated with substance abuse during this period was simply a lack of information and limited understanding on the parts of the medical establishment and U.S. society concerning the consequences of drug use and addiction. For example, early physicians thought morphine could be used as a cure for opium addiction. Ironically, heroin, developed commercially in 1898, was then thought to be a cure for morphine addiction. Cocaine, with its stimulating effects, was first thought to be a cure for alcoholism (Musto, 1999). With such a lack of knowledge surrounding drugs, insightful medical and policymaking decisions were virtually impossible. Fortunately, over time, the medical establishment's understanding of drug use and addiction would

become more advanced; however, America's social and political knowledge of this issue would progress much more slowly.

A significant shift in public perception and, consequently, the strategies designed to address substance use occurred when American society began to associate drug use with certain minority and ethnic groups (Courtwright, 1982; Musto, 1999). At the turn of the twentieth century there was a strong association between opium use and Chinese immigrants, and between cocaine use and African American males from the southern United States (Helmer, 1975; Musto, 1999). Racist and xenophobic sentiments, long a part of American history, contributed to an emerging social consciousness against drug users and subsequent campaigns to construct antidrug legislation. When drug use was associated with mainstream drug users and the dominant culture, it was not considered a serious social problem. However, once attention concerning substance use was directed toward less socially desirable members of society (e.g., immigrants and minorities), the American public's perception and reaction to drug use changed (Musto, 1999). Drug use and addiction were no longer seen as some type of personal misfortune. Instead, these phenomena were interpreted in more pejorative terms, as the self-indulgent and immoral behavior of a nonnativist lower class. This negative association of substance use with ethnic minorities was an important social factor in motivating a political response toward early antidrug legislation in the United States (Helmer, 1975; Lindesmith, 1957). It was in this context that the U.S. government passed its first comprehensive federal antidrug policy—the Harrison Narcotic Act of 1914.

THE HARRISON NARCOTIC ACT

The Harrison Narcotic Act, named after its author, Representative Francis Burton Harrison, was the final product of many years of legislative and political mediation among drug policy advocates, international drug control representatives, and powerful pharmaceutical and medical interests of the period (Musto, 1999). Adamant drug policy reformers and prohibitionists, such as Hamilton Wright, invested a great deal of personal energy in seeing the Harrison Act to fruition. Previous attempts and efforts to pass some type of comprehensive antinarcotic legislation, including an earlier and stricter version of the Harrison Act, the Foster Bill of 1910, had met with opposition and defeat (Musto, 1999). Too many interest groups simply wanted too many consolations and contingencies attached to an antinarcotic bill to pass such a measure. Finally, in June 1913, the chairman of the National Drug Trade Conference, a powerful drug lobby of the time, signed a draft of the proposed bill, which was then intro-

duced into the first session of the 63rd Congress by Representative Harrison (Musto, 1999). The bill would eventually pass and become the first landmark antidrug policy in U.S. history.

Within the Harrison Narcotic Act, there were two sections that would prove the most significant for controlling drug use in the United States. The first section stated that anyone engaged in the production or distribution of narcotics (i.e., opiates or cocaine; antimarijuana legislation would come later) must register with the federal government and keep records of all transactions with drugs. This section also mandated that all parties handling drugs, through either purchase or sales, must pay a tax. The second section of the act stated that unregistered persons could only purchase drugs by a prescription from a physician and that such a prescription must be for legitimate medical use (Belenko, 2000). The Harrison Narcotic Act was ostensibly a revenue measure that required persons who prescribed or distributed specified drugs to register and buy tax stamps. It also provided that possession of narcotics by any individual that had failed to register as a narcotics retailer or practicing physician was unlawful unless prescribed by a physician in good faith. The reason that this act was legislated within the context of a revenue measure, rather than a penal code, had to do with issues surrounding constitutionality and the powers of the federal government versus those of the state to enforce police powers on individuals (Pittman & Staudenmeier, 1994).

While the Harrison Narcotic Act was originally legislated as a means of raising revenues and regulating over-the-counter sales and medical prescriptions of opiates and cocaine (McWilliams, 1992), its enforcement by the U.S. Treasury Department's Narcotics Division quickly devolved into an exercise in drug prohibition and punishment. In their interpretation of the second section of the act, officials from the Narcotics Division deemed the prescription of drugs to drug addicts by physicians unlawful. As a result, agents from the U.S. Treasury Department began arresting physicians who supplied patients with opiates and cocaine for the purpose of treating an existing drug addiction (Musto, 1999). At this point in American history, this type of government intervention into the private lives of citizens was viewed as highly unconstitutional. This prompted litigation by arrestees of the Harrison Narcotic Act, challenging its propriety. Two crucial Supreme Court decisions in 1919 (*United States v. Doremus* and *Webb et al. v. United States*) upheld the constitutionality of the tax and denied maintenance supplies for addicts with no intention of ceasing drug use (Belenko, 2000). A 1922 case (*United States v. Behrman*) effectively ruled that physicians could not supply drugs to addicts for any reason (Dickson, 1968). According to Lindesmith (1965), "The Supreme Court decisions up to 1922 made it impossible for doctors to treat addicts in any way

acceptable to law enforcement officials" (p. 7). In 1925, the decisions of these cases were ultimately reversed (*Linder v. United States*); however, their effects on the medical establishment and its treatment of drug addicts were profound. In the two decades following passage of the Harrison Narcotic Act, "an estimated 25,000 physicians were arraigned on narcotics charges and 3,000 served prison sentences (Williams, 1938, as cited in Peyrot, 1984, p. 88).

While the Harrison Narcotic Act did not technically criminalize the use and possession of narcotics in the United States, its passage and subsequent legal interpretation would provide the foundation for the punitive antidrug policies that would follow. This was the first step in the history of U.S. drug policy toward defining drug use as a criminal act and, therefore, a social problem to be addressed by law enforcement and the criminal justice system. In less than a decade, the Harrison Narcotic Act transformed drug addicts in the United States from patients to criminals (Durrant & Thakker, 2003). By 1930, 35 percent of all convicts in America would be confined as a result of the criminalizing effect of the Harrison Narcotic Act (Booth, 1996). The interpretation of drug use and addiction as a public health issue, as exemplified by many of the European nations of the time, would not occur in the United States. The Harrison Narcotic Act would remain the basis of narcotics regulation in the United States for the next 50 years.

HARRY ANSLINGER AND THE FEDERAL BUREAU OF NARCOTICS

In 1930, the Narcotics Division of the U.S. Treasury Department was transformed into the Federal Bureau of Narcotics (FBN) and Harry J. Anslinger was named as commissioner (McWilliams, 1992). Anslinger was a staunch antidrug advocate. During his tenure as FBN commissioner (1930–1962), he was instrumental in promoting law enforcement and the criminalization of drug use (Musto, 1999). Some historians refer to Anslinger as America's first "drug czar" (McWilliams, 1992); noted sociologist Howard Becker (1963) characterized him as a "moral entrepreneur," (p. 147) and the FBN's work as a "moral enterprise" (p. 145). Anslinger's position as Commissioner of Narcotics allowed him great influence on America's early response to drug use and addiction. His prohibitionist and punitive ideals toward drug use coupled with his leadership of a public organization dedicated to federal narcotics enforcement helped institutionalize a criminal justice response to drug use in the United States.

During these formative years, Anslinger contributed significantly to the social construction of early U.S. drug policy. In order to maintain a high level

of antidrug sentiment among the public, Anslinger was constantly campaigning against the evils of drug use and abuse. Anslinger used youth as a device to elicit a negative public response to drug use; he aggressively propagandized drug use as a killer of innocent children. As an example of Anslinger's antidrug rhetoric, in 1937 he and a colleague wrote,

> The sprawled body of a young girl lay crushed on the sidewalk the other day after a plunge from the fifth story of a Chicago apartment house. Everyone called it suicide, but actually it was murder. The killer was a narcotic known to America as marijuana . . . a narcotic . . . as dangerous as a coiled rattlesnake. (Anslinger & Cooper, 1937, p. 150)

Anslinger also unscrupulously promoted marijuana use as the impetus for criminal and deviant behavior among minorities and the lower class, particularly Mexican immigrants in the American southwest (Bonnie & Whitebread, 1974). This discourse was instrumental in influencing public and political perceptions of drug use in the United States during this period. In essence, drug use and drug users were depicted as immoral and dangerous, reinforcing in the minds of Americans the need for increased antidrug legislation and law enforcement. These sentiments would also serve Anslinger well in his bureaucratic and professional goals of promoting the function of the FBN, its status as a government agency, and its value to a society becoming inundated with perceived drug problems (Dickson, 1968). If Anslinger could effectively construct a drug problem in the United States, he could perpetuate his and the FBN's political existence. These conditions would provide the social and political contexts for the passage of America's next significant antidrug policy—the Marijuana Tax Act of 1937.

The Marijuana Tax Act required every person who dealt with marijuana in any manner (i.e., imported, manufactured, sold, prescribed, etc.) to pay a special tax. Importers and manufacturers of marijuana were required to pay a $24-per-year tax; physicians, only $1 per year (Belenko, 2000). Furthermore, the Marijuana Tax Act required every person having contact with marijuana to register with federal authorities. Any individual that did not comply with the provisions of the act became subject to criminal penalties (Belenko, 2000).

The Marijuana Tax Act was intentionally kept separate from the Harrison Narcotic Act because of fears that it would be submitted to the same claims of unconstitutionality that plagued the Harrison Narcotic Act in its formative years. Anslinger and other federal antimarijuana advocates did not want to chance having the Marijuana Tax Act compromised by legal contestations. In an additional step to ensure the act's legitimacy, Anslinger and the U.S. Treasury's General Counsel Herman Oliphant strategized to model the Marijuana Tax

Act after an already Supreme Court–tested policy—the National Firearms Act of 1934 (Musto, 1999). The National Firearms Act stipulated that certain "firearms could only be transferred upon payment of a transfer tax. As peculiar as this tax may [have] seem[ed], it was held constitutional by the Supreme Court in March 1937" (Musto, 1999, p. 222). As a result, Anslinger and Oliphant decided to propose the Marijuana Tax Act as a transfer tax and revenue-collecting measure to the U.S. Congress. The act, which effectively criminalized marijuana use in the United States, passed through Congress with hardly a debate (McWilliams, 1992).

STRICTER PENALTIES

Despite the early attempts of antidrug legislators and law enforcement to eliminate drug use in the United States, it would continue to persist as a social problem. Following the Second World War, U.S. antidrug policymakers chose to address the country's drug problem by severely increasing the penalties levied for drug offenses (Brecher & the Editors of Consumer Reports, 1972). In 1951, the Boggs Act was passed, which imposed a mandatory minimum sentence of two to five years for a first conviction of narcotics possession (Booth, 1996). A second offense carried a mandatory 5 to 10 year penalty without chance of parole or reduced sentence. These measures were advocated by Commissioner Anslinger and endorsed by nearly every member of the U.S. Congress; however, some prominent professional organizations, namely the American Bar Association (ABA) and the American Medical Association (AMA), had reservations regarding the propriety of such punitive measures (McWilliams, 1992). The concerns of the ABA and AMA led the U.S. Senate, in 1955, to create a congressional subcommittee to review the efficacy of existing federal drug policies (i.e., Harrison Narcotics Act, Marijuana Tax Act, and Boggs Act). The subcommittee's investigation concluded that drug trafficking in the United States was a greater problem than first imagined and that heroin use was particularly problematic (Belenko, 2000). This perceived failure of the Harrison Narcotic Act, Marijuana Tax Act, and Boggs Act to effectively deal with drug use in U.S. society did not lead to a change in policy or philosophy. Instead, and much to the chagrin of the professional organizations, the U.S. Senate recommended tougher, more punitive antidrug measures.

In 1956, the U.S. government passed the Narcotic Control Act, imposing even stricter penalties on drug users, doubling the maximum sentencing guidelines mandated by the Boggs Act. Consequently, a first-time conviction for drug possession could result in a maximum sentence of 10 years; a second conviction could result in a prison term of 20 years. The Narcotic Control Act

also included a death penalty provision (at the discretion of the jury) for heroin sales to anyone under the age of 18 years by any individual over the age of 18 years (Musto, 1999). One goal of these punitive policy measures was to communicate to American society that the government was seriously addressing the drug problem and that "severe measures were at last being taken against addiction" (Brecher & the Editors of Consumer Reports, 1972, p. 56). These unprecedented levels of legal punishment for drug use effectively institutionalized drug use as a criminal justice problem in the United States. This social and political history of early drug policy construction represents the foundation of America's response to drug use as a social problem, a foundation predicated upon prohibitionist ideals and a need to punish individual drug users. The institutionalization of drug use as a criminal justice problem also reinforced the moral and legal interpretations of drug use in the minds of the American public. The construction of drug use as a moral and legal problem would ultimately have a direct effect on the clinical treatment of drug addicts in the United States.

SUBSTANCE ABUSE TREATMENT PHILOSOPHIES IN THE UNITED STATES

In the United States, abstinence-based treatment protocols dominate the substance abuse treatment system. In most drug treatment programs, total abstinence is the only measure of success; anything less is regarded as a treatment failure. A proportion of this treatment philosophy can be attributed to the disease model of addiction. In the disease model, addiction is seen as a chronic, progressive, and potentially fatal illness (Sheehan & Owen, 1999). The addicted person is incapable of controlling or moderating his or her drug use. This disease is incurable; however, it may be arrested if an addicted person "totally abstains from any addictive substance" (Miller & Hester, 1995, p. 83). The abstinence-only philosophy is also the basis of the Twelve-Step recovery model of Alcoholics Anonymous, which permeates virtually every substance abuse treatment program in the United States (Doweiko, 2006).

Despite the popularity of the disease model within the substance abuse treatment community, it plays a relatively minor role in the social and political responses to substance abuse in the United States. It is a philosophy supported by some health care providers, most substance abuse treatment professionals, and many recovering alcoholics and drug addicts; however, outside of these circles, it does not have much credibility. As emphasized earlier, most Americans believe drug use to be immoral and criminal. However, rather than refute the credibility of the disease and Twelve-Step models, these conflicting interpreta-

tions of substance abuse actually complement each other when determining a clinical course of treatment for drug addicts. When deciding which treatment protocol is most appropriate for drug addicts, moral and legal interpretations of substance abuse also prescribe abstinence as the only acceptable treatment outcome. The relative influence of these interpretations is not only evident in the abstinence-only treatment philosophy; it defines a zero-tolerance mentality existent within many substance abuse treatment programs in the United States. Wide acceptance of the disease model would presume a greater tolerance toward drug use among substance abusers, particularly those whose illness has reached an advanced stage requiring formal treatment. However, the reality is that most inpatient and residential treatment programs have zero tolerance for any substance use during a treatment episode, often resulting in the expulsion of the patient from treatment. While this treatment of patients may be necessary to preserve the integrity of an abstinence-based treatment environment, it does little to help the patient who continues to suffer from an incurable illness. From a medical perspective, the abandonment of a patient with a chronic illness for relapsing seems highly unethical. In the United States, physicians tend to avoid this ethical dilemma because few physicians actually treat drug addiction. This can be attributed to a number of factors, including the repercussions of the Harrison Narcotic Act, the early institutionalization of drug use as a criminal justice problem, the stigmatization and avoidance of drug addicts as a patient population, the inadequate training and education of physicians in addiction medicine, and the apathy of the American medical establishment to aggressively pursue ownership of substance abuse as a social problem.

The moral and legal interpretations of drug use and addiction have not only encouraged an abstinence-only, zero-tolerance treatment model, they have discouraged the federal legislation and clinical adoption of any harm-reduction interventions in the United States; the one notable exception has been methadone maintenance treatment. Harm reduction is a public health philosophy that recognizes and accepts that substance use is going to occur in society; however, rather than punish drug use, harm reduction attempts to reduce and minimize the harmful consequences associated with substance use (Erickson & Butters, 1998). The aversion to a public health model/harm-reduction approach to substance use in the United States is a direct result of the social construction of early drug policy and the institutionalization of drug use as a criminal justice problem. The goals and objectives of a prohibition-criminalization paradigm run counter to the goals and objectives of a harm-reduction approach (Erickson & Butters, 1998). Antidrug policies founded on law enforcement and punishment are designed to reduce the number of drug users in a society rather than

minimize the harm associated with substance use (MacCoun & Reuter, 2001). In the United States, the resistance to harm-reduction strategies is so strong that most political representatives risk severe remonstration at the mere suggestion of reducing criminal penalties for drug use or providing an alternative to punishment when dealing with drug offenders. As an example, in 1993 the Surgeon General of the United States, Joycelyn Elders, made a suggestion at a public meeting that the U.S. government should study the impact that legalizing drugs would have on U.S. society (Labaton, 1993). Dr. Elders, the country's highest ranking medical officer, did not advocate the legalization of drugs; she merely suggested studying the problem. The fallout from this statement was quite significant, eliciting public and political outrage, as well as repudiation from President Clinton denying any intentions to legalize drugs. Dr. Elders's controversial opinions on drug policy (and later, public sex education) led to her dismissal one year later.

As mentioned above, one substance abuse treatment intervention that appears to contradict the American resistance to harm reduction is methadone maintenance treatment (MMT). MMT is an opioid substitution therapy intended to help heroin addicts avoid opioid withdrawal symptoms, engage in substance abuse treatment longer, and lead healthier and more productive lives (Graff & Ball, 1979; Senay, 1985). Methadone was first developed in Germany during World War II as a substitute painkiller to morphine (Zweben & Payte, 1995). The development of MMT as a substance abuse treatment in the United States was pioneered by Drs. Dole and Nyswander during the 1960s. This occurred primarily as a result of the disapproval among members of the American Bar Association and American Medical Association of the highly punitive antidrug measures legislated in the 1950s, as mentioned previously (Musto, 1999). While the U.S. Senate was reviewing the efficacy of federal drug legislation, the ABA and AMA commissioned their own joint committee to study the issue of narcotics addiction. In 1958, this joint committee issued its report recommending that an outpatient facility prescribing narcotics for the maintenance of heroin addicts be established on a controlled experimental basis (Belenko, 2000). This recommendation paved the way for Dole to receive a grant from the Health Research Council of New York City to establish a research unit to investigate the feasibility of methadone maintenance treatment (Joseph & Appel, 1993).

MMT was enthusiastically received by the Nixon administration (1969–1974) during a period of intense concern regarding a perceived heroin epidemic and consequent rise in drug-related crime (Musto, 1999). Maintenance was seen as the most economical and successful way to treat thousands of heroin addicts, effectively reducing both drug use and associated crime. Heroin addicts

were actively recruited into methadone maintenance programs (Rosenbaum, Murphy, & Beck, 1987). In the United States during the 1970s and 1980s, methadone-treated patients numbered over 75,000 (Ball et al., 1987). By the 1990s, the United States had over 750 outpatient methadone maintenance programs treating approximately 115,000 patients on any given day (Parrino, 1993). Currently, it is estimated that approximately 160,000 persons receive MMT services in the United States (Firoz & Carlson, 2004). Contrary to the image these figures may portray, MMT in the United States is not a popular program. Medically, MMT is considered a pharmacological intervention; however, among most substance abuse treatment professionals, it is considered simply substituting one addictive substance for another. Politically, MMT is seen as a way to reduce drug-related crime and increase employment among drug addicts. Some authors (Erickson & Butters, 1998; MacCoun & Reuter, 2001) have stated that, in the United States, there is much more concern with the dangerous behavioral consequences and drug-related harm inflicted on non–drug users, than with the health and welfare of those actually afflicted with substance use disorders. From this perspective, a more critical interpretation of MMT is that it is utilized as a means to keep drug addicts and their unhealthy behaviors away from middle- and upper-class society, rather than a necessary treatment for heroin addiction. This interpretation, albeit cynical, appears more consistent with the social and political responses to drug use and addiction throughout American history and as an explanation for MMT as a harm-reduction exception in the American substance abuse treatment formulary.

POLICY ALTERNATIVES

Unlike the United States, most countries have not chosen to deal with drug use and addiction from a strictly prohibitionist perspective. Many societies acknowledge and accept the reality that drug use occurs and that attempts to fully eliminate drug use through punitive measures are unrealistic. These societies have attempted to balance a criminal justice response to drug use with harm-reduction strategies, such as maintenance treatment, needle exchange, safe injection sites, and the decriminalization of small amounts of prohibited substances (MacCoun & Reuter, 2001; Mosher and Atkins, 2007). In the United States, the prohibition-criminalization paradigm has resulted in over 500,000 drug-related imprisonments; this represents approximately 25 percent of the U.S. prison population. At a federal level, over half of all U.S. federal prisoners (approximately 55%) are incarcerated for drug law violations (Bureau of Justice Statistics, 2007). Despite a legacy of punitive criminal justice and vigorous law enforcement, drug use and addiction remain a social problem in

the United States. America's resistance to accepting a public health model of substance use has perpetuated the U.S. government's continued promotion of a "war on drugs" agenda, focusing the majority of resources on supply-side drug policies. Most drug policy advocates and scholars agree that current drug policies in the United States have a limited effect on curtailing drug use and addiction and that some type of reform is necessary.

The most extreme proposals for drug policy reform in the United States come from proponents of drug legalization. Drug legalization advocates feel that legalizing all drugs would counteract problems of drug-related crime, criminal justice and law enforcement costs and corruption, and the limiting of personal liberties (Jonas, 1991; Mosher & Atkins, 2007; Nadelmann, 1991). Some also see potential tax and health benefits associated with the legalization and government regulation of illegal substances (Nadelmann, 1991). While this discourse is academically stimulating, it is unrealistic that the United States, or any other country for that matter, will ever seriously consider legalizing all drugs. Drug legalization rhetoric may actually serve to reinforce a prohibition-criminalization paradigm in what is predominantly an ideologically conservative American public. Furthermore, American social policy tends to change incrementally, rather than in revolutionary-type movements (DiNitto, 2007). This implies that any movement away from the current status quo and toward drug policy reform will require relatively small policymaking initiatives and advancements.

According to Peyrot (1984), because the criminal justice response to drug use and addiction was the first response to become institutionalized in the United States, it virtually secured its position as the preferred approach to dealing with drug use and addiction in American history, to the extent of precluding any subsequent responses from replacing it. As a result, clinical approaches predicated on a medical interpretation of addiction as an illness or disease have only built on the preexisting moral and legal interpretations of this problem. Any attempts by the medical or substance abuse treatment professions to supplant the criminal justice system as the primary institution for dealing with drug use and addiction in the United States have not been successful. The limited acceptance of the public health and disease models of addiction also limits the alternatives available when contemplating drug policy reform. While many scholars and clinicians would like to see a revolutionary paradigm shift from prohibition-criminalization to public health and harm reduction, realistic policy alternatives must be considered. According to Peyrot's theory, successful drug policy modifications or changes must consider the role of the criminal justice system, as well as its inclusion in collaborative strategies across treatment and service systems.

One current collaborative intervention in the United States that achieves this goal is the drug court movement. Through a process known as "therapeutic jurisprudence" (Rosenthal, 2002, p. 154), drug courts effectively integrate the values of both the criminal justice system and the substance abuse treatment community. The first drug court opened in the United States in 1989, and currently there are more than 2,000 in operation serving more than 120,000 clients (Huddleston, 2007). Drug courts are designed to provide nonviolent drug offenders with an intensive court-based treatment alternative to incarceration (Nolan, 2002). Ideologically, the criminal justice system regards mandated substance abuse treatment as a reduced form of punishment while the substance abuse treatment community sees it as a positive step away from incarceration and into a therapeutic situation. Expansion of the current U.S. drug court system is a policy alternative that serves the interests of the criminal justice system and substance abuse treatment community, as well as drug users and addicts.

Expansion of substance abuse treatment services within the criminal justice system would also fit Peyrot's (1984) prescription. While some substance abuse treatment services already exist within the U.S. prison system, an expansion of services for nonviolent drug offenders both incarcerated and re-entering society would be a progressive step. Another policy issue that affects low-income drug offenders transitioning out of prison is their ineligibility for social welfare benefits. In 1996, as part of a national movement toward welfare reform, the U.S. government passed legislation denying public assistance (i.e., income maintenance benefits and food stamps) to any persons convicted of a drug felony (Personal Responsibility and Work Opportunity Reconciliation Act, 1996). This has had a particularly detrimental effect on low-income women transitioning back to their communities, as well as their children and families (Allard, 2002). A progressive policy alternative could have the public assistance eligibility status of nonviolent drug offenders reinstated upon completion of a substance abuse treatment program in prison. Another option that institutes the criminal justice system as the arbiter of substance abuse treatment services is to construct prison facilities exclusively for nonviolent substance abusers dedicated to rehabilitation rather than incarceration of drug users and addicts.

While these policy alternatives including the criminal justice system are far from optimal for most American drug policy reformers, they do represent options that may be politically salient to a bipartisan U.S. Congress. A more traditional harm-reduction policy that appears feasible in the United States is needle (or syringe) exchange. Currently, nearly every U.S. state has some type of needle exchange policy and program; however, the federal government has refused to pass any legislation or fund any programs supporting needle

exchange (Ip, 2007; MacCoun & Reuter, 2001). A consequence of this is that the U.S. government communicates to the American people that intravenous drug users and people with HIV/AIDS are not worthy of federal assistance, essentially perpetuating the stigmatization of these groups. Hopefully, unanimity at a state level will eventually have a positive influence on federal policymaking decisions.

Finally, if Americans want to take a significant first step toward reforming the current prohibition-criminalization paradigm and instituting a public health model of substance use they should at least consider examining the decriminalization (or depenalization; MacCoun & Reuter, 2001) of small amounts of marijuana. By decriminalizing small amounts of marijuana (e.g., half an ounce; 14 grams), drug users and addicts would not be subject to criminal justice penalties for the personal use of marijuana. This would not legalize marijuana use, nor relieve marijuana traffickers from arrest, prosecution, and incarceration. It would redefine personal marijuana use as an unhealthy behavior rather than a criminal act. In the United States, marijuana use accounts for 75 percent of all illegal drug use (Substance Abuse and Mental Health Services Administration, 2007). Decriminalizing small amounts of marijuana would lower criminal justice and law enforcement costs, as well as reduce perceptions among some Americans of the government's intrusiveness into personal liberties (MacCoun & Reuter, 2001). Furthermore, according to MacCoun and Reuter, depenalizing the possession of small amounts of marijuana would not increase marijuana use in U.S. society.

CONCLUSION

The statement by law historian Rufus King that opened this chapter was made in 1953 during a period of heightened punitive U.S. policymaking against drug users and addicts. His position implies that the prohibition-criminalization paradigm instituted in the United States was a mistake. Through antidrug policies, such as the Harrison Narcotic Act, Marijuana Tax Act, Boggs Act, and Narcotic Control Act, American policymakers effectively removed the care of drug users and addicts from the medical establishment and institutionalized drug use and addiction as criminal justice problems. This also served to construct drug users and addicts as criminals in the minds of the American people. If Americans are ever to change from moral and legal interpretations of drug use to a public health perspective, it must be preceded by a perceptual conversion among members of mainstream society to view drug users and addicts as useful and productive people rather than malcontents and deviants (Erickson & Butters, 1998). In the mean time, policy alternatives that integrate the

criminal justice system with helping professions (e.g., substance abuse treatment, medicine, and social welfare) may prove to be the most effective in transcending social and political proscriptions toward American drug users and addicts.

REFERENCES

Aldrich, M. R. (1988). Historical notes on women addicts. In *Youth Environment Study* (pp. 1–6). San Francisco, CA: Project MENU.

Allard, p. (2002). Life sentences: Denying welfare benefits to women convicted of drug offenses. Washington, DC: The Sentencing Project.

Anslinger, H. J., & Cooper, C. R. (1937, July). Marihuana: Assassin of youth. *American Magazine, 124*(1), 18–19, 150–153.

Ball, J. C., Corty, E., Petroski, S. P., Bond, H., Tommasello, A., & Graff, H. (1987). Treatment effectiveness: Medical staff and services provided to 2,394 patients at methadone programs in three states. In *National Institute of Drug Abuse: Research Monograph Series No. 76* (pp. 175–181). Rockville, MD: U.S. Department of Health and Human Services.

Becker, H. S. (1963). *Outsiders: Studies in the sociology of deviance*. New York: The Free Press of Glencoe.

Belenko, S. R. (Ed.). (2000). *Drugs and drug policy in America: A documentary history*. Westport, CT: Greenwood Press.

Berridge, V., & Edwards, G. (1987). *Opium and the people: Opiate use in nineteenth century England*. New Haven, CT: Yale University Press.

Boggs Act of 1951, Pub. L. No. 82-255, 65 Stat. 767 (November 2, 1951).

Bonnie, R. J., & Whitebread, C. H. (1974). *The marihuana conviction: A history of marihuana prohibition in the United States*. Charlottesville: University Press of Virginia.

Booth, M. (1996). *Opium: A history*. New York: St. Martin's Press.

Brecher, E. M., & the Editors of Consumer Reports. (1972). *Licit and illicit drugs*. Boston, MA: Little, Brown.

Bureau of Justice Statistics. (2007). *Drugs and crime facts: Drug law violations*. Retrieved May 4, 2008, from http://www.ojp.usdoj.gov/bjs/dcf/correct.htm

Courtwright, D. T. (1982). *Dark paradise: Opiate addiction in America before 1940*. Cambridge, MA: Harvard University Press.

Dickson, D. T. (1968). Bureaucracy and morality: An organizational perspective on a moral crusade. *Social Problems, 16*, 143–156.

DiNitto, D. M. (2007). *Social welfare: Politics and public policy* (6th ed.). Boston, MA: Allyn and Bacon.

Doweiko, H. E. (2006). *Concepts of chemical dependency* (6th ed.). Pacific Grove, CA: Brooks/Cole.

Durrant, R., & Thakker, J. (2003). Substance use and abuse: Cultural and historical perspectives. Thousand Oaks, CA: Sage.

Erickson, p. G., & Butters, J. (1998). The emerging harm reduction movement: The de-escalation of the war on drugs. In E. L. Jensen & J. Gerber (Eds.), *The new war on drugs: Symbolic politics and criminal justice policy* (pp. 177–196). Cincinnati, OH: Anderson.

Firoz, S., & Carlson, G. (2004). Characteristics and treatment outcome of older methadone-maintenance patients. *American Journal of Geriatric Psychology, 12*(5), 539–541.

Foster Bill of 1910, S. 6810, 61st Cong. (1910).

Graff, H., & Ball, J. C. (1979). The methadone clinic: Function and philosophy. *International Journal of Social Psychiatry, 22*(2), 140–146.

Harrison Narcotic Act of 1914, Pub. L. No. 63-223, 38 Stat. 785 (December 17, 1914).

Helmer, J. (1975). *Drugs and minority oppression*. New York: Seabury.

Huddleston, W. (2007). From the chief executive's desk. *NADCP News, 14*(2), 1, 4–6.

Ip, I. (2007). The quintessential catch-22: The US approach to needle exchange in HIV AIDS prevention. In M. L. Finkel (Ed.), *Truth, lies, and public health: How we are affected when science and politics collide* (pp. 87–101). Westport, CT: Praeger.

Jensen, E. L., & Gerber, J. (1998). The social construction of drug problems: An historical overview. In E. L. Jensen & J. Gerber (Eds.), *The new war on drugs: Symbolic politics and criminal justice policy* (pp. 1–24). Cincinnati, OH: Anderson.

Jonas, S. (1991). The US drug problem and the US drug culture: A public health solution. In J. A. Inciardi (Ed.), *The drug legalization debate* (pp. 147–160). Newbury Park, CA: Sage.

Joseph, H., & Appel, p. (1993). Historical perspectives and public health issues. In M. W. Parrino (Ed.), *State methadone treatment guidelines* (pp. 11–24). Rockville, MD: U.S. Department of Health and Human Services, Center for Substance Abuse Treatment.

King, R. G. (1953). The Narcotics Bureau and the Harrison Act: Jailing the healers and the sick. *The Yale Law Journal, 62*, 736–749.

Labaton, S. (1993, December 8). Surgeon General suggests study of legalizing drugs. *New York Times*, A23.

Linder v. United States, 268 U.S. 5 (April 13, 1925).

Lindesmith, A. R. (1957). *Opiate addiction*. Evanston, IL: Principia Press of Illinois.

Lindesmith, A. R. (1965). *The addict and the law*. Bloomington, IN: Indiana University Press.

MacCoun, R. J., & Reuter, p. (2001). *Drug war heresies: Learning from other vices, times, and places*. New York: Cambridge University Press.

Marijuana Tax Act of 1937, Pub. L. No. 75-238, 50 Stat. 551 (August 2, 1937).

McWilliams, J. C. (1992). Through the past darkly: The politics and policies of America's drug war. In W. O. Walker III (Ed.), *Drug control policy: Essays in historical and comparative perspectives* (pp. 5–41). University Park: The Pennsylvania University Press.

Miller, W. R., & Hester, R. K. (1995). Developmental history of models of alcohol and other drug abuse behaviors. In *Curriculum modules on alcohol and other drug problems for schools of social work*. Washington, DC: Center for Substance Abuse Prevention.

Mosher, C. J., & Atkins, S. (2007). Drugs and drug policy: The control of consciousness alteration. Thousand Oaks, CA: Sage.

Musto, D. F. (1999). *The American disease: Origins of narcotic drug control* (3rd ed.). Oxford, England: Oxford University Press.

Nadelmann, E. A. (1991). The case for legalization. In J. A. Inciardi (Ed.), *The drug legalization debate* (pp. 17–44). Newbury Park, CA: Sage.

Narcotic Control Act of 1956, Pub. L. No. 84-728, 70 Stat. 567 (July 18, 1956).

Nolan, J. L. (2002). Preface. In J. L. Nolan (Ed.), *Drug courts: In theory and in practice* (pp. vii–xiv). Hawthorne, NY: Aldine de Gruyter.

Parrino, M. W. (1993). Introduction. In M. W. Parrino (Ed.), *State methadone treatment guidelines* (p. xxxi). Rockville, MD: U.S. Department of Health and Human Services, Center for Substance Abuse Treatment.

Personal Responsibility and Work Opportunity Reconciliation Act of 1996, Pub. L. No. 104–193, §115, 110 Stat. 2180 (August 22, 1996).

Peyrot, M. (1984). Cycles of social problem development: The case of drug abuse. *The Sociological Quarterly, 25*, 83–96.

Pharmacy Act of 1868, 31 & 32 Victoria, c. 121, Parliament of the United Kingdom.

Pittman, D. J., & Staudenmeier, W. J. (1994). Twentieth century wars on alcohol and other drugs in the United States. In p. J. Venturelli (Ed.), *Drug use in America: Social, cultural, and political perspectives*. New York: Jones & Bartlett.

Pure Food and Drug Act of 1906, Pub. L. No. 59-384, 34 Stat. 768 (June 30, 1906).

Robinson, M. B., & Scherlen, R. G. (2007). Lies, damned lies, and drug war statistics: A critical analysis of claims made by the Office of National Drug Control Policy. Albany: State University of New York Press.

Rosenbaum, M., Murphy, S., & Beck, J. (1987). Money for methadone: Preliminary findings from a study of Alameda County's new maintenance policy. *Journal of Psychoactive Drugs, 19*(1), 13–19.

Rosenthal, J. T. A. (2002). Therapeutic jurisprudence and drug treatment courts: Integrating law and science. In J. L. Nolan (Ed.), *Drug courts: In theory and in practice* (pp. 145–171). Hawthorne, NY: Aldine de Gruyter.

Rouse, J. J., & Johnson, B. D. (1991). Hidden paradigms of morality in debates about drugs: Historical and policy shifts in British and American drug policies. In J. A. Inciardi (Ed.), *The drug legalization debate* (pp. 183–214). Newbury Park, CA: Sage.

Senay, E. C. (1985). Methadone maintenance treatment. *International Journal of the Addictions, 20*(6 & 7), 803–821.

Sheehan, T., & Owen, p. (1999). The disease model. In B. S. McCrady & E. E. Epstein (Eds.), *Addictions: A comprehensive guidebook* (pp. 268–286). New York: Oxford University Press.

Substance Abuse and Mental Health Services Administration. (2007). *Results from the 2006 National Survey on Drug Use and Health: National findings* (Office of Applied Studies, NSDUH Series H-32, DHHS Publication No. SMA 07–4293). Rockville, MD: U.S. Department of Health and Human Services.

United States v. Behrman, 258 U.S. 280 (March 27, 1922).

United States v. Doremus, 249 U.S. 86 (March 3, 1919).

Webb et al. v. United States, 249 U.S. 96 (March 3, 1919).

Zweben, J. E., & Payte, T. (1995). Methadone maintenance programs. In J. Jaffe (Ed.), *Encyclopedia of drugs and alcohol* (vol. 2, pp. 672–677). New York: Simon & Schuster Macmillan.

This page intentionally left blank

Chapter **2**

The Philosophy of Denial in Alcohol Studies: Implications for Research

Lorraine T. Midanik, PhD

In documentation and discussions of the development of the disease model of alcoholism, and most specifically Alcoholics Anonymous (AA), Al-Anon, and Adult Children of Alcoholics (ACA)/Codependency, very little has been written about how *denial* as a phenomenon came to be the central tenet of the disease ideology. Interestingly, *denial* was never originally mentioned in the Big Book of AA nor was it listed as a specific symptom or even part of a symptom in Jellinek's early work that became the scientific underpinning of the disease model (Jellinek, 1946, 1947, 1952). However, as argued by Kurtz (1979) denial was always an assumption underlying the identification and definition of alcoholism by AA.

> Denial, after all, had been earmarked by Alcoholics Anonymous to be the characteristic symptom and deep core of alcoholism. The clever use of language in service to denial has throughout history subverted true communication and so fostered alienation and the separations of mistrust. (p. 193)

Even though the relationship between denial and alcoholism is not explicit in early work in the alcoholism field, there is a strong connection between the two concepts. Yet, without it specifically being mentioned, this begs the question of why and how denial emerged in the alcohol field and also why it has retained its prominence and legitimacy over time.

Sometimes when two concepts are closely associated, such as alcoholism and denial, they begin to be viewed as universal truths. This phenomenon was discussed by Dwight Heath in his forward to Kai Pernanen's book titled *Alcohol*

in Human Violence (Pernanen, 1991). Heath points out that within specific social contexts, we tend to make linkages between words (in this case, alcohol and violence) that come to be known as common truths and over time are mistakenly taken to somehow represent reality. Pernanen's (1991) book focuses on multiple conceptualizations of the relationship between alcohol use and human violence, which serves to counteract partially the prevailing causative paradigm and to investigate more thoroughly the rather complex and intricate linkages between the two areas.

Alcoholism and denial—or more specifically the common notion that alcoholics or heavier drinkers often and predictably deny the extent of their use of alcohol, their subsequent alcohol-related problems, and their alcoholism per se—can be seen as another example of a somewhat universally accepted truth that, to some clinicians and researchers, does not necessarily require empirical verification. This assumption of denial based on a disease definition of alcoholism (the predominant model in the alcohol field) affects not only the ways in which studies of alcoholics or alcohol use are conducted but also the stance taken by researchers regarding self-report validity issues and the methods they select to demonstrate validity (Midanik, 1989).

There are two bumper stickers that have been prominent in the San Francisco Bay Area (in California, United States) for some time; both reflect not only the rebelliousness of the residents of this geographic area but also speak to the problem of unquestioned dominant paradigms. The first one, "Subvert the Dominant Paradigm," takes a social action perspective and implies a nonacceptance of the status quo by recommending that we overthrow it and replace it perhaps with one of our own. The second one merely says "I Survived the Dominant Paradigm," thus emphasizing the resiliency of some individuals to continue on with their lives despite nonacceptance of the predominant world view. Regardless of whether one subverts or survives the paradigm, it is first important to examine the assumptions behind each perspective, to question its dominance, and to explore alternative views that can only serve to understand better the phenomenon we choose to study. The predominant view in the alcohol field is the disease model, which includes this assumption of denial. In fact, the concept of denial has become so popular in the recent decade that it is not unusual to hear someone use the phrase "I'm in denial" as a way to apologize for not remembering some significant event or person.

The purpose of this chapter is to explore the concept of denial in more depth. It will be argued that denial, a very specific and extremely important cornerstone of the disease model, has permeated more than the clinical work for which it was originally intended. First, we will discuss the larger issue of truth telling and provide a context in which denial is one component. Second, we will

look historically at how denial as a defense mechanism has been defined over time. Then we will explore how this concept became part of the disease model of alcoholism and has been adopted by other Twelve-Step movements as well as the general field of alcohol studies, and how denial is infused in treatment, treatment evaluation, and prevention studies. We will discuss the political aspects of using denial in the alcohol field and assess how denial as an explanation for behavior has been a central force in the research world, particularly methodological work, in terms of how measurement issues are conceptualized and assessed by researchers. Finally, we will argue that belief in this philosophy of denial has compromised much methodological research in the alcohol field by researchers who have vested interests in the outcome, that is, that self-reports of alcohol use and alcohol problems are valid or not.

TRUTH TELLING, LYING, AND DECEPTION

Perhaps a good place to begin our assessment of what factors contribute to the importance of truth telling in research at different historical times is to consider how the alternatives, namely lying and deception, are viewed in our society and how they relate to denial. By taking a broader perspective, we can start to place denial and its effect on research and treatment in the context of larger philosophical issues around how lying is handled on a daily basis in our society.

It has been argued fervently that denial is a way of life that permeates individual lives by allowing traumatic events to be handled more effectively so that individuals can continue their lives in the least disruptive way. Political psychologists connect denial experienced by individuals as a result of early childhood and the interaction of children with their parents. During this time, avoidance of painful experiences serves a fundamental protective function that unfortunately may keep an individual in a fantasy world, unable, or unwilling, to face reality (Milburn & Conrad, 1996).

Beyond denial, there is the overall issue of lying and its pervasiveness in everyday life. Consider the following paragraphs from Sissela Bok's book entitled *Lying: Moral Choice in Public and Private Life* (Bok, 1978).

> Should physicians lie to dying patients so as to delay the fear and anxiety which the truth might bring them? Should professors exaggerate the excellence of their students on recommendations in order to give them a better chance in a tight job market? Should parents conceal from children the fact that they were adopted? Should social scientists send investigators masquerading as patients to physicians in order to learn about racial and sexual biases in diagnosis and treatment? Should government lawyers lie to Congressmen who might otherwise oppose a

> much-needed welfare bill? And should journalists lie to those from whom they seek information in order to expose corruption?
>
> We sense differences among such choices; but whether to lie, equivocate, be silent, or tell the truth in any given situation is often a hard decision. Hard because duplicity can take so many forms, be present to such different degrees, and have such different purposes and results. Hard also because we know how questions of truth and lying inevitably pervade all that is said or left unspoken within our families, our communities, our working relationships. Lines seem most difficult to draw, and a consistent policy out of reach. (pp. xv–xvi)

As children we are often told to be truthful; it is a value, an ethic, of our culture. We hear witnesses in courtrooms agreeing to "tell the whole truth and nothing but the truth." And, children are often told the story of George Washington who, despite possible negative consequences, confessed to chopping down a cherry tree (Saarni & Lewis, 1993). Yet, as noted in the preceding Bok quote, the line between telling the truth and lying is a difficult, if not impossible, one to draw. Despite this difficulty and the messages that we receive daily to the contrary, we somehow tend to operate under the assumption that the truth is both achievable and desirable in all situations.

Several books have been written about lying and deception from both a philosophical position (Fingarette, 1969, 1988) and a psychological perspective (Ekman, 1992; Ford, 1996; Lewis & Saarni, 1993). Despite the different perspectives represented by the authors writing in this area, they overwhelmingly agree that lying is pervasive in our society. Ford (1996) cites a U.S. poll from which 90 percent of the respondents claimed that they were deceitful. Their lies, in order of frequency, were about their true feelings, income, accomplishments, sex life, and age (Patterson & Kim, 1991). While Saarni and Lewis (1993) agree that it is commonplace in our society to lie, they ask the larger question of what contributes to this behavior within our culture. Factors, situational and individual, that foster lying and deception on an everyday basis are important to consider when assessing why these behaviors occur.

Regardless of whether deception is toward others or oneself, or whether there is or is not awareness of the deception, lying is very commonplace in our society. To demonstrate the pervasiveness of lying in our society, Ford (1996) compiled a list of 25 verbs, 21 nouns, and 5 adjectives that connote deceit. What is striking about this list is the multiple levels of deceit that are represented. Ford (1996) argues that the word "lying" is laden with emotion; thus, we need softer words, euphemisms, to describe different levels of the same phenomenon. Given the diverse terminology used to label deceit, how can one define the entire range of behaviors associated with telling lies?

It is simplistic to define lying as the opposite of truth. This assumes that proof, however defined, of the falsity of what is said or written constitutes a lie. Rather, Bok (1978) argues that it is the *intention to mislead* that differentiates a lie from an untruth. Establishing intent is often a very difficult if not impossible task, thus the demarcation between the two is perplexing and exceedingly complex. Lying is also a complicated subject to discuss because of the *gradations* involved. We tend to categorize the seriousness of lies based on the consequences or potential consequences of these actions. Thus, an individual who lies in court about committing a serious crime is judged much more harshly than someone who lies about his or her educational background on a job application. How we choose to define and clarify the gradations of lies has potentially important consequences on a societal level as well as on an individual level.

Bok (1978) also makes a distinction between different types of lying that include white lies and excuses. She contends that lying in ubiquitous; we lie when we overstate an individual's ability in a letter of recommendation, and we, as researchers, lie when we provide placebos in experiments. Yet while we defend some of these lies as harmless (hence, white lies), they are still intentional falsehoods despite the fact that we do not intend to hurt anyone in the process.

Perhaps equally important is the perception of individuals who think that someone is lying to them, and who can become resentful and disappointed because they feel manipulated. Moreover, they may perceive a shift of power to the perceived liar, which would be particularly problematic to deceived individuals who were originally in more powerful positions.

Saarni and Lewis (1993) provide a taxonomy of lying that is built on two dimensions: first, deception toward another person or toward oneself; and, second, whether the individual is aware or unaware of the deception. Based on these dimensions, Saarni and Lewis define three types of deception: (1) ordinary deception toward others committed with self-awareness; (2) deception toward others that requires some degree of self-deception; and (3) self-deception even in the absence of another, that is, the need for illusion.

The first type of deception, lying with self-awareness, most commonly occurs as a way to avoid retribution of others for behaving or not behaving in a particular way. The person committing the deception knows what is expected, knows what others expect, and chooses to lie as a way to avoid some type of punishment or anger. If this deception is disclosed, the recipients are offended for two reasons: failure to do what was expected as well as breach of trust. Saarni and Lewis (1993) also contend that these kinds of lies can be adaptive. For example, a person may well know that driving after drinking can increase

one's risk of a traffic accident. Yet when asked by a significant other if he or she had been driving after consuming alcohol, the response may be negative in order to avoid any arguments or reprisals.

The second type of deception involves deceiving others but being unaware or at least partially unaware of doing this (some self-deception). This is a more complicated type of deception given that the degree of self-deception is difficult to determine. Saarni and Lewis (1993) give an example of a man who tells his wife that he has not had an extramarital affair when in fact he has had an ongoing relationship with another woman. While he is very aware of his deception, he convinces himself that his failure to be honest with his wife is for her own good—it would hurt her.

The third type of deception, self-delusion, allows individuals to avoid bad or negative feelings about themselves and provides an alternative to the psychic costs of shame, humiliation, or embarrassment. Thus, losing one's job because of continued absences due to prolonged drinking bouts may be too difficult for an individual to acknowledge. It may be easier to create the illusion that the commute was too long, the supervisor was not fair, or the work was boring as better reasons that do not require that individuals lower their self-esteem. Interestingly, Saarni and Lewis (1993) speak to the advantages of self-delusion and argue that

> There is no question that self-deception—the creation of illusions, "looking at the world through rose-colored spectacles" has its advantages. Because of this, we cannot readily accept the idea that all forms of self-deception or other similar psychic phenomena like denial or forgetting are necessarily maladaptive. (p. 13)

Truth Telling and Research

The previous discussion is not intended to cover the entire field of philosophy of truth telling, lying, and deception but rather to address areas typically not discussed in these works. That is, the intent is to provide a brief background in this area so that methodological research aimed at determining who provides and who does not provide accurate information can be integrated within the larger context of truth telling across situations and environments in our society.

Connections previously made between lying and research have focused primarily on two distinct and important areas: research fraud and deceptive social research. In writing about research fraud, seminal books have been published that describe in detail the work of scientists, some of them quite famous, who created their data, consciously and overtly misinterpreted their data, or stole their data in order to become well-known in their respective fields (Broad &

Wade, 1982; Gould, 1981). These works contain fascinating accounts of the extent to which a few researchers will go to achieve fame at the possible expense of their reputations and academic positions. However, the focus is on researchers and what they chose to do or not do about their research and subsequent publications. The approach here is quite different. It is not on fraud committed by researchers per se, but rather on the research process itself and the difficulties of obtaining valid information directly from human subjects. Within this context, I ask the larger question of how the broader frameworks of lying and truth telling affect methodological research focused on the veracity of subjects' reports.

Like research fraud, much has been written about the conduct of social research projects that incorporate deceptive means to obtain data from participants. Deception in these instances ranges from lying or overtly misleading subjects about the actual purpose of the study to deluding mental health facilities about the condition of inpatients—for example, pseudo-patient studies (e.g., Rosenhan, 1973). While there are many cases of deceptive research, the classic examples in the United States that eventually led to the National Research Act of 1974, which requires national guidelines for the protection of human subjects, include the Tuskegee Syphilis Experiment (Jones, 1993) and the Obedience to Authority studies of Stanley Milgram (Milgram, 1974). In the former study, begun in 1932, the focus was on studying the natural history of syphilis in a population of very poor, rural, black males who had the disease. Throughout the 50-year-long study, the subjects were told that they were receiving treatment when they were, in fact, being given medical tests, some of which were quite painful (e.g., spinal taps), and when penicillin became the treatment of choice during the 1940s, this was withheld from the study population. The Obedience to Authority study also used deception in determining how far research subjects would go to obey orders by someone who appeared to have authority over them. Milgram found that with ample prodding, most research subjects would administer what they believed to be lethal shocks to unknown recipients if told to do so by someone who was perceived to be in authority. In both studies, the focus is on the lack of truth telling (or deception) of researchers in the recruitment of their subjects and in the conduct of their studies, particularly in terms of their safety and protection. Veracity of the subjects' reports or behaviors is clearly not the focus.

In the alcohol field, truth telling and research typically focuses on the validity of self-reports of respondents. Misreporting of alcohol consumption and alcohol problems is often viewed as lying—intentional misrepresentations of the truth because of denial. We assume that either individuals cannot admit, or refuse to admit, the problem or problems to themselves (denial) or that they

refuse to admit these problems to the researcher (denial or social desirability issues) or both.

Not only do we explain misreporting by these constructs, but there is also an implicit expectation that misreporting will occur given that denial is an integral component of the disease model. This self-fulfilling prophecy leads us to develop new and better ways to either help the individual overcome or break through denial, or find other, perhaps more clever, ways to get the truth, since it may not be readily available when conventional approaches are used. The results of the misreporting, termed "lies," is frustration and anger within certain research communities and ultimately a general mistrust and skepticism of all self-reported behavior.

The irony in this argument is that our expectations seem unrealistic in light of the world, specifically the research world in the social sciences in which we live and operate. If lying is so pervasive, why are we so surprised when we suspect under- or overreporting during the research process? Given the preceding discussion, it is somewhat difficult to believe that as researchers we are so astounded when we find that subjects' answers to questions do not either match our expectations or, more importantly, are not the same as responses obtained from other sources such as official records or collateral reports.

HISTORICAL LOOK AT DENIAL

To understand the definition of denial and how it differs from other untruths, it is first important to examine closely how it was formulated. This section will briefly review definitions of denial from a psychoanalytic perspective to provide a background for ways in which it has permeated the alcohol field.

Psychiatric definitions of denial refer to both the processes used by the individual who is denying and more specifically to what is or can be denied (internal or external states). Further, denial is placed under the larger umbrella of defense mechanisms, which are defined as "psychological strategies by which persons reduce or avoid negative states such as conflict, frustration, anxiety, and stress" (Holmes, 1984, p. 347). Beginning with Freud in 1894 (Slap, 1977), the concept of defense mechanisms began appearing in the literature as a basic defining principle of hysterical phenomena. By 1926, Freud argued for some categorization of defense mechanisms, which he believed may specifically be associated with certain forms of psychiatric illnesses (Laplanche & Pontalis, 1973). He further emphasized the importance of studying mechanisms of defense because they were the foundation on which psychoanalysis was built (S. Freud, 1914a, 1914b). Freud saw defense mechanisms as the key to interpreting conflict and hence central to the psychoanalytic understanding of neu-

rotic and psychotic patients. However, it was Anna Freud who defined denial in more depth and broadened its meaning to include its positive role particularly for children, whose options for handling problematic and potentially dangerous situations are more limited (A. Freud, 1946).

Specific defense mechanisms discussed by Freud and others include denial, repression, suppression, projection, displacement, regression, identification, compensation, reaction formation, sublimation, rationalization, and intellectualization. While a thorough discussion of these defense mechanisms goes beyond the scope of this chapter, it is important to recognize that defense mechanisms have three common characteristics. First, all of them are used to reduce or totally avoid negative emotional states such as anxiety or conflict. Second, when used, they distort reality, and third, individuals who use defense mechanisms are not consciously aware that they are using them (Holmes, 1984).

Definitions of denial vary somewhat by their specificity or lack of it. Originally termed scotomatization by Freud (Hinshelwood, 1989), denial is generally defined as one strategy to reduce or avoid negative states by not focusing on the threat-provoking aspects of a situation and by reinterpreting or reframing the situation so as to render it is less threatening. Other terms used for denial include redefinition and reappraisal (Holmes, 1984). There is some confusion over whether Freud originally intended denial to refer only to a defense against the claims of external reality, given that repression is a defense mechanism that applies to defending against internal issues such as instinctual demands or ideas (Eidelberg, 1968). However, it is generally agreed that denial includes selective attending to some aspect of reality in which "the ego refuses to become aware of an unpleasant or unwanted aspect of reality; in some instances the facts are accepted but their significance is denied" (Slap, 1977, p. 28).

If denial is being used as a defense mechanism because the patient is reinterpreting a threatening situation to a less threatening one, then it is assumed that one purpose of treatment would be to invoke reality into the patient's consciousness. From a psychoanalytic perspective this would involve locating the unresolved conflict and moving that conflict, or parts of that conflict that are unacknowledged, from the unconscious to the conscious in a safe environment—presumably treatment.

There are two major issues that arise when trying to translate and apply psychoanalytic definitions of denial to nonpsychoanalytic situations. The first issue is that denial as well as other mechanisms of defense are commonly used by most people since it is presumed that human beings strive to reduce negative or anxiety-provoking states (Holmes, 1984). Because use of defense mecha-

nisms is so ubiquitous in the general population, it is more difficult to differentiate pathology from normalcy. The question then becomes, at what point does denial become problematic? Given its pervasiveness, one would assume that problematic (or pathological) denial is a matter of degree but when and how this line gets drawn is unclear. Second, because psychoanalysts are writing from their clinical perspectives, they describe pathological denial as patients' refusal to look at significant and major aspects of their own lives. However, in less extreme cases (when denial is presumed to be used), it is important to keep in mind who is applying this label to whom. To the extent that denial is the label used to explain individuals whose realities differ from others, it is important to look at the vested interests of the individuals involved, the relative power between them, and the possible gain of specific individuals when the label of denial is applied (Brissett, 1988).

DENIAL IN THE ALCOHOL FIELD

The previous section provided a brief psychoanalytic history of denial and its application in the field of psychiatry. Because that section was not meant to describe the vast array of defense mechanisms and other intrapsychic phenomena included in psychiatric diagnosis and treatment, the attention to denial can be misleading. It is one of several concepts in the realm of psychiatric and psychological tradition. However, in the alcoholism literature, denial is a key component of identification, treatment, and recovery of the alcoholic (Brissett, 1988). The emersion of denial in the alcohol field will be traced in this section in terms of four interrelated and overlapping forces: AA, Al-Anon, the ACA/Codependency Movement, and the development of Employee Assistance Programs (EAPs).

Alcoholics Anonymous and Denial

While denial as a formal construct was not a part of the early alcohol literature until it was implicitly included in the formal development of AA, T. D. Crothers, in his treatise on the symptoms of inebriety, discussed it broadly as "egotism" as early as 1911:

> In many instances the most ingenious reasons are given to confirm and sustain his confidence in their reality, and no pressure or advice by friends can make any impression or indicate the real character of his mental condition. Persons with good judgment on all other matters will have the most delusional impression of their own conditions, and assert with great confidence that outside influences and undue anxiety and annoyance by friends are active causes of his present stage. This peculiar egotism is a species of general paralysis, particularly of the

> high brain centers. It literally means a progressive anesthesia of certain areas of the brain with unconsciousness of the real condition. (Crothers, 1911, p. 56)

Despite this claim of "progressive anesthesia" caused by excessive alcohol use, it is not until Jellinek's studies beginning in the mid 1940s that we begin to see some references to how deception may be part of the disease concept of alcoholism. Jellinek's *Grapevine Study* and subsequent research resulted in the delineation of four phases of alcohol addiction based on 43 symptoms (Jellinek, 1952). Within this categorization, Jellinek did not formally place denial as a separate symptom in his array of progressive symptoms. However, its existence is subsumed under symptom 9, "rationalization of drinking behavior" in the crucial phase, which Jellinek describes as follows:

> he produces the well-known alcoholic "alibis." He finds explanations which convince him that he did not lose control, but that he had a good reason to get intoxicated and that in the absence of such reasons he is able to handle alcohol as well as anybody else. These rationalizations are needed primarily for himself and only secondarily for his family and associates. The rationalizations make it possible for him to continue with his drinking, and this is of the greatest importance to him as he knows no alternative for handling his problems. (Jellinek, 1952, p. 680)

This concept of rationalization comes up again as the final symptom (43) in the chronic phase. It is commonly referred to as the breakdown of the rationalization system developed throughout the stages of alcoholism to justify one's behavior. This is an extremely important phase in Jellinek's nosology as it defines the process of "hitting bottom," the only point at which alcoholics can be able to receive the help they need. "Hitting bottom" is described as the point where

> the rationalizations become so frequently and so mercilessly tested against reality that the entire *rationalization system fails* (43) and the addict admits defeat. He now becomes spontaneously accessible to treatment. Nevertheless, his obsessive drinking continues as he does not see a way out. (Jellinek, 1952, p. 683, italics in the original)

Other researchers have differed with respect to where denial fits into the phases of addiction. Paredes (1974) argues that denial and "deceptive maneuvers" are more important in the early stages of alcoholism when the individual's family, work, and place in the community are still reasonably intact. While acknowledging that denial is involved throughout the course of alcoholism, Amodeo and Liftik (1990) see rejection of a diagnosis as its most common form and of particular importance in the middle stage of alcoholism. They outline three types of denial exemplified in the following statements:

> [T]here's nothing wrong with the way I drink . . . OK, so I drink. But I don't have a problem with it . . . If my drinking gets out of hand sometimes, it never leads to any big problem. (p. 132)

Curiously, as stated earlier, denial was never formally acknowledged as a key concept in the disease model. This may have been because the early founders of AA fervently worked to separate their fellowship from the influence of professionals in psychiatry or psychology. This was evident in the AA literature and in its descriptions of how alcoholism develops and the specific stages that one must pass through to "hit bottom" and hopefully move on to the recovery process.

In the third edition of *Alcoholics Anonymous: The Story of How Many Thousands of Men and Women Have Recovered from Alcoholism* (AA, 1976), the 44 personal stories are divided into three sections: (1) "Pioneers of A.A." (12 stories of men and women who entered AA in the late 1930s and 1940s); (2) "They Stopped in Time" (17 stories representing members who more recently joined AA); and (3) "They Lost Nearly All" (13 stories reflecting AA's changing demographics). In this edition, as in an earlier one published in 1955, changes were made to these stories to "represent the current membership of Alcoholics Anonymous more accurately, and thereby to reach more alcoholics" (AA, 1976, p. xii). Yet, while not explicitly discussed, denial as a concept is implied throughout the stories in this volume, which, of course, fits the sequencing of historical events recounted by the authors before the discovery that they were alcoholics. Examples of "implicit denial" include the following:

> I didn't think I was an alcoholic. I thought my problem was that I had been married to a drunk for twenty-seven years. And when my husband found A.A., I came to the second meeting with him. I thought it was wonderful, simply marvelous, for him. But not for me. Then I went to another meeting, and I still thought it was wonderful-for him, but not for me. (AA, 1976, p. 321)

> The only importance in all of this lies in the fact that at twenty-three I was just as sick as I was at thirty-three, when I came into A.A., but at that time I apparently had no place to go because I had no drinking problem. . . . I'm sure they could not have convinced me my sickness was within myself, nor could they have shown me the need for self-analysis that A.A. has shown me is vital if I am to survive. So I had no place to go. Or so it seemed to me. (AA, 1976, p. 546)

Al-Anon and Denial

Beyond the stages of the disease model and its clinical application, Al-Anon also is a formidable force that has integrated denial as both a necessary key element and sufficient condition for achieving membership in this group. In 1969,

Al-Anon Family Group Headquarters printed a pamphlet titled *Alcoholism: A Merry-Go-Round Named DENIAL* taken from a speech delivered by Reverend Joseph Kellermann at the Second Annual Workshop of the Connecticut Al-Anon Groups in Milford, Connecticut (Kellermann, 1969). In this pamphlet, Rev. Kellermann presents alcoholism as a tragic three-act play with four characters: the alcoholic, the enabler, the victim, and the provoker. The pivotal component of this play is denial manifested by the alcoholic in at least two forms. First, Kellermann contends that alcoholics are basically immature and dependent, yet "he may act in an independent way in order to *deny* this fact" (p. 2, italics in original). Second, alcoholics deny any responsibility for the results of their drinking behavior. Further, alcoholics hide the amount of drinking they do and repeatedly deny how important alcohol has become in their lives.

Beyond the many ways that alcoholics deny, Kellermann moves from the alcoholic to the enabler role to further elucidate the powerful effects of denial. An enabler is defined as a person who rescues alcoholics from their predicaments (a role that also includes the so-called helping professions). In the early literature (as in this pamphlet), the role of the enabler and the provoker (referring to the spouse) can be separate roles. Later literature combines these roles so that the spouse becomes the person who not only rescues the spouse but also harbors bitterness and resentment toward the alcoholic, which exacerbates an already troubled marriage. In his pamphlet, Kellermann makes the claim that the individual in the enabler role "denies the alcoholic the process of learning *by correcting his own mistakes*" (p. 5, italics in original). Thus, enablers are active participants in the denial process.

One of the most interesting parts of this pamphlet is Kellermann's misuse of the term "denial." Whereas defense mechanisms are clearly defined as occurring on a subconscious level beyond awareness, Kellermann argues explicitly that "The real problem is that the alcoholic is well aware of the truth which he so strongly denies" (p. 7). Thus, denial, as defined in this literature as well as in other alcohol literature, is a conscious process that is the central core of the disease process—"The key word in alcoholism is '*Denial*'" (p. 3, italics in original). This is also reflected in other Al-Anon literature. In a content analysis of Al-Anon stories published in two editions of *Al-Anon Faces Alcoholism* (Al-Anon, 1965, 1985), denial was directly referred to in a story that was included in the 1985 edition but not in the previous edition (Martin, 1992).

Adult Children of Alcoholics (ACA)/Codependency and Denial

In reviewing the linkages between the Twelve-Step movements and denial, there is clearly a progression from implicit to explicit use of the term. The lit-

erature in AA never quite uses the term "denial" but clearly describes such psychological processes as part of its disease symptomatology. Al-Anon takes this one step further and includes denial in some of its literature. As a self-help organization, Al-Anon does not seem opposed to using the concept of denial as an explanation of the behavior and psychological processes of a significant other who needs to be enlightened. The ACA/Codependency movement is at the other end of the continuum. Denial is an important and highlighted component of codependency. The term is readily used and acknowledged.

Cermak (1986) proposed *DSM-III* criteria defining "codependent" as a type of personality disorder. With one exception, Cermak's criteria for codependent personality disorder lacks any timeframe references; it also fails to specify how many times a specific behavior needed to occur before a diagnosis is justifiable. The first of his "E" criteria is the "excessive reliance on denial." To be diagnosed as codependent, three or more of the "E" criteria need to be met; thus, a person does not have to excessively rely on denial to be a codependent. Yet Cermak (1986), in his description of these specific criteria, argues that the similarity between the denial experienced by codependents of their codependency is exactly the same as the denial experienced by the person who is chemically dependent. He further maintains that

> Denial may be seen as an impaired strategy for achieving security. In the face of a threat, narrowing one's awareness can create the appearance of safety. . . . For active chemical and co-dependents, however, denial continues to give the false impression of security. It is rarely acknowledged and relinquished until the pain and emptiness of their ever narrower and more isolated life becomes too much to bear. (p. 22–23)

In a later book that focused on recovery for ACAs, Cermak (1988) devotes an entire chapter to denial titled "A Time to See: Pulling Down the Walls of Denial." Within this chapter, Cermak outlines the warning signs of denial by three levels: level I—denying your parents' alcoholism; level II—denying the effect of parental alcoholism; and level III—excessive use of willpower (while acknowledging parental alcoholism and its effects). What becomes quite clear in Cermak's descriptions of all three levels is that unless the ACAs/Codependents admit to some level of pathology (as defined by Cermak), they are experiencing some level of denial. It is not just any awareness that is the opposite of denial, but it is a well-specified awareness defined by the professionals in the field.

This literature, with its emphasis on becoming aware of the symptoms of codependency in order to begin the recovery process, has also been criticized by several researchers. Asher and Brissett (1988) contend that the spouses of alcoholics (primarily women) get labeled as codependents based on their

"retrospective reinterpretation of their lives with their alcoholic husbands, guided and legitimated by rehabilitation personnel" (p. 331). Taking a social constructionist approach, Asher and Brissett (1988) further argue that this labeling process has served to render these women's identities as deviant and thus medicalize their so-called condition. Thus, the enabler role, described and popularized by the Al-Anon movement, has further evolved and medicalized to the term "codependent" that now, like alcoholism, appears to be an objective medical condition with a legitimate diagnosis. Another perspective would be to view this behavior as one of several in a repertoire of coping behaviors for these women. However, this approach pathologizes these women by rendering them "sick."

Employee Assistance Programs (EAPs) and Denial

In his 1952 article in which he definitively outlines the four key phases of alcohol addition, Jellinek made a critical comment in his discussion of the 43rd and final symptom of alcoholism. As discussed earlier, he contends that the alcoholic must have a failure of her rationalization system during which she can no longer hide from reality and must admit defeat. This phenomenon of "hitting bottom" was interpreted as being the most crucial step in order to begin the recovery process. Yet in Jellinek's discussion of this symptom, he opens the door for other strategies.

> Formerly, it was thought that the addict must reach this stage of utter defeat in order to be treated successfully. Clinical experience has shown, however, that this "defeat" can be induced long before it would occur of itself and that even incipient alcoholism can be intercepted. As the latter can be easily recognized it is possible to tackle the problem from the preventive angle. (Jellinek, 1952, p. 683)

This recognition that the treatment process can begin without "hitting bottom" (or what is sometimes called "raising the bottom") became the rationale for different types of treatment approaches that included court-referral treatment programs and EAPs (Weisner & Room, 1984). The main goal of this approach was early case finding: the idea that some group or institution could actively seek out clients and, through strong persuasion (coercion) tactics, convince them to choose treatment as opposed to another alternative that was much less desirable (e.g., jail or unemployment). The foundation for this approach was the idea that the denial system of the alcoholic was permeable even at earlier stages. Programs found ways to codify their expertise in breaking through this denial and used as their primary strategy a technique called constructive confrontation that is also sometimes called intervention or confrontation.

The rationale for using such a strategy as constructive confrontation at an earlier stage of alcoholism lies in expanding the definition of an alcohol-related problem beyond the individual to a workplace issue (Trice & Roman, 1972). "The basic assumptions of constructive confrontation are that alcohol and drugs have no place in work organizations and that work organizations may legitimately expect unimpaired performances from their employees" (Trice & Roman, 1972, p. 174). Essentially, Trice and Sonnenstuhl (1988) argue that this process borrows from the anthropological literature on studies of drinking within different subcultures in which alcohol-related problems are rare. These studies point to the strength of these cultures to exert strong social controls over their members whenever drinking begins to interfere with role obligations. Thus, constructive confrontation can be defined as a management tool, or creation of a management culture, to maintain strong control over its employees by finding mechanisms to change their behavior (use of alcohol or drugs) if it affects their work performance.

Trice and Sonnenstuhl (1988) define constructive confrontation as a two-part process. The constructive aspect involves discussions that emphasize support and concern about the welfare of the employee, encourage the employee to conform to the workplace standards and policies so that group membership is not jeopardized, and provide concrete suggestions concerning how the employee can achieve a good performance record again. The confrontation part of the process reminds the employee of the work performance standards, informs the employee that if the standards are not met, some type of disciplinary action or sanction will be taken, and "establishes some social distance between the deviant employee and those group members who are meeting expectations" (p. 160).

While this approach has its roots in EAPs (and earlier occupational alcoholism programs) its ability to break through denial has also been utilized by more conventional treatment programs to recruit clients at an earlier stage of alcoholism. This is exemplified in Vernon Johnson's book (1973) titled *I'll Quit Tomorrow*. Johnson referred to the then new strategy to get alcoholics into treatment called "confrontation," which could purportedly break through an assumed denial system and allow the alcoholic to recover. The basic assumption behind this technique was that "even at his sickest, the chemically dependent person can accept reality *if it is presented to him in a receivable form*" (Johnson, 1973, p. 49, italics in original). This "receivable form" is an unexpected group meeting of the alleged alcoholic and his significant others during which time the individuals in the group present the alcoholic with the so-called reality of what his drinking and resultant behaviors have been and how they personally have been affected. It is also assumed that denial of reality is a major part of the

illness itself, necessitating therefore reality to be presented from a source other than the client. Johnson (1973) outlined seven principles for a successful intervention. Essentially, during this process significant others provide evidence that link behavior to drinking. The goal of this process is to move the individual from denial to acceptance of a new reality, the reality of the participants in the intervention. This reality dictates that drinking is the cause of that individual's problems and that, ultimately, treatment is the solution.

This strategy of intervention called confrontation or constructive confrontation was developed to allow treatment personnel to break through (or penetrate) the denial system of an alcoholic at an earlier stage, thereby allowing him to receive help for an alcohol problem before hitting bottom. This approach became the mainstay of occupational programs whose goals were to identify employees with alcohol problems based on job performance and to use the job as leverage to encourage (or coerce) those employees into alcoholism treatment early on in the process. The earliest forms of these occupational programs focusing on identifying employees with alcohol problems were called Occupational Alcoholism Programs (OAPs). OAPs first began in the 1940s, with 6 programs identified in 1945 and more than 4,000 in the United States by 1979. Googins and Godfrey (1987) list the basic rationale for OAPs, which includes an increased recognition that alcohol problems span a wide range of populations, including employed groups; that the costs of alcoholism to employers is high; that the workplace is appropriate for intervention; that treatment of alcohol problems can be effectively integrated into the work environment; and that alcoholism is a disease that is treatable based on the tenets of AA. The idea that alcoholics could be identified and referred to treatment early on in their disease progress was the basic philosophy behind OAPs. Without this emphasis and support for raising the bottom by creating a crisis through confrontation instead of waiting for alcoholics to hit bottom (as prescribed by the disease model), early case finding as a program strategy would not have been an effective tool and therefore sellable to management.

However, creating a crisis for an alcoholic in order to break through the denial system would not be possible if there was not a strong assumption that denial of an alcohol problem is a clear symptom of alcoholism in its early forms. Allen (1984) makes the strong claim that the one factor that "almost all problem drinkers have in common is that of denial" (p. 259). When faced with this denial during the assessment process, counselors are instructed to view this as a symptom of the disease of alcoholism. In other words, denial of one's excessive alcohol use and the problems it causes is to be expected. Counselors are also told that despite this denial, they will still be able to make accurate assessments if they have sufficient knowledge about substance abusers. Thus,

differences that may arise between the counselors' judgments of their employees' problems associated with alcohol and the perceptions of the employees of their own problems associated with alcohol are to be attributed to psychological processes—namely their denial system. What is perhaps one of the most interesting aspects of the use of denial in the case-finding process is the focus on individual attribution for alcohol-related problems and the assumed innocence of any structural, work-related environmental factors that might contribute to alcohol use.

> Denial of the drinking problem, or denial that drinking has any negative effects on job performance, which is often the critical barrier to improvement, can be gently dissolved by objective performance feedback that allows for a conclusion of self-attribution. (Chalmers, 1984, p. 305, italics in original)

Beyond the employees themselves, denial is also discussed as a problem of everyone around them including family members, friends, supervisors, and coworkers, leading them to ignore the problem. In relation to the workplace, the so-called cure for this form of denial is a training program to increase awareness (Scalon, 1991). Campbell and Graham (1988) actually expand the term to include "managerial denial" as a separate chapter in their EAP guide for managers. Within this framework, the managers become strongly implicated in the disease process itself and are thus included in the medicalization and labeling of codependency as a form of deviancy. Within this chapter, they also include a section titled "Breaking through Denial" (of the managers), which further suggests that managers, like their employees with alcohol problems, suffer from the same disease process and therefore need some intervention to break through their denial systems. However, unlike the employee with alcohol problems, constructive confrontation is not the answer for the manager. The treatment for the manager is education in the form of learning about the disease and learning how to document its individual effects in the workplace so that user denial can be broken.

EXPANDING DEFINITION OF DENIAL IN THE ALCOHOL FIELD

That specific forces and groups such as AA, Al-Anon, ACA/Codependency, and EAPs have embraced denial as one of their major defining constructs is not terribly surprising. What is perhaps more intriguing is how its definition has been massaged and expanded over time, going well beyond its original meaning. Hartocollis (1968), for example, is clearly ambivalent about the unconscious nature of denial. He asserts what might be termed "selective denial," meaning

that even though alcoholic patients may minimize the large amount of alcohol that they drink this "does not mean that these alcoholic patients are unaware of the extent to which they use alcohol—they may even boast of the amount of their drinking if with fellow drinkers" (p. 50). Wallace (1986) also questions the unconscious nature of denial, which he further terms "tactical denial." By calling denial a tactic and by defining it as a "common device" used by alcoholics to remain blind to the impact of alcohol on their work, health, reputation, and relationships, Wallace implies a conscious intent that differs considerably from the original definition. This is similar to Paredes (1974) alignment of denial with deceptive maneuvers used by alcoholics to conceal the extent of their drinking.

Haberman (1963) added a sociocultural dimension to the meaning of denial in his study of demographic differences between families who denied or admitted drinking problems. While there were few demographic differences between those who admitted or denied alcohol problems in their family, "Negro Baptists and parents with drinking sons more frequently denied problem drinking" (p. 141). Haberman (1963) concludes that class or cultural differences may account for whether or not alcohol problems are admitted. In a later article, Haberman (1970) used the term "denial" to simply mean saying "none" to an item on a questionnaire in a sample of New York City residents that asked respondents "would you say that you do *too little* or *too much*, or about the *right* amount of drinking alcoholic beverages?" (p. 710, italics in original).

While originally in the psychiatric literature denial was defined as one type of defense mechanism, denial in the alcoholism field has expanded far beyond its original meaning. "Denial includes a variety of ego defense mechanisms, such as rationalization, projection, and avoidance" (Amodeo & Liftik, 1990, p. 131). Anderson's (1981) treatise on denial also expands the definition to include simple denial, minimizing, blaming (projection), rationalizing, intellectualizing, diversion, and hostility. He argues that in order to protect self-esteem, alcoholics are more likely to choose defensiveness as opposed to coping, which he believes is a healthier way to handle threatening situations.

Another way in which denial has been conceptualized in the alcoholism field was presented by Paredes (1974), who noted that denial was part of a highly complex process that he likened to a dramatic performance (similar to *Alcoholism: A Merry-Go-Round Named DENIAL*). In order for alcoholics to maintain their drinking behavior and continue to refuse the label of "alcoholics," Paredes (1974) contends that they must continue to perform in fairly elaborate ways to maintain a status quo in their social and work relationships. Further, Paredes (1974) elaborates that "denial and the accompanying rationalizations and prevarications are part of a creative process requiring imaginativeness and

ingenuity" (p. 25). Thus, it is clear that Paredes also questions the unconscious nature of denial as it was originally intended by taking a somewhat interactional approach.

Finally, Tarter, Alterman, and Edwards (1984) conceptualize alcoholic denial by using a biopsychological approach. Within this framework, they argue that denial can be viewed as part of a cognitive-physiological theory that includes disturbed arousal regulation, cognitive impairment that hinders the ability to discriminate interoceptive cues and physiological states, and cognitive impairment that underestimates emotion-laden events. This unique framework removes denial from the psychiatric perspective and defines it as an interaction of the physical with psychological components and implications. With this approach, the authors argue that interventions aimed at improving cognitive awareness and modifying cognitive style are appropriate as well as interventions that enhance arousal ability, such as relaxation and biofeedback.

Denial Scales

Along with the expanded and somewhat dynamic definitions of denial have come a series of denial scales that purportedly measure this construct. One of the first attempts to quantify denial was by developed by Moore and Murphy (1961) in their study of 100 male veterans who were hospitalized as alcoholic inpatients. The degree of denial was rated at admission and discharge by reviewing treatment reports, notes by nurses and staff members, psychological test results, staff conference opinions, and social service contacts with families. Moore and Murphy utilized a 5-point scale based on the criteria ranging from "Patient saw no reason to decrease drinking while hospitalized" to "strongly wishes to remain abstinent" (pp. 599–600). Of interest and not surprisingly, this scale is disease model–based with no allowance for any rational choice to continue to drink. Differences among each level revolve around varying degrees of awareness by the patient that drinking is harmful and that he or she must stop drinking. It is presumed that once patients are fully aware of their drinking problems (no longer denying), the next step will be a commitment to abstinence. Because the framework is diseased based, it once again implies a linear progression of illness with denial as the single most crucial barrier to recovery.

Crawford (1982) also used a 5-point rating scale of acceptance/denial for alcohol patients obtained by staff. While the wording is slightly different, the assumptions behind each rating are fairly similar to Moore and Murphy (1961) and fit into the disease model perspective. In addition to the acceptance of alcoholism and a commitment to abstinence, Crawford (1982) also adds the dimension of active participation in sobriety. While this is not defined per se, it

also fits into an overall assumption of the disease model that sobriety can only be achieved through constant attendance at Twelve-Step meetings with active participation.

A third denial rating scale, the Denial Rating Scale (DRS) (Goldsmith & Green, 1988), was also developed on a clinic sample and it follows a similar way of assessing denial—namely the so-called awareness factor. However, the DRS differs slightly from the other scales in that it takes a broader approach in an effort to capture both the affective and intellectual understandings of each client concerning his or her alcoholism. In almost a self-actualization approach, the highest level, Level 8, can only be achieved if clients engage in a specific level of self-exploration that allows them to talk about what they are thinking and feeling. Like the other two scales, there is an assumption of linearity with "control" as a key factor. Level 8 can only be achieved if clients admit they cannot control their alcohol use, they are aware that alcohol is a problem in their lives, their problems go beyond alcohol and extend to other parts of their lives, they are willing to self-explore behavioral and affective levels, and they continue to have the self-image of an alcoholic.

While the interrater reliability scores were adequate, the authors attempted to establish construct validity in two ways: by assessing what factors were associated with the initial denial rating and by assessing the association between factors and changes in denial. That the authors assumed these analyses were tests of construct validity is quite puzzling. First, their results indicate that involuntary referrals had higher denial ratings than those who were voluntarily admitted to their program. This is clearly an interesting and expected finding, but it does not bring us any closer to a determination of whether this scale is measuring denial and, more importantly, of whether denial, as they have defined it, is as much the issue as is the fact that clients may conceptualize their alcohol issues differently than the predominant treatment modality at the clinic. Labeling these noncompliant patients as deniers allows the treatment personnel the luxury of not having to reassess their own belief systems concerning alcohol problems, alcoholism, and alcohol treatment. Second, factors that are correlated with change in denial as an indicator of construct validity are very unusual, given that presumably the treatment in the clinic is to break through denial. Given this, changes in the scale should indicate a positive outcome as opposed to a validating the construct of denial. Finally, in an effort to develop a valid and reliable scale on alcoholic defensiveness, Ward and Rothaus (1991) factor analyzed a 94-item scale and found two strong factors: denial and rationalization. What is particularly interesting about their scale is the assumptions behind the following 23 items, which comprise the denial factor. It is interesting to note that all of these items are from a disease model perspective. Thus, if

individuals in the authors' treatment program do not adhere to this model, they would score higher on this denial scale. Consider individuals who have other major problems going on in their lives (e.g. death of a loved one, loss of a home in a fire). Would it not be reasonable for them to answer "No" to items that stipulate that alcohol is one of the biggest problem and still not be in denial? If some individuals have a treatment goal that is not complete abstinence, does this mean that they are in denial? Further, if respondents to this scale have considerable resources, is it also reasonable to assume that continued drinking will lead them to the "gutter" as implied in one of the items?

POLITICAL INVESTMENT IN DENIAL

From the previous sections, it is clear that there are several sets of assumptions about denial that influence how it is has been applied in the alcohol field. First, denial is assumed to be present and a major factor when working with any or all alcoholics. Because denial is considered to be part of the disease of alcoholism, its presence legitimates the label and vice versa when excessive drinking or alcohol problems are suspected or reported. There is no room in this perspective for truth telling from the drinker himself. No stories of alcoholics are offered in the treatment literature that demonstrate early recognition of drinking problems or of excessive consumption by the alcoholic himself. One can only suppose that the label of alcoholic might, in some instances, not be appropriate based on how alcoholism is defined. Further, there is no room in the literature for alcoholics going beyond denial. That is, it is also assumed that alcoholics cannot overstate their drinking problems or their alcohol consumption. The presumption is that there is a continuum with denial on one end and truth telling on the other. Overreporting rarely if ever exists.

Second, the definition of denial in alcohol studies has been expanded well beyond its original meaning to include a wide variety of defense mechanisms as well as undesirable behavior (e.g., hostility) that somehow hampers the identification, treatment, or recovery from alcoholism. Thus, denial has taken on a much broader meaning and becomes the catchall word for any behavior that prevents the adoption of the tenets of the disease model system.

Third, as the denial scales indicate, measurement of denial is directly based on assumptions behind the disease model that attribute all problem areas to alcohol misuse. Individuals who disagree with these assumptions score high on these scales (are labeled "deniers"), but it is unclear whether the scale scores represent real denial, or having a different perspective on one's life than the researcher!

There are several political implications for denial as the primary defining principle of alcoholic behavior. First, because denial is purported to occur early in the disease process, it provides the rationale for earlier intervention. The earlier the intervention can be applied, the more clients can be amassed to assure a lucrative treatment industry. It fits well with an expanding treatment ideology in which

> denial legitimates treating drinkers involuntarily; it insulates and protects the industry from those outsiders who, being products of a cultural denial, are unable to see the magnitude and complexity of the alcohol problem, and it rationalizes and thereby mutes the criticism of the industry by these same misguided outsiders. (Brissett, 1988, p. 397)

It also allows the treatment system an out when treatment fails. Claims can be made that the alcoholic's denial system was too strong and could not be penetrated yet!

Another political issue raised by the use of denial as a main construct in the alcohol field is that there is a strong presumption that individuals around the drinker (e.g., family, counselors, etc.) are not only more knowledgeable about the alcohol problems but also have possession of the true reality, rendering the drinker's reality as false (Brissett, 1988). The assumption is most often that denial will contaminate self-reports. Moreover, the implications of whose reality is correct are enormous when one considers larger issues of social control versus individual control.

DENIAL IN ALCOHOL RESEARCH

The previous discussion has been directed at clinical treatment for alcoholics and research on alcoholic denial also conducted in clinical programs. Treatment evaluation studies in the alcohol field rely heavily on self-reports to assess baseline at treatment entry and then follow-up information usually immediately following the end of a treatment program and for at least one and sometimes several time periods following treatment. Because alcoholic self-reports are suspect by many in the research community, it behooves most alcohol treatment evaluators to provide some measure of self-report validity so that results from the study are not (or are less) suspect.

There is also a vested interest on the part of treatment evaluators and their funding agencies to show that self-reports are almost uniformly valid (albeit under certain conditions), as opposed to less valid, so that findings are taken seriously by the larger scientific community and by the leaders in the alco-

hol field as well. Thus, study after study and review after review report that alcoholics give valid self-reports despite the lack of a gold standard (Babor, Steinberg, Anton, & Del Boca, 2000). Less work is spent on delineating what demographic or social factors may predict less accurate reporting, on developing new ways to improve self-reports, or on why the validity question is being asked in the first place.

Just as importantly, there is little information on what evaluators sometimes call "unanticipated consequences" in validity studies in the alcohol field. That is, because denial is so prominent in alcohol research, little attention is paid to the possibility that overreports may occur, even though it is occasionally mentioned in the literature (Babor et al., 2000). In the early 1970s for my doctoral dissertation, I attempted to develop a model to predict early dropout from a comprehensive alcoholism treatment program and included a validity study for the 65 new entrants (Midanik, 1979). Each of the new entrants was asked in detail about his alcohol consumption during the past 24 hours and then a breath sample was obtained. The subjects did not know at the time of the interview that they would be asked to give a breath sample.

The results were quite surprising. Despite how self-reported blood alcohol concentrations (BACs) were calculated (including very liberal and very conservative estimates of elimination rates), a comparison of self-reported BAC and BAC as derived from breath tests showed that from 23 percent to 57 percent of this sample overreported their recent alcohol consumption (defined as + or – 0.05) (Midanik, 1982). Based on the literature at the time and currently, this finding was counterintuitive to the uniformly held belief that alcoholics deny and thus underreport their drinking.

Multiple explanations can account for these findings including how the questions were asked, the accuracy of the instrument used to validate the responses, assumptions that I made concerning the estimates of BACs from self-reports, and the context of the interview. Perhaps most importantly these findings occurred because the respondents, who were predominantly homeless, chronic alcoholics, were in desperate need of the food, shelter, and hopefully treatment that the program would provide. Thus, by overstating their alcohol consumption level, many probably felt that this ensured entrance into the program (Midanik, 1989).

This phenomenon is not brand new in the broader social science literature or in the alcohol field. Bradburn and Sudman (1980) have described in depth the role demands of the good respondent who assesses quickly the socially desirable behavior within a specific context. The hello-goodbye effect as described in the clinical psychology literature refers to how clients tend to overpresent their symptoms at entrance to treatment and underpresent their symptoms at

termination of treatment (Cave, 1999). Pernanen (1974) wrote from a personal communication from W. Schmidt, that there were instances where individuals reported alcohol consumption that was physiologically impossible without resulting in death for the respondent. Finally, Weisner and Room (1984) noted that the circumstances of treatment may strongly influence the way clients present themselves, particularly in reference to criminal justice clients (who are not drunk drivers) coming to the county alcohol treatment system. Thus, within different treatment contexts there are gains to be made by presenting oneself as being a heavier or a lighter drinker and as having few or multiple alcohol-related problems.

What is disturbing about these findings is that because they run counterintuitive to denial being the guiding force in the alcohol field, little additional research has been done in this area. If new entrants to an alcohol treatment program tend to overreport their recent alcohol consumption, there are important implications for treatment personnel who base treatment decisions on these self-reports. Further, overreports, particularly at baseline, can bias treatment evaluation studies by inflating success rates (presuming there was an opposite bias after treatment). Yet despite the implications of these findings, little interest has been shown by researchers in the alcohol field to explore this area.

BEYOND DENIAL

It is difficult, if not impossible, to imagine the alcohol field without the concept of denial as its major, but not only, legitimate explanation for why treatment was not as successful as hoped or for why our research findings do not fit with either conventional wisdom or with some outside criterion that purportedly is a more valid measure. In a world without the convenience of denial, AA stories would look very different. There would be an early recognition of a problem with alcohol and a full confession that the alcoholic made a rational choice to continue to drink despite this awareness. Treatment providers working with alcoholics might have to acknowledge that their insights may not have a universal allure and simply may not be applicable to everyone deemed to have alcohol problems. Al-Anon groups might have to consider other roles beyond the enabler to describe partners of alcoholics who choose to continue their behavior, admit to it, and thus, make their choices.

However, we do live in a world where denial is an important mainstay in the alcohol field. It has provided a central ideology for the disease model, and in doing so, it has become a kind of shorthand for those who are not quite at the recovery level. Lowney (1999) refers to this overall process as the religion of recovery with five core principles:

> (1) that one's own needs must come before the demands of society, (2) that families emotionally and spiritually wound children during the socialization process, (3) that those scars carry into adulthood, causing us to be stuck in debilitating behavioral patterns, (4) that to heal, we must let go of these private wounds by sharing them with others, and (5) that these patterns are symptomatic of the disease that ails us—codependency. Talking to others similarly hurt, who have "been there," can break the hold of the past that has had such power over the individual and permit the miracle of recovery to commence. (p. 89)

Lowney (1999) argues convincingly that the roots of this individualistic movement are in carnivals and revival meetings in which social problems were displayed as freaks in the sideshow, and the solution to these social problems involves public confession and public acceptance of a strong religious belief system. Recovery, thus, is good, and consequently denial, as the primary identified barrier to recovery (somewhere between steps 3 and 4 above), is considered to be bad.

The ultimate cost of this individualistic ideology is often not discussed. To do so means questioning the status quo and being considered an opponent of this very popular treatment modality that views denial as the enemy even though it has been argued that there may be a positive aspect to denial (Lazarus, 1983). However, this large investment in denial as the main defining feature of alcoholism has allowed clinicians and other people involved in treatment programs along with those who conduct research in the alcohol field to maintain a certain amount of power over clients. One purpose of this chapter was to question the assumption that denial needs to be the central core of alcohol treatment and to argue for more discussion on how denial has shaped research agendas. A reexamination of this assumption can only serve to further treatment and research in this field.

REFERENCES

Al-Anon World Services. (1965). *Al-Anon faces alcoholism.* Virginia Beach, VA: Al-Anon World Services.

Al-Anon World Services. (1985). *Al-Anon faces alcoholism.* Virginia Beach, VA: Al-Anon World Services.

Alcoholics Anonymous (1976). Alcoholics Anonymous: The story of how many thousands of men and women have recovered from alcoholism (3rd ed.). New York: Alcoholics Anonymous World Services.

Allen, S. (1984). Interviewing and problem assessment in the academic work environment. In R. W. Thorenson & E. P. Hosokawa (Eds.), *Employee assistance programs in higher education* (pp. 255–266). Springfield, IL: Charles C. Thomas.

Amodeo, M., & Liftik, J. (1990). Working through denial in alcoholism. *Families in Society*, March, 131–135.

Anderson, D. J. (1981). *The psychopathology of denial* (Professional Education Series #9). Minneapolis, MN: Hazelton Foundation.

Asher, R., & Brissett, D. (1988). Codependency: A view from women married to alcoholics. *International Journal of the Addictions, 23*(4), 331–350.

Babor, T. F., Steinberg, K., Anton, R., & Del Boca, F. (2000). Talk is cheap: Measuring drinking outcomes in clinical trials. *Journal of Studies in Alcohol, 61*(1), 55–63.

Bok, S. (1978). Lying: Moral choice in public and private life. New York: Pantheon Books.

Bradburn, N. M., & Sudman, S. E. A. (1980). *Improving interview method and questionnaire design*. San Francisco, CA: Jossey-Bass.

Brissett, D. (1988). Denial in alcoholism: A sociological interpretation. *Journal of Drug Issues, 18*(3), 385–402.

Broad, W., & Wade, N. (1982). Betrayers of the truth: Fraud and deceit in the halls of science. New York: Simon & Schuster.

Campbell, D., & Graham, M. (1988). *Drugs and alcohol in the workplace*. New York: Facts on File Publications.

Cave, S. (1999). *Therapeutic approaches in psychology*. New York: Routledge Modular Psychology.

Cermak, T. L. (1986). *Diagnosing and treating co-dependence*. Minneapolis, MN: Johnson Institute Books.

Cermak, T. L. (1988). A time to heal: The road to recovery for adult children of alcoholics. Los Angeles, CA: Jeremy p. Tarcher, Inc.

Chalmers, D. K. (1984). Evaluation research. In R. W. Thoreson & E. P. Hosokawa (Eds.), *Employee assistance programs in higher education* (pp. 285–310). Springfield, IL: Charles C. Thomas.

Crawford, R. J. (1982). The use of videotape in the erosion of denial in alcoholism: Pilot. *New Zealand Medical Journal, 95*(701), 82–84.

Crothers, T. D. (1911). Inebriety: A clinical treatise on the etiology, symptomology, neurosis, psychosis and treatment and the medico-legal relations. Cincinnati, OH: Harvey Publishing.

Eidelberg, L. (1968). #414 Denial (disavowal). In *Encyclopedia of psychoanalysis* (pp. 101–102). New York: The Free Press.

Ekman, P. (1992). Telling lies: Clues to deceit in the marketplace, politics, and marriage New York: Norton.

Fingarette, H. (1969). *Self-deception*. New York: Humanities Press.

Fingarette, H. (1988). *Heavy drinking: The myth of alcoholism as a disease*. Berkeley: University of California Press.

Ford, C. V. (1996). *Lies!Lies!!Lies!!! The psychology of deceit*. Washington, DC: American Psychiatric Press.

Freud, A. (1946). *The ego and the mechanisms of defense*. New York: International Universities Press.

Freud, S. (1914a). *On dreams*. London: Heinemann.
Freud, S. (1914b). *Psychopathology of everyday life*. New York: The Macmillan Company.
Googins, B., & Godfrey, J. (1987). *Occupational social work*. New Jersey: Prentice-Hall.
Goldsmith, R. J., & Green, B. L. (1988). A rating scale for alcoholic denial. *Journal of Nervous and Mental Disease, 176*(10), 614–620.
Gould, S. J. (1981). *The mismeasure of man*. New York: W. W. Norton.
Haberman, P. W. (1963). Differences between families admitting and denying an existing drinking problem. *Journal of Health and Human Behavior, 4*, 141–145.
Haberman, P. W. (1970). Denial of drinking in a household survey. *Quarterly Journal of Studies on Alcohol, 31*, 710–717.
Hartocollis, p. (1968). Denial of illness in alcoholism. *Bulletin of Menninger Clinic, 32*, 47–53.
Hinshelwood, R. D. (1989). *A dictionary of Kleinian thought*. Northvale, NJ: Jason Aronson.
Holmes, D. S. (1984). Defense mechanism. In R. J. Corsini (Ed.), *Encyclopedia of psychology* (pp. 347–350). New York: John Wiley & Sons.
Jellinek, E. M. (1946). Phases in the drinking history of alcoholics: Analysis of a survey conducted by the Official Organ of Alcoholics Anonymous. *Quarterly Journal of Studies on Alcohol, 7*, 1–88.
Jellinek, E. M. (1947). Recent trends in alcoholism and in alcohol consumption. *Quarterly Journal of Studies on Alcohol, 8*(1), 1–42.
Jellinek, E. M. (1952). Phases of alcohol addiction. *Quarterly Journal of Studies on Alcohol, 13*, 673–684.
Johnson, V. E. (1973). *I'll quit tomorrow*. New York: Harper and Row.
Jones, J. H. (1993). Bad blood: The Tuskegee Syphilis Experiment. New York: Free Press.
Kellermann, J. L. (1969). *Alcoholism: A merry-go-round named denial* [brochure]. Virginia Beach, VA: Al-Anon Family Groups.
Kurtz, E. (1979). *Not God: A history of Alcoholics Anonymous*. Center City, MN: Hazelden.
Laplanche, J., & Pontalis, J. B. (1973). *The language of psycho-analysis*, (Trans. Donald Nicholson-Smith). New York: W. W. Norton.
Lazarus, R. S. (1983). The costs and benefits of denial. In S. Breznitz (Ed.), *The denial of stress* (pp. 1–30). New York: International Universities Press.
Lewis, M., & Saarni, C. (1993). *Lying and deception in everyday life*. New York: The Guilford Press.
Lowney, K. S. (1999). Baring our souls: TV talk shows and the religion of recovery. New York: Aldine de Gruyter.
Martin, J. E. (1992). The evolution of Al-Anon: A content analysis of stories in two editions of its "Big Book." *Contemporary Drug Problems, 19*(4) Winter, 563–585.
Midanik, L. T. (1979). *Early dropout in two sites of a comprehensive alcoholism treatment program*. Unpublished doctoral dissertation, the Johns Hopkins University, School of Public Health, Baltimore, MD.

Midanik, L. T. (1982). Over-reports of recent alcohol consumption in a clinical population: A validity study. *Drug and Alcohol Dependence, 9*(2), 101–110.

Midanik, L. T. (1989). Perspectives on the validity of self reported alcohol use. *British Journal of Addiction, 84*, 1419–1424.

Milburn, M. A., & Conrad, S. D. (1996). *The politics of denial.* Cambridge, MA: MIT Press.

Milgram, S. (1974). Obedience to authority: An experimental view. New York: Harper & Row.

Moore, R. A., & Murphy, T. C. (1961). Denial of alcoholism as an obstacle to recovery. *Quarterly Journal of Substance Abuse, 22*, 597–609.

Paredes, A. (1974). Denial, deceptive maneuvers, and consistency in the behavior of alcoholics. *Annals of the New York Academy of Sciences, 233*, 23–33.

Patterson, J., & Kim, P. (1991). *The day America told the truth.* New York: Prentice-Hall.

Pernanen, K. (1974). Validity of survey data on alcohol use. In R. J. Gibbins, Y. Israel, H. Kalant, R. E. Popham, W. Schmidt, & R. G. Smart (Eds.), *Research advances in alcohol and drug problems* (vol. 1, pp. 355–374). New York: John Wiley and Sons.

Pernanen, K. (1991). *Alcohol in human violence.* New York: The Guilford Press.

Rosenhan, D. L. (1973). On being sane in insane places. *Science, 179*, 250–258.

Saarni, C., & Lewis, M. (1993). Deceit and illusion in human affairs. In M. Lewis & C. Saarni (Eds.), *Lying and deception in everyday life* (pp. 1–29). New York: The Guilford Press.

Scalon, W. F. (1991). Alcoholism and drug abuse in the workplace. New York: Praeger.

Slap, J. W. (1977). Psychoanalytic theory of defenses and defense organization. In B. B. Wolman (Ed.), *International encyclopedia of psychiatry, psychology, psychoanalysis, and neurology* (vol. 4, pp. 26–30). New York: Aesculapius Publishers.

Tarter, R. E., Alterman, A. I., & Edwards, K. L. (1984). Alcoholic denial: A biopsychological interpretation. *Journal of Studies on Alcohol, 45*(3), 214–218.

Trice, H. M., & Roman, p. M. (1972). *Spirits and demons at work: Alcohol and other drugs on the job.* Ithaca, NY: New York State School of Industrial and Labor Relations, Cornell University.

Trice, H. M., & Sonnenstuhl, W. J. (1988). Constructive confrontation and other referral processes. In M. Galanter (Ed.), *Recent developments in alcoholism* (vol. 6, pp. 159–170). New York: Plenum Press.

Wallace, J. (1986). Alcoholism from the inside out: A phenomenologic analysis. In N. J. Estes & M. E. Heinemann (Eds.), *Alcoholism: Development, consequences, and interventions* (pp. 3–14). St. Louis, MO: The C.V. Mosby Company.

Ward, L. C., & Rothaus, p. (1991). The measurement of denial and rationalization in male alcoholics. *Journal of Clinical Psychology, 47*(3), 465–468.

Weisner, C., & Room, R. (1984). Financing and ideology in alcohol treatment. *Social Problems, 32*(2), 167–188.

This page intentionally left blank

Chapter **3**

The Spirituality of Recovery and the Twelve Steps of Alcoholics Anonymous

Rev. Robert Davis Hughes, III, PhD

The *Twelve Steps of Alcoholics Anonymous*, first written by Bill W. (Wilson) in December 1938 and published in "The Big Book," *Alcoholics Anonymous* in 1939,[1] have become the dominant gold standard in defining the spirituality of recovery. Nowhere is this more apparent than in the fact that programs and treatment centers using other methods, especially secular ones, almost always have to define themselves as non-Twelve-Step approaches. Even these programs usually teach meditation or yoga as tools of recovery. The Twelve-Step community is overwhelmingly the largest single group of successfully recovering persons, and its definition of what it offers as "a program of spiritual recovery," that is "spiritual, but not religious," has not only defined the space in recovery circles but also set a tone for a whole generation's approach to questions of religion and spirituality, often in ways the founders never intended.

HISTORY

Bill W.'s own description of the historical sources of inspiration for his writing is most complete in the essays in *Alcoholics Anonymous Comes of Age: A Brief History of A.A.*, the publication of the addresses at the 20th Anniversary Convention of AA (Alcoholics Anonymous) in St. Louis in July 1955.[2] Culling from the various sources there, we can summarize as follows.

Ebby T. was a friend of Bill W.'s who first brought him a message of hope in November 1934. A friend of Ebby, never named, as far as I know, had been desperately seeking help for his own alcoholism, and visited Dr. Carl Jung

in Zurich in 1930. Jung, the most friendly of the patriarchs of psychiatry to religion and spirituality, had told this person that he was an alcoholic of the hopeless type, who could only be cured by a deep and life-changing spiritual experience akin to religious conversion.[3] In 1934, Bill W. had also received a diagnosis of "hopeless alcoholic" from Dr. William D. Silkworth, a New York psychiatrist working with alcoholics, who became one of the great friends of AA. He described alcoholism as an allergy leading to a compulsive obsession, the beginnings of the disease concept of alcoholism.

In August 1934, having been prepared by the friend who had seen Dr. Jung, Ebby got sober through contact with the Oxford Groups, an English evangelical movement founded by Frank Buchman. The major American exponent of these groups at the time was the Rev. Samuel Shoemaker, at that time Rector of Calvary Church, New York. He became both one of the great friends of AA and also the greatest evangelist of the Episcopal Church in that era. A word of caution is needed here. "Evangelical" at that time certainly meant a brand of protestant Christianity that emphasized a vital and personal experience of God in conversion and a subsequent life of moral earnestness. It did not, however, have associations with what has become known as the religious right in our own time. Indeed, it was usually aligned with progressive and reformist views on social issues. That was certainly true of Shoemaker. The Oxford Groups emphasized a well-established formula: admit you are licked; ask God for help; take stock and confess your defects to another person in confidence; make restitution for the harm you have done; practice sacrificial self-giving; and pray to whatever God you think is there for the power to do all that.[4]

Ebby visited Bill W. in November of 1934 and shared the story of his recovery. By this time Bill was looking for any answer, struggling with the God problem as a result of his scientific training; he decided to have a go, and visited the mission Calvary Church operated on 23rd Street, where Ebby had been lodged. There he responded to the altar call and gave his first testimony, to the initial embarrassment of Ebby! Hopeful at last, Bill returned home but got drunk again the following morning. He had, however, grasped a ray of hope, and he checked himself back into Towns Hospital under the care of Dr. Silkworth. Ebby visited him there and renewed the message of the Oxford Groups. Bill eventually cried out, "If there is a God, let him show Himself! I am ready to do anything, anything."[5] Immediately, he had a major ecstatic experience. Although he felt his life was changed, he still had his modernist doubts; these were quieted by Dr. Silkworth's medical view that such experiences were objectively possible; Bill also read the classic *Varieties of Religious Experience* by philosopher and psychologist William James (who laid out the archetypal description of the born-again experience in modernist, deistic terms), which was deeply

influenced by the Romantic movement.[6] In addition to James's account of the experience of a second birth, Bill W. was deeply influenced by James's description of "complete hopelessness and deflation at depth" as conditions for such an experience.[7] The result, then, is a blend unique to its own time: principles of Christian spirituality expressed in a classical evangelical form, embodied in the Oxford Groups and the ongoing counsel of Sam Shoemaker on the one side, but interpreted by the modernist, psychological approach of Bill W.'s own skepticism and the influence of Carl Jung, William James, and William Silkworth. The initial result was the "six steps" derived from the Oxford Groups but freed from any specific dogmatic content.[8]

The next major event was Bill W.'s trip to Akron and the conversation with the alcoholic Dr. Bob S. (Smith) and his acceptance of the program, which would, in years to come, lead to the treatment of over 5,000 alcoholics in St. Thomas Hospital in Akron by Dr. Bob and his nurse, Sister Ignatia. The beginning of AA is officially dated from this conversation in May 1935, and Dr. Bob's last drink on June 10. The personal piety of Dr. Bob and Sr. Ignatia, another of the great friends of AA, and the influence of the Akron Oxford Groups with which the first Akron group was affiliated, produced a more recognizable version of Christian orthodoxy that included the four Oxford principles: absolute honesty, absolute purity, absolute unselfishness, and absolute love.[9] Indeed, the Akron group remained affiliated with the Oxford Groups longer than the New York group, and the Akron tradition in AA to this day still maintains echoes of this connection.[10]

The situation was more complex in New York. As Bill describes it, there were three subgroups. First were the conservatives, who wanted to maintain the explicitly Christian and biblical focus. Second were the liberals, who wanted to keep the references to God and spirituality but to free the program from any commitment to religious dogma or doctrine. This was partly in response to the observation (still true, I believe) that the purely religious approach produced few cases of significant, long-term sobriety. The third group, the radicals, were a handful of atheists and agnostics who wanted no reference to God at all. They argued for a purely psychological approach, allowing the alcoholic, once lured in, to take the God bit or leave it alone. The groups had agreed that a book, later published as *Alcoholics Anonymous* in 1939, was necessary to tell their story and broaden the audience for recovery. As Bill was drafting the steps, which ended up being expanded from 6 to 12, in December of 1938, the conflict among these groups over the God content reached a pitch, with the radical side being led by Henry, an avowed atheist. As the publication date approached, anxious conversations ensued, until finally a compromise was reached. In step 2, God was described as "a Power greater than ourselves," the language about kneeling

was removed from step 7, and the references to God in steps 3 and 11 were expanded with the now famous phrase "as we understood Him" (Anonymous, 58–60).

While this looks like the triumph of the liberal position, it actually reflects the original Oxford formula as reported by Ebby. Indeed, approval of the book quickly came not only from Sam Shoemaker (who by this time was wrestling with the problems of cult of personality in the Oxford Groups, which would eventually lead to his breaking with them); acclaim also came from the greatest preacher of the time, Henry Emerson Fosdick, in a favorable book review; and a favorable opinion from the Church Committee on Publications of the Roman Catholic Archdiocese of New York was also welcome.[11] This latter stream became embodied in the person of Fr. Ed Dowling, S.J., of St. Louis, in 1940. This was after the publication of "The Big Book" but substantially before the definitive commentary on the steps and traditions, *Twelve Steps and Twelve Traditions*, first published in 1953. Fr. Ed became another of the great friends of AA, and the influence of Sam Shoemaker continued as well. Further work needs to be done on this later text, but the commentary in the 1953 book reflects a good deal of classical spiritual wisdom and even an air of Ignatian influence that can probably be traced to Fr. Dowling. The purpose of toning down the God language in the steps was not to decrease the expectation of real conversion and vital spiritual experience, or even what was seen as the normal return of most members to a practice of a particular religion, but rather the desire to widen "our gateway so that all who suffer might pass through, regardless of their belief or *lack of belief*."[12] Confirmations of the approach came from successful recoveries of practicing Hindus in India and approval of the steps by Thai Buddhists (with a suggestion that "God" be translated "good," but acknowledging that the phrase "as we understood Him" makes the steps acceptable).[13]

Thus the concept was born of a program that was to embody classical principles of Christian conversion compatible with both a Protestant/Evangelical and also a Catholic/Ignatian reading (and, as subsequent events have shown, an Orthodox/Patristic reading) on the one hand, and a modernist, psychological reading open to persons of no faith at all on the other. This tension remains in the movement, usually damped down by the maxim "spiritual but not religious." Within AA itself it is now scarcely controversial and causes few arguments. But as the program is presented to outsiders, some religious conservatives see only a New Age cult, while militant atheists see religion coming in the back door. My own sense is that a postmodern reading is most helpful: the Twelve Steps (and the equally important Traditions) are deliberately written (perhaps as a gift of God) to allow for alternative readings. As a result, we should perhaps not speak

so much of Twelve-Step spirituality as Twelve-Step spiritualities. That not only allows the doubting newcomer to enter, but also allows the earnest recovering seeker to find more defined religious paths of spirituality that are one way of living compatibly with the program.[14]

ANALYSIS OF THE TWELVE STEPS

The definitive original statement of the Twelve Steps is chapter 5 of "The Big Book," *Alcoholics Anonymous* and the definitive commentary is *Twelve Steps and Twelve Traditions* for AA.[15] Other groups, such as Al-Anon and Narcotics Anonymous, which also use the Twelve Steps, have produced their own versions and commentaries, definitive for their reading and practice of the steps. Nothing substitutes for reading the original sources, especially for the person seeking recovery. What follows is an introduction to the spiritual rhythm of the steps, and for simplicity's sake, based on AA only. First, some preliminary points.

Step 1 begins with "We," and implicitly the other 11 do as well. The "We" is the some one hundred people who had found recovery at the time "The Big Book" was first published. It now encompasses a recovery community of several million worldwide. But the point is crucial—this is not a "self-help" program but a program of a therapeutic community. One current expression of this is the combination of therapeutic community treatment (as, for example, in the De Hoop model) with Minnesota model Twelve-Step treatment, a combination currently having great success in Egypt.[16] This "We" reminds the newcomer that the group is an essential component of recovery, a new community of those who are succeeding in recovery following this path. It is not a program of spiritual or moral discipline to be undertaken alone. Even in extreme circumstances in which a person must be solitary for a time, it is a program taken in the company of the worldwide "We." This is an important corrective both for the isolation that is often the result of the disease and for Western solipsism (extreme individualism). It also reminds the newcomers that a social context for their new life of sobriety already exists.

Second, the steps are in the past tense and are suggestions. That means some people have already walked this road to recovery successfully. It also means that admitting, for example, that we are now powerless with unmanageable lives does not mean we need to stay that way forever. The suggestion rubric means, as so often is said in meetings, "take what works for you, and leave the rest, at least for now." It allows each person to find his own path on this broad road. The resultant community is thus more like a postmodern family than a military unit, and indeed the organizational principles in the traditions reinforce that.

A remarkable characteristic of AA fellowship is that of universal support. No one is liked by everyone; no one really in recovery, however, would wish another member would not succeed but go back out. It is the only organization I know of in which everyone really roots for everyone else. Restored fellowship and right relationships begin with the group but eventually extend to the family, the workplace, and the larger social environment as long as people in these locales accept the requirements of the new life of the recovering person, and ideally enter recovery themselves for treatment of codependency issues. In AA, Al-Anon, and other Twelve-Step groups, the concept of sponsorship is a primary focus of this fellowship. The sponsor is a person chosen by the recovering person, usually with more sobriety time than she has, who serves as a kind of *anamcara* (Irish for "soul-friend"),[17] who listens to the story of newly recovering persons and guides them in their practice of the steps while introducing them into the fellowship of the recovery program.

Third is the statement "we claim spiritual progress rather than spiritual perfection."[18] In its immediate context this is designed to reduce the fear expressed as "What an order! I can't go through with it." At the same time, it has deep resonances with a tradition of spiritual growth that found its Christian expression in Gregory of Nyssa's definition of perfection itself as an endless journey of progress, Luther's *simul justus et peccator* (at the same time justified and sinner), and John Wesley's idea of Christian perfection, which was, as we now know, deeply influenced by a reading of Gregory. The one type of spirituality this challenges is the kind that expects moral perfection as an immediate gift following conversion. That approach to recovery from addiction often produces dramatic but short stretches of dryness (abstinence from drinking or drugging). It is my experience that mature pastors, even in denominations that lean in that direction, have come to recognize this problem and the need for the more extensive "discipling" the steps provide. Perhaps the most essential point is this: deep recovery requires spiritual growth, but also spiritual growth for the truly addicted requires recovery, including abandoning the idea that drug-induced states of euphoria are genuine spiritual experiences. Addiction is increasingly seen as a kind of blanket over any other psychological issues preventing therapeutic healing or further developmental growth; it is primary and must be dealt with before other issues can be addressed and normal developmental growth resumed. Even more so for the spiritual growth, the deeper tides of the spiritual life cannot be enjoyed by an addicted person until the primary spiritual issues of addictive disease are addressed.[19]

Turning to the steps themselves, they fall into three groups that express a natural spiritual rhythm certainly familiar in classical Christian expressions

but also held in common with many other traditions: conversion (steps 1–3), cleansing (steps 4–9), and building a new life (steps 10–12).

The first three steps express a process of conversion. Like all good theories of conversion, this must address conversion *from* and conversion *to:* an honest appraisal of the unsatisfactory life one wishes to leave, the realistic hope for a new and different life, and a commitment of trust to undertake that new life. So, step 1 requires from the addict an honest admission of powerlessness over the drug and the resultant disease, and of the havoc of unmanageability in one's life that results. This step is the keystone of recovery, and also the one that has been most controversial. Because the program was originally defined by Bill W.'s apprehension of James's definition of "deflation in depth," the original sweeping application of powerlessness to the lives of the privileged with overweening egos resulted, and conversion was seen largely as repentance. Critics noted that this approach was not suitable for persons whose lives had not allowed them to develop such well-formed egos. Some proposals, usually by well-intended "civilians" (what recovering people call others without the disease) with the interests of the oppressed and the abused at heart, actually compromised the notion of powerlessness to the point of endangering recovery.[20] My own sense is that accepting healing and liberation as legitimate modes of conversion helps more, by allowing the oppressed to develop the selves they have been denied while still acknowledging the powerlessness over the primary obsession of the disease as simply empirically true. Indeed, all conversions involve some of all three modes (repentance, healing, liberation), but different circumstances suggest different balances. The virtue to be learned in this first honest look at one's circumstances is humility, not as humiliation (though the life affected by the disease often brings some of that) but simply as an honest and truthful acceptance of the reality of who one is and the nature of the life one is currently leading.[21] It does, in at least one limited area of life, require acceptance of defeat, and a kind of surrender, but the surrender is not to hopelessness, nor certainly to any oppression and abuse, but to what comes next.

What comes next is "We came to believe that a Power greater than ourselves could restore us to sanity." Step 2. Having admitted the hopelessness of our situation in step 1, "we" now come to hope in something or someone else. Having been defeated by a power greater than we are, we look for one on our side, as it were. Here the tension inherent in the original history is obvious—the Oxford Group side clearly meant to point here to the Christian concept of God, while the more radical New York group wanted no such reference. "Higher Power," as we have seen, became the compromise. The experience of most people in Twelve-Step recovery is that any old sense of a Higher Power will do for a start,

even the group itself, but that this usually evolves into a more specific direction later, though not always. What is essential is the connection between humility and hope in these first two steps. Only true humility leads to the possibility of a realistic hope.

Having come to this belief, or at least having agreed to make the experiment of this belief, "We made a decision to turn our will and our lives over to the care of God *as we understood Him*." In many groups today that final masculine is optional. This is the initial birth of faith out of the dialectic between humility and hope, faith as a trusting commitment in the form a turning over of our will and lives to whatever of God we understand. The experience of many is that this is enough, that whatever Higher Power there is, is happy to work with the feeblest and least understood efforts to turn around in a new direction, and it is "turn around" that is the classic definition and root meaning of "conversion." Many who find the concept of surrender difficult are helped when they see that the step does not require them to surrender will and life to a God who becomes their authoritarian dictator but rather to the *care* (from Latin *caritas*, Greek *agapé*, love) of such a Higher Power, who will take care of them, not possess them or dictate to them.

These first three steps are dominated by what the text later calls three pertinent ideas:

(a) That we were alcoholic and could not manage our own lives.
(b) That probably no human power could have relieved our alcoholism.
(c) That God could and would if He were sought.[22]

The task for soul-friends, sponsors, pastors, therapeutic counselors, or even treatment during these stages is not to try to turn the person around (that is the job of the Higher Power), but rather to *make space* for the person to turn around when he is ready. This will include consistent feedback about the reality he actually faces; help in breaking through the denial about that, which is the first symptom of the disease; assistance in breaking away from communities and relationships that reinforce the denial; and offering the first glimpses of the possibility of a hopeful new life. Recovery groups themselves are often the most helpful wardens of such space.

If we have been stuck in the mud and covered with slime, the first thing we want after being rescued is a good shower. That is the essence of the cleansing in steps 4 to 9, which fall naturally into three groups. In step 4 we make an *examination of conscience*, a fearless and searching moral inventory of ourselves, looking especially for deep character flaws associated with the disease and the actions flowing from them, as well as any deeply held sources of guilt and, equally important, virtues and other assets we may not be fully utilizing.

One of the true strokes of genius, even in the description of this step, is the recognition of resentment as the "number one offender," most likely to get us drunk again. This is not appropriate anger, but anger that is stored and cherished to the point of idolatry. While dealing with resentment, we also note that we are taking our own inventory, not anyone else's. We are responsible only for cleaning up the mess on our side of the street.[23] Or, as it is put in *Twelve Steps and Twelve Traditions*, "It is a spiritual axiom that every time we are disturbed, no matter what the cause, there is something wrong with us."[24] The emphasis is clear: the one thing in a situation, however unacceptable, over which I am surely not powerless, is my own attitudes and behavior. The growing ability of recovering persons to take adult responsibility for their own emotional lives and actions is one of the great gifts of the program.

Having completed this inventory, step 5 asks us to admit "to God, to ourselves, and to another human being the exact nature of our wrongs." Any who have done this will acknowledge both the additional honesty and increased objectivity that is called forth by actually saying these things out loud to another human being; there is also as a rule a deeper sense of being forgiven if someone pronounces it. For those who practice a religion that includes rites of oral *confession*, the label that classical spiritual traditions put on this act, that is recommended; but it should be recalled that this admission came directly from the Oxford Groups, where the tradition was more evangelical: any trustworthy person will do.[25] It is most helpful to recall in steps 4 and 5 that we are looking for "the exact nature of our wrongs," the flaws and defects that are the wellspring of our disease and insanity, much more than a laundry list of everything we have ever done that is wrong or stupid.

At this stage, counselors, therapists, sponsors, and pastors make room for the struggle of the inventory, and support the recovering person through the difficulties while preparing themselves, and any community they influence, to welcome back a newly cleansed and forgiven fellow sufferer. Those authorized by faith communities or called in some other way will also be prepared to hear a 5th step, where the principle art combines a firm commitment to encourage rigorous honesty, completed by a clear ability to assure the penitent of forgiveness and a welcome back to the human race. A deep sense of one's own ongoing defects helps. This is the point at which standard Minnesota Model treatment usually ends, and the newly recovering person is sent back into his old environment with an after-care plan or is encouraged, where appropriate, to undergo extended treatment or a halfway house first.

Having identified and admitted the nature of our wrongs, we turn to what the tradition calls *amendment of life*, in steps 6 and 7. Here we rely on our Higher Power to remove the defects of character we have uncovered in the

previous steps, especially at first those most likely to get us drunk. Again, with great insight, the founders made this process into two steps—first working on the willingness to have these defects removed and facing our own attachment to them. The notion of willingness, as opposed to willfulness, becomes very pertinent here.[26] Indeed, most recovering persons find they need to practice these two steps over and over, as the removal of one set of defects and attachments uncovers another. Then, in step 7, we ask God to remove the defects. This is the first point in the program in which we have actually asked God for something. It is also critical to understand this is not a program of moral self-improvement. Though recovering persons will have plenty of work of their own to do, here we ask God to do it, to remove "every single defect of character which stands in the way of my usefulness to you and my fellows."[27] This is part of what is called the 7th step prayer. The importance of these steps is underlined by the failures many alcoholics have experienced when faced with moralistic demands that have been put upon them, demands they have failed to meet over and over until either despair or rebellious rage become the options. The deep surrender of these steps becomes not a defeat but a kind of relaxing into the care to which we committed ourselves in step 3. The task of counselors, sponsors, and pastors at this point, often different from those who have guided someone through the first five steps, is to reinforce both courage in facing the detachment and deep relaxation in letting go.

In most classic spiritual traditions, the final step of cleansing is *restitution*. Part of taking responsibility for having made a mess is the willingness to clean it up as far as is possible and prudent. Step 8 asks us to "make a list of all persons we had harmed, and become willing to make amends to them all." Rigorous honesty, thoroughness, and willingness continue to be significant. No limits are placed on this list, though it is useful to distinguish people we have actually harmed from those who simply observed and were embarrassed by our jackass behavior. We strive to become willing to make amends to all we have truly harmed, without qualification. Counselors, sponsors, and pastors at this point can encourage thoroughness and accuracy and can help to develop the willingness. The key to the latter emerges most clearly in situations in which the recovering person feels deeply wronged by the other person. Again, the emphasis must be on our own side of the street without seeking to control the action or reaction on the other. Neither here, nor anywhere else in the program, however, should forgiveness be identified with permission to continue abusive behavior. Amends does not include willingness to be further victimized.

That latter notion is one of the prudential elements that a good sponsor, counselor, or pastor can underline in supporting someone in doing step 9 amends, "wherever possible, except when to do so would injure them or oth-

ers." This needed prudence has four elements: (1) Am I focused only on my self and my own actions? (2) Is the timing right? The temptation to try to make amends all at once or prematurely needs to be resisted. Each one requires careful thought and prayer. (3) Is the means chosen for the amends (direct conversation, letter, a quiet restitution) appropriate? (4) Can we be sure that the proposed amends will not harm the other person or anyone else? Once these are worked through, those in support roles can assist the recovering person in finding the courage to make the hard ones and can help them process the results afterwards.

Step 9 concludes the process of conversion and housekeeping, and it is at this point that recovering people are led to expect the beginnings of the deep consolations and benefits of sobriety expressed in what are called "the promises."[28] Here, a significant corner is turned. Up to this point, the focus has been on not using and staying clean and dry. Counselors and the group can support this, but the ultimate responsibility is with the recovering person. One frequently hears in meetings, "We don't promise to keep you sober, but we do promise to ruin your drinking," since no one who has come to understand the disease ever has really good experiences of further use of alcohol or other drugs. As the promises begin to come true, however, something deeper emerges—the new life of sobriety itself. The real turning point toward wellness is when the recovering person turns away from the fears, guilt, and shame of the life left behind and falls deeply in love with the new life of sobriety. Of great significance is giving up any resentment or regret that further successful use of alcohol or drugs is not possible. This is a deep shift from negative to positive regard for the new life, often embedded in the saying "You have to go to meetings until you want to; then you don't have to go anymore, but you will because you want to." No matter how long we are sober, the support of the group helps us maintain and deepen our new lives of sobriety. On a personal note, I have often observed persons succeed in getting clean and dry using other methods than the Twelve Steps, even for extended periods of time. I have never seen anyone turn this corner I have just described except by practicing Twelve-Step recovery, the turn from "I must not drink or use anymore," to "I love being sober." This is the first truly deep spiritual result of the program, and it makes all the others possible.

The remaining steps are designed to support and maintain this new life, though doing so is also necessary for ongoing sobriety. Step 10 continues, in a very Ignatian manner that may, at least in later works, reflect the influence of Fr. Dowling, the process of self-examination on a daily basis, and it contains the important wisdom, "when we were wrong, promptly admitted it." Many have learned that when we delay such admissions, we pay the price in indigestion and other distress. Step 11 encourages daily prayer and meditation "to improve

our conscious contact with God as we understood Him." Those who are able to do so are encouraged to return to, or newly undertake, the disciplines of a particular faith community, and the deeper blessings offered there now become truly available for the first time.[29] In any case, books of daily meditations are provided by various agencies, including AA itself.[30] Much of the spiritual wisdom of the program is condensed in these little books and in *Twelve Steps and Twelve Traditions*.

Step twelve has three parts. "Having had a spiritual awakening as the result of these steps," raises the issues of "spiritual awakening." The definitive discussion of this is Appendix II in "The Big Book."[31] The point most emphasized there is that the concept includes both the sudden, dramatic, Damascus Road types of experience and also the slower, more gradual experiences that are only noticed in retrospect. My own sense is that the best focus is on turning that corner from not drinking or using to being in love with sobriety. The other thing to note is that in focus here is a spiritual experience *as a result of these steps;* one of the hard things that must often be learned is that previous "spiritual experiences," especially under the influence of any mind-altering substances, may well be bogus. Even Bill W. had to learn this the hard way in his experiments with LSD.[32] Here, as in the previous step, the sponsor, counselor, or pastor may find it best to refer the recovering person to a spiritual director versed in the wisdom of at least one great tradition, unless they themselves are so qualified. Discernment can be a key gift at this point.

The second part of step 12 is the best known: "tried to carry this message to alcoholics." This is the "pass it on" step, and the one that led Bill W. to call Dr. Bob in Akron in May of 1935. There is much wisdom in the program about this, most important being avoiding so-called two-stepping: the practice of jumping from a first attempt at step 1 to an annoying and naive effort at carrying the message. Many organizations succumb to the temptation of harnessing the enthusiasm of the new recruit in proselytizing efforts. AA actually forbids this, and postpones carrying the message until the other steps have been worked and a spiritual awakening has occurred. At that point, however, continuing sobriety may depend on willingness to share the message. Care must also be taken for prudential concerns, including gender and sexuality issues, always going in pairs, not getting hooked into expectations for results. "We carry the message, not the alcoholic." Sponsorship itself is seen as one effective way of fulfilling this part of the step.

The third part is "to practice these principles in all our affairs." The tools and wisdom gained in AA or similar programs are for all of life, not merely a cordoned-off sobriety. Here ongoing participation in the group is especially helpful in combating the structures of mental mismanagement arising from

the denial inherent in the disease, the notorious "stinking thinking," which can often result in a dry drunk.

The 12 traditions largely govern the common life of the groups, but tradition 12, and its "backwash" into tradition 11 remind the recovering person that "Anonymity is the spiritual foundation of all our Traditions, ever reminding us to place principles before personalities." This probably grows out of the negative experience of personality conflicts in the Oxford Groups around the time AA was forming, but it also becomes a real protection against false pride in recovery. The traditions also embody the "third legacy" of AA: service, to the suffering alcoholic or addict, the group, the larger organization, and the world.[33] Our recovery is not for own sake alone, but so that we might be of use.

THE SERENITY PRAYER

The so-called Serenity Prayer is used at most meetings and has also assumed an important role in recovery spirituality. No one is quite sure where it came from, though the appearance on a Christmas card of Reinhold Niebuhr's appears definitive.[34] Its primary use is as a tool to discern powerlessness (the things I cannot change) from areas of responsibility (the things I can). Many recovering people have found that repeating it at times of great concern is of immense help and often brings actual serenity.

POSTMODERN CHALLENGES

The Romantic, deistic psychology of William James and Carl Jung was the means at hand in the late 1930s for reading the spiritual wisdom of the Christian tradition in a manner that opened the gateway to recovery of persons of religious faith other than Christian and to persons of little or no faith of any kind. This psychology is no longer widely accepted, at least in its original form, by the medical, scientific, and therapeutic communities, while the Romantic, universalistic deism of James and Jung is under serious critique by postmodern scholars of spirituality and religion, both those who practice a traditional religion and those who do not.[35]

Perhaps the biggest challenge is to recognize the need to move beyond the definition of conversion Bill W. picked up from James as being "deflation in depth"; to recognize that while deflation may still be an appropriate approach to the powerlessness of the disease for persons of relative privilege and strong ego, approaches of liberation and healing are more appropriate for the young, disempowered, oppressed, abused, and even postmodern persons not afflicted with an overweening autonomous ego. At the same time, alternative readings

shaped around the needs of those persons must not compromise the actual powerlessness and unmanageability that does result from the obsession inherent in the disease itself, which is no respecter of persons. Alternative *versions* (as opposed to alternative *readings*) that compromise on step 1 do not as often lead to successful, long-term recovery, in my experience.

The recovery community has largely absorbed the new neuropsychological knowledge of addiction and brain chemistry as a better explanation for the obsession of the disease than the original metaphor of "allergy" (not the later scientific analyses of allergic reactions as these relate to addictions). Newer methods of motivational interviewing show promise in closing the gap between the hard-core abstinence advocates and the proponents of harm reduction. The challenge on the spiritual side, then, is whether the recovery community can also let go of the modernist psychologism of Romantic deism present at the birth of AA and still maintain a community of authentic recovery *spiritualities;* these would be based on a family of acceptable readings of the steps that keep in common the core of what is needed for recovery but that align with the concrete spiritual practices of faith communities that include the original Christian womb of AA and also the broad range of other faiths and communities of spiritual practice, including new religions and persons of little or no faith, as the founders intended.

In large, urban areas with many groups, this may mean some meetings devoted to reading and practicing the steps in alignment with particular faith communities. In the Chicago area, for example, once there was a meeting for atheists and agnostics, there could also be one that read the steps as Christian, as long as both groups kept the traditions. For example, the Christian group *allows* the use of Christian language for "Higher Power" but does not require it; the only requirement for membership is still a desire to stop drinking. One could imagine groups aligned with other faith communities also, as long as they kept the traditions. My own sense is that these groups should be seen as supplementary meetings, with attendance at more inclusive meetings also recommended. The challenge in the inclusive meetings is greatest—how to move beyond the modernist expression of inclusivism as the spiritual generic to a postmodern family of spiritualities of concrete practice, each with an authentic reading and practice of the steps. That would give a whole new meaning to the dictum that the steps define a program of recovery that is "spiritual but not religious."

NOTES

1. Anonymous, *Alcoholics Anonymous* (New York: Alcoholics Anonymous World Services, 1939, 4th ed. 2001). Hereafter cited as *AA*.

2. Anonymous, *Alcoholics Anonymous Comes of Age: A Brief History of A.A.* (New York: Alcoholics Anonymous World Services, 1957). Hereafter, *AACOA*.
3. Bill W. later verified this conversation in correspondence with Jung in 1961. *AACOA*, ix.
4. *AACOA*, 58–59, 62–63.
5. *AACOA*, 63.
6. *AACOA*, 64. Romanticism is the nineteenth-century artistic and philosophical movement that emphasized refined emotions over the Enlightenment commitment to autonomous reason, which is the essence of modernism. Deism is the philosophy that there is a generic God or power available to philosophy, who is the real truth expressed mythically in revealed religions. See William James, *The Varieties of Religious Experience: A Study in Human Nature* (London: Longmans, 1903). The critique of James's modernism and Romanticism is explicit in Nicholas Lash, *Easter in Ordinary* (London: SCM, 1988); see also Grace M. Jantzen, *Power, Gender, and Christian Mysticism* (Cambridge, UK: Cambridge University Press, 1995); and Denys Turner, *The Darkness of God* (Cambridge, UK: Cambridge University Press, 1995). For an ongoing critique of Romanticism and the "Philosophia Perennialis" in spirituality, see Owen C. Thomas, *What Is It That Theologians Do, How They Do It and Why* (Lewiston, NY: Edwin Mellen, 2006).
7. *AACOA*, 64.
8. *AACOA*, 160.
9. Bill W.'s own description of these influences on the writing of the 12 steps is in *AACOA*, 160–65.
10. For a consistent reading of AA as fundamentally Christian, and of Bill W.'s experience of Christian conversion, see the several works of Dick B. (Richard Burns), available at http//www.dickb.com.
11. *AACOA*, 168–69; Dr. Fosdick's review is available in Appendix E: d, 322–24.
12. *AACOA*, 167, italics original.
13. *AACOA*, 80–81.
14. Postmodernism is a large body of diverse critical theory that questions the assumptions of modernism, especially the possibility of a grand, definitive narrative and an autonomous ego defined principally by reason. Postmodernism emphasizes the way different communities of interpretation can produce equally valid readings of the same text. The addresses of Fr. Dowling and Dr. Shoemaker are good examples of open-ended Christian readings of the steps; *AACOA*, 253–71.
15. *AA*, 58–71; Anonymous, *Twelve Steps and Twelve Traditions* (New York: Alcoholics Anonymous World Services, 1953), hereafter *12X12*.
16. For the De Hoop community in Holland see http://www.dehoop.org; "Minnesota Model" refers to the AA-based treatment that developed at Hazelden and other locations in that state; for information on the International Substance Abuse and Addiction Center of Studies treatment and training center in Egypt, see http://www.ahram.org.eg.

17. See the entry, "Direction, Spiritual," in Philip Sheldrake (Ed.), *New Westminster Dictionary of Christian Spirituality* (Louisville, KY: Westminster John Knox, 2005), 243–45 and the bibliography there.
18. *AA*, 60.
19. See Robert Davis Hughes III, *Beloved Dust: Tides of the Spirit in Christian Life* (New York: Continuum, 2008), for this image of spiritual tides and a reading of them from a Christian perspective that also includes much garnered from 12-step wisdom, and also an analysis of perfection as progress in the classic sources.
20. The earliest source I can find is Gail Unterberger, "Twelve Steps for Women Alcoholics," *The Christian Century*, December 6, 1989, 1150–52.
21. For an extended discussion of these issues about conversion and engagement with the psychological and theological literature, see Hughes, *Beloved Dust*, 71–248.
22. *AA*, 60.
23. *AA*, 64–67.
24. *12X12*, 90.
25. *AA*, 74.
26. For a brilliant exposition of this concept in psychology and spirituality, see Gerald May, *Will and Spirit: A Contemplative Psychology* (San Francisco: Harper & Row, 1982). Dr. May's identification of addictive disease as "the sacred illness of our time" found a definitive expression in Gerald May, *Addiction and Grace* (San Francisco: Harper & Row, 1988).
27. *AA*, 76.
28. *AA*, 83–84.
29. For my own account of the Christian expression of these possibilities, see Hughes, *Beloved Dust*, 249–396.
30. For the latter, Anonymous, *Daily Reflections* (New York: Alcoholics Anonymous World Services, 1990).
31. *AA*, 567–68.
32. A pair of definitive texts on this subject, from a Christian point of view, are R. C. Zaehner, *Mysticism, Sacred and Profane: An Inquiry into Some Varieties of Praeter-Natural Experience* (Oxford: Clarendon, 1957); and *Zen, Drugs, and Mysticism* (New York: Pantheon Books, 1973).
33. See *AACOA*, 139–221.
34. See *AACOA*, 196.
35. Again, see the references in note 6 above.

BIBLIOGRAPHY

Anonymous. *Alcoholics Anonymous*. New York: Alcoholics Anonymous World Services, 1939 (4th ed. 2001).

———. *Twelve Steps and Twelve Traditions*. New York: Alcoholics Anonymous World Services, 1953.

———. *Alcoholics Anonymous Comes of Age: A Brief History of A.A.* New York: Alcoholics Anonymous World Services, 1957.
———. *Daily Reflections.* New York: Alcoholics Anonymous World Services, 1990.
Hughes, Robert Davis III. Beloved Dust: Tides of the Spirit in Christian Life. New York: Continuum, 2008.
James, William. *The Varieties of Religious Experience: A Study in Human Nature.* London: Longmans, 1903. A good critical edition is that introduced by John Smith, (Cambridge, MA: Harvard University Press, 1981).
Jantzen, Grace M. *Power, Gender, and Christian Mysticism.* Cambridge, UK: Cambridge University Press, 1995.
Lash, Nicholas. *Easter in Ordinary.* London: SCM, 1988.
May, Gerald. *Will and Spirit: A Contemplative Psychology.* San Francisco: Harper & Row, 1982.
———. *Addiction and Grace.* San Francisco: Harper & Row, 1988.
Sheldrake, Philip (Ed.). *New Westminster Dictionary of Christian Spirituality.* Louisville, KY: Westminster John Knox, 2005.
Thomas, Owen C. *What Is It That Theologians Do, How They Do It and Why.* Lewiston, NY: Edwin Mellen, 2006.
Turner, Denys. *The Darkness of God.* Cambridge, UK: Cambridge University Press, 1995.
Unterberger, Gail. Twelve Steps for Women Alcoholics. *The Christian Century,* December 6, 1989, 1150–52.
Zaehner, R. C. *Mysticism, Sacred and Profane: An Inquiry into Some Varieties of Praeter-Natural Experience.* Oxford: Clarendon, 1957.
———. *Zen, Drugs, and Mysticism.* New York: Pantheon Books, 1973.

This page intentionally left blank

Chapter **4**

Addictions and Free Will: A Structured Exploration of the Boundaries of Personal and Societal Responsibility

Julian G. Martins, MA, MB, BS, and
Basant K. Puri, MA, PhD, MB, BChir, MRCPsych, MMath

In the 1960s . . . two Supreme Court holdings . . . set forth a legal distinction between status (having the disease of addiction) and behavior (the acts performed by the addicted person). I suggest that these judicial decisions remain relevant, that the "disease model" of addiction does not excuse all harmful addiction-induced behavior, and that the addict's acceptance of his or her disease and entering into therapy is just the bright line that the law seeks in order to establish responsibility.

—Cohen (2007)

Whether addiction is best understood as a brain disease or a moral condition . . . is often centered on the question of whether and to what degree we can justly hold addicted individuals responsible for their actions . . . [however,] it may be wise for societies to err on the side of holding addicted individuals responsible for their behavior . . . [but] it should be with the view to rehabilitation of the addicted person and protection of society rather than moral opprobrium.

—Hyman (2007)

Our penal system is highly counter-productive from a consequentialist perspective, especially in the USA, and yet it remains in place because retributivist principles have a powerful moral and political appeal . . . It is possible, however, that neuroscience will change these moral intuitions by undermining the intuitive, libertarian conceptions of free will on which retributivism depends.

—Greene and Cohen (2004)

The preceding quotations from recent work considering the boundaries of personal responsibility in addiction summarize the approach to be adopted in this brief overview of the subject. Notably, we are establishing a model of responsibility in addiction that seeks to both foster the development of personal responsibility and to also take into account recent findings from neuroscience. These seemingly juxtaposed positions—responsibility versus neuroscience—affect the roles of therapeutic, societal, and legal responses to addiction in either helping or hindering the development of the very personal responsibility we seek to foster.

It is beyond the scope of this chapter to provide a detailed analysis of the philosophical underpinnings of the nature of free will, but it may be helpful to provide a brief overview of the central concepts. Three principal models have been advanced to conceptualize free will: determinism, libertarianism, and compatibilism. In the deterministic model, free will is essentially an illusion, and human choices are a result of predetermined genetic, environmental, neurocognitive, and random influences over which the individual has no ultimate control. In the libertarian model, human choices are held to be entirely the responsibility of the individual concerned, independent of any outside influence. In the compatibilist model, which tries to reconcile these two opposites, acknowledgement is made of the fact that certain mental deficiencies—for example, in legal insanity and infancy or, in neuroscientific terms, lack of cognitive or affective control—might undermine the capacity for free choice. With respect to the logical response to individuals who transgress societal norms, in the deterministic model punishment makes no sense at all, whereas in the libertarian model, retributive punishment is easily justified on the grounds that the individual has willfully and freely chosen to act in a criminal fashion. Currently, the law tends to favor the libertarian perspective with mitigation being allowed only under extreme circumstances—for example, insanity, duress, and certain cases of extreme provocation. With advances in neuroscience, however—for example, the substantial impairments in cognitive control (the ability of an individual to suppress impulsive acts and rationally appraise alternative choices)—that are emerging from research in addiction, the boundaries of the legal response to such problems are likely to be shifted more to the compatibilist perspective, in which case retributive responses become harder to justify. Greene and Cohen (2004) argue for what they describe as a forward-looking consequentialist approach to these problems in which holding people personally responsible for their actions is useful only in so far as it serves to promote the individual's ability to regain self-control through appropriate access to treatment, and to provide the justification necessary for the protection of society through temporary containment. They argue that holding people

responsible simply to justify a retributive response is not scientifically tenable, as in reality there will always be certain factors that can be identified from a person's background that render this response suspect:

> Intuitively, we want to punish those people who truly deserve it, but whenever the causes of someone's bad behavior are made sufficiently vivid, we no longer see that person as truly deserving of punishment. This insight is expressed by the old French proverb: "to know all is to forgive all." (Greene & Cohen, 2004, p. 1783)

Against the background of these philosophical considerations, models of responsibility in addiction will be summarized together with their respective advantages and disadvantages. The overall shortcomings of these models will then be discussed, with particular reference to how societal responses that purport to tackle problems of an addictive nature might actually increase the overall burden of such problems through unforeseen systemic mechanisms. Finally, an alternative model of responsibility in addiction, as a further development of the aforementioned forward-looking consequentialist model, will be advanced, together with a discussion of possible new research methodologies that might be employed to lend scientific weight to the establishment of alternative approaches to the problem of addiction and related behavioral disorders.

MODELS OF RESPONSIBILITY IN ADDICTION

Addiction as a Disease

Modern definitions of addiction focus on the disorder as compulsive use despite ongoing negative consequences (American Psychiatric Association, 2000), rather than necessitating the presence of tolerance and withdrawal syndromes, as important drugs of abuse such as cocaine have little or no associated physical dependency syndromes. Despite the fact that this view of addiction is defined in somewhat tautological medical terms, researchers have amassed considerable evidence to support a disease model (Leshner, 1997; McLellan, Lewis, O'Brien, & Kleber, 2000) ranging from known risk factors, to a typical relapsing course, to associated genetic risk factors (Goldman, Oroszi, & Ducci, 2005; Hurd, 2006). Recent advances in cognitive neuroscience have begun to reveal the mechanisms that underpin the characteristics of addiction, notably how repeated episodes of drug taking consolidate behavior into compulsive use, how the risk of relapse from a drug-free state can persist for years, and how drug-related cues come to control behavior. Common to most if not all drugs of abuse is the release of dopamine from pathways in the brain that,

rather than stimulating pleasure directly as was once thought, serve to govern how the brain interprets signals of reward positively or negatively (incentive salience), which then shape how the brain remembers what was most rewarding (stimulus-reward learning) and how the brain then selects which rewarding goals to pursue (stimulus-action learning). There is increasing evidence that drugs of abuse lead to long-term neurological changes in synaptic plasticity, neural structure, gene expression, and protein synthesis that consolidate these patterns of behavior (see Hyman, 2005; Hyman, Malenka, & Nestler, 2006 for detailed reviews). Being an abnormally potent and consistent stimulus for dopamine release, as opposed to natural rewards such as food, shelter, finding a mate, and leisure activities, drugs of abuse usurp these natural rewards in a manner eloquently described by Hyman (2007, p. 10):

> Addictive drugs always signal "better than expected." Neural circuits "overlearn" on an excessive and grossly distorted dopamine signal . . . Cues that predict drug availability such as persons, places, or certain bodily sensations gain profound incentive salience and the ability to motivate drug seeking. Because of the excessive dopamine signal in the prefrontal cortex . . . drugs become overvalued compared with all other goals. Rational goals such as self-care, working, parenting, and obeying the law are devalued. In addition, normal aspects of cognitive control weaken; even if the addicted person wants to "cut down." prepotent cue-initiated drug-seeking responses are extremely difficult to suppress. If the person is successful in delaying drug seeking (or is, for external reasons unable to seek drugs), intense craving may result . . . Because the changes in synaptic weight and synaptic structure that underlie memory are among the longest-lived alterations in biology, the ability of drug-related cues to cause relapses may persist for many years, even a lifetime.

In addition to drug-induced changes resulting in reduced cognitive control, preexisting deficits in prefrontal lobe cortex function resulting either from injury (Damasio, 2007) or from developmental origins, particularly in the area of poor regulation of mood (Charland, 2007), both increase the risk for initiation, maintenance, and relapse of drug-taking, lending further weight to the disease model of addiction.

The modern definition of addiction as compulsive use despite negative consequences has led to the inclusion of certain categories of non-drug-related compulsive behavior under the umbrella of the "behavioral addictions" such as gambling (Potenza, 2006) and compulsive sexual activity (Bancroft & Vukadinovic, 2004; Goodman, 2008; Grant, Brewer, & Potenza, 2006; Mick & Hollander, 2006). This remains controversial, however, particularly in the situation of behaviors that attract strong moral opprobrium, such as sex offending that may be associated with compulsive sexual activity in a propor-

tion of cases, as concern is often expressed from statutory agencies that such a formulation may result in diminution of the offender taking full responsibility for his or her actions. This concern has, on occasion, required offenders to sign agreements prior to entry into therapeutic programs to the effect that sex offending is not an illness, it is a choice. Whilst in the majority of cases this is undoubtedly the case, and the goal of encouraging the taking of full personal responsibility is essential for long-term recovery, such categorical stances might be counterproductive in that they could inhibit an offender's ability to feel free to disclose behavior that he or she perceived to be significantly underpinned by compulsive elements. On the other hand, in other circumstances, particularly when the perceived risk to the public is high, statutory agencies abandon the choice model and call upon the medical model to justify other interventions.

This dualism in approach is strongly illustrated by the fact that, in the United States, a diagnosis of mental illness under the category of *DSM-IV* paraphilia was used as the initial justification for enacting civil commitment laws to detain violent sex offenders indefinitely following the end of their sentences, which, following a U.S. Supreme Court judgment *Kansas v. Hendricks* (1997), have been extended to include those without evidence of mental disorder (La Fond, 2000, 2008). In the United Kingdom, the prevailing view is that sex addiction plays no part in sex offending, although recently there has been some acknowledgement that medical components may play a role in sex offending in terms of the possible benefits of selective serotonin reuptake inhibitor (SSRI) antidepressants and of hormone treatment. More specifically, there has been increasing recognition that conventional cognitive behavioral group therapy programs do not sufficiently address the needs of a subgroup of offenders who exhibit what is described as obsession with sex or deviant sexual interest. As a result, a new therapeutic program, the Healthy Sexual Functioning Program, comparing specialized psychological intervention with SSRIs in a randomized controlled trial methodology is currently underway in the United Kingdom. This program seeks to address the compulsive elements that may underpin sex offending in a proportion of cases (*HSF-SSRI Trial*, n.d.).

The potential advantage of addiction being seen as chronic, relapsing brain disorder is that it is able to reduce the stigma associated with these problems, allow earlier access to more effective treatment programs, and avoid the overuse of ineffective and costly prison sentences (Carter & Hall, 2007). In fact, not only is prison ineffective at tackling drug problems, it is also a high-risk environment for drug initiation. A recent UK study showed that even in the absence of psychological vulnerability, a prison sentence substantially increases the risk of acquiring a new drug habit on leaving prison that did not exist before simply

because of factors such as boredom and the ubiquitous availability of drugs in penal establishments (Boys et al., 2002).

Contrasting with this there have been some unwelcome disadvantages of the disease model of addiction, notably the rapid detoxification regimen that offered hopes of a long-term cure for heroin addiction only to be associated with a substantially increased risk of death from overdose following drug resumption (Hall, 2000). Other examples include: the neurosurgical ablation of the nucleus accumbens to treat addiction on subjects in penal settings in Russia and China where informed consent is doubtful (Hall, 2006), calls for compulsory immunization (Hall, Carter, & Morley, 2004); genetic testing (Sherman, 2006) to target those vulnerable to drug use; and, the more widespread use of coercion and civil commitment for these problems (Hall et al., 2004). Finally, from the point of view of taking responsibility for behavior, the disease model could undermine the development of this responsibility and inadvertently worsen the prognosis for change. These issues will be further discussed later.

Addiction as a Moral Condition or Free Choice

Other authors argue that addiction is a moral condition in that addicts are making a free choice to behave in the way that they are because self-control can be achieved in certain contexts, for example, under the threat of coercion (Satel, 1999), or in the instance of returning Vietnam veterans who in the vast majority of cases were able to simply (apparently) discontinue their heroin addiction indefinitely (Robins, Davis, & Goodwin, 1974; Robins & Slobodyan, 2003), with some doing so through a process of maturing out (Winick, 1962). They also cite that addiction involves a complex series of voluntary acts to achieve the goal of drug taking and cannot be simply regarded as impulsive. An extreme example of this position has been put forward by Foddy and Savulescu (2007), who claim that addictive desires are merely pleasure-orientated desires not qualitatively different from any other preference except in strength, their tendency to tap into direct biological sources of pleasure, and the tendency for them to be socially unacceptable. These authors do not argue that addictive choices are moral ones per se, simply that they are chosen freely because they are a source of pleasure among many other alternatives. To justify this position they argue that there is a long history of the medicalization of behaviors deemed socially unacceptable, for example the so-called disease of drapetomania described by Cartwright (1851) to characterize the behavior of slaves that ran away from their masters. They also provide justification in terms of the fact that not enough is known about the neurobiology of normal desire in that the neuroadaptive changes described in addiction may actually be similar for

any normal preference if that were to be studied in as much detail as addictive preferences have.

Although this position has the advantage of promoting a strong expectation of personal responsibility for behavior, it carries with it the risk of an excessive retributive response to addictive problems. Also, the arguments put forward in favor of this perspective have scientific flaws in that it is well known that the circumstances in which an addiction is acquired strongly influence the specific cues that trigger relapse; therefore, the data on the low incidence of relapse in returning Vietnam veterans is not valid as indicative of self-control in these subjects in that back in the United States the veterans were simply not exposed to the kinds of cues that might have triggered a relapse. In addition, it is becoming increasingly recognized that semiautomatized complex behavioral sequences can be enacted following exposure to drug-related cues through the mechanism of stimulus-action learning described previously. Finally, data from life-course perspective studies of drug and alcohol use, to be described later in this chapter, reveal a picture of a chronic relapsing condition with very significant morbidity and mortality (Hser, 2007) and with an increased risk of relapse in untreated natural recovery groups (Moos & Moos, 2007), which makes it hard to equate addiction with a purely free choice model.

Addiction as a Social Ill

Levy (2007) argues that in addition to considering factors that relate to the individual addict's capacity for self-control or otherwise, due weight needs to be given to the myriad of influences from the social environment that either ameliorate or exacerbate addictive problems. For example, there is a consistent relationship between the relative price of alcohol as determined by taxation level and the extent of alcohol-related harm in any given society (see Chaloupka, Grossman, & Saffer, 2002 for a review of studies). As a possible mediator of this relationship, Levy (2007) gives the example of studies that have examined the ability of normal subjects to exercise self-control in the context of a continuing demand for restraint. Essentially, the longer subjects have to exercise self-control in one task, the less likely they will be able to persist in a subsequent independent task involving the need to exert self-control. This phenomenon has been termed *ego depletion* (Baumeister 2002; Baumeister, Bratslavsky, Muraven, & Tice, 1998) and illustrates that self-control is a limited resource on exposure to the continuing need for self-restraint. The advantages of this approach are that it clearly points to various social policy objectives that could be implemented to reduce the overall burden of addictive problems in a given society by reducing the availability of harmful substances and opportunities

to enact addictive behaviors, but again the disadvantage is that it could serve to deflect from the development of individual responsibility of the addicted person for their recovery.

Addiction as a Variable Lack of Rational Capacity

Morse (2007) argues for a position between the extremes of the brain disease and free choice models by promoting the concept of variable lack of rational capacity in addiction. This concept is similar to that of diminished cognitive control as described by Hyman (2007). Morse, however, sees this lack of rational capacity as a more fluid concept that might wax and wane depending on a variety of factors such as the extent of neurocognitive brain maladaptation as a result of the drug taking itself, the individual's affective state, the presence or absence of social support, or other factors. He recognizes that during periods of active drug taking, an addict's rational capacity might be severely impaired, but during periods of abstinence, the addict should be held responsible for future episodes of nonresponsible behavior; a phenomenon known as *diachronous responsibility* in philosophy. Other authors have enlarged on this concept, stating that just because there is strong evidence for a brain disease element in addiction this does not obviate the addict from all responsibility. Cohen (2007) cites other disease states where this is the case. For example, epilepsy is undoubtedly a brain disease that requires sufferers to nevertheless curtail certain behaviors such as driving if this would pose a risk to themselves or others. Similarly, he cites the case of HIV-infected individuals who may have to refrain from certain forms of sexual activity in order to prevent them from harming others. Cochrane (2007) adopts a similar interactional perspective, arguing that trying to claim that addiction is either a brain disease or a moral free choice is a false dichotomy because both perspectives are useful to achieve worthwhile goals; the neurobiological in terms of greater understanding of the addict's predicament and opportunities for the development of new effective therapies, the moral in so far as it can help to motivate addicts to muster sufficient rational capacity to take responsibility for their actions.

What is increasingly clear, however, is that good rational capacity or self-efficacy, the self-belief that an addict has the capacity within herself to take responsibility and maintain abstinence, is consistently one of the best predictors of long-term abstinence in numerous studies. For example, in the well known study Project MATCH (matching alcoholism treatments to client heterogeneity) comparing Motivational Enhancement Therapy (MET), Cognitive Behavioral Therapy (CBT), and Twelve-Step Facilitation (TSF), readiness to

change and self-efficacy were the strongest predictors of long-term drinking outcome (Project MATCH Study Group, 1998).

Consideration of the many studies that have been published by Project MATCH provides some valuable insights as to how self-efficacy might be promoted in differing groups of people with addictive problems. The self-efficacy study was originally designed to test the hypothesis that it would be possible to allocate subjects presenting with alcohol-related problems into one of the three previously described treatment conditions on the basis of their characteristics at recruitment. For example, it was predicted that subjects with high initial anger would respond best to MET, subjects with psychiatric symptoms to CBT, and subjects with strong network support for drinking to TSF, an intervention that encouraged participants to attend Alcoholics Anonymous (AA). Overall results did not support the matching hypothesis in that broadly speaking the outcome was similar in all of the three groups studied. However, more fine-grained statistical analyses revealed that the subjects with the most anger at presentation did respond preferentially to MET (Project MATCH Study Group, 1998), that subjects with the highest levels of network support for drinking did respond better to TSF (Project MATCH study group, 1998), and, crucially, that subjects with low initial self-efficacy (i.e., those one would expect to fare the worst) responded best if they received CBT (Witkiewitz, van der Maas, Hufford, & Marlatt, 2007) and if the therapist was able to establish a strong therapeutic alliance with the client (Ilgen, Tiet, Finney, & Moos, 2006). Self-efficacy was also found to both mediate the relationship between AA attendance and positive drinking outcome as well as AA attendance itself having positive effects on the development of self-efficacy (Bogenschutz, Tonigan, & Miller, 2006; Connors, Tonigan, Miller, & MATCH Research Group, 2001).

However, a note of caution is necessary concerning the Twelve-Step approach, as there is evidence that, because of the strong emphasis on spiritual beliefs in AA, atheist or agnostic alcoholics are far less likely to initiate or sustain contact with AA than those with preexisting religious beliefs (Tonigan, Miller, & Schermer, 2002). In addition, the first three steps "we admitted we were powerless over alcohol—that our lives had become unmanageable," "came to believe that a Power greater than ourselves could restore us to sanity," and "made a decision to turn our will and our lives over to the care of God as we understood Him" together with AA's understanding of alcoholism as a disease (Alcoholics Anonymous, 1953) could have the tendency to undermine the sense of personal responsibility for one's actions in the long-term despite increases in self-efficacy in the short-term. There is some evidence that this

might be the case, as self-efficacy and not AA attendance substantially reduced the risk of relapse in a 16-year follow-up study of alcoholics (Moos & Moos, 2007). Alternative self-help organizations not using the Twelve-Step approach have more recently emerged, namely SMART Recovery (http://www.smartrecovery.org/) and Rational Recovery (http://www.rational.org/), that are based on secular and scientific principles. Drawing on research evidence of the use of MET, CBT, and Rational Emotive Behavior Therapy, SMART Recovery (Self Management and Recovery Training) views addictive behavior as a dysfunctional choice rather than a disease, although it acknowledges that certain people for genetic and neurobiological reasons may have a predisposition to addiction. It divides the process of recovery into four phases—building motivation, coping with urges, problem-solving, and lifestyle balance—and aims to provide participants with the cognitive tools to deal with these challenges as well as support through facilitated meetings. Rational Recovery places even greater emphasis on self-efficacy and is based on a cognitive technique of challenging the so-called addictive voice (internal distorted thinking that supports intoxication). It does not view addiction as a disease but as a voluntary choice, and it discourages the adoption of the forever recovering alcoholic persona that can be a consequence of prolonged AA attendance. As yet, these two secular approaches have not been studied in as nearly as much detail as AA participation, and it remains to be seen as to whether they, in combination with professional interventions, offer the opportunity for greater development of self-efficacy in the long-term than Twelve-Step approaches.

PROBLEMS WITH THESE MODELS

The aforementioned models of responsibility in addiction have certain weaknesses. With the exception of the variable rational capacity model, they tend to view addiction in exclusive rather than inclusive terms; in other words, addiction is either completely a brain disease or a completely free choice, whereas the consensus view that is emerging is that the risk for developing an addiction is multifactorial. In addition, these models tend to underemphasize the developmental and life-course perspective on addictive problems; they tend to focus on relapse prevention and negative outcomes rather than on what promotes a good life and long-term positive outcomes. Finally, they also tend to underacknowledge the reciprocal relationship between societal responses, social policy, and legal perspectives on the likelihood of positive versus negative outcomes in addictive behaviors. These problems will be briefly reviewed in the following sections, followed by a description of an alternative comprehensive model of responsibility in addiction.

The Developmental and Life-Course Perspective on Addiction

One of the first studies to examine the developmental antecedents of drug use was the classic study by Shedler and Block (1990), who followed up a cohort of subjects from preschool to 18 years to examine what factors predicted subsequent drug use. They found that the subjects fell into one of three groups: the experimenters who had the best psychological adjustment at 18, the abstainers who tended to be anxious and lacking in social skills, and the frequent drug user group who showed a distinct behavioral profile of interpersonal alienation, poor impulse control, and emotional distress. Membership of these groups was predicted by the quality of parenting received in early childhood, and therefore these authors argued that drug use was a symptom, not a cause, of an underlying developmental problem of self-regulation stemming from suboptimal parenting experiences. Since this original study, the notion that experimenters were better adjusted than abstainers has been refuted (Milich et al., 2000; Tucker, Ellickson, Collins, & Klein, 2006), but numerous other studies have confirmed the association between adverse early life experience including abuse, social disadvantage, poor parenting, parental substance misuse, and delinquent peer affiliation with subsequent problems with behavioral self-regulation and substance misuse (Castro, Brook, Brook, & Rubenstone, 2006; Doherty, Green, Reisinger, & Ensminger, 2008; Fergusson, Horwood, Boden, & Jenkin, 2007; Fothergill & Ensminger, 2006; Kliewer & Murrelle, 2007; Marcenko, Kemp, & Larson, 2000; McCauley et al, 1997; Roche, Ahmed, & Blum, 2008; Wong et al., 2006). In addition, these problems appear to be additive, with no one single factor exerting a predominant influence (Melchior, Moffitt, Milne, Poulton, & Caspi, 2007). In short, there is robust data to indicate that adverse early life experience predicts the initiation of substance abuse in later life. With respect to models of responsibility, therefore, there is a need to broaden the concept of responsibility from the individual to parents, society, and policy makers, as these factors increase the likelihood of addiction in young people via the mechanisms of poor parenting, socioeconomic disadvantage, and inadequate educational opportunities.

In contrast to these factors responsible for initiation of drug use, a separate set of factors connected broadly to the concept of individual responsibility and self-efficacy appear to operate regarding maintenance of cessation of drug use once it has started. Data from life-course perspective studies of substance misuse provide valuable insights in this regard. Using a sophisticated longitudinal statistical methodology called growth mixture modeling in a 33-year follow-up study of heroin usage, three groups of addicts were identified within the sample: early quitters, stable high-level users, and late decelerated users (Hser,

Huang, Chou, & Anglin, 2007). The early quitter group had a later onset of drug use than the other two groups, lending support to the notion that early intervention is important to prevent long-term severity of addiction as has been identified by other workers (Anthony & Petronis, 1995). In a separate analysis of this same cohort, self-efficacy and reduced psychological distress were predictive of long-term stable recovery from heroin addiction, whereas childhood factors of disadvantage and family problems were not predictive of ongoing use (Hser, 2007).

These data indicate that a developmental and life-course perspective is essential when planning therapeutic, social, and legal responses to the problem of addiction, as different influences operate at multiple potential branch points in the trajectories of change throughout the lifetime of someone suffering from an addiction. It is imperative that such research methodologies are adopted more comprehensively if progress is to be made in tailoring interventions that are appropriate at each stage in the developmental progression of addiction.

Relapse Prevention and Negative Outcomes versus the Good Life and Positive Outcomes

The model outlined earlier examining the influence of the social environment as it relates to ego depletion has quite profound implications for the approach to addictive behaviors and indeed offending behavior in general as it may serve to explain why relapse-prevention strategies might ultimately be self-defeating, as the ability to exercise self-restraint over prolonged periods in the absence of altering the fundamental desire is problematic. Indeed, a recent study examining eight-year recidivism in sex offending following a well-designed randomized controlled trial of a relapse-prevention program found no beneficial effect (Marques, Wiederanders, Day, Nelson, & van Ommeren, 2005). In contrast, new approaches to the treatment of sex offending, collectively known as the good lives model are showing great promise (Ward, Mann, & Gannon, 2007). These approaches seek to reformulate this troublesome behavior as a dysfunctional way of meeting basic human needs or goods. The approach is based on working with offenders to establish a set of positive, pleasurable, prosocial life goals that build motivation for change in ever-increasing positive feedback loops as new goods such as jobs, appropriate relationships, and hobbies are acquired. This serves to devalue and reduce the intensity of the desire previously attached to deviant sexual behavior, thus placing less stress on the limited resource of self-control. A sole emphasis on relapse prevention, often accompanied by the use of extensive avoidant coping strategies and resulting social exclusion leads to profound deficits in social goods, and this does nothing to devalue the devi-

ant desire, thus increasing the risk that self-control will eventually break down (for a criticism of the Marques study see Marshall & Marshall, 2007). A similar good lives social inclusion approach to the management of sex offending is known as circles of support and accountability (COSA), whereby offenders, often at substantially high risk of reoffending, are offered regular support and mentorship by a group of especially trained community volunteers who both work with the offender to identify appropriate positive goods that can be achieved and serve to challenge any distorted thinking that might lead to relapse. Results of evaluations so far have been highly impressive, showing a 70 percent reduction in recidivism compared to a matched comparison group with communities stating that they felt safer knowing that a COSA group was functioning in their area (Wilson, Picheca, & Prinzo, 2005). These findings have been replicated using the same approach in the United Kingdom (Quaker Peace & Social Witness, 2003).

Similar models have been put forward in general criminology and in studies of the prolonged lifetime use of heroin where the concept of the loss of social capital (Sampson & Laub, 1992, 1993) as a result of imprisonment contributes significantly and progressively to the difficulty in establishing abstinence. This model also offers an explanation as to why early intervention and cessation of drug use before too much social capital has been lost is associated with long-term recovery (Hser, 2007). In addition, as mentioned before, life-course studies show that positive alcohol- and drug-free long-term recovery is promoted by self-efficacy and approach-focused, as opposed to avoidant, coping strategies (Hser, 2007; Moos & Moos, 2007).

Generally speaking, it is easier for all human beings to motivate themselves toward a positive goal, doing something, rather than an avoidance goal, not doing something. In relation to the data on ego deletion, this has important implications with respect to treatment intervention studies, and social and legal policy in relation to addictive behaviors, to ensure that the balance is weighted in favor of the realistic opportunity to achieve positive goals rather than on excessive demands for the addict to comply with negative, risk-informed, avoidance behaviors and punitive control that are likely to be counterproductive in the long term.

The Reciprocal Influence of the Social and Legal Context

As well as being mindful of developmental, life-course, and negative versus positively focused interventions, the profound influence of societal reactions, social policy, and legal frameworks with respect to the individual expression of addictive behavior has to date been grossly underestimated. Very often, and

particularly in relation to behaviors that cause society the most anxiety and opprobrium, excessive focus is attached to the punitive management and containment of those identified as exhibiting the behavior, with little regard as to how this approach might deter those at an early stage of these behaviors from seeking appropriate help to prevent further harm.

There is concrete evidence to suggest that this is occurring with respect to the emotive issue of child sex offending in the United States. In the summer of 1996 in Vermont, the Stop It Now! organization, a charity that seeks to prevent child abuse by offering confidential telephone support to those worried that a child might be being abused and to potential abusers who are contemplating committing an offense, launched its pilot site. Initially almost 60 percent of the calls were from sexual abusers or those at risk of abuse. When news of Megan's Law (open access community notification on the whereabouts of known sex offenders in the United States) emerged in the Vermont press, the number of phone calls from these groups fell to zero. This indicates that a social policy purportedly designed to protect children from the threat of strangers was now inhibiting others from disclosing about abuse in the home, which in any case accounts for 80–96 percent of all cases of child abuse. In addition, abusers themselves were going underground, further increasing the risk to children (Fitch, 2006). The Stop It Now! organization described the problem thus:

> The specter of notification, the accompanying shame, the potential vigilante response from the community, the inability to restore life to some level of normalcy post-release, the potential for the humiliation of other family members besides the abuser him/herself are all deterrents [from voluntary disclosure by victims and abusers] in a very real way. (Deborah Dobovan Rice of Stop It Now!, written communication, quoted in Fitch 2006, p. 37)

This data would suggest that there may be a reciprocal relationship between the perceived level of vilification and punitive control of known sex offenders and the willingness of both victims to disclose an offense (particularly in the largest category of intrafamilial abuse) and people worried about their sexual behavior from coming forward for help that might prevent abuse from occurring. Although the situation in the United Kingdom does not entail widespread community notification as in the United States, being placed on the sex offenders register is often reported in the press at the time of sentencing and is associated with significant social stigma. There is also continuing pressure from various quarters for the introduction of U.S.-style notification, and it is therefore likely that similar fears of disclosure by victims and potential abusers are operating in the United Kingdom too. As an illustration of the scientific irrationality of these policies, a hypothetical model is presented below based

on UK Home Office data on the frequency of child sex offending in England and Wales, the numbers of registered child sex offenders in England and Wales, and the 16 percent prevalence of the concurrent level of abuse of children in the United Kingdom under the age of 16 (Cawson, 2000). The number of predicted offenses committed by registered offenders has been multiplied by a factor of three, as on average it has been found that there are three victims for each index offense (Beech, Fisher, & Beckett, 1998). Even despite this loading, the model clearly suggests that if the overall burden of child sexual abuse is to be reduced, then policy needs to switch significantly toward more investment in preventative intervention (see Table 4.1 and Figure 4.1) alongside a more positively orientated good lives and COSA model of support and supervision of known offenders that is less likely to deter victims and potential abusers from voluntary disclosure. Moreover, a recent report by the National Society for the Prevention of Cruelty to Children (NSPCC) in the United Kingdom highlights the need for primary prevention:

> The NSPCC recommends that treatment services must be available for individuals who come forward because they are worried about their own thoughts, feelings and behaviors. This would ensure that there is a primary prevention element in the provision of services. (NSPCC, 2006, p. 21)

A similar model could be hypothesized with respect to the reluctance of heroin users to present early for treatment because of fears of prosecution. Given the importance of early intervention for reducing the risk of developing a severe, life-course persistent trajectory of drug use, new social and legal policy needs to be subject to this kind of analysis as to its likely effect on the overall burden of addiction prior to implementation.

6PR: An Alternative Model of Responsibility in Addiction

In order to encompass the multiple perspectives necessary to ensure a reasoned, balanced, scientific, and humane response to the problem of addiction, this chapter will conclude with a proposed list of factors and key questions against which any new therapy, social policy, or legal framework should be tested prior to implementation. The factors to be discussed have been given the acronym 6PR, which stands for: personal, public, positive, pragmatic, preventative, and panoramic responsibilities.

1. Personal responsibility
    a. To what extent and by what mechanisms does the proposed intervention promote the development of self-efficacy so as to foster the ability of the addict to manage his own behavior successfully over the life-course?

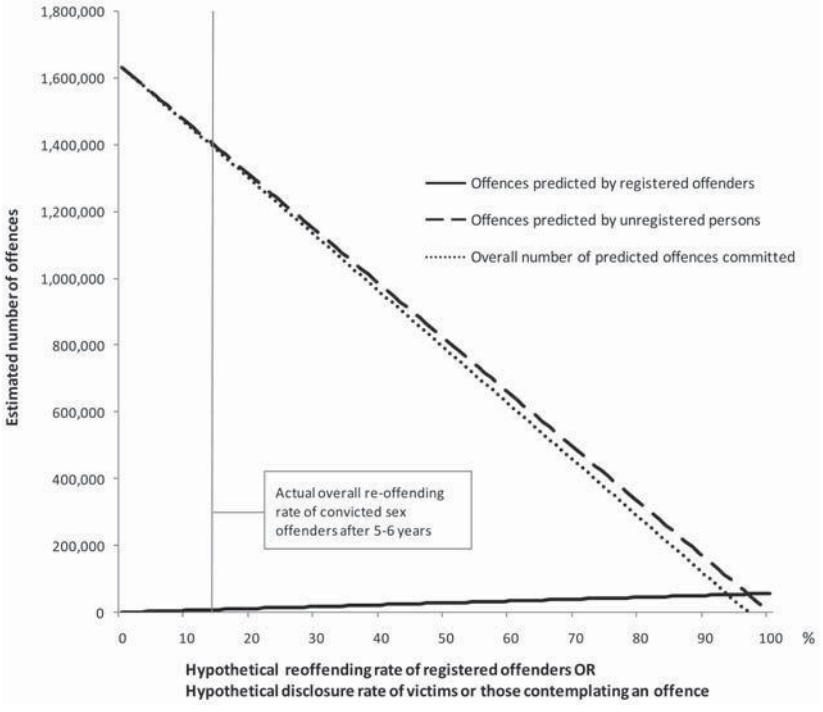

FIGURE 4.1: Hypothetical model of child sex offending assuming a reciprocal relationship between the perceived level of punitive control of registered offenders and spontaneous disclosure by victims or those contemplating an offense.

b. Are there any adverse effects from other influences that are likely to counteract the intended results of the intervention or diminish the addict's capacity for self-efficacy?

2. Public responsibility

   a. Will the proposed social policy or legal intervention exacerbate the problem in question?
   b. Will the proposed social policy or legal intervention reduce the overall burden of the problem in question?

3. Positive responsibility

   a. Is the proposed intervention based on a good lives model?
   b. Is the proposed intervention based on increasing the likelihood of a positive outcome and not just the avoidance of a negative one?

Table 4.1
Figures for Child Sex Offending in England and Wales for 2006–2007 on Which the Graphical Representation of the Hypothetic Model in Figure 4.1 is Based

Figures for 2006–2007	
Total population in England and Wales[a]	53,728,800
Child population < 16 years in England and Wales[a]	10,236,100
Percentage of population under 16	19.05%
Total number of sex offenses committed in England and Wales[b]	57,542
Number of child sex offenses committed in England and Wales[b]	35,024
Conviction rate for child sex offenses	0.000652
Predicted number of offenses from conviction rate	6,673
Concurrent sexual abuse in UK children < 16 years[c]	16%
Estimated actual number of offenses	1,637,776
Estimated number of offenses committed by unregistered persons	1,631,103
Estimated percentage of offenses committed by unregistered persons	99.59%
Number of sex offenders register in England and Wales	29,973
Estimated number of child sex offenders on register[d]	18,244
Average number of victims per index offense[e]	3
Actual overall reoffending rate of convicted sex offenders[f]	13.7%

[a]Data from http://www.statistics.gov.uk.
[b]Data from Table 2.04, http://www.homeoffice.gov.uk/rds/crimew0607.html.
[c]Cawson (2000).
[d]Data on the age of the victim is not recorded on the register, so this figure is estimated from the ratio of the number of child sex offenses to the total number of sex offenses committed.
[e]The numbers of predicted offenses by registered offenders has been multiplied by a factor of three (Beech et al., 1998).
[f]Hanson and Morton-Bourgon (2005) report an overall recidivism rate of 13.7 percent from 82 studies following up 29,450 offenders over an average of five to six years.

4. Pragmatic responsibility
    a. Is the proposed intervention likely to produce the maximum benefit for the maximum number of people?
    b. Does the proposed intervention avoid excessive retributive elements that may exacerbate the overall problem?
5. Preventative responsibility
    a. Does the proposed intervention increase or decrease the likelihood that those with addictive behaviors will present at the earliest opportunity for help?

   b. Does the proposed intervention increase or decrease the likelihood that victims of addictive behavior will be able to disclose?

6. Panoramic responsibility
   a. Has the proposed intervention been examined for its potential impact in all relevant areas to ensure there are no adverse unforeseen consequences?
   b. Is the scientific method used to evaluate the intervention sufficiently wide ranging to model all the relevant variables?

In order to answer these questions scientifically, it may be necessary to integrate and further develop methodologies to accommodate the multiple influences at work. For example, the promising techniques of growth mixture modeling in the life-course analysis of addictive behavior may need to be integrated with new techniques in social network analysis (Butts, 2008; Hall & Valente, 2007) in order to simultaneously model individual developmental trajectories against societal influences so as to begin to answer the difficult and emotive questions that addictive behavior raises as rigorously as possible. Armed with this new information, and alongside advances in the cognitive neuroscience of addictive problems, there are grounds for optimism in tackling one of mankind's most troublesome and baffling afflictions.

REFERENCES

Alcoholics Anonymous. (1953). *Twelve steps and twelve traditions.* New York: Alcoholics Anonymous World Services.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Anthony, J. C., & Petronis, K. R. (1995). Early-onset drug use and risk of later drug problems. *Drug and Alcohol Dependence, 40*(1), 9–15.

Bancroft, J., & Vukadinovic, Z. (2004). Sexual addiction, sexual compulsivity, sexual impulsivity, or what? Toward a theoretical model. *Journal of Sex Research, 41*(3), 225–234.

Baumeister, R. F. (2002). Ego depletion and self-control failure: An energy model of the self's executive function. *Self and Identity, 1*(2), 129–136.

Baumeister, R. F., Bratslavsky, E., Muraven, M., & Tice, D. M. (1998). Ego depletion: Is the active self a limited resource? *Journal of Personality and Social Psychology, 74*(5), 1252–1265.

Beech, A., Fisher, D., & Beckett, R. (1998). Step 3: An evaluation of the prison sex offender treatment programme. London: Home Office.

Bogenschutz, M. P., Tonigan, J. S., & Miller, W. R. (2006). Examining the effects of alcoholism typology and AA attendance on self-efficacy as a mechanism of change. *Journal of Studies on Alcohol, 67*(4), 562–567.

Boys, A., Farrell, M., Bebbington, p. , Brugha, T., Coid, J., Jenkins, R., et al. (2002). Drug use and initiation in prison: Results from a national prison survey in England and Wales. *Addiction, 97*(12), 1551–1560.

Butts, C. T. (2008). Social network analysis: A methodological introduction. *Asian Journal of Social Psychology, 11*(1), 13–41.

Carter, A., & Hall, W. (2007). The social implications of neurobiological explanations of resistible compulsions. *American Journal of Bioethics, 7*(1), 15–17.

Cartwright, S. A. (1851). Report on the diseases and physical peculiarities of the Negro race. *The New Orleans Medical and Surgical Journal, 7*, 691–715.

Castro, F. G., Brook, J. S., Brook, D. W., & Rubenstone, E. (2006). Paternal, perceived maternal, and youth risk factors as predictors of youth stage of substance use a longitudinal study. *Journal of Addictive Diseases, 25*(2), 65–75.

Cawson, p. (2000). Child maltreatment in the United Kingdom: A study of the prevalence of child abuse and neglect. London: NSPCC.

Chaloupka, F. J., Grossman, M., & Saffer, H. (2002). The effects of price on alcohol consumption and alcohol-related problems. *Alcohol Research & Health, 26*(1), 22–34.

Charland, L. C. (2007). Affective neuroscience and addiction. *American Journal of Bioethics, 7*(1), 20–21.

Cochrane, T. I. (2007). Brain disease or moral condition? Wrong question. *American Journal of Bioethics, 7*(1), 24–25.

Cohen, p. J. (2007). Addiction, molecules and morality: Disease does not obviate responsibility. *American Journal of Bioethics, 7*(1), 21–23.

Connors, G. J., Tonigan, J. S., Miller, W. R., & MATCH Research Group. (2001). A longitudinal model of intake symptomatology, AA participation and outcome: Retrospective study of the Project MATCH outpatient and aftercare samples. *Journal of Studies on Alcohol, 62*(6), 817–825.

Damasio, A. (2007). Neuroscience and ethics: Intersections. *American Journal of Bioethics, 7*(1), 3–7.

Doherty, E. E., Green, K. M., Reisinger, H. S., & Ensminger, M. E. (2008). Long-term patterns of drug use among an urban African-American cohort: The role of gender and family. *Journal of Urban Health, 85*(2), 250–267.

Fergusson, D. M., Horwood, L. J., Boden, J. M., & Jenkin, G. (2007). Childhood social disadvantage and smoking in adulthood: Results of a 25-year longitudinal study. *Addiction, 102*(3), 475–482.

Fitch, K. (2006). Megan's Law: Does it protect children? An updated review of evidence on the impact of community notification as legislated for by Megan's Law in the United States. London: NSPCC.

Foddy, B., & Savulescu, J. (2007). Addiction is not an affliction: Addictive desires are merely pleasure-oriented desires. *American Journal of Bioethics, 7*(1), 29–32.

Fothergill, K. E., & Ensminger, M. E. (2006). Childhood and adolescent antecedents of drug and alcohol problems: A longitudinal study. *Drug and Alcohol Dependence, 82*(1), 61–76.

Goldman, D., G. Oroszi, & Ducci, F. (2005). The genetics of addictions: Uncovering the genes. *Nature Reviews Genetics, 6*, 521–532.

Goodman, A. (2008). Neurobiology of addiction. An integrative review. *Biochemical Pharmacology, 75*(1), 266–322.

Grant, J. E., Brewer, J. A., & Potenza, M. N. (2006). The neurobiology of substance and behavioral addictions. *CNS Spectrums, 11*(12), 924–930.

Greene, J., & Cohen, J. (2004). For the law, neuroscience changes nothing and everything. Philosophical transactions of the Royal Society of London. *Series B, Biological Sciences, 359*, 1775–1785.

Hall, W. (2000). UROD: An antipodean therapeutic enthusiasm. *Addiction, 95*(12), 1765–1766.

Hall, W. (2006). Stereotactic neurosurgical treatment of addiction: Minimizing the chances of another 'great and desperate cure.' *Addiction, 101*(1), 1–3.

Hall, J. A., & Valente, T. W. (2007). Adolescent smoking networks: The effects of influence and selection on future smoking. *Addictive Behaviors, 32*(12), 3054–3059.

Hall, W., Carter, L., & Morley, K. I. (2004). Neuroscience research on the addictions: A prospectus for future ethical and policy analysis. *Addictive Behaviors, 29*(7), 1481–1495.

Hanson, R. K., & Morton-Bourgon, K. E. (2005). The characteristics of persistent sexual offenders: A meta-analysis of recidivism studies. *Journal of Consulting and Clinical Psychology, 73*(6), 1154–1163.

Hser, Y-I. (2007). Predicting long-term stable recovery from heroin addiction: Findings from a 33-year follow-up study. *Journal of Addictive Diseases, 26*(1), 51–60.

Hser, Y-I., Huang, D., Chou, C-P., & Anglin, M. D. (2007). Trajectories of heroin addiction: Growth mixture modeling results based on a 33-year follow-up study. *Evaluation Review, 31*(6), 548–563.

Hser, Y-I., Longshore, D., & Anglin, M. D. (2007). The life course perspective on drug use: A conceptual framework for understanding drug use trajectories. *Evaluation Review, 31*(6), 515–547.

HSF-SSRI trial. (n.d.). Retrieved from http://public.ukcrn.org.uk/search/StudyDetail.aspx?StudyID=2325.

Hurd, Y. L. (2006). Perspectives on current directions in the neurobiology of addiction disorders relevant to genetic risk factors. *CNS Spectrums, 11*(11), 855–862.

Hyman, S. E. (2005). Addiction: A disease of learning and memory. *American Journal of Psychiatry, 162*(8), 1414–1422.

Hyman, S. E. (2007). The neurobiology of addiction: Implications for voluntary control of behavior. *American Journal of Bioethics, 7*(1), 8–11.

Hyman, S. E., Malenka, R. C., & Nestler, E. J. (2006). Neural mechanisms of addiction: The role of reward-related learning and memory. *Annual Review of Neuroscience, 29*, 565–598.

Ilgen, M., Tiet, Q., Finney, J., & Moos, R. H. (2006). Self-efficacy, therapeutic alliance, and alcohol-use disorder treatment outcomes. *Journal of Studies on Alcohol, 67*(3), 465–472.

Kansas v. Hendricks, (1997). Retrieved January 21, 2009, from http://openjurist.org/521/us/346

Kliewer, W., & Murrelle, L. (2007). Risk and protective factors for adolescent substance use: Findings from a study in selected Central American countries. *Journal of Adolescent Health, 40*(5), 448–455.

La Fond, J. Q. (2000). The future of involuntary civil commitment in the U.S.A. after Kansas v. Hendricks. *Behavioral Sciences & the Law, 18*(2–3), 153–167.

La Fond, J. Q. (2008). Sexually violent predator laws and the liberal state: An ominous threat to individual liberty. *International Journal of Law and Psychiatry, 31*(2), 158–171.

Leshner, A. I. (1997). Addiction is a brain disease, and it matters. *Science, 278*(5335), 45–47.

Levy, N. (2007). The social: A missing term in the debate over addiction and voluntary control. *American Journal of Bioethics, 7*(1), 35–36.

Marcenko, M. O., Kemp, S. P., & Larson, N. C. (2000). Childhood experiences of abuse, later substance use, and parenting outcomes among low-income mothers. *American Journal of Orthopsychiatry, 70*(3), 316–326.

Marques, J. K., Wiederanders, M., Day, D. M., Nelson, C., & van Ommeren, A. (2005). Effects of a relapse prevention program on sexual recidivism: Final results from California's sex offender treatment and evaluation project (SOTEP). *Sexual Abuse: A Journal of Research and Treatment, 17*(1), 79–107.

Marshall, W. L., & Marshall, L. E. (2007). The utility of the random controlled trial for evaluating sexual offender treatment: The gold standard or an inappropriate strategy? *Sex Abuse, 19*, 175–191.

McCauley, J., Kern, D. E., Kolodner, K., Dill, L., Schroeder, A. F., DeChant, H. K., et al. (1997). Clinical characteristics of women with a history of childhood abuse: Unhealed wounds. *Journal of the American Medical Association, 277*(17), 1362–1368.

McLellan, A. T., Lewis, D. C., O'Brien, C. P., & Kleber, H. D. (2000). Drug dependence, a chronic medical illness: Implications for treatment, insurance, and outcomes evaluation. *Journal of the American Medical Association, 284*(13), 1689–1689.

Melchior, M., Moffitt, T. E., Milne, B. J., Poulton, R., & Caspi, A. (2007). Why do children from socioeconomically disadvantaged families suffer from poor health when they reach adulthood? A life-course study. *American Journal of Epidemiology, 166*(8), 966–974.

Mick, T. M., & Hollander, E. (2006). Impulsive-compulsive sexual behavior. *CNS Spectrums, 11*(12), 944–955.

Milich, R., Lynam, D., Zimmerman, R., Logan, T. K., Martin, C., Leukefeld, C., et al. (2000). Differences in young adult psychopathology among drug abstainers, experimenters, and frequent users. *Journal of Substance Abuse, 11*(1), 69–88.

Moos, R. H., & Moos, B. S. (2007). Treated and untreated alcohol-use disorders: Course and predictors of remission and relapse. *Evaluation Review, 31*(6), 564–584.

Morse, S. J. (2007). Voluntary control of behavior and responsibility. *American Journal of Bioethics, 7*(1), 12–13.

NSPCC. (2006). The NSPCC's response to the home office child sex offenders review. London: NSPCC.

Potenza, M. N. (2006). Should addictive disorders include non-substance-related conditions? *Addiction, 101*(Suppl. 1), 142–151.

Project MATCH Study Group. (1998). Matching alcoholism treatments to client heterogeneity: Project MATCH three-year drinking outcomes. *Alcoholism, Clinical and Experimental Research, 22*(6), 1300–1311.

Quaker Peace & Social Witness. (2003). Circles of Support and Accountability in the Thames Valley: Interim report. London: Quaker Communications.

Robins, L. N., Davis, D. H., & Goodwin, D. W. (1974). Drug use by U.S. army enlisted men in Vietnam: A follow–up on their return home. *American Journal of Epidemiology, 99*, 235–249.

Robins, L. N., & Slobodyan, S. (2003). Post-Vietnam heroin use and injection by returning US veterans: Clues to preventing injection today. *Addiction, 98*(8), 1053–1056.

Roche, K. M., Ahmed, S., & Blum, R. W. (2008). Enduring consequences of parenting for risk behaviors from adolescence into early adulthood. *Social Science & Medicine, 66*(9), 2023–2034.

Sampson, R. J., & Laub, J. H. (1992). Crime and deviance in the life course. *Annual Review of Sociology, 8*, 63–84.

Sampson, R. J., & Laub, J. H. (1993). Life-course desisters? Trajectories of crime among delinquent boys followed to age 70. *Criminology, 41*(3), 603–626.

Satel, S. L. (1999). What should we expect from drug abusers? *Psychiatric Services, 50*(7), 861.

Shedler, J., & Block, J. (1990). Adolescent drug use and psychological health. A longitudinal inquiry. *The American Psychologist, 45*(5), 612–630.

Sherman, W. (2006, February 12). Test targets addiction gene. *New York Daily News*, 28.

Tonigan, J. S., Miller, W. R., & Schermer, C. (2002). Atheists, agnostics and Alcoholics Anonymous. *Journal of Studies on Alcohol, 63*(5), 534–541.

Tucker, J. S., Ellickson, p. L., Collins, R. L., & Klein, D. J. (2006). Are drug experimenters better adjusted than abstainers and users?: A longitudinal study of adolescent marijuana use. *Journal of Adolescent Health, 39*(4), 488–494.

Ward, T., Mann, R. E., & Gannon, T. A. (2007). The good lives model of offender rehabilitation: Clinical implications. *Aggression and Violent Behavior, 12*, 87–107.

Wilson, R. J., Picheca, J. E., & Prinzo, M. (2005). Circles of Support & Accountability: An Evaluation of the Pilot Project in South-Central Ontario. Ottowa, ON: Correctional Service of Canada.

Winick, C. (1962). Maturing out of narcotic addiction. *Bulletin on Narcotics, 14*, 1–7.

Witkiewitz, K., van der Maas, H. L., Hufford, M. R., & Marlatt, G. A. (2007). Nonnormality and divergence in posttreatment alcohol use: Reexamining the Project MATCH data "another way." *Journal of Abnormal Psychology, 116*(2), 378–394.

Wong, M. M., Nigg, J. T., Zucker, R. A., Puttler, L. I., Fitzgerald, H. E., Jester, J. M., et al. (2006). Behavioral control and resiliency in the onset of alcohol and illicit drug use: A prospective study from preschool to adolescence. *Child Development, 77*(4), 1016–1033.

Chapter **5**

Religiosity as Protective against Addictions in Adolescents

Patrick McNamara, PhD, and James Burns, PhD

There is now substantial evidence supporting the claim that religiousness can protect against a variety of addictions in adolescents (see Smith, 2003, for review). We believe that the evidence is strong enough to justify a search as to how religion confers its protective effects on adolescents. What does religion do for adolescents that allows them to avoid addiction? In what follows, we first review key findings in the literature demonstrating protective effects of religion against addictions in adolescents. We then summarize potential mechanisms for this protective effect. Among these potential mechanisms are the interactions between religious beliefs/practices and the developing brain. Because these latter interactions have been neglected in the literature, we focus on these protective interactions in an effort to identify potential mediating pathways for religion's effects on adolescent risk for addiction.

DEFINITIONS OF RELIGIOSITY

Studies of adolescent substance use have used various definitions of *religiosity* (Wallace & Williams, 1997). In most studies of the impact of religiosity on health-related outcomes, including substance use and addictions more generally, religiosity was measured with only a single question or a single subscale indexing frequency of attendance at religious services. Recent theoretical work, however, suggests that the broader construct of religiousness is multidimensional, including facets of belief, private religious practices, participation in ritual services, and coping (Fetzer Institute, 1999; George, Larson, Koenig, & McCullough, 2000; McNamara, 2002). For purposes of this chapter, we will define religiosity as composed of two major factors: perceived importance of a

belief in God (faith) and religious *practices* associated with faith-related institutions (rituals, prayer, worship, etc.).

RELIGIOSITY CONFERS A PROTECTIVE EFFECT AGAINST ADDICTION IN ADOLESCENTS

Recent reviews of the literature on potential protective effects of religiosity against addiction in both adults and adolescents have demonstrated that greater religiosity, as measured by participation in religious services or by strength of religious convictions, is associated with lower risk of use of addictive substances (Blyth & Leffert, 1995; Booth & Martin, 1998; Galanter, 2006; Gorsusch, 1995; Koenig, McCullough, & Larson, 2001). Findings from the National Study of Youth and Religion (Smith & Faris, 2002) support a strong inverse relationship between religiosity and addictions in adolescence. After controlling for a host of potential confounders, the survey of a national sample of 2,478 teens found that religious high school seniors were less likely to smoke, drink, or use illicit substances than their less religious counterparts. Brown, Schulenberg, Backman, O'Malley, and Johnston (2001), also examined a nationally representative sample of high school classes who graduated between 1976 and 1997 (N = 188,000). The researchers, who were interested in whether the correlates of tobacco use changed across time, surveyed a random sample of 15,000 and 19,000 high school seniors from approximately 135 high schools each year. The investigators found a highly consistent association between lower cigarette use and greater religious involvement across the 22-year study period. There is little doubt therefore that an inverse relation exists between religious involvement and engagement in risky behaviors associated with addiction.

Religious involvement of course is complex and means many different things to as many different people. What aspects of religious involvement are most effective for protecting against addiction? One of the strongest predictors of protective effects of religion in adolescence is the variable frequency of attendance at religious services (Adlaf & Smart, 1985; Amey, Albrecht, & Miller, 1996; Foshee & Hollinger, 1996; Hadaway, Elifson, & Petersen, 1984; Wallace & Williams, 1997). Attendance at ritual services either yields a direct protective benefit or it may do so indirectly, perhaps through reinforcing religious beliefs or through extending the social networks of participants, encouraging prosocial attitudes leading to engagement in prosocial behaviors such as those motivated by learned altruism. There is some evidence that both of these factors may be operative. For example, strength of belief in religious tenets is strongly and inversely related with substance abuse (Bahr, Maughan, Marcos,

& Li, 1998; Barnes, Farrell, & Banerjee, 1994; Brown & Gary, 1991; Jessor, Chase, & Donovan, 1980; Jessor & Jessor, 1977; Resnick et al., 1997). Jessor and colleagues (Jessor et al., 1980; Jessor & Jessor, 1977) have found that a scale on the perceived importance of religion was inversely related to measures of problem drinking in regional and national samples of high school students. It is not clear, however, that this effect of perceived importance was independent of attendance at religious services. Nevertheless, these studies clearly show that both belief and attendance are critical to the positive effect of religiosity.

MECHANISM OF THE PROTECTIVE EFFECT

In his recent systematic review of the literature on religion's protective effects on adolescents, Smith (2003) identified several key factors that could potentially mediate religion's constructive effects: moral directives, spiritual experiences, role models, community and leadership skills, coping skills, cultural and social capital, network closure, and extracommunity links. He summarized these factors as clustering into three major domains of functioning: learned competencies; moral order, and social/organizational ties. In this chapter we focus on the first of these three domains of functioning: namely, learned competencies.

LEARNED COMPETENCIES IN PROTECTION AGAINST RISK: THE KEY ROLE OF IMPULSIVITY AND POOR DECISION-MAKING/PLANNING IN ENHANCING RISK

Two of the most robust proximate predictors of adolescent risk behaviors are impulsivity and poor decision-making and planning (Boyer, 2006; Hawkins et al., 2000). Impulsivity and poor decision-making reflect the failure to learn or to acquire the crucial set of skills that allows one to stop, think, and plan for the most optimal outcomes under conditions of stress. Stanford and colleagues (Stanford, Greve, Boudreaus, & Mathias, 1996) demonstrated, with a reasonably large sample of adolescents and young adults ($N = 1{,}100$), that highly impulsive individuals were more likely than less impulsive individuals to engage in a number of risk-taking behaviors (e.g., peer aggression, drunk driving, and drug use). Several investigators (Beyth-Marom, Austin, Fischhoff, Palmgren, & Jacobs, 1993; Furby & Beyth-Marom, 1992; Steinberg, 2006) have pointed out that adolescents and adults are equally aware or knowledgeable concerning the potential for adverse consequences in risky situations, but adolescents tend to impulsively discount the costs of risky situations and fail to plan or to take steps to avoid risky situations. Adolescent impulsivity and decreased ability to

be playful may be further related to a decreased recognition of being addicted as well as a challenge to recovery for the stages of change necessary for sufficient movement away from addictive surroundings and behaviors. This is known to require a contemplative state that precedes action (Prochaska, DiClemente, & Norcross, 1992).

Studies of the relation between risk taking and cost versus benefit analyses in adolescents suggest that young people who score high on traits such as impulsivity, novelty seeking, and reward dependence tend to discount long-term costs and unduly augment short-term benefits and thus are more likely to engage in risky behavior (e.g., Fromme, Stroot, & Kaplan, 1993; Goldberg & Fischhoff, 2000; Horvath & Zuckerman, 1993; Lavery, Siegel, Cousins, & Rubovits, 1993; McBride, Weatherby, Inciardi, & Gillespie, 1999; Singer, Dai, Weeks, & Malave, 1998; Thorton, Gibbons, & Gerrard, 2002). We suggest that the effects of these traits on risk of substance abuse or for addiction more generally may be moderated by religiosity levels and cognitive functions that regulate impulsivity levels. There is a large independent literature on the cognitive functions implicated in control of impulsivity and poor decision-making when individuals are under stress. Those cognitive functions (or learned competencies) are typically called the executive cognitive functions (ECFs).

EXECUTIVE COGNITIVE FUNCTIONS VERSUS IMPULSIVITY IN ADOLESCENCE

Executive Cognitive Functions (ECFs) refer to such functions as planning, initiation, impulse control, cognitive inhibition, attention, monitoring, and adjustment of nonroutine and goal-directed behaviors. ECFs allow for inhibition of impulses, delay of gratification of wishes, and thus accurate appraisal of long-term future rewards versus immediate short-term and more salient rewards. When ECF development is delayed, self-control and self-regulation are also delayed and can be significantly derailed. Thus, risky behaviors are more likely. A substantial body of research supports our conjecture. For example, early self-regulatory abilities as measured by ECF tasks significantly predict later cognitive and social competencies (Metcalfe & Mischel, 1999; Mischel, 1983; Mischel & Ayduk, 2004; Mischel, Shoda, & Peake, 1988; Mischel, Shoda, & Rodriguez, 1989; Sethi, Mischel, Aber, Shoda, & Rodriguez, 2000).

We can get some idea of normative development of ECFs in typically developing children via administration of neuropsychologic tests that tap various executive processes such as the Stroop task, the go/no-go or stop signal tasks, verbal fluency measures, and card sort tasks. The standard Stroop task requires respondents to name the ink color of either 100 patches of the colors red, blue,

and green or 100 color names printed in a discrepant ink color. The subject has to inhibit the impulse to read the name rather that to report the color, and thus the outcome measure is the difference in the time required to complete the two tasks (color words minus color patches). In the Stop-signal task, respondents are told to stop responding with button presses to a visual stimulus (letter X or O) when hearing a tone (stop signal). The outcome measure is the minimum delay between the onset of stimulus and tone necessary to stop the response, the so called stop-signal reaction time (SSRT). In verbal-fluency tasks, respondents are asked to produce all the words they can think of in a given category like animals (semantic fluency) or as many words as they can starting with a certain letter (phonetic fluency). All tasks are timed. Levin et al. (1991) found an increased ability to inhibit responses with increasing age on the go/no-go test in a normal sample. Impulsive errors (commissions) declined steadily up to adolescence and then plateaued. Williams, Ponesse, Schachar, Logan, and Tannock (1999) reported similar findings for this task. Using a matching to sample type task, Welsh, Pennington, & Grassier (1991) also found that impulsive responding matured by age 10. Brocki and Bohlin (2004) assessed a variety of ECFs in children and adolescents and then conducted dimensional analyses on performance scores. The dimension interpreted as speed/arousal seemed to be the first one to reach maturity, with the most active period of development occurring around the age of eight. The withholding dimension of inhibition revealed maturity around age 10, along with the first developmental spurt on the fluency factor. The second developmental spurt on working memory/fluency continued into adolescence.

In short, for most typically developing children, inhibitory ECFs as measured by stop signal and Stroop-like tasks develop up to the time of puberty and then level off, while working memory and verbal fluency skills continue to mature into the adolescent period.

These neuropsychologic results are consistent with a host of recent neuroimaging studies of the developing brain (reviewed in Blakemore, den Ouden, Choudhury, & Frith, 2007, and Paus et al., 2001). Both gray and white matter volumes increase in most brain regions until puberty. After puberty, gray matter volumes do not change significantly, while white matter volumes continue to increase. Giedd et al. (1999) observed a linear increase in gray matter volume in the parietal and frontal regions between four years of age and the approximate age of onset for puberty. In girls, the asymptote for these anatomical developments was at 10.2 years in the parietal cortex and 11.0 years in the frontal cortex. For boys, the asymptotic ages were 11.8 years and 12.1 years for the two respective areas.

These neuroanatomical and neuropsychologic data suggest that trajectories involving acquisition of new inhibitory ECF capacities begin to decline with

the onset of puberty. Thus, the child must cope with pubertal changes and hormonal surges with whatever inhibitory ECFs he or she has developed up until pubertal onset. Any additional resources that can enhance ECF capacities, therefore, would presumably help the child deal with pubertal changes. We suggest that one such resource is religion. This supposition predicts that children who utilize religious practices to self-regulate impulsivity levels after puberty will be better able to regulate impulsivity levels than children who do not utilize religious practices.

To show how religion can be used as a resource to self-regulate impulsivity levels, it will be necessary to go into greater detail on the neurobiology of addiction and of impulsivity more generally.

BRAIN BASES OF ADDICTION: FRONTAL LOBES, DOPAMINE, AND IMPULSIVITY

The prefrontal cortex (PFC) constitutes approximately one-third of the human cortex and is the last part of the human brain to become fully myelinated in ontogeny, with maturation occurring in late childhood/early adolescence (Huttenlocher & Dabholkar, 1997). The PFC receives projections from the mediodorsal nucleus and gives rise to the primary motor cortex, as well as premotor, supplementary motor, and the dorsal and orbital sectors of the prefrontal (proper) lobes. All of these PFC areas send inhibitory efferents onto their sites of termination in other areas of the brain and spinal cord, thus suggesting a supervisory or regulative role for the PFC.

Impairment in prefrontal cortical function in humans is functionally implicated in virtually every major neuropsychiatric disorder, including depression (Starkstein & Robinson, 1991), schizophrenia (Lewis, Cruz, Eggan, & Erickson, 2004), obsessive-compulsive disorder (Tek & Ulug, 2001), bipolar disorder (Haznedar et al., 2005), Parkinson's disease (Starkstein & Merello, 2002), Huntington's disease (Troster & Woods, 2003), the disinhibitory impulsivity syndromes (Berlin, Rolls, & Iversen, 2005), addictions (Winstanley, Theobald, Dalley, Cardinal, & Robbins, 2006), and several others besides, such as memory retrieval dysfunction and the dementias (Cummings & Mega, 2003). The frontal lobes mediate what are believed to be distinctively human mental capacities, such as language generativity (Miller & Cummings, 1999), autobiographical memory retrieval (Wheeler, Stuss, & Tulving, 1997), theory of mind (Baron-Cohen, 2004) empathy (Adolphs, Baron-Cohen, & Tranel, 2002), working memory (Goldman-Rakic, 1987), executive functions (Goldberg & Bougakov, 2005), impulse control (Berlin et al., 2005), volition (Passingham, 1995) and possibly even the sense of self (Northoff & Bermpohl, 2004).

NEUROCHEMISTRY OF FRONTAL LOBES

The frontal lobes are densely innervated by dopaminergic (DA) fibers originating in the ventral tegmental area (VTA) and the substantia nigra (SN). The nigrostriatal system indirectly influences the frontal lobes through the basal ganglia. The mesocortical system originates in the VTA and terminates in the ventral striatum, amygdala, nucleus accumbens, and the frontal lobes. This latter mesocortical system is crucially important for understanding human behavior as its stimulation appears to be intrinsically rewarding. All drugs/substances of addiction, for example, appear to derive their addicting properties by their abilities to potently stimulate this frontal dopaminergic system. Dopamine neurons of the VTA and SN have long been associated with the reward and pleasure systems of the brain. Virtually all of the known addictions (including cocaine, heroin, amphetamines, alcohol, food, and sex) exert their addictive actions, in part, by prolonging the influence of dopamine on target neurons (Wise, 2005). VTA DA neuron responses appear to be necessary to facilitate formation of associations between stimuli that predict reward and behavioral responses that obtain reward (Schultz et al., 1995). The orbital frontal cortex integrates the most complex level of associations of reinforcement with both stimuli and responses (Rolls, 2004). In summary, stimulation of dopaminergic terminals in the mesolimbic-frontal systems constitutes the substrate for a most potent reward/reinforcing system and thus the substrate for addiction. PFC inhibitory efferents that terminate onto orbitofrontal, limbic, VTA DA and brainstem arousal centers constitute the substrate for control of impulsivity levels. To the extent that religion can engage PFC inhibitory circuits, it can function to control impulsivity levels.

RELIGION AND THE FRONTAL LOBES

Religiosity has traditionally been linked to the temporal lobes (e.g., Bear & Fedio, 1977; d'Aquili & Newberg, 1993; Persinger, 1987; Ramachandran, Hirstein, Armel, Tecoma, & Iragui, 1997). But most of the evidence for a role of the temporal lobes in religious experience was based on observations of the behaviors of a small subset of temporal lobe epileptics who exhibited the interictal behavioral syndrome (Bear & Fedio, 1977; Dewhurst & Beard, 1970;. The so-called syndrome included hyperreligiosity as one of its signs. D'Aquili and Newberg (1993) very sensibly assume that all the major association areas of the cortex generate some aspect of the total religious experience. In their models, the temporal lobes attach meaning and significance to events, while posterior parietal sites participate in construction of both the sense of self and the accom-

panying sense of the dissolution of the self during mystical states. With respect to the frontal lobes, d'Aquili and Newberg (1993) reviewed a number of studies that apparently established a link between sustained attention associated with the practice of meditation and electroencephalography (EEG) theta waves above the prefrontal cortex. The EEG data therefore suggests that sustained meditation results in activation of prefrontal networks. Newberg, Alavi, Baime, Mozley, and d'Aquili (1997) later confirmed these EEG data using single photon emission computed tomography (SPECT) imaging techniques. Regional cerebral blood flow changes were studied in six highly experienced meditators while they meditated. Results demonstrated significantly increased blood flow to the inferior frontal and dorsolateral prefrontal cortical regions while the subjects engaged in intense meditation. More recently and using functional magnetic resonance imaging (fMRI) techniques, Azari et al. (2001) later reported greater dorsolateral frontal, dorsomedial frontal, and medial parietal cortex activation during religious recitation in self-described religious subjects.

In summary, it appears that religious practices do indeed engage prefrontal neural networks and therefore can potentially help regulate impulsivity levels. We now need to suggest how this is accomplished at the cognitive level.

THE INTERACTION OF RELIGIOUS AND PFC ECFS: IMPLEMENTATION INTENTIONS

We postulate that one key way in which religiousness protects against addiction and other risky behaviors throughout childhood is via practice in formulating *implementation intentions* or the ability to translate a motivated intention ("I will not engage in premarital or unprotected sex") into an implementation plan ("I will seek a partner in my religious community who agrees with me about premarital sex" or "If my boyfriend pressures me for sex, then I will insist on condom use and I will present to him a condom"). All religions emphasize development of moral character, of putting into practice a suite of moral behaviors and inhibiting sinful or sanctioned behaviors. A host of religious practices are recommended as aids in this character formation process. These religious and spiritual practices are recommended as aids against temptation and tools to help implement good behaviors. We contend that religious practices also help the individual form implementation intentions.

RELIGIOUSNESS AND IMPLEMENTATION INTENTIONS

Spiritual practices can enhance the ability to generate effective implementation intentions. Spiritual and religious practices focus the individual's attention

on moral character, right action, follow-through, actions not words, trustworthiness, steadfastness, and so on. Religious practices such as prayer, religiously orientated meditation, devotional worship, attendance at liturgical or ritual celebrations, study of scripture, engaging in charitable practices, and so on are action sequences designed to realize an overall goal of moral character formation (Batson, Schoenrade, & Ventis, 1993; McNamara, 2002). Religious practices like prayer and meditation, for example, are seen as tools to facilitate generation of what we now call implementation intentions in service to that goal. In fact, it is difficult, if not impossible, to fully engage simultaneously in prayer and an addictive behavior(s).

All religious traditions appear to utilize petitionary prayers and rituals directed at supernatural agents to solicit help, strength, and guidance. Similarly, the sacred texts and scriptures are generally read for guidance on how to behave in order to implement the intention to become a good member of the religious group. Likewise, within theistic traditions, participation in ritual services are understood to be occasions to obtain particular powers/graces (e.g., through communion with God) to become a better participant (e.g., Christian). In an exhaustive review of characteristics and functions of rituals, Boyer and Liénard (2006) have pointed out that goal demotion is characteristic of many and perhaps all religious rituals. Performance of the ritual is focused on correct performance or implementation of each step in the ritual itself rather than on its putative overriding purpose. In summary, among all its other effects, religion appears to train and assist people in creating effective implementation intentions in order to enhance adherence to behavioral and ritual standards of the group and to enhance moral character formation.

PROTECTIVE EFFECTS OF IMPLEMENTATION INTENTIONS

It has been demonstrated repeatedly that forming an implementation intention enhances many health protective behaviors including smoking cessation efforts, cancer checks, exercise regimens, dietary changes, and even safe driving practices (Armitage, 2004, 2006; Cohen & Gollwitzer, 2007; Milne, Orbell, & Sheeran, 2002; Orbell, Hodgkins, & Sheeran, 1997; Orbell & Sheeran, 2000; Prestwich, Lawton, & Conner, 2003; Sheeran & Orbell, 1999, 2000; Sheeran & Silverman, 2003; Verplanken & Faes, 1999). These implementation intentions apparently heighten the accessibility of anticipated situational cues that are often used by individuals to halt an impulsive choice, reconnect to an original superordinate goal and go with a longer-term behavioral plan (Gollwitzer, 1990, 1993, 1996, 1999; Gollwitzer & Schaal, 1998; Heckhausen,

1991; Heckhausen & Gollwitzer, 1987; Sheeran, 2002; Sheeran, Milne, Webb, & Gollwitzer, 2005). More specifically, implementation intentions require people to specify the situations in which they will perform an intended target behavior and the strategies that they will use in order to ensure that their goals/intentions are realized (e.g., superordinate goal: "I want to avoid sexually transmitted diseases while remaining sexually active"; implementation intention: "If I am pressured to have unprotected sex with my boyfriend, then I will stop everything and initiate a conversation on use of condoms"). Again, when examining successful relapse prevention techniques, the ability to remain goal oriented and intentional underlie the ability to be abstinent and or to maintain it after relapse (Prochaska & DiClemente, 1986).

Implementation intentions trigger action initiation in an automatic fashion when the specified situation cues are met (e.g., Brandstätter, Lengfelder, & Gollwitzer, 2001; Gollwitzer & Brandstätter, 1997; Orbell & Sheeran, 2000; Webb & Sheeran, 2004). The more situational cues people specify in their implementation intention (i.e., the more "if" statements that people generate in their implementation intentions), and the greater the number of strategies for performing the behavior that people generate (i.e., the more "then" statements that people generate in their implementation intentions), the more likely they will be to successfully achieve their goals. Based on the foregoing literature review we believe it is reasonable to suppose that religiosity and executive control processes such as impulse control and the planning and generation of implementation intentions mutually interact during development in such a way as to yield effective protection against addiction and risky behaviors in children and adolescents.

REFERENCES

Adlaf, E. M., & Smart, R. G. (1985). Drug use, religious affiliation, feelings, and behavior. *British Journal of the Addictions, 80*, 163–171.

Adolphs, R., Baron-Cohen, S., & Tranel, D. (2002). Impaired recognition of social emotions following amygdala damage. *Journal of Cognitive Neuroscience, 14*(8), 1264–1274.

Amey, C. H., Albrecht, S. L., & Miller, M. K. (1996). Racial differences in adolescent drug use: The impact of religion. *Substance Use and Misuse, 31*, 1311–1332.

Armitage, C. J. (2004). Evidence that implementation intentions reduce dietary fat intake: A randomized trial. *Health Psychology, 23*(3), 319–323.

Armitage, C. J. (2006). Evidence that implementation intentions promote transitions between the stages of change. *Journal of Consulting and Clinical Psychology, 74*(1), 141–151.

Azari, N. P., Nickel, J., Wunderlich, G., Niedeggen, M., Hefter, H., Tellmann, L., et al. (2001). Neural correlates of religious experience. *European Journal of Neuroscience, 13*(8), 1649–1652.

Bahr, S. J., Maughan, S. L., Marcos, A. C., & Li, B. (1998). Family, religiosity, and the risk of adolescent drug use. *Journal of Marriage and the Family, 60,* 979–992.

Barnes, G. M., Farrell, M. P., & Banerjee, S. (1994). Family influences on alcohol abuse and other problem behaviors among black and white adolescents in a general population sample. *Journal of Research on Adolescence, 4,* 183–201.

Baron-Cohen, S. (2004). The cognitive neuroscience of autism. *Journal of Neurology, Neurosurgery, and Psychiatry, 75*(7), 945–948.

Batson, C. D., Schoenrade, p. , & Ventis, W. L. (1993). *Religion and the individual: A social-psychological perspective*. New York: Oxford University Press.

Bear, D. M., & Fedio, p. (1977). Quantitative analysis of interictal behavior in temporal lobe epilepsy. *Archives of Neurology, 34,* 454–467.

Berlin, H. A., Rolls, E. T., & Iversen, S. D. (2005). Borderline personality disorder, impulsivity, and the orbitofrontral cortex. *American Journal of Psychiatry, 162,* 2360–2373.

Beyth-Marom, R., Austin, L., Fischhoff, B., Palmgren, C., & Jacobs, M. (1993). Perceived consequences of risky behaviors. *Developmental Psychology, 29*(3), 549–563.

Blakemore, S. J., den Ouden, H., Choudhury, S., & Frith, C. (2007). Adolescent development of the neural circuitry for thinking about intentions. *Social Cognitive and Affective Neuroscience, 2*(2), 130–139.

Blyth, D. A., & Leffert, N. (1995). Communities as contexts for adolescent development: An empirical analysis. *Journal of Adolescent Research, 10*(1), 64–87.

Booth, J., & Martin, J. E. (1998). Spiritual and religious factors in substance use, dependence, and recovery. In H. G. Koenig (Ed.), *Handbook of religion and mental health* (pp. 175–200). San Diego, CA: Academic Press.

Boyer, T. W. (2006). The development of risk-taking: A multi-perspective review. *Developmental Review, 26*(3), 291–345.

Boyer, p. , & Liénard, p. (2006). Why ritualized behavior? Precaution systems and action parsing in developmental, pathological and cultural rituals. *Behavioral and Brain Sciences, 29,* 1–56.

Brandstätter, V., Lengfelder, A., & Gollwitzer, p. M. (2001). Implementation intentions and efficient action initiation. *Journal of Personality and Social Psychology, 81,* 946–960.

Brocki, K. C., & Bohlin, G. (2004). Executive functions in children aged 6 to 13: A dimensional and developmental study. *Developmental Neuropsychology, 26*(2), 571–593.

Brown, D. R., & Gary, L. E. (1991). Religious socialization and educational attainment among African Americans: An empirical assessment. *Journal of Negro Education, 60,* 411–426.

Brown, T. N., Schulenberg, J., Bachman, J. G., O'Malley, p. M., & Johnston, L. D. (2001). *Consistency and change in correlates of youth substance use, 1976 to 1997* (Monitoring the Future Occasional Paper No. 49). Ann Arbor, MI: Institute for Social Research, the University of Michigan.

Cohen, A. L., & Gollwitzer, p. M. (2007). The cost of remembering to remember: Cognitive load and implementation intentions influence ongoing task performance. In M. Kliegel, M. McDaniel, & G. Einstein (Eds.), *Prospective memory: Cognitive, neuroscience, developmental, and applied perspectives* (pp. 367–390). Mahwah, NJ: Erlbaum.

Cummings, J. L., & Mega, M. S. (2003). *Neuropsychiatry and behavioral neuroscience* (2nd ed., revised). New York: Oxford University Press.

d'Aquili, E. G., & Newberg, A. (1993). Religious and mystical states: A neuropsychological model. *Zygon: A Journal of Religion and Science, 28,* 177–200.

Dewhurst, K., & Beard, A. W. (1970). Sudden religious conversions in temporal lobe epilepsy. *British Journal of Psychiatry, 117,* 497–507.

Fetzer Institute. (1999). Multidimensional measurement of religiousness/spirituality for use in health research: A report from the Fetzer Institute/National Institute on aging working group(. Bethesda, MD: Fetzer Institute.

Foshee, V. A., & Hollinger, B. R. (1996). Maternal religiosity, adolescent social bonding, and adolescent alcohol use. *Journal of Early Adolescence, 16,* 451–468.

Fromme, K., Stroot, E. A., & Kaplan, D. (1993). Comprehensive effects of alcohol: Development and psychometric assessment of a new expectancy questionnaire. *Psychological Assessment, 5,* 19–26.

Furby, L., & Beyth-Marom, R. (1992). Risk taking in adolescence: A decision making perspective. *Developmental Review, 12,* 1–44.

Galanter, M. (2006). Innovations: Alcohol and drug abuse: Spirituality in Alcoholics Anonymous: A valuable adjunct to psychiatric services. *Psychiatric Services, 57,* 307–309.

Geschwind, N. (1983). Interictal behavioral changes in epilepsy. *Epilepsy, 24*(Suppl. 1), 523–530.

George, L. K., Larson, D. B., Koenig, H. G., & McCullough, M. E. (2000). Spirituality and health: What we know, what we need to know. *Journal of Social and Clinical Psychology, 19,* 102–116.

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., et al. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience, 2*(10), 861–863.

Goldberg, E., & Bougakov, D. (2005). Neuropsychologic assessment of frontal lobe dysfunction. *Psychiatric Clinics of North America, 28*(3), 567–580.

Goldberg, J., & Fischhoff, B. (2000). The long-term risks in the short-term benefits: Perceptions of potentially addictive activities. *Health Psychology, 19,* 299–303.

Goldman-Rakic, p. (1987). Circuitry of primate prefrontal cortex and regulation of behavior by representational memory. In V. Mountcastle & F. Plum (Eds.), *Higher cortical function: Handbook of physiology* (pp. 373–417). New York: American Physiological Society.

Gollwitzer, p. M. (1990). Action phases and mind-sets. In E. T. Higgins & R. M. Sorrentino (Eds.), *The handbook of motivation and cognition: Foundations of social behavior* (vol. 2, pp. 53–92). New York: Guilford Press.

Gollwitzer, p. M. (1993). Goal achievement: The role of intentions. *European Review of Social Psychology, 4,* 141–185.

Gollwitzer, p. M. (1996). The volitional benefits of planning. In p. M. Gollwitzer & J. A. Bargh (Eds.), *The psychology of action: Linking cognition and motivation to behavior* (pp. 287–312). New York: Guilford Press.

Gollwitzer, p. M. (1999). Implementation intentions: Strong effects of simple plans. *American Psychology, 54,* 493–503.

Gollwitzer, p. M., & Brandstätter, V. (1997). Implementation intentions and effective goal pursuit. *Journal of Personality and Social Psychology, 73*(1), 186–199.

Gollwitzer, p. M., & Schaal, B. (1998). Metacognition in action: The importance of implementation intentions. *Personality and Social Psychology Review, 2,* 124–136.

Gorsuch, R. L. (1995). Religious aspects of substance abuse and recovery. *Journal of Social Issues, 51*(2), 65–83.

Hadaway, C. K., Elifson, K. W., & Petersen, D. M. (1984). Religious involvement and drug use among urban adolescents. *Journal for the Scientific Study of Religion, 23,* 109–128.

Hawkins, J. D., Herrenkohl, T. I., Farrington, D. P., Brewer, D., Catalano, R. F., Harachi, T., et al. (2000). *Juvenile Justice Bulletin. Predictors of youth violence.* Washington DC: Office of Juvenile Justice and Delinquency Planning.

Haznedar, M. M., Roversi, F., Pallanti, S., Baldini-Rossi, N., Schnur, D. B., & Licalzi, E. M., et al. (2005). Fronto-thalamo-striatal gray and white matter volumes and anisotropy of their connections in bipolar spectrum illnesses. *Biological Psychiatry, 57,* 733–742.

Heckhausen, J. (1991). Adults' expectancies about development and its controllability: Enhancing self-efficacy by social comparison. In R. Schwarzer (Ed.), *Self-efficacy: Thought control of action* (pp. 107–126). Washington, DC: Hemisphere.

Heckhausen, H., & Gollwitzer, p. M. (1987). Thought contents and cognitive functioning in motivational vs. volitional states of mind. *Motivation and Emotion, 11,* 101–120.

Horvath, p. , & Zuckerman, M. (1993). Sensation seeking, risk appraisal, and risky behavior. *Personality and Individual Differences, 14,* 41–52.

Huttenlocher, R. P., & Dabholkar, A. S. (1997). Regional differences in synaptogenesis in human cerebral cortex. *Journal of Comparative Neurology, 387*(2), 167–178.

Jessor, R., Chase, J. A., & Donovan, J. E. (1980). Psychosocial correlates of marijuana use and problem drinking in a national sample of adolescents. *American Journal of Public Health, 70,* 604–613.

Jessor, R., & Jessor, S. L. (1977). *Problem behavior and psychosocial development.* New York: Academic Press.

Koenig, H. G., McCullough, M., & Larson, D. B. (Eds.) (2001). *Handbook of religion and health: A century of research reviewed.* New York: Oxford University Press.

Lavery, B., Siegel, A. W., Cousins, J. H., & Rubovits, D. S. (1993). Adolescent risk-taking: An analysis of problem behaviors in problem children. *Journal of Experimental Child Psychology, 55,* 277–294.

Levin, H. S., Culhane, K. A., Hartmann, J., Evankovich, K., Mattson, A. J., Harward, H., et al. (1991). Developmental changes in performance on tests of purported frontal lobe functioning. *Developmental Neuropsychology, 7,* 377–395.

Lewis, D. A., Cruz, D., Eggan, S., & Erickson, S. (2004). Postnatal development of prefrontal inhibitory circuits and the pathophysiology of cognitive dysfunction in schizophrenia. *Annals of the New York Academy of Sciences, 1021,* 64–76.

McBride, D. C., Weatherby, N. L., Inciardi, J. A., & Gillespie, S. A. (1999). AIDS susceptibility in a migrant population: Perception and behavior. *Substance Use and Misuse 34*(4–5), 633–652.

McNamara, p. (2002). The motivational origins of religious practices. *Zygon: A Journal of Religion and Science, 37*(1), 143–160.

Metcalfe, J., & Mischel, W. (1999). A hot/cool system analysis of delay of gratification: Dynamics of willpower. *Psychological Review, 106,* 3–19.

Miller, B. L., & Cummings, J. L. (1999). *The human frontal lobes: Functions and disorders*. New York: The Guilford Press.

Milne, S., Orbell, S., & Sheeran, p. (2002). Combining motivational and volitional interventions to promote exercise participation: Protection motivation theory and implementation intentions. *British Journal of Health Psychology, 7,* 163–184.

Mischel, W. (1983). Delay of gratification as process and as person variable in development. In D. Magnusson & V. P. Allen (Eds.), *Human development: An interactional perspective* (pp. 149–165). New York: Academic Press.

Mischel, W., & Ayduk, O. (2004). Willpower in a cognitive-affective processing system: The dynamics of delay of gratification. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 99–129). New York: Guilford Press.

Mischel, W., Shoda, Y., & Peake, p. (1988). The nature of adolescent competencies predicted by preschool delay of gratification. *Journal of Personality and Social Psychology, 54,* 687–696.

Mischel, W., Shoda, Y., & Rodriguez, M. L. (1989). Delay of gratification in children. *Science, 244,* 933–938.

Newberg, A. B., Alavi, A., Baime, M., Mozley, p. D., & d'Aquili, E. G. (1997). The measurement of cerebral blood flow during the complex cognitive task of meditation using HMPAO-SPECT imaging. *Journal of Nuclear Medicine, 38,* 95.

Northoff, G., & Bermpohl, F. (2004). Cortical midline structures and the self. *Trends in Cognitive Sciences, 8*(3), 102–107.

Orbell, S., Hodgkins, S., & Sheeran, p. (1997). Implementation intentions and the theory of planned behavior. *Personality and Social Psychology Bulletin, 23,* 945–954.

Orbell, S., & Sheeran, p. (2000). Motivational and volitional processes in action initiation: A field study of the role of implementation intentions. *Journal of Applied Social Psychology, 30,* 106–143.

Passingham, R. E. (1995). *The frontal lobes and voluntary action*. New York: Oxford University Press.

Paus, T., Collins, D. L., Evans, A. C., Leonard, G., Pike, B., & Zijdenbos, A. (2001). Maturation of white matter in the human brain: A review of magnetic resonance studies. *Brain Research Bulletin, 54*, 255–266.

Persinger, M. A. (1987). *Neuropsychological bases of God beliefs*. New York: Praeger.

Prestwich, A., Lawton, R., & Conner, M. (2003). The use of implementation intentions and the decision balance sheet in promoting exercise behaviour. *Psychology and Health, 18*, 702–721.

Prochaska, J. O., & DiClemente C. C. (1986). Towards a comprehensive model of change. In U. Miller & N. Heather (Eds.), *Treating addictive behaviors* (pp. 3–27). New York: Plenum Press.

Prochaska, J. O., DiClemente, C. C., & Norcross, J. (1992). In search of how people change. *American Psychologist, 47*, 1102–1114.

Ramachandran, V. S., Hirstein, W. S., Armel, K. C., Tecoma, E., & Iragui, V. (1997). The neural basis of religious experience. *Society for Neuroscience Abstracts, 23* (Part 2), 1316.

Resnick, M. D., Bearman, p. S., Blum, R. W., Bauman, K. E., Harris, K. M., & Jones, J. (1997). Findings from the National Longitudinal Study on Adolescent Health. *Journal of the American Medical Association, 278*, 823–832.

Rolls, E. T. (2004). The functions of the orbitofrontal cortex. *Brain and Cognition, 55*(1), 11–29.

Schultz, W., Romo, R., Ljungberg, T., Mirenowicz , J., Hollerman, J., & Dickinson, A. (1995). Reward-related signals carried by dopamine neurons. In J. Houk, J. Davis, & D. Beiser (Eds.), *Models of information processing in the basal ganglia* (pp. 118–130). Cambridge, MA: The MIT Press.

Sethi, A., Mischel, W., Aber, J. L., Shoda, Y., & Rodriguez, M. L. (2000). The role of strategic attention deployment in development of self-regulation: Predicting preschoolers' delay of gratification from mother-toddler interactions. *Developmental Psychology, 36*, 767–777.

Sheeran, p. (2002). Intention-behaviour relations: A conceptual and empirical review. In M. Hewstone & W. Stroebe (Eds.), *European review of social psychology* (vol. 12, pp. 1–36). Chichester, UK: John Wiley & Sons.

Sheeran, p. , Milne, S., Webb, T. L., & Gollwitzer, p. M. (2005). Implementation intentions and health behaviours. In M. Conner & p. Norman (Eds.), *Predicting health behaviour: Research and practice with social cognition models* (pp. 276–323). Buckingham, UK: Open University Press.

Sheeran, p. , & Orbell, S. (1999). Implementation intentions and repeated behavior: Augmenting the predictive validity of the theory of planned behavior. *European Journal of Social Psychology, 37*, 231–250.

Sheeran, p. , & Orbell, S. (2000). Using implementation intentions to increase attendance for cervical cancer screening. *Health Psychology, 19*, 283–289.

Sheeran, p. , & Silverman, M. (2003). Evaluation of three interventions to promote workplace health and safety: Evidence for the utility of implementation intentions. *Social Science and Medicine, 56*, 2153–2163.

Singer, M., Dai, H., Weeks, M. R., & Malave, D. (1998). AIDS risk perception among women drug users in Hartford, CT. *Women, Drug Use, and HIV Infection, 27,* 67–85.

Smith, C. (2003). Theorizing religious effects among American adolescents. *Journal for the Scientific Study of Religion, 42*(1), 17–30.

Smith, C., & Faris, R. (2002). Religion and American adolescent delinquency, risk behaviors, and constructive social activities. Chapel Hill, NC: National Study of Youth and Religion.

Stanford, M. S., Greve, K. W., Boudreaus, J. K., & Mathias, C. W. (1996). Impulsiveness and risk-taking behavior: Comparison of high school and college students using the Barratt Impulsiveness Scale. *Personality and Individual Differences, 21*(6), 1073–1075.

Starkstein, S. E., & Robinson, R. G. (1991). The role of the frontal lobes in affective disorder following stroke. In H. S. Levin, H. M. Eisenberg, & A. L. Benton (Eds.), *Frontal lobe function and dysfunction* (pp. 288–303). New York: Oxford University Press.

Starkstein, S. E., & Merello, M. (2002). *Psychiatric and cognitive disorders in Parkinson's disease.* Cambridge: Cambridge University Press.

Steinberg, L. (2006). *Adolescence* (8th ed.). New York: McGraw-Hill.

Tek, C., & Ulug, B. (2001). Religiosity and religious obsessions in obsessive-compulsive disorder. *Psychiatry Research, 104*(2), 99–108.

Thorton, B., Gibbons, F. X., & Gerrard, M. (2002). Risk perception and prototype perception: Independent processes predicting risk behavior. *Personality and Social Psychology Bulletin, 28,* 986–999.

Troster, A. I., & Woods, S. P. (2003). Neuropsychological aspects of Parkinson's disease and parkinsonian syndromes. In R. Pahwa, K. E. Lyons, & W. C. Koller (Eds.), *Handbook of Parkinson's disease* (pp. 127–157). New York: Dekker.

Verplanken, B., & Faes, S. (1999). Good intentions, bad habits, and effects of forming implementation intentions on healthy eating. *European Journal of Social Psychology, 29,* 591–604.

Wallace, J. M., Jr., & Williams, D. R. (1997). Religion and adolescent health-compromising behavior. In J. Schulenberg, J. L. Maggs, & K. Hurrelmann (Eds.), *Health risks and developmental transitions during adolescence* (pp. 444–468). New York: Cambridge University Press.

Webb, T. L., & Sheeran, p. (2004). Identifying good opportunities to act: Implementation intentions and cue discrimination. *European Journal of Social Psychology, 34,* 407–419.

Welsh, M. C., Pennington, B. F., & Grassier, D. B. (1991). A normative study of executive function: A window on prefrontal functioning in children. *Developmental Neuropsychology, 7,* 131–149.

Wheeler, M. A., Stuss, D. T., & Tulving, E. (1997). Toward a theory of episodic memory: The frontal lobes and autonoetic consciousness. *Psychological Bulletin, 121,* 331–354.

Williams, B. R., Ponesse, J. S., Schachar, R. J., Logan, G. D., & Tannock, R. (1999). Development of inhibitory control across the lifespan. *Developmental Psychology, 35*, 205–213.

Winstanley, C. A., Theobald, D. E. H., Dalley, J. W., Cardinal, R. N., & Robbins, T. W. (2006). Double dissociation between serotonergic and dopaminergic modulation of medial prefrontal and orbitofrontal cortex during a test of impulsive choice. *Cerebral Cortex, 16*, 106–114.

Wise, R. A. (2005). Forebrain substrates of reward and motivation. *Journal of Comparative Neurology, 493*(1), 115–121.

This page intentionally left blank

Chapter **6**

Analysis of Intervention, Treatment, Policy, and Law That Contradict Ethical Criteria

Ann N. Dapice, PhD

Intervention in addiction through prevention, treatment, policy, and law requires responses guided by scientific knowledge. A considerable body of research demonstrates the physiology of addiction—whether the addiction is a substance such as food, nicotine, alcohol, or cocaine, or a behavior such as gambling, internet pornography, or stalking. Research also demonstrates the role of genetics, pre- and postnatal environment—or both. Studies show the wide variety and numbers of addictions that exist worldwide (Saah, 2005). Growing evidence indicates that what we call addiction is the result of once adaptive behaviors that no longer serve humans well (Dapice, 2006; Dapice, Inkanish, Martin, & Brauchi, 2002; Popkin, 2007). It is imperative that we analyze responses to addiction in order to determine to what extent they are knowledge-based and ethical.

When an illness causes people to act in ways that result in danger to themselves and others, what are adequate responses? If we cannot, or do not, stop addictive behaviors that are a danger to people and society, whose responsibility and obligation is it to protect all who are endangered? Our jails are full of the addicted and the mentally ill—who are often also addicted—and our statistics from treatment and intervention remain unsatisfactory. Progress on brain imaging and genetics now provides us with much new information, but older science has too often been ignored in addiction treatment, policies, and laws.

Until recently, the medical model has generally not blamed individuals who suffer from disease. It has been expected that individuals who have an illness

will attempt to obtain treatment. Whether treatments are successful or not, those with a disease will not be held responsible for the success or failure of treatments. For example, if individuals are diagnosed with cancers not related to known behaviors, and they undergo treatment for the cancers, relapses of the cancer will be seen to be either the result of ineffective treatment or a sign of the fact that no effective treatments are yet available. This is especially true in that cancer treatment is mainly carried out by physicians and nurses—not left to cancer victims to treat by themselves. In cancer and other diseases, patients are normally given a prognosis for treatment and a likely success rate for cure related to the kind of treatment. While the American Medical Association declared addiction to be a disease in 1956, treatment has most often not followed a disease model (Interlandi, 2008). In addition to the science of addiction, the science of moral development, values, and personality types provides information that is critical in understanding how we react to the problem of addiction.

HUMAN BEHAVIOR THAT AFFECTS RESPONSES TO ADDICTION

Studies of moral development over several decades (Kohlberg, 1984) have indicated that most people do not develop past lower stages of moral reasoning. Of six stages, stage 1 individuals focus on avoiding punishment and deferring to power. Stage 2 individuals seek to satisfy themselves and occasionally others. Stage 3 individuals behave for approval of others. Stage 4 individuals are oriented to authority and fixed rules. Stage 5 individuals define right action after examination. Stage 6 individuals use ethical principles such as the Golden Rule to decide ethical conduct. The addicted, their families, counselors, researchers, and policy and law makers can all be seen to be at one of six stages. Kohlberg and others in cross-cultural studies observed most people to be at stage 3—behavior for the approval of others (the peer pressure stage), with fewer individuals at lower stages and fewer still at higher stages.

Applied to addiction, a person at stage 1 will be dissuaded from activities of addiction out of avoidance of punishment and deference to power. Family members, counselors, researchers, and policy/law makers could all reason at this, the punishment stage. At stage 2 people will act just to satisfy themselves. An interesting thing happens, however, because at whatever stage the addicted individual can reason, acting on the addiction and maintaining the addiction moves the individual to the behaviors of stage 2, satisfying self—in spite of possible punishment (stage 1), in spite of lack of approval from others (stage 3), in

spite of laws (stage 4)—and even if the individual can reason at the a higher stages of 5 or 6, stages which generally correlate with altruistic behavior.

Kohlberg's work was based on conscious reasoning in reply to a situational dilemma. Dapice (1997) studied more than 6,000 individuals using the Values Unfolding Instrument (VUI), which was developed to allow access to less conscious values. Results from analyzing responses to the VUI showed that initial values stated by individuals unfolded to deeper values only six percent of the time and quite different values and themes developed in the unfolding process. Nearly all values first listed showed concern for others (e.g., love, compassion, fairness, etc.—ideal values) but unfolded to values that are concerned with self (real values). Factors such as gender, race, occupation, and religion made no difference in the results. When there was a conflict in values between self and altruism, people's behavior matched their unfolded self values (e.g., feeling good, success, status, feeling loved and appreciated). The large majority (93 percent) is self-oriented at the less conscious level but consciously believe themselves to be otherwise. They may act in ways that help others, but only if doing so accomplishes values important to them—feeling good, success, being liked and loved, and so forth. It is important to note, however, that seven percent not only unfolded more altruistic values but their anonymously documented behaviors paralleled their anonymously written results by their increased altruistic behaviors.

Such self values can play out in a number of ways in addiction. The most common unfolded values theme in the values research is feeling good. This could be called the dopamine or pleasure pathway value. These values themes also have negative versions, so in factor analysis the feeling good theme correlates with not having to struggle or have difficulty. Security versus fear could be called the fight/flight cortisol theme. Two other themes are: harmony and calm versus anxiety and stress, and excitement and stimulation versus boredom (Dapice, 1997, pp. 25–26). These have correlations to the functions of a number of neurotransmitters in the brain and body.

The Myers-Briggs Type Inventory (MBTI) includes four different continuums (Myers, 1980): Extroverts (E) focus on the outer world, people, and things; introverts (I) prefer an inner world of ideas and impressions. Sensing (S) types focus on the present and concrete information, while intuitive (N) types prefer the future, patterns, and possibilities. Thinking (T) types base decisions on logic and objective analysis of causation; feeling (F) types focus on values, the subjective, and person-considered concerns. Judging (J) types want a planned and organized life with decisions made; perceiving (P) types (like a flexible and spontaneous approach to life, preferring to leave their options

open. These four preferences yield 16 different types, for example, an ESFP would be extroverted, sensing, feeling, and perceiving (Myers, 1980).

One-size-fits-all addiction treatment ignores differences not only in diagnoses, or situations where there are multiple diagnoses, but also differences in Myers-Briggs psychological types (Myers, 1980). Whether client or counselor, differences in psychological type need to be assessed. Testing psychological type in an outpatient setting is easily and readily done. An individual's type does not provide an excuse, but it does give understanding of the different counseling needs to be met. Here are just a few of the clinical observations related to type.

Clinical experience shows that extroverts often enjoy attending Twelve-Step and other support groups, while introverts feel exposed and exploited or will feel like extroverts talk even when they have nothing important to say. Extroverted counselors will want to practice as their peers do (Kohlberg stage 3) so that they will be liked and appreciated by others. Introverts are more likely to counsel in a way that makes sense to them.

Sensing types will have difficulty with change, whether client or counselor. The saying, "If it ain't broke, don't fix it" is true here. For sensing types, things rarely are seen to be broken—even if everyone else can see that they are. Since sensing types are the majority of the population in the United States (Keirsey & Bates, 1978), this becomes a real challenge for counselors where a need for change is indicated. Intuiting types, however, relish change, and often will change from one modality to another—even when it is not required. This can be confusing to a vulnerable client.

Thinking types will be uncomfortable in talking about feelings, yet some treatment programs will insist that using a thinking preference is only denial. In fact, it is the failure to deal with both mental and emotional issues that cause problems. Feeling types will generally be more comfortable in talking about and labeling their feelings but, as noted before, may not be able to understand the consequences of their actions. They will often say, "I didn't intend for that to happen."

Judging types, client or counselor, will often respond using arbitrary power and control—as in "my way or the highway!" A counselor's or client's preference for gray areas (perceiving type) will be seen as lack of clarity to a judging type where things are only seen in terms of black and white. Meanwhile, a perceiving type will not respect a client or a professional who uses status or position to determine how treatment should occur. To a perceiving type, respect is always earned, never automatically given.

Clinical observation over a number of years testing clients for the 16 different psychological types indicated that no one type is more likely to be suc-

cessful in treatment than another. However, if counselors and clients do not understand the differences in type, there may be early failures—if for no other reason than that they will not understand, and thus, trust, each other.

Moral development, values research, and Myers-Briggs types involve motivations and behaviors that affect all in addiction—the addicted, the families, counselors, and policy and law makers. People with different Myers-Briggs types often have perceptions of reality that make it difficult for them to understand each other. Applying ethical principles in response to the addicted will be difficult for those at lower levels of moral and values development—where satisfying self is the main focus, consciously or otherwise.

ETHICAL THEORIES TO GUIDE INTERVENTION, POLICY, AND LAW

In *psychological egoism*, all actions are seen to be selfishly motivated and, unlike the Dapice research mentioned previously, there are no exceptions (Feinberg, 1987). People are perceived as only doing things for others when it satisfies them. *Ethical egoism* states that not only do people act in their own self interests but that they *should* act according to their own interests. The good that makes up people's interests may be defined as happiness or pleasure, or it may be of a more enlightened nature. Ethical egoism is considered to be the business ethic. Because the helping professions are expected to work at higher level of moral reasoning and action, a conflict can occur when employed by a for-profit organization. In *utilitarian theory*, a choice will be made that results in the greatest total good for everyone, regardless of the impact on the decision-maker (Hospers, 1982). It is the ethic of science. Scientific study can involve action that may result in good for many but not for all. For example, while using control groups, sick people may die when they are not being treated. *Deontological theory* is the professional ethic. The Golden Rule is the most common example of deontology and has been found as a stated ideal across cultures. A frequent misinterpretation of the Golden Rule is that immature people often understand and act on "Do unto others as they've done unto you" (Kohlberg stage 1–revenge) or "Do unto others so they'll do for you" (Kohlberg stage 2). Egocentric individuals may make decisions based on their own preferences. An example of this in addiction treatment it would be: "It worked for me so everyone should do what I did." In addiction, the goal should be what will best help the addicted client, not the preference of the professional, or a one-size-fits-all treatment. The needs of clients, not professionals, should be reflected in the professional codes of ethical conduct.

More recent ethical theories include:

1. *Rawls's distributive justice* (Rawls, 1971), which says that rights to liberty should be compatible with the liberty of others, and equities should be arranged to the advantage of all.
2. *Frankena's theology of obligation* (Albert, Denise, & Peterfreund, 1988), which says that results *must be good,* not just that we want or *intend* to do good. We should: (a) not do harm, (b) prevent harm, (c) remove harm, and (d) do and promote good.
3. *Firth's ideal observer* (Firth, 1970) states that there are five characteristics of moral decision-making. We should: (a) not have personal gain or interest (disinterestedness), (b) obtain as much information as possible about a client's problem and situation (omniscience), (c) try to understand consequences of decisions as if they are happening to us (omniprecipience), (d) react in the same manner to the same actions (consistency), and (e) not be sick, overly tired, or stressed when making moral decisions (normality).

Each of these theories shows that practicing in a professionally ethical way is not as simple as following a set of rules set out by one's profession. However, most of us were taught the universalizing Golden Rule as a standard. All major religions, as well as philosophy, have taught this standard. These modern ethical guidelines help us do the Golden Rule better. As we learn more about the science of any problem, we are challenged to develop better responses through treatment, policies, and laws in order to meet our ethical obligations.

SCIENTIFIC RESEARCH TO GUIDE RESPONSES TO THE PROBLEM OF ADDICTION

Tobacco and food are in competition for first place as fatal forms of substance abuse (Tanner, 2004). Obese individuals have the same processes occurring in their brains as do users of cocaine (Brookhaven National Laboratory, 2004). Obesity is a primary factor in cardiovascular disease and type 2 diabetes. Nicotine remains in first place as a cause of what is called "preventable death" —known ethically as "preventing harm" —yet use of nicotine has often been condoned and encouraged through reward systems in mental health and drug rehabilitation facilities, other addiction settings, and prisons. Recently, U.S. federal funding has required that smoking not be allowed in federally funded mental health, addiction facilities, or prisons. Yet well-intentioned counselors can still be heard to say, "We can't take their last drug away." This is a major concern since nicotine use is the leading cause of relapse to alcohol and other drugs (Mathews-Larson, 1991). New studies show the relationship of smoking to increased incidence of type 2 diabetes (Willi, Bodenmann, Ghali, Faris, & Cornuz, 2007, pp. 2654–2664), increased antisocial behavior on the

part of children whose mothers smoked during pregnancy (Button, Maughan, & McGuffin, 2005), and its harmful impact on users and those around them (Fields, 2008; Yam, 2007).

Since the 1980s we have had scientific knowledge about genetically shaped EEG brain waves, showing that reduced amplitude correlates with alcohol dependence. The chromosomal region that affects the P300 electrical brain wave has been identified and correlated with alcohol craving and predisposition to relapse. These EEG abnormalities occur in alcoholics and their children who have not yet used alcohol (Begleiter & Porjesz, 1988; Propping, Kruger, & Mark, 1981; Tabakoff & Hoffman, 1988; Volavka, Pollock, Gabrielli, & Mednick, 1985). Studies have shown that relapse to alcohol, cocaine, and opioid dependence can be predicted by brain waves and that these are better predictors of relapse than other variables (Bauer, 2001).

A growing body of knowledge shows the relationship of stress, cortisol, and the predisposition to self medication. A special issue on stress and drug use by the National Institute of Drug Abuse (2002) summarized the results of stress in laboratory experiments, severe prenatal stress, stress early in life, and posttraumatic stress disorder (PTSD). In 1999, Koob observed that heavy drinking not only depletes the brain's supplies of neurotransmitters necessary for feelings of wellbeing and pleasure (dopamine, serotonin, GABA, and opioid peptides), but also promotes the release of cortisol. Release of cortisol causes tension and depression, in turn causing the individual to drink more, leading to an ongoing vicious cycle. Imaging now allows the observation of damage from child abuse, neglect, sexual abuse, and verbal abuse to the cerebellar vermis in the brain. The damaged brain attempts to suppress electrical irritability physiologically, and the individual attempts to alleviate the irritability through abuse of alcohol and drugs (Anderson et al., 1999; Teicher, 2002).

Meanwhile, it has been shown that hypoglycemia affects up to 95 percent of alcoholics, causing them to become irritable, angry, depressed, hostile, and crave carbohydrates in the form of food or alcohol. Consumption of carbohydrates in various forms can relieve symptoms temporarily until surges of insulin cause the cycle to continue. In the alcoholic community this phenomena has been known as dry drunk and can be observed in individuals who have never used alcohol. A related statistic is that one in four deaths among sober alcoholics is due to suicide. There is little improvement in the suicide rate of alcoholics after sobriety. Addiction treatment requires more than just removing the substance or behavior (Bell & Martin, 2002; Mathews-Larson, 1991).

The need for effective addiction treatment that promotes physical healing and repairs physiological damage is not new and has been studied for decades. Bill Wilson, the cofounder of Alcoholics Anonymous (AA), first established

the link between alcoholism and hypoglycemia and the need for biochemical treatment using niacin and B vitamins to regulate blood sugar. His own experience suffering depression long after sobriety caused him to research the need for these vitamins in treating alcoholism. However, he was advised by his board not to incorporate this information into AA practice (Mathews-Larson, 1991). In spite of changing attitudes that begin to see addiction as a legitimate illness, as opposed to a moral failing, there is resistance from some in the addiction community who are wedded to Twelve-Step programs (Interlandi, 2008, p. 38).

Success rates from Twelve-Step and all but a few rehabilitation programs range between three and eight percent after four years (Mathews-Larson, 1991; National Institute on Alcohol Abuse and Alcoholism, 1996; Polick, Aarmor, & Bracker, 1980). A person diagnosed with cancer, given that prognosis, would likely dictate a last will and testament and make a desperate attempt to find research or treatment that is more successful. In cancer treatment, as in other medical treatment, it is ethically and legally required that such information, the prognosis, be given to victims of disease. It is important that addiction counselors and agencies do the same. When children have a serious disease such as cancer, the parents are legally forced to have them treated based on scientific research if they do not choose to do so on their own. It is curious that we do hold similar rules for underage addicted children who likewise have court ordered treatment—but it is not usually treatment that is based on scientific research.

According to the largest survey of Americans' drinking ever conducted (Dawson, 1996) only about one in four alcohol-dependent individuals enters treatment, including AA. According to AA, 1 in 10 of those who come to AA continues as long as a year. In the largest trial of psychotherapy so far (National Institute on Alcohol Abuse and Alcoholism, 1996), few alcoholics abstained for even as long as a year following treatment: 9 percent for outpatient treatment, and 35 percent for those who first had inpatient treatment. Another study (Krystal, Cramer, Krol, Kirk, & Rosenheck, 2001) examining the benefits of Naltrexone for 627 randomly assigned veterans with chronic, severe alcohol dependence found no significant difference in outcomes between the Naltrexone groups and the placebo group. Even though Naltrexone is approved by the FDA for treatment of alcoholism, the researchers concluded that their research does not support such treatment. After hearing the low treatment success rates at a conference, one individual said, "Well, it worked for me—I'm one of the few! It's not my problem if it doesn't work for everyone else."

Addicts are often told that they have to bottom out first—advice that can be a death sentence. In a personal communication (September 19, 2003), Harold

Stecker , a psychologist who has worked for decades with the mentally ill and addicted, stated that given the reality that alcoholism is active in 10 percent of the population, the numbers that actually enter treatment, the numbers that relapse, and the death rates, 19 out of 20 bottom out through death. Whatever the intent of advice about bottoming out, the reality is that most destroy their lives, often the lives of others, and the longer alcoholism continues without treatment, the greater the damage is to the brain, making treatment even more difficult. Such advice does harm and not good. It would be the same as telling cancer patients to wait for surgery, chemotherapy, and radiation treatment until they had pain sufficient to motivate them to endure treatment. Since pain is generally a late symptom in all cancers, this would ensure death. It is important to begin providing clients with the prognosis of addiction, as well as the success rates for treatment in facilities.

Successful treatment and intervention require responses that are appropriate to the disease. One would not send a cancer patient to a Twelve-Step program for treatment of the cancer without an ensuing lawsuit. A cancer victim might attend a cancer support group, but that would not be the only treatment provided. As Thomas McLellan of the University of Pennsylvania has said, "The specialist doesn't discharge you to a church basement. If he did, we would call it malpractice" (Interlandi, 2008, p. 42).

Research informs us that at a minimum, successful treatment requires: (1) balancing the affected EEG brain waves and neurotransmitters (Bauer, 2001; Begleiter & Porjesz, 1988; Koob, 1999; Propping, Kruger, & Mark, 1981; Tabakoff & Hoffman, 1988; Volavka et al.,1985), (2) addressing brain damage from substance abuse and cortisol toxicity, (Amen, 2005; Anderson, et al., 1999; National Institute of Drug Abuse, 2002; Teicher, 2002), (3) educating about conditions of hypoglycemia, and (4) supplying missing biochemicals such as L-Glutamine, B vitamins, and other substances needed by the brain (Amen, 2005; Bell & Martin, 2002; Mathews-Larson, 1991).

An example of research-directed treatment is FDA accepted cranial electrotherapy stimulation (CES). Studied in double- and triple-blind experiments for decades (Klawansky, Yeung, Berkey, & Shah, 1995), it works well for anxiety (Overcash & Siebenthall, 1989), depression (Marshall & Izard, 1974; Passini, Watson, & Herder, 1976), and PTSD, as well as cognitive damage from substance abuse (Smith, 2007, p. 45; Smith & O'Neill, 1975). Since it does not have to cross the blood-brain barrier it has no serious or lasting side effects. Electro-medicine stimulates and balances neurotransmitters while normalizing brain waves (Heffernan, 1997)—including the P300 craving wave (Braverman, Smith, Blum, & Smayda, 1990). By the 1980s, 80 percent of clients using CES treatment were still sober after seven years. CES has been studied and found

effective in a variety of addictions including alcohol, cocaine, benzodiazepines, heroin, marijuana, methadone, and nicotine (Jarzembski, 1985; Patterson, 1986). It is an inexpensive, self-administered microcurrent delivered by a 9-volt battery.

Addiction nutrition is also important whether the addiction is to food, a drug, or behavior. L-Glutamine is used sublingually to quickly regulate blood sugar and related cravings for carbohydrates, tobacco, and alcohol (Mathews-Larson, 1991). Carl Anderson, researcher at Harvard University Medical School, reported in a personal communication (September 10, 2004) that L-Glutamine is missing in the brains of smokers but not in the brains of those who can take or leave tobacco products. The kinds of foods we eat are critical to brain health. It is important to know the kinds of foods we should not eat in excess and how to supplement with nutrients what we do not normally receive in our diets (Amen, 2005; Mathews-Larson, 1991).

When treatment is not effective, it is important to test for levels of cortisol and to perform other laboratory blood work. A brain scan may be necessary to determine if there are other conditions in the brain not identified. Brain scans can demonstrate numerous physiological and anatomical conditions that otherwise will remain unknown and prevent effective treatment. While scans are expensive, the cost does not begin to compare with that of the addiction when not successfully treated. Treatment requires knowledge specific to the individual. Psychiatrist Daniel Amen states that studying brain scans and behavior has revealed that there is no typical person with alcoholism or drug addiction (Amen, 2005, pp. 51, 233). Except for brain imaging and recent progress in genetics, much research indicating the physiology of addiction and necessary interventions has been available for decades, yet finding addiction facilities that respond to these needs is often a challenge.

In addiction treatment there is a history of ignoring the mental and physical aspects of addiction while focusing on emotional and spiritual aspects. Much of this understanding is due to false dichotomies of Western thought that mental, emotional, physical, and spiritual aspects are somehow separate from each other. Brain imaging and physiological research show that the mental, emotional, physical, and spiritual are all processed through the brain. Neurotransmitters do not only exist in the brain but throughout the body. Humans are mental-emotional-physical-spiritual beings and these are all connected (Dapice, 2006, p. 251). Each is an aspect of the whole person. When people are ill, their reasoning, emotions, and spirits are all necessarily affected, and often they lose status and support from society as well. Becoming stuck in one aspect allows the areas ignored to become sicker.

Each aspect needs to be addressed: (1) mental—substance damage to the brain, examination of consequences, accurate information about addiction;

(2) emotional—stress, anxiety, depression, PTSD, anger, irritability, and grief; (3) physical—substance and cortisol damage to organs, EEG and neurotransmitter normalization, nutrition specific to addiction; (4) spiritual—practices individualized to the client's belief system that give meaning but are not compulsive or destructive and that move the person from despair to hope. In effective treatment of addiction, each aspect will improve with treatment that responds to the disease. The goal is the balancing of the mental, emotional, physical, and spiritual aspects for health (Dapice, 2006, pp. 256–259).

It is clear that treatments provided for even a narrower range of addictions such as alcohol, methamphetamines, and opiates have not generally responded to the science of addiction—some of which is decades old. It is also true that scientific findings often change as we develop better tools and understanding. Available knowledge is ongoing, and professionals are expected to continually update their knowledge and skills. Having not responded well to existing knowledge about specific addictions in the past, how do we take action regarding a much larger array of addictions such as food, gambling, shopping, and pornography?

Example after example of current response to addiction shows that professional omniscience—or updated knowledge—is missing. There is a reluctance to change even in the face of evidence that present actions are not working. Prevention of harm is often missing when there is evidence that child abuse, violence, traumatic events—as well as inherited and old once adaptive brains—are making people sick. Good intentions are too often not examined for results. Given the earlier discussed research on moral development, less conscious values, and personality types, none of this is a surprise. Since large numbers (the majority) of the population are at lower stages of moral and values development and focused on themselves, since large numbers (the majority) of the population are sensing and do not want change, since large numbers (the majority) of the population are extroverted (Keirsey & Bates, 1978) and want to do what their peers do even if it is not based on scientific knowledge, the surprise would be that responses to addiction are going well. That does not change the reality of addiction that we are faced with, however.

ETHICAL OBLIGATIONS OF ORGANIZATIONS, COMMUNITIES, AND GOVERNMENTS

Beyond addicted clients and counselors, ethical analyses are important in evaluating what organizations, agencies, communities, and governments are accomplishing when it comes to addiction and all who suffer as a result of the addiction. Are we accomplishing good and how is it measured? Agencies too often count how many they have served but do not evaluate the impact of the

service. In the human sciences we know far more about the prevention of harm and removing it than is actually acted on. How do we promote good? These are all policy questions that can be measured and should be part of all moneys and grants given to service providers—private or public. Most often they are not.

What are the results of treatment over time? It is often difficult to find statistics on a treatment facility's long-term success rates. Professionals casually talk about best practices at addiction conferences but too often do not demonstrate how and why they are best, the scientific rationale behind the practices, and what they have accomplished in long-term results. Accrediting bodies for licensed counselors have not only the ability but the obligation to ascertain that not only is new research being presented to those who work in the addiction field but that there is evaluation of what attendees learn through pre- and posttesting. It is a moral imperative that all education, whether courses and programs on addiction in universities or continuing education programs, be updated with the latest research—and how to implement the knowledge. Unfortunately, there has often been a schism between researchers in universities and counselors in the field, but this does not meet the needs of the addicted—only the needs of the researchers and counselors.

Those who make policies and laws need to reckon with the reality of stage 2 behavior of the addicted. In addiction, the effect of the drug on the pleasure pathway hijacks the addicted brain and moves the person to behaviors of stage 2, in spite of possible punishment (stage 1), in spite of lack of approval from others (stage 3), in spite of laws (stage 4)— even if the individual can reason at a higher stage 5 or 6, as some well known authors and statesmen have shown. One-size-fits-all laws ignore not only the levels of moral development but also personality types. In a more individualized approach, if the desire is to punish an extrovert, solitary confinement will be used, and if the desire is to punish an introvert, a lot of group activities will be used. The opposites will serve as rewards. Because we ignore research, we put useless dollars into programs, rewards, and punishments that cannot, by their nature, work.

CONCLUSION

If only for financial reasons, we cannot continue the treatments, policies, and laws of the present that are not working. Variously attributed to Einstein and Benjamin Franklin—as well as AA's "Big Book," it is said that "Insanity is doing the same thing over and over again and expecting different results." The human and financial costs worldwide are staggering. Brain processes once protective can now be destructive in a world with excesses of food, nicotine, alcohol, and illegal drugs—and excesses of opportunity for gambling, electronic pornography, and stalking.

Damage done to brains through stress and abuse and addiction is now visible for all to see through brain imaging. We must use our old and often broken brains to do the creative work they are still capable of in order to find brakes to use in a time of destructive excesses. Addiction endangers us all and must be stopped for the good of the addicted and the good of all. Finding brakes for our old and broken brains is an ethical imperative.

REFERENCES

Albert, E., Denise, T., & Peterfreund, S. (1988). *Great traditions in ethics* (6th ed.). Belmont, CA: Wadsworth.

Amen, D. G. (2005). *Making a good brain great.* New York: Harmony Books.

Anderson, C. M., Polcari, A. M., McGreenery, C. E., Maas, L. C., Renshaw, p. F., & Teicher, M. H. (1999). Cerebellar vermis blood flow: Associations with psychiatric symptoms in child abuse and ADHD. *Society for Neuroscience Abstracts, 25*(Part 2), 1637.

Bauer, L. (2001, September). EEG shown to reliably predict drug and alcohol relapse potential. *Neuropsychopharmacology, 25*(3), 332–340.

Begleiter, H., & Porjesz, B. (1988). Potential biological markers in individuals at high risk for developing alcoholism. *Alcoholism: Clinical and Experimental Research, 12*(4), 488–493.

Bell, L., & Martin, K. (2002). A natural prescription for addiction. *Counselor, 3*(4), 40–44.

Braverman, E., Smith, R., Blum, K., & Smayda, R. (1990). Modification of P300 amplitude and other electrophysiological parameters of drug abuse by cranial electrical stimulation. *Current Therapeutic Research, 48*(4), 586–596.

Brookhaven National Laboratory. (2004). *Exposure to food increases brain metabolism.* Retrieved September 21, 2004, from http://www.bnl.gov/bnlweb/pubaf/pr/2004/bnlpr041904.htm.

Button, T., Maughan, p. , & McGuffin, p. (2005). The relationship of maternal smoking to psychological problems in the offspring. *Early Human Development, 83*(11), 727–732.

Dapice, A. (1997). To thine own self . . . Values and ethics in a complicated world. Skiatook, OK: Institute of Values Inquiry.

Dapice, A. (2006). The medicine wheel. *Journal of Transcultural Nursing, 17*(3), 251–260.

Dapice, A., Inkanish, C., Martin, B., & Brauchi, p. (2002, September). Killing us slowly: When we can't fight and we can't run. *Native American Times.* Retrieved May 25, 2004, from http://www.dlncoalition.org/related_issues/killing_us_slowly.htm.

Dawson, D. A. (1996). Correlates of past year status among treated and untreated persons with former alcohol dependence: United States, 1992. *Alcoholism: Clinical and Experimental Research, 20*, 771–779.

Feinberg, J. (1987). Psychological egoism. In G. Sher (Ed.), *Moral philosophy* (pp. 5–15). San Diego, CA: Hartcourt Brace Jovanovich.

Fields, R. D. (2008). White matter matters. *Scientific American, 298*(3), 59–60.

Firth, R. (1970). Ethical absolutism and the ideal observer. In W. Sellers & J. Hospers (Eds.), *Readings in ethical theory* (pp. 212–214). Englewood Cliffs, NJ: Prentice-Hall.

Heffernan, M. (1997). The effect of variable microcurrents on EEG spectrum and pain control. *Canadian Journal of Clinical Medicine, 4*(10), 2–8.

Hospers, J. (1982). *Human conduct* (2nd ed.). San Diego, CA: Harcourt Brace Jovanovich.

Interlandi, J. (2008, March). What addicts need. *Newsweek, 151* (9), 37–42.

Jarzembski, W. B. (1985). Electrical stimulation and substance abuse treatment. *Neurobehavioral Toxicology and Teratology, 7*(2), 119–123.

Keirsey, D., & Bates, M. (1978). *Please understand me* (3rd ed.). Del Mar, CA: Prometheus Nemesis.

Klawansky, S., Yeung, A., Berkey, C., & Shah, N. (1995). Meta-analysis of randomized controlled trials of cranial electrotherapy stimulation: Efficacy in treating selected psychological and physiological conditions. *Journal of Nervous and Mental Diseases, 183*(7), 478–485.

Kohlberg, L. (1984). *The psychology of moral development: Essays on moral development* (vol. 2). San Francisco, CA: Harper and Row.

Koob, G. (1999). Alcohol stimulates release of stress chemicals. *Scripps Research Institute*. Retrieved March 10, 2008, from http://www.atforum.com/SiteRoot/pages/addiction_resources/webvol_4.pdf.

Krystal, J. H., Cramer, J. A., Krol, W. F., Kirk, G. F., & Rosenheck, R. A. (2001). Naltrexone in the treatment of alcohol dependence. *New England Journal of Medicine, 345*(24), 1734–1739.

Marshall, A. G. & Izard, C. E. (1974). Cerebral electrotherapeutic treatment of depressions. *Journal of Consulting and Clinical Psychology, 42*(1), 93–97.

Mathews-Larson, J. (1991). *Seven weeks to sobriety*. New York: Villard Books.

Myers, I. B. (1980). *Gifts differing.* Palo Alto, CA: Consulting Psychologists Press.

National Institute of Drug Abuse. (2002, April 8). *NIDA addiction research news*. Retrieved May 25, 2004, from http://www.drugabuse.gov/MedAdv/02/NS-04.html.

National Institute on Alcohol Abuse and Alcoholism. (1996). *NIAAA reports Project MATCH main findings*. Retrieved May 25, 2004, from http://www.niaaa.nih.gov/NewsEvents/NewsReleases/match.htm.

Overcash, S. J., & Siebenthall, A. (1989). The effects of cranial electrotherapy stimulation and multisensory cognitive therapy on the personality and anxiety levels of substance abuse patients. *American Journal of Electromedicine, 6*(2), 105–111.

Passini, F. G., Watson, C. G., & Herder, J. (1976). The effects of cerebral electric therapy (electrosleep) on anxiety, depression and hostility in psychiatric patients. *Journal of Nervous and Mental Disease, 163*(4), 263–266.

Patterson, M. A. (1986). *Hooked? Net: The new approach to drug cure*. London: Faber & Faber.

Polick, V., Aarmor, D., & Bracker, H. (1980). *The course of alcoholism, four years after treatment*, Santa Monica, CA: Rand Corp.

Popkin, B. (2007, September). The world is fat. *Scientific American, 297*(3), 91–93.

Propping, p. , Kruger, J., & Mark, N. (1981). Genetic disposition to alcoholism. An EEG study in alcoholics and their relatives. *Human Genetics, 59*, 51–59.

Rawls, J. (1971). *A theory of justice*. Cambridge, MA: Harvard University Press.

Saah, T. (2005). The evolutionary origins and significance of drug addiction. *Harm Reduction Journal.* 2(8). Retrieved March 5, 2008, from http://bmc.ub.uni-potsdam.de/1477–7517–2-8/.

Smith, R. B. (2007). Cranial electrotherapy stimulation: Its first fifty years, plus three: A monograph. Mustang, OK: Tate Publishing.

Smith, R. B., & O'Neil, L. (1975). Electrosleep in the management of alcoholism. *Biological Psychiatry, 10*(6), 675–680.

Tabakoff, B., & Hoffman, p. L. (1988). Genetics and biological markers of risk for alcoholism. *Public Health Reports, 103*(6), 690–698.

Tanner, L. (2004). *Study: We're eating ourselves to death.* Associated Press. Retrieved September 21, 2004, from www.cbsnews.com/stories/2004/03/09/health/main604956.shtml.

Teicher, M. H. (2002). Scars that won't heal: The neurobiology of child abuse. *Scientific American, 286*(3), 68–75.

Volavka, J., Pollock, V., Gabrielli, W. F., & Mednick, S. A. (1985). The EEG in persons at risk for alcoholism. *Recent Developments in Alcoholism, 3*, 21–36.

Willi, C., Bodenmann, p. , Ghali, W., Faris, p. , & Cornuz, J. (2007). Active smoking and the risk of type 2 diabetes. *Journal of the American Medical Association, 298*(22), 2654–2664.

Yam, p. (2007). Tobacco turnoff. *Scientific American, 297*(5), 16.

This page intentionally left blank

Part II

MODELS

This page intentionally left blank

Chapter **7**

Family Issues in the Field of Addiction Treatment

Giuseppe Carrà, MD, MSc, PhD, Massimo Clerici, MD, PhD, Hamid Ghodse, MD, PhD, DSc, Flavio Poldrugo, MD, PhD, and Nady el-Guebaly, MD

SUBSTANCE USE DISORDERS AND FAMILY INTERVENTIONS

Substance use and aspects of family life and relationships have long been connected to the initiation, exacerbation, and relapse of the spectrum of substance use disorders. In the literature, drug misuse is seen as both a "problem *of* the family" and a "problem *for* the family" (Bancroft, Carty, Cunningham-Burley, & Backett-Milburn, 2002, p. 15). Examples include the way in which poor parent-adolescent relationships consistently predict adolescent drug use across cultures and time or how negative communications and poor coping strategies within a marriage may initiate and perpetuate drug use while close relationships with healthy families-of-origin may buffer the abuser from relapse (Rowe & Liddle, 2003). The evidence that points to traumatic family experiences, such as childhood neglect, homelessness, abuse, loss, and bereavement, increasing the likelihood that a person will go on to have drug problems (Kumpfer & Bluth, 2004), can be seen as a problem *of* the family. As 60 to 80 percent of people who misuse drugs live or are in regular contact with their family (Stanton & Heath, 2005), drug misuse can also be said to be a problem *for* the family. The impact on family members may differ depending on the roles

and responsibilities within the family (Lewis & Williams, 1994). The impact may be psychological (e.g., depression and anxiety), physical (raised blood pressure and ulcers) (Velleman et al., 1993), social (feelings of isolation and work, family and social difficulties) (Hudson, Kirby, Firely, Festinger, & Marlowe, 2002), and financial.

Involvement of family members and carers in the assessment and treatment process may also facilitate a successful outcome for the user (Clerici et al, 1988). Furthermore, it is crucial to address the needs of carers and other family members, such as stigmatization and feelings of exclusion (Best, Campbell, & O'Grady, 2006).

In contrast, a number of reviews of family-based interventions over the last 30 years concluded that while several strategies were identified as promising, the relative dearth of empirical studies precluded the demonstration of efficacy or effectiveness of some modalities (Copello, Velleman, & Templeton, 2005; NICE, 2007; O'Farrell & Fals-Stewart, 2003). Within the last 10 years, more rigorous investigation of the impact of these interventions have been published, spurred by the availability of several manualized couple/family-based approaches. Three preponderant theoretical perspectives for family strategies in addiction have emerged (Fals-Stewart, O'Farrell, & Birchler, 2004). Table 7.1 compares the various tenets involved:

a. The family disease approaches whereby addiction is conceptualized as a family illness suffered not only by the addicted individual but by family members viewed as codependent. Each member must address his respective disease process individually. This tenet, while popular, has limited empirical evidence.
b. The family system approaches, whereby general systems theory is applied to the family interactions becoming organized around the addiction and maintaining a dynamic balance between the addiction and family functioning. Related strategies have included:
    - Unilateral engagement techniques educating the family about intervening as a group to confront or motivate their affected relative to become aware of his problem and need for remedial help. In this early phase, the concerned family member is not involved.
    - Inclusive systems techniques, preferred by many family therapists, aiming at modifying family dynamics and interactions that dominate the family's need for their relative's addiction
c. The behavioral approaches whereby it is assumed that family interactions reinforce the addictive behavior and therapy aims at breaking this reinforcement and fostering behaviors conducive to abstinence.

Table 7.1
Marriage and Family Therapy of Alcoholism

Model	Family disease (e.g., Twelve-Step, Al-Anon)	Family systems: (a) Unilateral (b) Systems engagement (e.g., CRAFT, PTC, Johnson Intervention)		Behavioral marital (BCT)
Theory	Addiction is a family illness of codependence	Family can either reinforce or discourage drinking	Addiction has "functional benefit" for family system	Relationship conflict may trigger or perpetuate addiction
Target group	Concerned significant other (CSO)	CSO	Family seen with addict	Addict with spouse
Goals	Personal well-being of family members	Influencing addict to enter treatment	Relationships change underlying addiction	Abstinence and stable relationships
Interventions	Detachment and focus on personal growth	Reinforce nondrinking communication and treatment invitation	Changes in roles, communication, and problem-solving	Abstinence contracts, increasing positives, and communication skills
Main evidence	Rychtarik and McGillicuddy (1998) manualized controlled study (CSO waiting list)	Miller, Meyers, and Tonigan (1999): manualized CRAFT; Johnson, Barber and Gilbertson (1998): promising PTC	Steinglass, Bennett, Wolin, and Reiss (1987): review of strategies	Fals-Stewart et al. (2004): more abstinence, less problems, more cost-effective

CRAFT: Community reinforcement and family training; PTC: Pressure to change.

However, despite methodological weaknesses in this area, a number of conclusions can be advanced that support wider use of family-focused interventions in routine practice. A slightly different conceptualization framework reviewed three types of interventions: those aimed at involving the family in engaging the substance misuser in treatment; those focused on jointly involving family members and substance-misusing relatives in the treatment of the latter; and those focused on affected family members in their own right (Copello et al., 2005). There is evidence that working with family members affected by substance misuse can trigger treatment entry for the substance misusers (Garrett et al., 1998). Furthermore, family involvement in treatment is effective

in a number of formats such as community reinforcement and family training (CRAFT) a manualized treatment program that includes training in domestic violence precautions, motivational strategies, positive reinforcement training for carers and their significant other, and communication training. (Meyers, Miller, Hill, & Tonigan, 1999) as well as network methods (Copello et al, 2002; Galanter, 1993). Finally, family interventions that see the family members as their main target have been shown to enhance coping mechanisms and improve attitudes (Copello, Templeton, Krishnan, Orford, & Velleman, 2000). Particularly, the five-step intervention seeks to help families and carers in their own right, independent of relatives who misuse drugs. It focuses on three key areas: stress experienced by relatives, their coping responses, and the social support available to them. Step 1 consists of listening and reassuring the carer, step 2 involves providing relevant information, step 3 counseling about coping, step 4 counseling about social support, and step 5 discussion of the need for other sources of specialist help. This intervention consists of up to five sessions. Though it has been shown to be feasible, and beneficial, for specialist drug and alcohol services to deliver a brief intervention to family members (Templeton, Zohhadi, & Velleman, 2007), organizational issues mean that routine delivery of such an intervention may not yet be possible, until full recognition is given to the view that addiction problems are best dealt with in a more holistic way that takes into account the family context within which most people live.

Among *adolescent populations*, cost-effective family systems approaches are now shown to significantly reduce drug use, achieve better retention rates, reduce comorbid externalizing problems, and improve school performance as well as overall family function. A major comparison of the available strategies has been the National Institute of Drug Abuse (NIDA) Cannabis Youth Treatment Study (CYT), a multisite randomized clinical trial of five state-of-the-art approaches. The family approaches included multidimensional family therapy (MDFT), a developmentally and ecologically oriented approach to reducing adolescent drug use and related problems by intervening in the multiple systems that maintain these symptoms. MDFT reduced adolescents' substance use by 27 percent from intake to discharge in the CYT study, and 65 percent had no past month use at the three-month follow-up (Liddle, 2002).

The second approach, the family support network (FSN), combined individual cognitive behavior therapy (CBT) and motivational enhancement therapy sessions for the adolescent with parent psychoeducational groups, home visits, and case management services. FSN reduced past month use by 44 percent between intake and the end of treatment and similar to MDFT had 64 percent no past month use at the three-month follow-up (Dennis et al., 2002).

A third approach, multisystemic therapy (MST), a social ecological approach to altering the multiple risk factors that create and maintain adolescent substance abuse and delinquency, was compared in a four-year outcome trial with community services as usual. The percentage of urines free of marijuana among adults receiving MST (55 percent) was higher than the comparison group (28 percent) (Henggeler, Clingempeel, Brondino, & Pickrel, 2002).

The main tested behavioral approach in this age group is functional family therapy, which aims at altering family maladaptive patterns that reinforce the adolescent's problems and establishing more effective problem-solving approaches (Waldron, Slesnick, Brody, Turner, & Peterson, 2001). This approach, particularly combined with CBT, demonstrated significant reductions in drug use between intake to treatment and a seven-month follow-up assessment.

In the adult groups, the preponderance of evidence has been provided by a series of studies involving behavioral couples therapy (BCT). Suited to a predominantly nuclear family structure in North America, BCT has gained prominence for enlisting the family's help after the alcohol or drug-dependent person has sought treatment. In recognition of the destructive cycle between substance abuse and relationship dissatisfaction, instability, and irritability, the main objectives of BCT are to eliminate abusive drinking and drug abuse and to engage the couple/family's support for the patient's effort to change as well as restructuring to that effect the couple/family's interactions patterns. BCT involves the spouse or partner expressing active support for the person who misuses drugs in reducing drug use, including via the use of behavioral contracts. Couples are helped to improve their relationship through more effective communication skills and are encouraged to increase positive behavioral exchanges through acknowledgement of pleasing behaviors and engagement in shared recreational activities The number of sessions administered have ranged between 15 to 20 sessions for individual couples over five to six months or brief group sessions for three to four couples together over 9 to 12 weeks. BCT has been fully manualized (Fals-Stewart et al., 2004). BCT is only suitable for couples who are committed to their relationships (partners for at least one year and, if briefly separated, attempting to reconcile). They must be able to acquire new information and skills and must both share a primary goal of abstinence. Aggression is an exclusion criterion and so are addicted couples, which are more enabling of each other. This in reality has had the unfortunate result of eliminating more female patients in a relation with a using male. Couples-based interventions were consistently associated with abstinence both at end of treatment and at 6- and 12-month follow-up for people with primary stimu-

lant or heroin dependence (Fals-Stewart, Birchler, & O'Farrell, 1996; Kelley & Fals-Stewart, 2002; Winters, Fals-Stewart, O'Farrell, Birchler, & Kelley, 2002). Most of the published outcome studies have focused on individual marital therapy. A pilot group BCT therapy program, based on the experience with 38 couples so far seen, 3 or 4 couples at a time, compared to 33 couples who refused the approach, demonstrated sizable improvement at the six-month follow-up (el-Guebaly, Richard, Currie, & Hudson, 2004).

CULTURE AND FAMILY THERAPY

One of the most significant limitations of this treatment literature, however, lies in the fact that most studies have been conducted in specialist research centers in the Western world, using approaches that may be difficult to apply in most settings. Family interventions within other cultural contexts must account, among other factors, for significantly different structures, power division, religions, and attitude toward drugs and the role of self-help. Families in multicultural societies for more than one generation will also develop their unique blended perspectives. Within the constraint of this update, a few examples of the impact of culture are presented.

Families are the conveyors of traditional cultural norms, rules, and values and will accordingly shape attitudes toward substance use. For instance, smoking among Pakistani and Bangladeshi men is an accepted tradition, contributing to group cohesion and identity. Consequently, young boys grow to become smokers. Contrastingly, smoking among women is not acceptable and carries a cultural taboo and stigma. When transplanted into multicultural societies, like in the United Kingdom, offspring of these families criticize their fathers for smoking (Bush, White, Kai, Rankin, & Bhopal, 2003).

Each family has its own culture and its own dynamic, and, when substance abuse occurs, the response of different family members is not necessarily uniform. For example, the family may split into rejecting and rescuing factions, leading to family conflict. Wider cultural factors undoubtedly also affect the family's response to substance abuse. In traditional societies, accustomed to folk methods of confronting difficulties and problem-solving, a professional therapist may meet considerable resistance, which can be alleviated if the position of an older person in supervising treatment is recognized and acknowledged. To understand the experience of a family from a very different culture requires a mind that can be open to new constructions of the pattern of family life, both internally and externally and in terms of custom and expectation. For example, respected kinship and authority structures in Asian and African extended families are strikingly different to those in Western families, and, in countries where

the family is the nucleus of society, it often plays a significant role in bringing the abuser to treatment. Certainly, substance abusers themselves perceive family support as most important for remaining in treatment (Oyefeso, Jones, & Ghodse, 1996). A review of research studies testing the effectiveness of culturally adapted family-based interventions reported an increase in treatment retention by 40 percent compared to generic family interventions (Kumpfer, Alvarado, Smith, & Bellamy, 2002). This is of particular importance as the use of services by ethnic minorities tends to be low.

Family attitudes toward drinking and the drinker may also be harnessed toward prevention and recovery. It has been recognized that in some family-centered European cultures (like the Italian, Greek, Portuguese, and Spanish), exerting familial informal social control with regard to drinking is successful in preventing alcohol abuse. Families act as the primary groups for alcohol-related problem intervention. The Italian experience in extended family recovery started 30 years ago. The number of Clubs of Treated Alcoholics (CAT), a program originally established in Croatia (Room, 1998), is four times greater than groups of Alcoholics Anonymous. The CAT considers addiction as a family and community style, thus involving family and community members early into treatment. Changes in alcoholics' social functioning should be visible within the community in order for it to change (Patussi, Tumino, & Poldrugo, 1996). Modalities of treatment mainly involve extended family and community reinforcement training, but other components like BCT are also present (Poldrugo, 2003).

EXPRESSED EMOTIONS AS ATTITUDINAL INDICATOR

Recently, emotional attitudes toward the patient of key relatives have been associated with maladaptive coping mechanisms. These attitudes are also shaped by culture. People with schizophrenia from families that express high levels of criticism, hostility, and emotional overinvolvement, according to the Expressed Emotion (EE) Index, have more frequent relapses than do people with similar problems from families that tend to be less expressive. Little is known about EE as predictor of relapse for substance-related disorders. Alcoholic patients with high EE spouses, when compared with their counterparts with low EE spouses, were more likely to relapse, had a shorter time to relapse, and drank on a greater percentage of days in the 12 months after starting behavioral marital therapy (O'Farrell, Hooley, Fals-Stewart, & Cutter, 1998).

In a pilot trial in Milan (Carrà & Clerici, 2004), frequencies of high EE were investigated by the Camberwell Family Interview in relatives of people with heroin dependence in outpatient and residential treatment and were compared

with those of relatives of schizophrenic patients in standard outpatient care. Thirty patients affected by *DSM-IV* opioid dependence were recruited before entering an outpatient program, and 47 key relatives were identified; 12 other patients were recruited before entering a residential program, and 24 of their key relatives were identified. A random comparison sample of 30 patients with schizophrenia were recruited along with 45 of their key relatives. Relatives with higher EE appeared to be positively correlated to the severity of the opiate condition. Indeed, the relatives scores of the residential group were similar to those of schizophrenia. The EE score did not, however, predict the potential for relapses in a 24-month follow-up. A trial is currently being conducted about the effectiveness of manualized psychoeducation treatment in improving knowledge about addiction and lowering the EE profile as well. Following nine 90-minute weekly sessions, the EE profile was lowered in 18 among 35 retested relatives (34.6 percent; $p = 0.002$) at six-month follow-up. The family's knowledge about addiction increased as well. The Italian cultural approach to psychoeducation seems to favor an open discussion about presented topics and belief systems. On the other hand, a confrontational approach is not often accepted and may result in further emotional suffering.

CONCLUSIONS

This update summarizes the growing empirical evidence supporting the effectiveness of couple- and family-based therapies in the management of substance-related disorders. It also highlights the importance of cultural sensitivity and adaptation to enhance accessibility and retention of families and to improve the outcome of these interventions.

Research and guidelines in cultural adaptation are sparse because of the complexities presented by the diversities of culture, yet cultural competence is an important therapeutic ingredient. A pilot study using an EE index is presented for its promising comparative cultural potential. More related studies are encouraged. So far, the most sound recommendations appear to be those drafted by NICE (2007), emphasizing the need to offer guided self-help, to facilitate contact with support groups, to provide information and education about drug misuse, and to explore and promote effective coping behaviors.

REFERENCES

Bancroft, A., Carty, A., Cunningham-Burley, S., & Backett-Milburn, K. (2002). *Support for the families of drug users: A review of the literature.* Edinburgh: Scottish Executive Interventions Unit.

Barber, J. G., & Gilbertson, R. (1998). Evaluation of a self-help manual for the female partners of heavy drinkers. *Research on Social Work Practice, 8,* 141–151.

Best, D., Campbell, A., & O'Grady, A. (2006). *The NTA's First Annual User Satisfaction Survey 2005.* London: NTA.

Bush, J., White, M., Kai, J., Rankin, J., & Bhopal, R. (2003). Understanding influences on smoking in Bangladeshi and Pakistani adults: Community-based, qualitative study. *British Medical Journal, 326,* 962–967.

Carrà, G., & Clerici, M. (2004). Expressed Emotion and substance-related disorders in outpatient and residential treatment: A case control study. *World Psychiatry, 3*(Suppl. 1), 100–101.

Clerici, M., Garini, R., Capitanio, C., Zardi, L., Carta, I., & Gori, E. (1988). Involvement of families in group therapy of heroin addicts. *Drug and Alcohol Dependence, 21,* 213–216.

Copello, A., Orford, J., Hodgson, R., Tober, G., & Barrett, C., on behalf of UKATT Research Team. (2002). Social behaviour and network therapy: Basic principles and early experiences. *Addictive Behaviors, 27,* 345–366.

Copello, A., Templeton, L., Krishnan, M., Orford, J., & Velleman, R. (2000). A treatment package to improve primary care services for the relatives of people with alcohol and drug problems. *Addiction Research, 8,* 471–484.

Copello, A., Velleman, R., & Templeton, L. (2005). Family interventions in the treatment of alcohol and drug problems. *Drug and Alcohol Review, 24,* 369–385.

Dennis, M. L., Titus, J. C., Diamond, G., Babor, T., Donaldson, J., Godley, S. H., et al. (2002). The Cannabis Youth Treatment (CYT) experiment: Rationale, study design and analysis plans. *Addiction, 97*(Suppl. 1), 16–34.

el-Guebaly, N., Richard, R., Currie, S., & Hudson, V. (2004). Behavioral couple therapy: Individual and group interventions. *World Psychiatry, 3*(Suppl. 1), 100–101.

Fals-Stewart, W., Birchler, G. R., & O'Farrell, T. J. (1996). Behavioral couples therapy for male substance-abusing patients: Effects on relationship adjustment and drug-using behaviour. *Journal of Consulting and Clinical Psychology, 64,* 959–972.

Fals-Stewart, W., O'Farrell, T. J., & Birchler, G. R. (2004). Behavioral couple therapy for substance abuse: Rationale, methods and findings. *National Institute on Drug Abuse, Science and Practice Perspectives, 2,* 30–41.

Galanter, M. (1993). Network therapy for substance misuse clinical trial. *Psychotherapy, 30,* 251–258.

Garrett, J., Landau, J., Shea, R., Stanton, M. D., Baciewicz, G., & Brinkman-Sull, D. (1998). The ARISE intervention—using family and network links to engage addicted persons in treatment. *Journal of Substance Abuse Treatment, 15,* 333–343.

Henggeler, S. W., Clingempeel, W. G., Brondino, M. J., & Pickrel, S. G. (2002). Four-year follow-up of multi-systemic therapy with substance abusing and substance dependent juvenile offenders. *Journal of the American Academy of Child and Adolescent Psychiatry, 41,* 868–874.

Hudson, C. R., Kirby, K. C., Firely, M. L., Festinger, D. S., & Marlowe, D. B. (2002). Social adjustment of family members and significant others (FSOs) of drug users. *Journal of Substance Abuse Treatment, 23,* 171–181.

Kelley, M. L., & Fals-Stewart, W. (2002). Couples- versus individual-based therapy for alcohol and drug abuse: Effects on children's psychosocial functioning. *Journal of Consulting and Clinical Psychology, 70,* 417–427.

Kumpfer, K. L., Alvarado, R., Smith, P. , & Bellamy, N. (2002). Cultural sensitivity and adaptation in family-based prevention interventions. *Prevention Science, 3,* 241–246.

Kumpfer, K. L., & Bluth, B. (2004). Parent/child transactional processes predictive of resilience or vulnerability to "substance abuse disorders." *Substance Use and Misuse, 39,* 671–698.

Lewis, I. D., & Williams, C. (1994). Grandparents parenting the second time around. *Association of Black Nursing Faculty Journal, 5,* 110–111.

Liddle, H. A. (2002). *Multidimensional family therapy for adolescent cannabis users.* Cannabis Youth Treatment (CYT) series (vol. 5). Rockville, MD: Center for Substance Abuse Treatment.

Meyers, R., Miller, W., Hill, D., & Tonigan J. (1999). Community reinforcement and family training (CRAFT): Engaging unmotivated drug users in treatment. *Journal of Substance Abuse Treatment, 10,* 291–308.

Miller, W. R., Meyers, R. J., & Tonigan, J. S. (1999). Engaging the unmotivated in treatment for alcohol problems: A comparison of three strategies for intervention through family members. *Journal of Consulting and Clinical Psychology, 67,* 668–697.

NICE. (2007). *Drug misuse: Psychosocial interventions.* NICE clinical guideline 51. London: National Institute for Health and Clinical Excellence.

O'Farrell, T. J., & Fals-Stewart, W. (2003). Alcohol abuse. *Journal of Marital and Family Therapy, 29,* 121–146.

O'Farrell, T. J., Hooley, J., Fals-Stewart, W., & Cutter, H. S. (1998). Expressed Emotion and relapse in alcoholic patients. *Journal of Consulting and Clinical Psychology, 66,* 744–752.

Oyefeso, A., Jones, M., & Ghodse, H. (1996). Ethnic monitoring in specialist drug services: A population-based analysis. *Journal of Substance Misuse, 1,* 91–96.

Patussi, V., Tumino, E., & Poldrugo, F. (1996). The development of the Alcoholic Treatment Club System in Italy: Fifteen years of experience. *Contemporary Drug Problems, 23,* 29–42.

Poldrugo, F. (2003). Homogenizing alcoholism treatment across Europe. In W. W. Fleischhacker & D. J. Brooks (Eds.), *Addiction: Mechanisms, phenomenology and treatment* (pp. 97–111). Vienna: Springer.

Room, R. (1998). Mutual help movements for alcohol problems in an international perspective. *Addiction Research & Theory, 6*(2), 131–145.

Rowe, C. L., & Liddle, H. A. (2003). Substance abuse. *Journal of Marital and Family Therapy, 29,* 97–120.

Rychtarik, R. G., & McGillicuddy, N. B. (2005). Coping skills training and 12-step facilitation for women whose partner has alcoholism: Effects on depression, the partner's drinking, and partner physical violence. *Journal of consulting and clinical psychology, 73*(2), 249–261.

Stanton, M. D., & Heath, A. W. (2005). Family/couples approaches to treatment engagement and therapy. In J. H. Lowinson, p. Ruiz, R. B. Millman, et al. (Eds.), *Substance abuse: A comprehensive textbook* (4th ed., pp. 680–689). Philadelphia: Lippincott Williams & Wilkins.

Steinglass, p. , Bennett, L., Wolin, S., & Reiss, D. (1987). *The alcoholic family*. New York: Basic Books.

Templeton, L., Zohhadi, S., & Velleman, R. (2007). Working with family members in specialist drug and alcohol services: Findings from a feasibility study. *Drugs: Education, Prevention and Policy, 14,* 137–150.

Velleman, R., Bennett, G., Miller, T., Orford, J., Rigby, K., & Tod, A. (1993). The families of problem drug users: A study of 50 close relatives. *Addiction, 88,* 1281–1289.

Waldron, H. B., Slesnick, N., Brody, J. L., Turner, C. W., & Peterson, T. R. (2001). Treatment outcomes for adolescent substance abuse at 4 and 7 months assessments. *Journal of Consulting and Clinical Psychology, 69,* 802–813.

Winters, J., Fals-Stewart, W., O'Farrell, T. J., Birchler, G. R., & Kelley, M. L. (2002). Behavioral couples therapy for female substance-abusing patients: Effects on substance use and relationship adjustment. *Journal of Consulting and Clinical Psychology, 70,* 344–355.

This page intentionally left blank

Chapter **8**

Codependence from a Family Systems Perspective

Mary Theresa Webb, PhD, and Ann Fabean, PhD

BACKGROUND

Thirty years ago, the chemical dependence/addiction field and the family systems field were beginning to explore how alcoholism and other chemical dependencies affect families. While those working in the addiction field noted certain behaviors of addicts and family members that were characteristic of the disease and started treating the whole family in family weeks, marriage and family counselors lacked assessment tools to determine the extent to which alcoholism and other addictions led to dysfunctional family patterns. Addiction professionals were the first to recognize that the dysfunctional patterns they observed in addicts' families contributed to the perpetuation of their denial systems and enabled addictions to continue. They labeled the co-addict, spouse, or parent of the addict *codependent*.

Dr. Timen Cermak, a psychiatrist, attempted to define a codependent personality disorder with appropriate diagnostic criteria (Cermak, 1986) so that treatment could be reimbursed by insurance He identified codependence as a personality disorder most resembling narcissism and echoism with intergenerational enmeshment of personal boundaries. He further classified two levels of codependence, primary and secondary. Although his theory had not been accepted as a mental health disorder when their studies began, other diagnoses, such as mixed personality disorder or dependent personality disorder, come closest. Yet neither Cermak nor other mental health professionals have put the term in a broader family systems model.

In the 1970s and 1980s, some addiction professionals began to recognize traits in the family system that provided insights into the concept of family codependence. Virginia Satir (1967), Don Wegscheider (1979), and Sharon Wegscheider-Cruse classified and named the chemically dependent family roles and functions. They explored the intergenerational aspects of alcoholic families. Since then, others such as , John Bradshaw (1988), and Melody Beattie (1989) have expanded on their work. Pia Mellody, with Andrea and Keith Miller, in their book, *Facing Codependence* (Mellody, Miller, & Miller, 1989), traced the roots of codependence in family processes.

Reactions from other mental health professionals followed. Some criticized the broad use of the term *codependent* in the addictions field. They claimed the term was oversimplified, overused, and was founded in an antiquated disturbed personality hypothesis (Horney, 1950).

Yet it was Karen Horney who named some of the characteristics that lead to codependence in her book, *The Neurotic Personality of Our Time* (Horney, 1937). She examined childhood histories of a number of neurotic persons and concluded that "the common denominator in all of them is an environment showing the following characteristics in various combinations: a lack of warmth and affection, actions or attitudes on the part of parents that arouse hostility" (Horney, 1937, p. 68). She discussed what professionals today call a *double bind*. This term means that children conditioned by their parents to feel guilty for expressing hostile feelings, particularly anger or for being oppositional when caught, also felt inadequate when they did rebel and broke a rule, no matter how unfair or incongruous the family rule might be.

Carter and McGoldrick (1989) in their book, *The Changing Family Life Cycle*, identified stressors that create disruptions of the family life cycle and produce dysfunctional symptoms. These stressors include family patterns, myths, secrets, and legacies handed down through multiple generations, as well as the unpredictable stressors of untimely death and chronic illness.

Meanwhile, respected family therapists such as Salvadore Minuchin recognized the impact a family has on an individual. "Family structure is the invisible set of functional demands that organizes the ways in which family members interact" (Minuchin, 1974, p. 51). In other words, he expressed how family members use their learned behavior in relating to and interacting with others outside the family environment, starting with conditioning in childhood.

Murray Bowen (1978), another renowned family therapist, focused his theories on determining a healthy family ego mass. In order to function in a healthy state, a family has to protect the whole system while, at the same time, respecting the autonomy of the individual within the system. This does not usually

happen in a system affected by chemical dependence (Zerwich & Michaels, 1989). Zerwich and Michaels concluded that low self-esteem is at the core of an addicted family's ego mass.

Froma Walsh (1982), in compiling her research on well-functioning family systems, reaffirmed Bowen's theories of a family needing a healthy ego mass. In an addicted family, the ego mass is skewed or unbalanced. The family roles identified by Satir (1967) and Wegscheider (1979) become necessary in order to rebalance the system.

Today, both addiction professionals and family therapists note that blaming, scapegoating, rigidity, emotional repression, shaming, poor self-esteem, excessive control, problems of intimacy, and blurring of generational boundaries form some of the pathology surrounding an addicted family. In an attempt to tighten up the concept of codependence to assist addictionologists, social workers, marriage and family counselors, psychologists, and psychiatrists, and to diagnose and treat individuals and family members affected by one person's addiction, we choose to define the term *codependence* as follows:

> Intergenerational patterns of living and problem-solving that are formed within a family system where there are or have been various dysfunctional patterns that include chemical abuse or dependence. The system then supports dependence and counterdependence in interpersonal relationships with a loss of individual autonomy and a distortion of reality.

We designed the Codependence Assessment Guide identifying eight constructs to treat affected persons holistically with subjective but quantifiable Likert Scales for therapists for treatment planning. Our model bridges the gap between the chemical dependence model that is based on the biological, psychosocial, and spiritual disease model of addiction and the family systems model or theory. We have refined and enlarged on our theory in the practice of therapy and in international training sessions. We have asked clients or trainees to prepare individual family genograms, diagrams of the intergenerational physical and mental illnesses, relationships, and patterns, in order to heal from codependence family patterns. Our tool was first presented in 1989 to the staff at the Staunton Clinic of Sewickley Valley Hospital, Pennsylvania. Since then we have taught it around the world and used it to help clients in therapy heal. We are indebted to the Beavers-Timberlawn Family Evaluation Scale (BT) for providing a methodology from which to work (Fredman & Sherman, 1987, pp. 33–35). In this chapter we present the eight constructs that follow with questioning strategies, expanding Fredman and Sherman's

measuring tools to include such items as chemical usage and attitudes that foster inappropriate use of chemicals. In addition, we included constructs of intimacy, triangulation, validation (which builds self-esteem), counterdependence, manipulation and control, shame, playfulness, and belief systems. We realize that normal families may exhibit some of these behaviors, therefore we designed our measuring tool and tested its validity to find just what range normal would be. We have revised this guide for laypersons to read and study to gain insight into their own nuclear and intergenerational family systems. (The full assessment guide for clinicians with the measuring scale will be available for download electronically on Amazon.com.)

CODEPENDENCE CONSTRUCTS: STRUCTURE OF THE FAMILY

Between Generations

Questioning Strategies

1. Can you explore or write about the traditions, patterns, or myths and how they got started in your family?.
2. What is/was your relationship with your grandparents?
3. To whom do/did you go in your family for guidance?
4. How do/did you see your grandparents or parents interacting or interfering in the life of your family?

Rationale

Interference and control by one or more senior family members who have set unhealthy patterns and traditions that must be followed leads to enmeshment or disengagement.

These reoccurring patterns, myths, and traditions are expected to be handed down from one generation to another. An example might be introducing alcohol as a rite of passage to those entering adolescence or upon graduating from high school. Some of these traditions or customs are abusive, such as wife inheritance, another tradition found in African tribal areas and today one of the leading factors in the spread of HIV/AIDS. In pathological systems, particularly alcoholic families, family members develop an identity that is that is not separate from that of the family and have malfunctioning hierarchical boundaries (Minuchin, 1974). In healthy systems, individuals learn about boundaries, and these boundaries are flexible. Boundaries are defined and explained in Pia Mellody's book, *Facing Codependence*, 1989).

Intimacy

Questioning Strategies

1. Are/were you relaxed with your family when you are/were together?
2. Can/could you share your feelings as well as thoughts with someone in your family or someone close to you?
3. Do/did you feel comfortable in close relationships?
4. Do/did all members of your family have a chance to express freely their thoughts, feelings, and wishes and have them accepted by others?
5. Did you have special times by yourself when you were growing up? Can you describe any of those times? Do you like being alone today?
6. Do/did good feelings about yourself depend on how others in your family system respond(ed) toward you?
7. Do you have any sexual problems? Can you explain what they are?

Rationale

If one is not able to share private thoughts, feelings, and wants because of the fear of being hurt or being abused this person lacks intimacy.

Erikson defined six progressive stages of growth from infancy to adulthood (Harder, 2002). These developmental stages are important to an individual's well-being and can make a difference where health or dysfunction are concerned. Erik Erikson's study of these stages showed the importance of each stage leading to the next. In the sixth stage, the stage that Erikson describes as intimacy versus isolation, a child grows into mature adulthood with a sense of well-being about himself both inside and outside around others. Intimacy in close relationships means that individuals can safely share private thoughts, feelings, and wants without the danger of being hurt. They can accept each other's weaknesses as well as appreciate and encourage another's strengths. Because emotional, physical, and sexual abuse occur in families where there is substance abuse, children who grow up in these families find themselves either dependent and intertwined or isolated.

Power and Control

Questioning Strategies

1. Does/did one person in the family attempt to make all decisions for other family members, to answer for anyone else, to tell anyone else what to do, or to accept the blame for someone else's behavior?
2. When something goes/went wrong, is/was someone else always to blame?

3. Are/were you responsible for another's behavior?
4. What is/was the worst thing that can/could happen if you don't/didn't fix the problems in your family?

Rationale

Next comes the issue of power and control in families and couple relationships and the issue of who exerts that power or control and how. Power can exhibit itself through an authoritarian parent or grandparent or through the seeming helplessness or dependency created by one family member's manipulation or abuse of one or more other family members. Sometimes power and control are manifested in destructive ways. In a codependent family, the chemically dependent person exerts the power, while the codependent spouse exerts the control. Controlling or being controlled is at the very core of codependency. In healthy families, the parents share power. Consensus and self-autonomy are fostered and encouraged.

Parental Coalition

Questioning Strategies

1. Who do/did you see as the dominant person or persons in your family system?
2. Can you describe how decisions are/were made in your family?
3. Do/did your mother and father argue very much? Do/did you ever feel that you have/had to side with one parent or another?
4. Do/did your parents ever talk about going on a date together?
5. Do/did you think your mother and father treat/treated you and each other fairly?

Rationale

In codependent families, parents cannot make shared decisions. One partner in the marriage is dependent and passive and the other dominates and acts as caretaker for the chemically dependent spouse. Cermak (1986) refers to this as the Narcissus/Echo pattern. Battles for power and control often ensue between parents. One child or another, at various times, gets caught in that power struggle, or all children are equally ignored in the ongoing conflict. In healthy family parents share decision making.

Triangulation

Questioning Strategies

1. Who do/did you think absorbs/absorbed the pain in your family?

2. Who in your family speaks/spoke for an issue between your parents?
3. Do/did you feel trapped in your family? If you do/did, explain how and when this happens/happened and what this is/was like.
4. Do/did you feel that you have/had to fix it or protect one of your parents from the other?

Rationale

Triangles are characteristics in families where there is unresolved conflict. Murray Bowen (1978) examined the phenomenon of triangulation in some depth. Triangles are prevalent in codependent families.

Either a child gets drawn up into in a conflicted parental relationship or an abused chemical or another form of addiction becomes the third party in the triangle.

CHEMICAL SUBSTANCES

Use of Chemical Substances

Questioning Strategies

1. Do/did you discuss chemical dependence or addiction in your family? Does/did anyone try to avoid talking about this issue? Or is/was there discomfort when the subject is/was brought up?
2. What is your favorite drink?
3. Did any of your grandparents have problems with alcohol or any other drug? When that person was abusing, who got more involved?
4. In what ways is your drinking or drug use different or like that of your grandparents?
5. Have you ever felt you needed to, or has anyone ever told you to, cut down on your drinking/drugging? Have you ever felt bad or guilty about your drinking and or drugging?
6. Do you need the drink or drug to steady your nerves or to relax?
7. Can you stop drinking/drugging after just one?

Rationale

Chemical abuse is indicated when someone is using a mindaltering chemical—alcohol, nicotine, marijuana, cocaine, or a prescribed drug—and that chemical is causing physical, emotional, or relational problems. Chemical dependency is indicated when the chemical being abused is causing that person to experience blackouts (with alcoholics), withdrawal symptoms, or inability to stop using the chemical. Appropriate use is defined as using prescription

medications under a physician's guidelines for appropriate use, or, in the case of alcohol, for special religious or family celebration functions. One or two ounces of alcohol a day may not cause problems for adult social use, but that depends on an individual's metabolism, weight, sex, and age, as well as other factors. For example, women can become alcoholic sooner than men because of a lack of a stomach enzyme called alcohol dehydrogenase, used by the body to digest alcohol (Frezza, Padova, Pozzato, Terpin, Bardona and Lieber, 1990).

Enabling

Questioning Strategies

1. Are/were you concerned about anyone in your family drinking or drugging too much?
2. Does/did anyone in your family act crazy, silly, or mean when drinking or drugging?
3. What rules do/did you have in your family around the use of alcoholic beverages or other mind-altering drugs?
4. Do/did you ever have any fights about one another's drinking or drug use?
5. Who drives/drove home after a party where there has/had been alcohol consumed?

Rationale

There is an interesting phenomenon that occurs in families where chemical abuse or dependence exists. The drinking or drug use is never talked about with the abuser directly. It is the family secret! Often a spouse or a child is expected to make sure there is an adequate supply in the house. An enabling spouse, parent, or sibling will deny there is any problem, or will use with the addict to keep him company. When chemical usage begins to be a problem, family members may talk among themselves. An enabler thinks that the right thing to do is protect and take care of another. Centuries of religious training and cultural conditioning reinforce doing to and for others before taking care of oneself. To be able to endure the suffering caused by someone else's behavior while under the influence of mind-altering chemicals is equated with Christian martyrdom (Cermak, 1986). Yet taking care of one's own self increases one's ability to be able to be concerned for others Family members of addicts need to stop enabling and care enough to come together with a professional to plan an intervention to express their how they feel about the drugging or drinking behavior with love when he or she is under the influence.

COMMUNICATION

Directness in Communication

Questioning Strategies

1. Does/did someone communicate aggressively both verbally and nonverbally (e.g., finger pointing or attacking or labeling another as "bad")?
2. Can/could you talk about things that matter to you in your family?
3. When there are/were discussions with family members do/did you feel comfortable in expressing your feelings openly and directly?
4. How do/did you go about planning for a family outing?
5. If something bothers/bothered you, whom do/did you tell?
6. How do/did you solve problems when they arise/arose in daily life, or are/were they ignored?

Rationale

In healthy families, each person has a sense of autonomy and importance in the family system. Communication is clear, direct, honest, and specific, and each person has a chance to express her opinions. Differences of opinion on issues are discussed and respected. In a codependent family, communication is indirect, often going through a third party. There are many "you" statements with judgmental overtones (I'm right and you're wrong). Differing opinions are not accepted. Cognitive distortions and irrational ideas abound. Sometimes family members interrupt or finish each others' sentences. Clear messages are not received or are misinterpreted. Communication contains judgmental or global statements—for example, the use of *should*, *ought*, *never*, or *always*, or personalizations, mind reading, and overgeneralizations.

Validation

Questioning Strategies

1. Do/did you or any of your family members talk of suicide or self-destructive behavior? If so, these individuals need/needed professional help.
2. Do/did your family members support one another? How?
3. Do/did your family members show more approval or disapproval of each other?
4. Can you name some ways your parents affirm/affirmed you as a person?

Rationale

In healthy family systems, nurturing and bonding give each member a positive sense of self-worth. In a codependent family system, messages conveyed are negative and critical. They are internalized and lead to a sense of inadequacy and wrongness about oneself. Codependent family systems are constantly invalidating each family member's perception of another's behaviors Adults raised in families where there is no validation look for validation by being people pleasers or controllers. They become depressed or exhibit psychosomatic illnesses to get attention.

Family members deny there are problems and need to put on a good public face ("I'm fine" or "We're all fine"). When together one or more family members will appear despondent or hopeless (e.g., eyes downcast, face sagging, or body slouched and hands limp). When discussing a family crisis, someone might respond: "It's not so bad" or, in the case of reoccurring bad dreams, "maybe I'm making it up," Someone else might say that the situation is hopeless or that life is not worth living.

Counterdependence

Questioning Strategies

1. Does/did the level of intensity in your family escalate?
2. Do/did sentences get cut off by interruptions?
3. Does/did one person try to counter or explain what another person says/said or does/did?
4. Are/were you comfortable talking with other family members?
5. Does/did someone always win and someone else always lose in your family discussions?
6. Do/did your discussions often end with your feeling frustrated or angry?
7. When you communicate. communicated with other persons in your family, do/did you feel that you have/had been heard? Describe how you know/knew that you've/you've been heard.
8. Do/did you have to explain or defend your thoughts or your feelings to others?

Rationale

Reacting to each other rather than listening to one another stifles effective communication. Such is often the case in codependent families. Communication about even the most routine matters often ends in shouting matches. One unhealthy way to relieve the tension between parents in these families is for

one person in the marriage to use chemical substances, food, or sex to relieve the tension. With constant interruptions, threats, and intimidations, messages are often cut off before they have a chance to be heard. This leads to internalized feelings of frustration and resentment. Children from these families may grow up into adulthood with posttraumatic stress syndrome.

FAMILY EMOTIONAL AFFECT

Range of Feelings Expressed

Questioning Strategies

1. Do/did you believe you or other family members use the various defense mechanisms in place of emotions?
2. Do/did you or family members have difficulty understanding what is meant when asked questions about feelings or when the answer is/was "I'm fine" or "we're fine"?
3. Can you discuss what feelings you are in touch with—now and in the past?
4. Is/was it safe to discuss feelings in your family?
5. Which feelings are/were okay to express and which feelings are/were not okay to express?

Rationale

In family systems where there is chemical dependence certain feelings are repressed, especially negative feelings, as well as feelings that might make an individual vulnerable, such as feeling hurt, sad, or scared Emotional affect is flat or frozen. Elaborate defense mechanisms are used to survive, such as projective identification, denial and avoidance, psychosomatic or exaggerated illness, fantasizing, intellectualizing, rationalizing, and minimizing.

Mood and Tone

Questioning Strategies

1. Can/could you or your family members smile and laugh comfortably, or does/did the laughter camouflage uneasiness?
2. Does/did one family member ridicule another or put the other person down either by her tone or via style of communicating?
3. Is/was there affection displayed by family members?
4. Is/was anger and hurt expressed in your family, and if so, how, and by whom? How are/were these emotions displayed?

Rationale

In healthy family systems, there is a fair exchange of mood and tone from affection and warmth to anger and hurt. In codependent systems, the playfulness often has an overtone of hostility, anger, and rage. There is a need to shame and demean. This toxic shame permeates the family system and is carried over from one generation to another. It is dehumanizing.

Manipulation and Control

Questioning Strategies

1. Do/did you usually do what is/was expected of you by family members whether you want/wanted to or not? How do/did you feel about it? Give an example.
2. Do/did you ever feel conned?

Rationale

As with power and control, the need to manipulate comes out of a sense of inadequacy and weak ego strength (Cermak, 1986). Since there is little trust in codependent family systems, empowering others is usually not an option. Manipulation is done either subtly or covertly with either blurred boundaries or disengagement. Underneath the layers of manipulation and control there is suppressed anger, hurt, and fear from living in a toxic shame-based family system.

Climate

Questioning Strategies

1. Do/did your family members seem relaxed with one another or do/did they seem uptight (e.g., do/did they sit on the edge of their chairs?)?
2. Does/did anyone have any nervous habits? Is/was there a certain rigidity of appearance?
3. Does/did anyone in your family have stress-related illnesses?
4. What happens/happened when there is/was a crisis in your family or in your relationships?
5. What ways do/did you find to relieve your stress when a crisis occurs?

Rationale

In normal families, times of anxiety and stress are unavoidable At other times, family members are relaxed and at ease with one another. In codepen-

dent families, stress and anxiety are constantly present. There is one crisis after another, and family members are under a state of constant tension and stress. Chemical and food abuse, compulsive exercising, smoking, and other compulsive, obsessive behaviors relieve the stress. Health problems such as cardiovascular diseases, back problems, intestinal disorders, ulcers, and other stress-related illnesses result.

Conflict Resolution

Questioning Strategies

1. When you have/had disagreements with others, how do/did you feel and how do/did you manage your feelings?
2. Do/did you avoid conflict in your relationships when they occur/occurred? Or do/did you discuss the issues around the conflict with the person whose views conflict/conflicted with yours?

Rationale

Having conflict and resolving it is normal in interpersonal relations. Real forgiveness can only occur when the conflicting parties hash out their differences and find some resolution. Conflict occurs whenever self-autonomy clashes with group expectations and goals or whenever one person's interests, wishes, and/or expectations clash with another's and there is an underlying "I win; you lose" competitive atmosphere. Anger, anxiety, resentment, blaming, and guilt continue until there has been some attempt to listen to each other, clarify the issues, and participate in a decision-making process.

In chemically abusive or codependent family systems, there is much unresolved intergenerational conflict. Conflict management styles range from blaming to intellectualizing, generalizing, or avoiding and pretending that everything is fine and there is no conflict. The intensity of these problems gets worse with each succeeding generation until all the family members find healing.

Trust

Questioning Strategies

1. Whom did you trust in your childhood family?
2. Whom do you trust now?

Rationale

As a result of unresolved conflict, power struggles, and inconsistent discipline and nurturing, dysfunctional and codependent family members learn either

not to trust or to trust blindly. Mistrust is rooted deeply in these families and is difficult to change. It is difficult even in the context of a counseling relationship.

ROLES

Family roles

Questioning Strategies

1. Can you identify yourself and family members in one of these roles either through the use of mobiles, role play, or reading any of the following authors' books?
2. What does being yourself mean to you? Is/was it okay to be yourself in your family?
3. Do/did you have a set role in your family?
4. Are you okay with who you believe you are now?

Rationale

Although Sharon Wegscheider-Cruse first defined and identified the roles of family members in the chemically dependent family as dependent, enabler, hero, scapegoat, lost child, and mascot, Don Wegscheider modified these and relabeled them victim, protector, caretaker, problem child, and family pet. Each role has set behaviors, feelings, and perceptions.

These roles were created to identify some of the typical behaviors family members may exhibit in codependent families as well as how the roles keep the family system in balance. Sometimes adults from codependent families rigidly define themselves by the role with which they identify, the role becoming a pathological label. In recent years family and marriage specialists have cautioned against assuming these roles are rigid and unchangeable since they can also be found in normal families. In normal families members move from one of these identified roles to another role more easily than in codependent families.

Blaming

Questioning Strategies

1. What function do/did you have in your family?
2. Do/did you ever think you cause/caused it when something goes/went wrong in your family?

Rationale

Blaming and attacking are common defense mechanisms for the chemically dependent or other addicted person. These defense mechanisms go along with the denial that surrounds the codependent family system. The blaming occurs because of repressed feelings of pain, hurt, and rejection that cannot be safely verbalized. When problems occur for the addicted person, others are blamed. This is really emotional abuse. Those accused can stay victimized in adult life. They survive by placating, protecting, or distracting. The one who placates is the family reconciler who tries to keep anger in check. The protector is the caretaker. The distracter uses diversionary tactics, such as humor, to relieve tension. Blaming and attacking by one family member of another can be observed through verbal use of "you" statements and aggressive body language such as finger pointing.

FAMILY RULES

Function

Questioning Strategies

1. What were some of the rules in your family when you were growing up?
2. Did you have any say in formulating these rules?
3. Were these rules clear?
4. How are rules made in your family now and what purpose do they serve?

Rationale

In healthy families, rules are flexible and set according to need. Children's concerns are expressed and heard, and both parents cooperate in leadership in the family. The rules parents set have a purpose of meeting the shared goals of their leadership. Rules in a dysfunctional family are inconsistent and frequently reflect parents' unfulfilled ego needs. In chemically affected families, not only are rules inconsistent but they are also usually rigid and closed, implicit but not explained ("My way is the only way"). There is no opportunity for other family members to express individual concerns. For the addict, "my way" varies according to "my" mood or the affect of "my" use of chemicals on "my" moods.

Honesty

Questioning Strategies

1. Do/did you always know why your parents tell/told you to do certain things?
2. Do/did you think the rules in your family are/were fair?

Rationale

The rules formulated in a healthy family are clear and concise, and they have a function, even though children in these families—particularly adolescents—may sometimes think the rules are not fair, whereas in codependent families rules are manipulative. These manipulative rules serve the addict's or the co-addict's neurotic needs at the moment.

Discipline

Questioning Strategies

1. How are/were you disciplined?
2. Who is/was the disciplinarian?
3. Do/did you always know why you are/were being disciplined?

Rationale

Every child needs clear boundaries for what is acceptable and unacceptable behavior. When the rules are clear and have a set function, children know an infraction of those rules means there will be consequences. In healthy families, those consequences are known to the child and are appropriate to that child's age level. The child might even have a say in setting those consequences. In codependent families, discipline is meted out inconsistently and frequently applied with anger. When one or both of the parents is abusing chemicals, and depending on the extent of the abuse, discipline is more likely to be physically or sexually abusive. It is chaotic and often violent.

Shame

Questioning Strategies

1. Can you describe what you like/liked or dislike/disliked about yourself or another member of the family?
2. Do/did family members put down one another? In what way?
3. Do/did you feel that you are/were abused or violated in any way? How?
4. Do/did anyone in your family have rage attacks?

Rationale

When children do something wrong, they feel a sense of shame. We experience shame when we violate our own or someone else's boundaries. Human beings learn by experience that they are not perfect and that they often fall short of their own and other's expectations. This is called healthy shame. This

kind of shame leads to a healthy conscience and a quality of humility. Shame in a nonnurturing, codependent family system is unhealthy or toxic. Such families are emotionally abusive and often sexually and physically abusive. Once a person's boundaries have been violated sexually, emotionally, physically, or spiritually (Mellody, et al., 1991) the child grows up feeling bad and rotten about herself.

PLAYFULNESS

Use of Humor

Questioning Strategies

1. Does/has anyone in your family ever teased you?
2. How does/did this make your feel?
3. When someone in your family tells/told jokes, do/did they make you laugh?
4. Can you tell a funny story about yourself right now?

Rationale

The ability to laugh with each other and oneself is important to personal and family health. The attitude of playfulness generated in family dynamics makes it fun to be together and with one another. Joking and teasing in healthy families is at no one else's expense. There are families where dry humor and sarcasm are frequently used to relieve tension. These are only attempts at humor and are not true humor. The more intense the climate in the family, the less humor there is. In family gatherings, there is frequent intense, serious talk. It is only with the use of alcohol or other mind-altering drug use or addiction that any lightening of intensity can take place, and then it may have tragic consequences and embarrassing repercussions to the family. The intensity is relieved at the expense of everyone's health.

Family Interaction

Questioning Strategies

1. Do/did your family's free time activities foster collaboration or one-upmanship?
2. How do/did you feel about family holidays and why?
3. What kind of games do/did you play when you get/got together?

4. Describe what happens/happened where and when your family gets/got together with relatives?
5. What do/did you do to have fun in your family?

Rationale

When healthy families play together, they collaborate to see that everyone has a good time and no one feels hurt or left out. Parents who encourage their children to play games competitively need to be careful that the competitive spirit is not carried over into family sibling rivalry and jealousy. This also applies to couples and how they spend their recreational time.

BELIEF SYSTEMS

Family Beliefs

Questioning Strategies

1. Are/were all of the members in your family loyal to a set of rules that are/were controlling and demanding of perfection?
2. Can/could anyone in your family ever do anything "good enough"?
3. Can/could you ever do anything "good enough" in the eyes of your family?
4. Do/did individuals in your family have boundaries?
5. Is/was it okay to discuss differences in beliefs and values?
6. Is/was it okay to have differences in beliefs and values accepted by the family?

Rationale

In the developmental process of an individual in a family system, internalization and externalization work together to formulate a belief system. If the external stimuli are negative and shame-bound, then the internalized process is one of negativity and shame. This permeates an individual's values and belief of self. Therefore, it is important for individuals and families to explore messages handed down through the generations that have affected values and beliefs.

Moral Standards

Questioning Strategies

1. What are/were your family's moral standards, and do you agree with them?

2. Are/were these interrelated with the family's denominational religious beliefs?
3. How are/were these morals enforced?
4. Does/did the family attend a church, synagogue, or other place of worship? Do/did they attend together or separately?
5. Is/was dialogue, questioning, or challenging of these beliefs or moral standards permitted?
6. How does/did your family respond to anyone who challenged, questioned, or disagreed?

Ratonale

For many, the adage that the family that prays together stays together is very significant. For such families, religion can be a stabilizing factor and can provide a positive moral code by which to live. (Note that moral codes can exist without religion, that religion is one, and a primary, source of moral code.) In functional, healthy families, a moral code can present certain morals and standards that require accountability while, at the same time, can permit dialogue about each individual's perspective on and perceptions of this moral code. Individuals are permitted to question and challenge and still be part of the system. In codependent family systems, religion or any moral code can be used to negate self-autonomy and insist on perpetuation of rigid, negative, and judgmental morality.

CONCLUSION: CHALLENGES

Codependent family members frequently exhibit symptoms of the family disease for a longer period of time than the addict does. The addict can become abstinent and receive individual and group support and treatment and be *in recovery*. Family members who do not understand that their reactive patterns or maladaptive behaviors need to be healed are not *in recovery*. They need as much support and treatment—if not more, in many cases—than the addict does. Attending Codependents Anonymous, Emotions Anonymous, Al-Anon, or ACOA Twelve-Step support groups as well as counseling can help codependent family members release their destructive family patterns, traditions, and behaviors so that they can function in healthier ways.

In the 1970s, 28-day treatment programs for addicts included an extra week for family members. However, in today's Western controlled-care climate, which allows only short outpatient treatment, family member treatment often is ignored. Even in Eastern or developing countries where therapeutic modalities of treatment exist, little is known or practiced for family recovery. In

African countries, women who live with alcoholic or HIV-infected males need special culturally sensitive care and support. Many family members who live or have lived with active addicts and who still suffer from depression or posttraumatic stress deserve the same attention and care as the addict.

REFERENCES

Beattie, M. (1989). *Co-dependent no more*. Center City, MN: Hazelden.

Bowen, M. (1978). Family therapy in clinical practice. Gason Arrowood.

Bradshaw, J. (1988). *On the family*. Health Communications.

Carter, B., & McGoldrick, M. (1989). *The changing family life cycle*. Allyn and Bacon, Division of Simon and Schuster.

Cermak, T. L. (1986). *Diagnosing and treating co-dependence* Johnston Institute and Hazelden, Center City.

Fredman, N., & Sherman, R. (1987). Handbook of measurements for marriage and family therapy. Brunner/Mazel.

Frezza, M., DiPadova, C., Pozzato, G., Terpin, M., Barona, E., & Lieber, C. S. (1990). High blood alcohol levels in women: The role of decreased gastric alcohol dehydrogenase activity and first class metabolism. *New England Journal of Medicine, 322*(2), 95–99.

Harder, A. F. (2002). *The developmental stages of Erik Erikson*. Retrieved January 21, 2009, from http://www.learningplaceonline.com

Horney, K. (1937). The neurotic personality of our time. W. W. Norton.

Harder, A.F. *The Developmental Stages of Erick Erikson* retrieved from www.learning online.com, 2002.

Kurtz, E. (1981). *Shame and guilt*. Hazelden.

Lederer, G. S., & Brown, F. H. (1991). The interlocking issues of gender and entitlement in alcoholic marriages. *Family Dynamics of Addiction Quarterly, 1*(3).

Mellody, P. , Miller, A., & Miller, K. (1989). *Facing codependence*. Harper and Row.

Minuchin, S. (1974). *Family and family therapy*. Harvard University Press.

Satir, V. (1967). *Con-joint family therapy*. Science and Behavior Books.

Walsh, F. (Ed.). (1982). *Normal family processes*. The Guilford Press.

Wegscheider, D. (1979). *If only my family understood me*. CompCare Publishers.

Wegscheider-Cruse, S. (1981). *Another chance*. Palo Alto, CA: Science and Behavior Books.

Zerwich, J., & Michaels, B. (1989). Nursing interventions for addicted patients. *Nursing Clinics of North America, 24*(1), 20–22.

Chapter **9**

Best Practices for Dual Diagnosis Treatment and Program Development: Co-occurring Mental Illness and Substance Disorders in Various Combinations

Kathleen Sciacca, MA

The literature suggests that between 40 and 60 percent of individuals presenting in mental health settings have a co-occurring substance abuse diagnosis, and 60 to 80 percent of individuals presenting in a substance abuse facility have a co-occurring mental health disorder (Mueser, Drake, Turner, & McGovern, 2006). An earlier review by the U.S. Alcohol, Drug Abuse and Mental Health Administration (ADAMHA) cited that at least 50 percent of the 1.5 to 2 million Americans with severe mental illness abuse illicit drugs or alcohol as compared to 15 percent of the general population (Ridgely, Osher& Talbott, 1987). This does not account for people with co-occurring disorders who are not seen in either system.

Receiving simultaneous treatment, in one setting—integrated treatment—has been shown to improve substance use outcomes, including abstinence or harm reduction from substance abuse. Clients who receive integrated treatment experience improvements in independent living, symptom management, competitive employment, and non-substance-using social contacts; these clients also expressed improved quality of life (Drake, McHugo, Xie, Packard, & Helmsletter, 2006). Unfortunately, research shows that 50 percent of people with co-occurring severe mental illness and substance disorders receive no care; 45 percent receive poor care; and only 5 percent receive evidence-based care (Drake et al. 2006).

This chapter will detail a treatment approach that has been integrated into a variety of program models across services and systems of care. This consensus-based best practice model incorporates evidence-based interventions for the

treatment of people who have varying profiles of co-occurring mental health and substance disorders. The discussion will be based in part on the author's experience as a consultant with the design and implementation of the model over the past 24 years across the United States and internationally.

There is a wide range of diagnosed co-occurring disorders that include mental illness and substance disorders. The following clinical profiles may be useful in identifying the target population within a given agency, service, or system. They are broad categories that are found across systems.

DIFFERENTIATING CATEGORIES OF PEOPLE WITH CO-OCCURRING MENTAL ILLNESS AND SUBSTANCE DISORDERS

There are two recognizable broad categories and one subcategory of people with co-occurring mental illness and substance disorders (Sciacca, 1991). These include:

1. **MICAA** (Mental Illness Chemical Abuse and Addiction): This clinical profile includes people who have a severe, persistent mental illness (SPMI) (Axis I), chemical abuse, or addiction. These clients are usually found in the mental health system. This acronym was introduced by the New York State Commission on the Quality of Care in 1987 (Sundram, Platt, & Cashen, 1986)
2. **CAMI** (Chemical Abusing Mentally Ill): This clinical profile includes people who have chemical dependency or abuse with co-occurring personality disorders (Axis II) and other emotional problems. Their mental health symptoms do not include SPMI. These clients are usually found in the substance abuse system.

A subcategory of CAMI is substance-induced acute symptoms, which may include psychotic symptoms such as delusions and hallucinations. These clients cross over from drug and alcohol services to mental health services, utilize both systems, and may be confused with the MICAA population. They do not have SPMI; rather, their acute symptoms are evoked by substance-induced chemical imbalances, making it difficult to differentiate these clients from people with active acute symptoms resulting from SPMI. The important difference is: When CAMI clients' acute symptoms go into remission, no negative or residual symptoms remain. Instead, they resume their usual behavior, attitudes, and activities.

The essential difference between MICAA and CAMI clinical profiles is that the MICAA client has a diagnosed severe persistent mental illness on Axis I (also termed major mental illness) that would be present without the use of

alcohol or drugs—for example, schizophrenia or bipolar disorder. The major mental illness is free-standing from substance disorders yet co-occurring with them. MICAA profiles include a broad range of co-occurring disorders that meet diagnostic criteria. Examples: schizophrenia and crack abuse; bipolar disorder and alcohol dependence; major depression and polysubstance abuse. Most MICAA clients require medication for the treatment of their mental illness.

The CAMI client does not have a severe persistent mental illness on Axis I. Mental health symptoms may be within the realm of personality disorders, Axis II—for example, antisocial personality disorder, borderline personality disorder, or acute symptoms that are substance-induced or mental health symptoms resulting from traumatic experiences. It is reported that the prevalence of personality disorders is at least 50 percent in substance-abusing populations (Nace, 1990). According to Nace (1990, p. 186), the most severe levels of substance abuse are associated with character pathology rather than serious mental illness.

Each of these systems of mental health and substance abuse has a responsibility for its particular population. The mental health system and specific programs need to include a screening tool to detect substance abuse in clients who are known to have a mental illness. In the best practice model presented here, the modified CAGE (cut down, annoyed, guilty, and eye-opener) questionnaire (Mayfield, McLeod, & Hall, 1974, Sciacca, 1990–2008) is implemented for that purpose. The substance abuse system and specific programs need to employ a screening tool that screens for mental health symptoms among clients who are known to have substance disorders. In the model presented here, the Mental Illness Screening Form (MISF) (Sciacca, 1990–2008) is used.

Where there may not be any clinical information, such as among people who are homeless, screening for both mental illness and substance disorders is necessary. There are a number of screening tools and evaluative tools that are used to screen for mental illness and substance disorders for this purpose. The screening tools noted here are recommended for use at the point of client intake into services.

Clients who present with active acute mental health symptoms or substance-induced acute symptoms could benefit from drug and alcohol testing to determine if a particular episode is substance related. For example, a MICAA client who presents with acute symptoms may have evoked her symptoms through substance use or withdrawal. Either condition may exacerbate mental health symptoms. Empirical evidence strongly supports the adverse effects of substance abuse on the course of severe mental illnesses. The consequences of substance abuse for clients include symptom exacerbation, increased hospital-

ization, medication noncompliance, disruptive behaviors, and decreased social functioning (Mueser, Bellack, & Blanchard, 1992).

The CAMI client who presents with acute symptoms may have substance-induced symptoms due to chemical imbalances. Examples include: cocaine-induced psychotic disorder with delusions; cocaine-induced psychotic disorder with hallucinations; cocaine-induced mood disorder, and so forth. In either case, the knowledge that drugs or alcohol are involved in any particular acute episode will assist in ascertaining the possibility of interaction effects and also serve to provide some assurance that co-occurring disorders are not overlooked. This information is valuable for both provider and client, particularly if there are repetitions that warrant further evaluation or if there are indications of the need for dual diagnosis treatment.

ADDITIONAL SYMPTOMS FOUND IN MICAA AND CAMI

People who have MICAA or CAMI profiles have a high incidence of traumatic experiences, including sexual abuse. It has been reported that one in three women and one in seven men is the victim of sexual assault before age 18. Clinical studies report 30–90 percent of women and 30–45 percent of men in substance abuse treatment have a history of sexual abuse. Victims of sexual assault and rape are much more likely to use alcohol and other drugs to cope with the trauma of their victimization (Kilpatrick, Edwards & Seymour, 1992). Traumatic experiences may result in various symptoms including substance abuse and mental health symptoms. The resulting mental health symptoms should be included in evaluations and pronounced within the client's overall diagnostic profile. In some cases, a narrative of the client's traumatic experience accompanies the client's diagnostic profile as a aside story, but the resulting symptoms are not included in the diagnostic profile. For example, resulting depression, dissociation, anxiety, panic disorder, and posttraumatic stress disorder (PTSD) are not included.

Addressing symptoms resulting from trauma and trauma issues is important when designing integrated programs. In a five-year study conducted by the U.S. Substance Abuse and Mental Health Services Administration (SAMHSA), the findings showed that when each of these three areas—mental health, substance abuse, and trauma issues—were addressed with women using an integrated model, those who received counseling addressing all three areas showed greater improvement than those who did not. In addition, women who participated in treatment decisions regarding their own care had better outcomes (Morrissey, Jackson, et al., 2005; Morrissey, Ellis, A. R., et al., 2005; Sciacca & Dobbins, 2001).

MICAA and CAMI Clients in Treatment

MICAA and CAMI clients can be treated together in treatment groups, and when group leaders are comfortable with both populations their differences may enrich the group process. The model of group treatment for co-occurring disorders outlined in this chapter is appropriate for the inclusion of MICAA *and* CAMI clients, as well as for treating these populations separately. The distinction in treatment for clients who differ in these profiles is based on the degree of structure, treatment intensity, and pace of the treatment.

MICAA clients do well with less intense, less frequent treatment groups, ancillary services, and programming, at a pace that corresponds to their participation. CAMI clients, on the other hand, do better with more frequent (though not more intense or confrontational) treatment groups, ancillary services, and programming, at a pace that matches their participation. It has been demonstrated that people with co-occurring disorders have improved outcomes when they receive simultaneous treatment for *all* of their symptoms (SAMHSA, 2002).

THE BEGINNING OF TREATMENT FOR CO-OCCURRING DISORDERS

The first treatment groups designed specifically for people who have co-occurring disorders began in 1984, in the New York State Psychiatric Care System (Sciacca, 1987a, 1991, 1996). Many clients attending a psychiatric day treatment program had severe, persistent mental illness and co-occurring substance abuse and dependence. They had been falling out of the system prior to that time, and they continued to do so as new interventions got under way.

Today we find these clients in large numbers in alternative systems, particularly among the homeless and in the criminal justice system. Early surveys reported that 10 to 20 percent of homeless persons were dually diagnosed with severe mental illness and alcohol or drug problems (Tessler & Dennis, 1989). In the criminal justice system, 16 percent of jail and prison inmates were estimated to have severe mental health and substance-abuse disorders. Among detainees with mental disorders, 72 percent also had a co-occurring substance-abuse disorder. (National Alliance for the Mentally Ill, 2003).

In the mental health and substance-abuse systems, MICAA clients were essentially unwanted, believed to be untreatable and costly to the systems, and presented clinical challenges not considered to be within the domain of mental health *or* substance abuse practitioners. CAMI clients may have received treatment for their addictive disorders alone or, with the added complexities

of differential diagnosis in the CAMI population, mental health symptoms may have been interpreted as substance related (which some of them may have been) rather than preexisting conditions or free-standing symptoms (the alternative possibility) or may not have been addressed at all. During this time, the concept of dual disorders emerged and grew in popularity. Treatment systems, however, including mental health and substance abuse, were unresponsive. The New York State Commission on the Quality of Care later corroborated this claim (Sundram et al., 1986).

It continues to be held that there are systems barriers in both systems and that clients are perceived as 'misfits' at every level—at the system policy level, at the program design level, at the clinical practice level, and at the clinician competency and training level—in terms of regulations, information systems, funding mechanisms and clinical credentialing and certification" (Minkoff & Kline, 2004, p. 66).

Through early attempts to provide group treatment interventions for MICAA clients, they initially responded angrily to any suggestion that they might have substance-abuse or dependency issues. Some found this suggestion to be derogatory and interpreted that staff were calling them drug addicts, which implied stigma and very negative connotations for them, or they protested that their simple pleasure of drinking alcohol would now be taken away from them. Conversely, CAMI clients in the substance-abuse system found suggestions that they may have mental health symptoms to be derogatory and implying the stigma of being called crazy and all that implied. It became clear that developing interventions required that practitioners: (a) accept clients where they were and (b) accept symptoms and conditions, and each client's level of readiness to address these or to engage in a process of change. Interventions were "nonconfrontational, and included a phase model of intervention identifying clients' movement along a continuum of change delineated by group-treatment phases.

Pronouncing that clients were at different phases and stages of readiness to address substance abuse was in direct contrast to the traditionally held readiness criteria for acceptance into substance-abuse treatment services. Potential clients were expected to acknowledge their substance disorders, be ready and motivated to accept treatment, and comply with abstinence. MICAA clients did not present themselves in that light; therefore, there was no alternative but to provide combined mental health and substance abuse interventions within the mental health system—integrated treatment.

From the beginning, in 1984, it was necessary to begin at the client's readiness and motivation and to view progress or change incrementally. This was later corroborated through consensus in the SAMHSA-Center for Mental Health

Services (CMHS). The "Standards of Care, Practice Guidelines, Workforce Competencies and Training Curricula" (Sciacca, 1998a) report (SAMHSA-CMHS, 1998) which recommended phase and stage models of intervention for working with people who have co-occurring disorders. The report articulated some of the following recommendations:

- For each disorder, assessment should include a definition of the stage of change or level of motivation.
- When mental illness and a substance-use disorder coexist, each disorder should be considered as primary, and integrated dual primary treatment should be provided; the treatment for each disorder should be matched to the diagnosis and the stage of change (SAMHSA-CMHS, 1998).
- Within each subtype of the treatment population, consumers are in different phases of treatment and at different stages of change with regard to their illnesses. Thus a comprehensive array of interventions that are phase and stage specific is required.

As the dual diagnosis group treatment model evolved in 1984, three phases of group participation emerged (Sciacca, 1991; Sciacca & Thompson, 1996). In phase 1, for example, clients are less prepared to self-disclose or discuss their personal situations; in phase 2, they begin to talk about themselves and may include some early discussion of their own behavior. Two readiness scales were designed to measure clients' starting points and progress along a continuum of incremental change (Sciacca, 1990–2008) for their substance abuse and mental health symptoms (see Tables 9.1 and 9.2).

Very frequently, clients were at different levels of readiness to address different symptoms. Treatment usually began with clients who were physically addicted to drugs or alcohol, who actively used substances, and who had active mental health symptoms. It proceeded from denial through recovery (Sciacca, 1987, 1991), and through stages of change—precontemplation through maintenance (Prochaska & DiClemente, 1984). Clear strategies and treatment content were correlated to each readiness level (Sciacca, 1987, 1991; Sciacca & Thompson, 1996), and stage matched tasks were identified for the stages of change (DiClemente 2007; Prochaska & Norcross, 2001).

As the field of dual diagnosis forged ahead, in 1986 the New York State Office of Mental Health initiated the MICAA Training Site for Program and Staff Development New York. State-wide, this program remained viable until 1992. Providers of all disciplines, from a variety of systems, participated in training events and program-development initiatives. This included the substance-abuse system where CAMI clients were regularly encountered. Interventions and dual diagnosis programs were designed with the same elements: noncon-

Table 9.1
Sciacca Comprehensive Service Development Readiness Scale for Substance Abuse

Assessment of Client's Readiness to Address Symptoms or Engage in Treatment for Substance Abuse:

1. Client is denying the scope or existence of his substance-abusing behavior and is unwilling to seek treatment or information.
2. Client is denying the scope or existence of his substance-abusing behavior but is willing to learn more about alcohol/drugs, substance abuse, and other topics.
3. Client is agreeing that he has a substance-abuse problem but believes that he can manage it without additional treatment or information.
4. Client is agreeing that he has a substance abuse problem, is willing to seek information, but has not demonstrated working on the substance-abuse problem itself.
5. Client is aware of the problems that substance abuse has caused, is willing to seek information, and is engaged in some form of treatment to work on these problems.

Please write the number indicating the level of treatment readiness alongside each of the substances client uses.

Alcohol ___	Barbiturates ___	Cocaine ___	Marijuana ___
Hallucinogens ___	Crack ___	Stimulants ___	Heroin ___
Inhalants ___	Opiates ___	Tranquilizers ___	Other _____

Comments: _____
_____
_____
_____
_____

Source: Dual/Multiple Diagnosis Clinical Assessment, K. Sciacca, copyright 1984, 2000, 2003, 2005, 2008.

frontation, and phase- and stage-specific interventions correlating to each client's readiness to engage in treatment (Sciacca, 1996). This work has since been replicated in numerous states and cities—for example: Tennessee (Sciacca, 1998b), Washington, DC (Sciacca, 1999), Michigan (Sciacca, 1997a), Georgia ("GA Seeks Statewide Implementation," 2003).

The challenge was to engage clients in discussion about a variety of related topics, including symptoms, without requiring them to acknowledge substance abuse, mental illness, or other behavioral problems. As this objective became clear, an impersonal approach, one that did not require clients to agree or admit to having either mental health or substance-abuse symptoms was necessary. One strategy that facilitated this process was the inclusion of educational materials from a variety of media on a variety of topics that clients discussed and critiqued. Materials such a fact sheets, videos, and outside speakers were

Table 9.2
Sciacca Comprehensive Service Development Readiness Scale for Mental Illness

Assessment of Client's Readiness to Address Symptoms or Engage in Treatment for Mental Health Symptoms:

1. Client is denying the scope or existence of her mental health symptoms and is unwilling to seek treatment or information.
2. Client is denying the scope or existence of her mental health symptoms but is willing to learn more about mental health issues and other topics.
3. Client is agreeing that she has mental health symptoms, but believes that she can manage them without additional treatment or information.
4. Client is agreeing that she has mental health symptoms, is willing to seek information, but has not demonstrated working on the problem itself.
5. Client is aware of the problem(s) that mental illness has caused, is willing to seek information, and is engaged in some form of treatment to work on these symptoms.

Please write the number indicating the level of treatment readiness alongside each of the mental health symptoms.

Depression ___	Delusions ___	Psychosis ___	Mania ___
Suicidal ideation ___	Violence ___	Hallucinations ___	Anxiety ___
Isolation ___	Intrusions ___	Thought disorders ___	Panic ___
Other _____			

Comments: _____
_____
_____
_____
_____

Source: Dual/Multiple Diagnosis Clinical Assessment, K. Sciacca, copyright 1984, 2000, 2005, 2008.

included in groups. The focus was on discussing the opinions of clients on a broad range of topics rather than clients' self-disclosure or didactic education.

Fact sheets providing information about mental illness, substances, addictive processes, interaction effects, HIV, family issues, stress-related issues, and other topics could be read aloud by participants and discussed from various points of view, without implying that the participants were users, and without requiring self-disclosure. As this work proceeded, the group treatment approach that began in 1984 evolved into the following comprehensive model, which adapted to clients' responses and needs (Sciacca, 1987, 1991; Sciacca & Thompson, 1996). This process was further evolved with the development of the pregroup interview (Sciacca, 1990–2008).

DUAL DIAGNOSIS BEST PRACTICES GROUP TREATMENT MODEL

Interactions with clients in dual diagnosis groups resulted in further adaptations to meet their treatment needs and to engage them in explorations to assist them to clarify their situations (Sciacca, 1987, 1996, 2007). The objectives of this approach included the following:

1. Assist clients in recognizing and accepting that they have symptoms of mental illness and substance disorders and dispel any associated shame, self-blame, guilt, and stigma;
2. Help clients learn and understand the real properties of mental illness, substance abuse, and dependence, and the interactions between co-occurring disorders;
3. Facilitate explorations of the benefits and disadvantages of behavior change, leading to accepting or rejecting treatment;
4. Help clients reach an understanding of their unique situations and make decisions regarding change;
5. Provide a client-centered, empathic listener, who elicits and attends to clients' points of view, beliefs, experiences, feelings, disappointments, aspirations, and goals; and
6. Offer a support group where clients can give and receive support in a safe, respectful environment and where they can embark on a process of change with established alliances.

ENGAGEMENT IN DUAL DIAGNOSIS GROUPS

Once clients are identified as potentially having dual/multiple disorders through screening, the group leader is made aware of their candidacy for the dual diagnosis group.

The leader will include them on a client disposition form (Sciacca, 1984) and refer to that list to set up pregroup interviews as openings occur in the group.

The client will be advised by her primary counselor, or by other staff, that there is a group or program that may be beneficial to her and that the leader would like to meet to discuss this. The client's potential to have this meeting will determine how formal (scheduled date and time to meet with the leader) or informal (meet with client in the waiting area or community room) the setting of the interview will be. This interview may take place in a variety of settings across systems. The interview usually does not exceed one-half hour.

The group treatment process begins with the pregroup interview (Sciacca, 1987a, 1991, 1990–2008), conducted by the group leader prior to the client's participation.

The pregroup interview (Sciacca, 1987, 1991, 1990–2008) may begin with an open-ended question, such as, "What are your thoughts about participating in group discussions?" This provides an opportunity for clients to discuss their understanding of groups; if pertinent, their previous experiences in groups; or whether or not they believe they have any need for such an intervention. This is an opportunity for the group leader to listen to and understand clients' perspectives. The leader can now follow up with client-centered interviewing (Rogers, 1946). Here is an example of how the client may respond: "I don't really like groups." The leader may reflect: "So you have attended groups before and did not find them very useful to you."

The client-centered approach entails following the client's thoughts for a while and conveying, through empathic, reflective listening, that the listener understands the client's perspective and feelings. The experience of having a good listener, one who is interested in the client's perspective and who conveys respect and understanding, can help build valuable rapport. "If the client feels that he is actually communicating his present attitudes, superficial, confused, or conflicted as they may be, and that his communication is understood rather than evaluated in any way, then he is freed to communicate more deeply" (Rogers, 1946, p. 6). Client-centered reflective listening sets the tone for collaboration between client and leader. In dual diagnosis treatment the client-centered "following" approach is complemented by directive strategies employed by the leader. Interventions are strategically correlated to the client's readiness level, the treatment phase (Sciacca, 1991), and are stage-matched to the client's stage of change (DiClemente, 2007; Prochaska & Norcross, 2001) in an effort to facilitate an exploration that may lead to a process of incremental progress along a continuum to acceptance, problem recognition, action, and recovery.

One objective of the pregroup interview is to establish a purpose for each client's participation. This may include a client who is mandated to treatment by the criminal justice system, a client who is in a mental health program and denies any use of substances, or a client who is in a substance-abuse program and denies any mental health symptoms. A purpose for the client's participation may include an expressed interest in learning or in providing support to other group members. The client may not acknowledge his symptoms of co-occurring disorders. Clients' perceptions of their situations are acceptable to the group leader. However, a group leader should not readily accept clients into groups who have not identified any purpose or reason for attending. These clients may proceed to behave accordingly, by tuning out or by reiterating their disdain, during group sessions. The client is given every opportunity to identify topics of interest other than mental health and substance disorders that may be more relevant, more useful, or more comfortable to discuss. An open question may also yield some ideas: "What would you like to get out of participating in

group discussion?" In addition to the client's interests, the leader also facilitates an exploration of possible contributions the client may make to the group and its members. Mandated clients are making a choice to attend treatment versus other alternatives, and the leader wants to acknowledge that choice.

THE FIRST ELEMENT OF THE INTERVIEW—SUPPORT

Clients are asked whether or not they can be supportive of others who may be working on change, even though they do not perceive themselves as having problems. (During actual groups, leaders and group members define the concept of support through interactions and discussions.) This is followed by an overview of other group members' readiness. For example, the leader may say: "In our group, we have people like you, who do not believe that they have any problems, (symptoms, etc.); we have some people who are exploring this possibility; we have others who are trying to change; and we have some who have successfully changed." This sets the tone for the various readiness levels clients will encounter in groups; the leader may have to repeat this premise in group when the topic arises.

If MICAA and CAMI clients attend group together, the leader informs clients of this and asks clients of each profile if they have any thoughts or concerns about this. For example, a CAMI client might be asked, "In our group, we have participants who have a serious mental illness, and some who take medication. Do you have any thoughts about that?" The reverse would be asked of the MICAA client: "In our group we have participants who have serious substance disorders who do not have a serious mental illness. Do you have any thoughts about that?" The leader would use language that is pertinent and comfortable to describe each profile to each client. The point is that clients should not simply find themselves among others who have symptoms that they do not identify with or feel uncertain as to why they are in group with others whom they experience as different. This works both ways, where MICAA clients may not identify with CAMI clients. This area of discussion reaches more broadly than the group alone. Here, the leader is opening dialogue and setting the tone for important changes that are necessary when we include dual diagnosis programming in general. The initiation for every client is that we are all supportive of one another, regardless of the differences we may have. Clients are reminded: This is a *support* group. We are all here to help one another, regardless of our differences. This may not exclude the need to address clients' perceptions of their differences from one another or their concerns. In general, disrespectful behavior originates from stigma and judgmental attitudes. This is the antithesis of the content and process of the dual diagnosis group model and the approach

of the leader. As participants learn the real properties of each of these disorders and they experience the respect of the leader, they grow to be empathic toward one another.

THE SECOND ELEMENT OF THE INTERVIEW—EDUCATION

The leader discusses the educational component. Here the leader fans out the educational topics beyond mental health and substance disorders. Dual diagnosis groups include a broad range of topics. The leader might discuss the inclusion of a number of topic areas: coping with stress; understanding relationships; mental health issues; how one goes about attaining goals; various substances and their effects; and understanding how people change. The client may express interest in learning more about a particular substance, giving the reason that a roommate is a user, or a parent or child has been addicted. This is acceptable as an identified interest in learning and a purpose for attending.

THE THIRD ELEMENT OF THE INTERVIEW—OUTSIDE SPEAKERS

The leader asks about the client's interest in the inclusion of outside speakers who address various topics in groups. The leader addresses any forms of consent that may be necessary to include outsiders.

THE FOURTH ELEMENT OF THE INTERVIEW—RECEPTIVITY TO LEARNING

The leader discusses the client's receptivity to learning, even with the awareness that she may not believe there is a problem or a reason for change. "Even though you do not think you have problems in any of these areas would you be open to learning from the materials presented and from others who attend the group?"

THE INTERVIEW IS SUMMARIZED

The leader summarizes the interview by highlighting the client's reasons for attendance. These reasons may include: willingness to help others or other contributions the client may make, interest in learning about a specific topic, or other previously identified reasons for group attendance.

MEASURING CLIENTS' READINESS TO CHANGE

In 1992, with the advent of the publication of *Motivational Interviewing* (Miller & Rollnick, 1991) the correlations across a number of the motivational interviewing concepts and practices and dual diagnosis best practices were surprisingly apparent (Sciacca, 1997b). Among them was the description and utilization of the stages of change (Prochaska & DiClemente, 1984). The stages of change provided a second way to conceptualize clients' readiness and motivation and an incremental process of change and became included in the dual diagnosis best practice model.

At the end of this interview, it is important when possible, to measure the client's readiness to address specific symptoms or to embark on changing particular behaviors. In some cases this may require more strategic discussion, as outlined in the preadmission interview (Sciacca, 1987b). Readiness to address mental health and substance-abuse symptoms are measured with the Sciacca Readiness Scale (Sciacca, 1990–2008) (see Tables 9.1 and 9.2). Later, as treatment goals are formulated, the stages of change (Prochaska & DiClemente, 1984) are included and applied.

Clients are frequently at different levels of readiness to address different symptoms or behaviors. For example, a client may think alcohol is problematic but find marijuana useful. Readiness is defined on a 1-to-5 scale (Sciacca, 1990–2008, with 1 as "least ready" and 5 as "participating in an action plan." In this example, we have a 1 for marijuana and a 4 for alcohol on the Substance Abuse Readiness Scale, Table 9.1. This would correspond to the stages of change with the precontemplation stage and the preparation stage, respectively.

Stating a willingness or desire to change does not yield a measure of action, but it may be indicative of the preparation stage. At the preparation stage, a collaborative change plan would be developed. When the client follows through on various elements of the plan, he is in the action stage. It is important to measure readiness early in the process and to employ interventions that facilitate change processes that are stage-matched.

Goal planning optimally includes a goal stated in behavior change terms. Here is an example for substance abuse: client will abstain from alcohol. Here is an example for mental health: client will spend less time in isolation and participate in more activities. Each goal includes the client's stage of change and readiness scale measure (Sciacca, 1990–2008) followed by stage-matched facilitations. One wants to avoid mismatching stages and processes. "Action oriented therapies may be quite effective with individuals who are in the preparation or action stages. These same programs may be ineffective or detrimental, however, with individuals in pre-contemplation or contemplation stages"

(Prochaska & Norcross 2001, p. 447). In the transtheoretical stage of change model, behavior change is conceptualized as unfolding over time and involves a progression through six stages. Both the stages of change and the Sciacca Readiness Scale are nonlinear, and clients may return to earlier stages or readiness levels.

APPLYING THE STAGES OF CHANGE

The perspective that has become common among addictive behavior researchers and clinicians subdivides the process of change into specific stages that attempt to capture critical tasks that need to be accomplished to modify these behaviors (DiClemente, 2007). Five stages of change are formally recognized in the stages of change literature, and the tasks for advancing through each of them are articulated (DiClemente, 2007; Prochaska, DiClemente, & Norcross, 1992).

- *Precontemplation* is the stage where the client has no intention to change behavior in the foreseeable future. Clients in this stage are unaware or underaware of their problems. Tasks: creation of concern and interest in change. Example: Facilitate awareness of the need for change and concern about the current pattern of behavior; envision the possibility of change. Increase client's awareness of risks and consequences of current behavior.
- *Contemplation* is the stage in which clients are aware that a problem exits, may be seriously thinking about overrcoming it, but have not yet made a commitment to take action. *Ambivalent* is a term frequently used to define the client's experience in this stage. The client may identify both positives and negatives, but there is no intention to change at this time. Clients can remain stuck in either the precontemplation or the contemplation stage. Tasks: decisional considerations and decision-making. Example: Facilitate analysis of the pros and cons and the costs and benefits of change, decision-making. Aim to tip decision balance in favor of change. Elicit reasons for change, risks of not changing, and help strengthen client's confidence and self-efficacy.
- *Preparation* is a stage where the clients are intending to take action. The negatives outweigh the positives, they have reached the decision to make a change. They may report some small changes, such as reduction in the behavior, but they have not yet reached the criteria for effective action such as abstinence. The client lacks a plan for change. Tasks: increase commitment and create a collaborative change plan. Assist client to find a change strategy that is acceptable, accessible and effective. Administer a functional analysis. Facilitate confidence building.
- *Action* is the stage that involves the most overt behavioral changes and requires commitment of time and energy. The client is participating in a plan

of action. This may include actions that the client has identified, changing a social network ,or that the provider has suggested, additional treatment, self-help and so forth. Tasks: initial modification of the behavior. Help facilitate the implementation of strategies for change, revising the plan as needed, sustaining commitment in the face of difficulties. Affirm client's commitment for change. Help client identify steps and skills for change. Help client identify additional resources that may be helpful. Include coping strategies for high-risk situations. Note: Preparation for maintenance entails assessment of triggers, as well as in preparation for action. A functional analysis may be administered. Alternative responses in high-risk situations and coping skills are recommended in both the action stage and the maintenance stage.

- *Maintenance* is the stage in which clients work to prevent relapse. The behavior or symptom is now in remission. Tasks: sustaining the new behavior and creating a stable new pattern. Example: assist client to sustain change over time and across situations. Accentuate positive benefits since changing behavior. Develop strategies to prevent relapse. Provide referrals and resources. Develop an overall plan of maintenance services and supports.

  The mechanisms needed to accomplish these tasks are called processes of change.

Two additional considerations are postulated.

- *Relapse*—Slipping back to previous behavior and re-entering the cycle of change.
  - Assure client that relapses are normal and can be overcome.
  - Help reframe slips into learning experiences, revise the maintenance plan and help client to renew determination and confidence in order to resume change efforts.
- *Termination*—Leaving the cycle of change.
  - Low temptation, high self-efficacy, new behavior is normative, multiple changes to sustain change.

The vast majority of addicted people are not in the action stage. Aggregating across studies and populations (Abrams, Follick, & Biener, 1988; Gottleib, Galavotti, McCuan, & McAllister, 1990), 10–15 percent of smokers are prepared for action, approximately 30–40 percent are in the contemplation stage, and 50–60 percent are in the precontemplation stage. If these data hold for other populations, then action-oriented programs may underserve, misserve or not serve the majority of their target population (Prochaska et al., 1992).

When addressing co-occurring disorders, it is likely that a client in substance-abuse treatment is in the precontemplation or the contemplation stage to address his mental health symptoms. The reverse is true for clients in mental health programs. Many of these clients do not generally see substance abuse

as a problem. In effect, dual diagnosis treatment requires that interventions are matched with the earliest stages in order to effectuate the possibility for behavior change.

This process of goal planning, stages of change, readiness measures, and incremental change includes continual updates and outcomes that are tracked on the client behavior change update form (Sciacca, 2000). If the client agrees to attend the group, she will participate in the following group process.

PHASES OF DUAL DIAGNOSIS GROUP TREATMENT

Three phases of group treatment have been identified (Sciacca, 1991; Sciacca, 1990–2008); each phase has goals and objectives.

Phase one: This phase includes exposure to information as the initial focus; there is no need for clients to self-disclose if they are not ready to do so. Clients are free to express their opinions about the topics presented through literature, videos, and speaker's ideas. The objectives include:

1. Developing a trusting atmosphere that is nonconfrontational and respectful of one another, including other members' symptoms and opinions;
2. Providing clients with information that provides the real properties of substance disorders, mental illness, and interaction effects;
3. Dispelling judgments, morality, and stigma;
4. Exposing clients to information regarding the risks and consequences of ingesting various substances, and interactions with mental health symptoms and medication; and
5. Addressing and discussing related topics such as relationships, social networks, stressors, family involvement, goals, aspirations, values, and processes of behavior change.

If clients gain a cognitive and experiential understanding of their situations through this process, it will eventually lead to acceptance of symptoms. This process is intended to facilitate a shift, from the client's exclusive or primary focus on the positive experiences with substance use, or the lack of awareness of any impact at all of substance disorders or mental illness (precontemplation stage), to the identification of positive *and* negative experiences resulting from substance use, including interaction effects, and recognition of risks and consequences of not addressing mental health symptoms (contemplation stage.)

Phase two: Client self-disclosure, and an exploration of positives and negatives, may eventually lead to problem recognition. When trust has developed—and shame, self-blame, and guilt are alleviated—clients begin to self-disclose. This may be minimal at first; the objective is to continue to explore and expand on this recognition.

In this phase, more attention is focused on each individual, and the format of the group may change. For example, leaders will begin groups by checking in with each member and discussing their pursuits and current issues. This is followed by a group theme, explored by everyone. Topics may continue to evolve around adverse effects of substance abuse, unchecked mental health symptoms, and interaction effects. Leaders will facilitate an exploration of each client's experiences in these areas. Strategies such as the pros and cons and the decision balance can be utilized among other themes that generate discussion around decision-making. Teaching the stages of change and facilitating group members' identification of their stages for a particular goal facilitates the recognition of change as a process, normalizes change—including ambivalence or feeling stuck—and further reduces shame, blame, and negativity.

Themes that relate to clients' values and strengths, including previous successes, afford discussion around confidence issues. The group ends with a summary, where the leader may affirm each member, review what occurred in the session, and provide an opportunity for each member's input and closure.

In phase two, group leaders who are also clinicians administer a co-occurring clinical assessment in individual sessions (Sciacca, 1990–2008); clients' responses inform a refined treatment plan. The objectives in phase two include the following:

1. To facilitate each client's identification of risks and consequences while the leader employs a nonjudgmental, client-centered, empathic approach;
2. To facilitate bringing risks and consequences into clear focus for each client;
3. To elicit the recognition and discussion of additional risks, consequences, and benefits of change;
4. To facilitate the client's resolution of ambivalence and to foster problem recognition (pros and cons, decision balance);
5. To assist clients in attaining the necessary confidence and hope that change is possible; and
6. To help clients learn about and understand how people change.

This open process empowers each client to explore and reach decisions about the effects of substance abuse on his mental health and well-being, and the effects of mental health symptoms on substance abuse symptoms. In effect, this understanding may help the client move toward behavior change.

Phase three: The client identifies a problem and begins planning for and executing behavior change (preparation and action stages). Here, the client may address only one of a number of symptoms, a particular substance used, and so forth. This is acceptable to the leader and to the group process.

The leader will listen for and reinforce and further develop discussions about change. In particular, commitment talk (e.g., "I will do this") leads to planning for change and correlates to outcome (Amrhein, Miller, Yahne, Palmer, & Fulcher, 2003).

A change plan is collaborative and will include the client, the leader, and possibly the group members. Moving into the action stage will include confidence building, and actions will be planned and prioritized according to their potential for success. The leader may check in and follow the client's behavior change pursuits during initial group go around and will continue to provide direction and support.

The change plan may include services outside the primary program, such as self-help programs, additional therapy, detoxification, or medication. The overall plan is followed by the group leader, who helps the client make necessary adjustments, including revising elements of the change plan that do not work for the client.

As clients experience success in one area of change, they frequently identify additional problematic behaviors and embark on other changes. Strategies and interventions now include appropriate increments of change that assist clients with symptom management, symptom remission, stability, acquiring coping skills, and relapse prevention. Facilitating bringing all symptoms into remission is the overall goal.

SUMMARY OF DUAL DIAGNOSIS GROUP PROCESS

1. Clients are critics, not students of the information presented or discussed.
2. Videos, fact sheets, and speakers are examples of educational materials used. One topic, alcohol, may be presented with different forms of media. Unless this is a time-limited group, this is not one-time learning.
3. Education and information are not purely didactic. There is a balance between information (cognition) and personal experience regarding all topics. Clients give their opinions based on their own experiences or the experiences of others whom they know.
4. Clients may validate or invalidate the information. There is no right or wrong answer.
5. Providers do not present themselves as experts. They employ an exploratory approach, and are open to learning with *and from* the clients and the information discussed.
6. Clients are not pressured to self-disclose. A client who recognizes adverse effects, and who decides to work on substance abuse/mental health symptoms, does so as a willing participant (intentional change), rather than one who might be bribed or coerced.

THE BENEFITS OF INTEGRATED DUAL DIAGNOSIS SERVICES, PHASES OF TREATMENT, STAGES OF CHANGE, AND CLIENT-CENTERED INTERVENTIONS

The following are some of the benefits of integrating dual diagnosis treatment, the stages of change, and client-centered interventions into agencies and services for people who have co-occurring disorders:

- Empathic, accepting, client-centered interventions improve the engagement potential for many dually diagnosed clients who have been disengaged from treatment because of rejection or lack of competent care.
- Client-centered interventions facilitate gaining knowledge and understanding of the client's true self including his understanding of symptoms and behaviors, readiness to engage in behavior change, goals, aspirations, and disappointments and broaden the interventions beyond medication and symptom-focused approaches.
- Group treatment has evolved as an effective model to treat dual disorders; the inclusion of the stages of change and related strategies and materials in dual diagnosis groups or in groups that exclusively adhere to stages of change topics is highly effective, adding important learning experiences within the psychoeducational component of treatment and enhancing the recovery process of dually diagnosed clients.
- Providers who treat co-occurring disorders learn a comprehensive set of strategies and skills that enhance their professional competence and have the potential to greatly improve the outcome of their clients.
- Clients who receive simultaneous treatment for mental health and substance disorders in one setting are able to participate in the program or system they are familiar and most comfortable with. They have the opportunity to address all of their symptoms and the resulting interaction effects and thereby learn whether or not co-occurring disorders are an obstacle to their recovery from substance disorders, mental illness, or both.

CLIENT-CENTERED INTERVENTIONS—ESSENTIAL AND EFFECTIVE

Employing a client-centered approach (Rogers, 1946) is essential and extremely beneficial to both the client and practitioner. From the onset, clients are listened to, understood, and communicated with beyond their clinical symptoms, to the real self. Rapport building may ensue and alliances may be formed. This is in direct contrast to previous experiences of clients being unwanted or being treated as misfits or being confronted about having symptoms that they may not agree with. The client-centered, reflective listening

approach begins with an engaging versus disengaging process, one in which the client feels understood and recognizes that she is an important part of the process.

The practitioner's approach in employing client-centered, dual diagnosis and stages of change concepts is transitioned to one who facilitates explorations leading to the client's movement along a continuum of incremental change and decision-making instead of one whose former approach was to feel pressured to make people change and usher them into action whether or not change was precipitated by external pressure, threat, or bribery. In any of the latter cases, change would likely last only as long as the external pressures did.

Participating with providers who are accepting and respectful, who are empathic and inclusive, and who allow the clients' process of change to unfold is both essential and invaluable to clients who have co-occurring disorders.

The benefits to clients who receive these interventions include genuine alliances and caring; a listener who wants to understand their perspectives and facilitate a respectful exploration of their ideas and feelings as they relate to their present issues and goals; and insight and understanding into the behavior change process. Most important is the recognition of the stages of change as a normal process of change.

Client-centered, reflective listening creates the necessary experience of safety and the opportunity for deepening the exploration and, thereby, the ability to potentially address the real and often underlying issues around behavior change. This can lead to resolving the deeper causes of being stuck. For example, a client may present a problem initially stated as "My wife wants to go back to school. She has enough to do, and I don't think she needs to, so we argue." Reflective listening may lead to the client's revelation of some deeper issues such as these later statements. "She might go back to school and decide she doesn't want anything anymore. She doesn't want any family." Now the client has moved from a cognitive presentation of a problem—disagreement as to whether or not his wife needs more education—to an underlying emotional issue, fear of abandonment.

Movement from cognitive presentations of problems to underlying emotional issues is frequently very rapid (anywhere from within 10 minutes to within one session and so on) even when reflective listening alone is employed.

Other important ways that client-centered, reflective listening is essential to this process includes the many kinds of thought processes a leader or practitioner may relate to during the course of engagement or treatment. For example, a client with transient psychotic delusions may talk about his wife and children being upstairs at the day center while he is attending the dual diagnosis treatment group downstairs. The client doesn't have a wife or children. The

practitioner can reflect the essence of the client's statement without engaging in invalidating the details. For example: "It seems that you have been thinking about your family a lot lately." This response can validate the client's statement. It may also lead to discussion about the client's actual family.

A client who has recently been diagnosed with a late-onset severe persistent mental illness transitions from a high-pressure lifestyle to participating in a day treatment program. He states to the therapist, "I am really enjoying just lying around the day center without anything at all to do." Here a therapist may get concerned that the client is lapsing into lethargy. Reflective listening can provide the following validation: "You seem to be appreciating some relief from the high-pressured lifestyle you have been living." Rather than opposing the use of day center as a way for the client sink into lethargy, this response opens the door to explore many other ways the client may relieve that stress yet still be able to function.

A client tells her therapist that she goes out with a man on weekends (one whom it seems may be engaging in some illegal drug use); they go dancing, and she states that she is really having a good time. Here again a practitioner may feel pulled into the precautions of the situation, but reflective listening can simply validate the underlying need. "You really need to have something fun and exciting going on in your life. It makes a real difference for you." This opens the possibility to explore alternatives for fun and excitement.

There are many examples of reflective listening interventions that convey respect, understanding, validate the client's needs, thoughts, and feelings, and facilitate the client's exploration. The practitioner remains connected to the client throughout providing thoughtful reflections. Reflective listening is never judgmental, and the practitioner always strives to convey to the client that he or she is understood. At the same time the practitioner also employs directive strategies such as pros and cons, decision balances that facilitate explorations and insights that may lead to problem recognition and the decision to change one's behavior.

READINESS TO CHANGE, PHASES OF TREATMENT, AND STAGES OF CHANGE

Designing services around a client's readiness to address symptoms is revolutionary for programs, providers, and clients. Service systems and individual programs that show progress only when a client reaches the action stage, particularly with clients who have co-occurring disorders, are likely to show failing results in the areas of program outcome, provider outcome, and client outcome.

When clients' progress is measured incrementally, programs, providers, and clients show real success and outcome is reversed. Matching interventions to client readiness provides the opportunity for improved outcomes. Providers and clients are working together at the client's level of readiness and stage of change, and the process of change is facilitated. The relationship between client and provider is greatly enhanced.

Rather than a client being viewed in negative terms, such as uncooperative or resistant, the client's stance or behavior is defined by his readiness or stage of change.

If a client is understood to be in the precontemplation stage and the provider is trying to get him to go into action to change behavior, the resulting responses and attitudes the client conveys will be understood by the nature that he is in precontemplation and not that he is uncooperative or opposing the provider. It is the stage of change that defines the client's relationship to the change process.

Where such clients formerly found themselves participating in programs that required participation in action plans and abstinence or symptom management, which inevitably set them up for failure, within the incremental change model the same clients can explore their perceived benefits and lack of concerns and move beyond the negative struggle.

In addition to the action-ready model, many providers have been taught through expediency that symptom activation or remission alone also equals successful outcome or failure. This medical model of symptom management is primarily symptom focused and impersonal. The focus can be placed on medication compliance or whether a client's particular symptom is active or in remission on a particular day. The client-centered approach to medication is not used; instead, clients are expected to comply with given regimens.

Here is another very important juncture where collaborative, client-centered interventions can give voice and participation to the client's pros and cons as to what is good about the medication what is not good about it and learn what the options are from a good listener. A client who participates in the process of medication adjustments is far more likely to reach a medication regimen that is least detrimental and most effective.

In all areas of treatment, the client who is in a relationship with providers who accept her readiness level, employ client-centered collaborative interventions, listen carefully and make a genuine effort to employ strategies to assist the client to move through the process of change, reaps the benefits of feeling connected to another human being who is genuinely concerned. The client's true self is involved; thereby a meaningful, inclusive, and empowering experience is possible.

NEW SKILLS AND APPROACHES, BENEFITS TO PRACTITIONERS

Practitioners who receive education and training to prepare for treating co-occurring disorders expand both their knowledge and their skills as they learn new approaches in assisting people to change. They are prepared to accept clients as they present for services and to employ interventions that are accepting, engaging, pertinent, and effective. Their task has changed from beingresponsible for making people change to being a facilitator of a thorough exploration of each client's situation, thereby affording them the opportunity to recognize problems and to reach the decision to change or not to change. This dramatically reduces the incidence of burnout. Instead, progress is charted realistically and incrementally along each client's process through the stages and phases beginning from denial to recovery and from precontemplation to maintenance.

Learning dual diagnosis treatment, the stages of change, and client-centered interventions results in knowledge, skill, and wisdom for each practitioner who subscribes to it, regardless of her clinical orientation or system of care. The acceptance of clients regardless of their clinical profile is essential.

Discarding negative interpretations of client behavior while promoting awareness of the difficulties and hardships that all symptoms impose on clients evokes empathy and opens the path to alliances and engagement. This allows the process of the examination of areas, such as the pros and cons, to go forward and facilitates an exploration that may result in the decision to change one's behavior.

THE BROADER IMPACT OF INTEGRATED TREATMENT FOR CO-OCCURRING DISORDERS

Programs and systems also benefit from the inclusion of these approaches. A departure from the premise that all clients are in the action stage, and systemic recognition of each client's level of readiness and stage of change (including the fact that the client may be at different stages of readiness to change different behaviors), changes a program from one that is failing to bring clients into action to a program that is charting successful outcome along a number of incremental and more realistic criteria. This benefits both programs and systems in their accountability and outcome.

Where the action-focused model leads to interpretations of client, provider, program, and systems failure, programs that utilize incremental strategies and measurements of change demonstrate success in each of these areas. From this perspective, the effectiveness and popularity of concepts such as readiness to

change and stages of change serve to revolutionize the way we view behavior change and how we document progress within our programs and for our clients, as well as providing strategies and interventions that correlate to client readiness and strategies and that are directive in facilitating movement through the process of change. The impact on all programs and systems that reach this realization is immense. Providers who learn this distinction are convinced that outcome measures that are exclusively focused on the action outcome are unrealistic, result in client opposition rather than movement, and staff burnout rather than gratification.

The field of dual diagnosis has grown tremendously since its beginning in 1984, yet there are far too few services available for far too many people who suffer with co-occurring disorders. Education, training, and integrated program development need to be approached more aggressively and simultaneously with systems change, staff development, and program implementation. The sequential model that frequently entails years of work with small groups of key providers who address systems changes before staff development, program development, and client care can ensue is unnecessary and wasteful (Sciacca, 2007). A simultaneous approach to systems issues, staff development, and integrated services implementation is far more effective and advantageous (Sciacca, 2007). The main objective is to prepare administrators, management, and staff to bring effective services to the clients who need this essential professional help. The integrated dual diagnosis approach discussed in this chapter does exactly that. Existing programs have the ability to design integrated program tracks along with clinical tools and program underpinnings and educate and train staff to provide effective interventions. In effect, each agency or service-delivery system has the responsibility to provide integrated care for the dually diagnosed clients it regularly encounters. Many of the clients are already in attendance; it is a matter of engaging these clients in meaningful discussions about their perceptions of their own situations and to provide information that may be valuable to assist them to review their ideas and experiences in the light of some new information.

It is our responsibility as providers in behavioral health care, social services, and primary health care to accelerate this process and to bring effective services to those who so sorely need them.

Dual Diagnosis Web site: http://pobox.com/~dualdiagnosis

REFERENCES

Abrams, D. B., Follick, M. J., Biener, L. (1988 , November). Individual versus group self-help smoking cessation at the workplace: Initial impact and 12-month out-

comes. In T. Glynn (Chair), *Four National Cancer Institute-funded smoking cessation trials: Interim results and emerging patterns*. Symposium conducted at the annual meeting of the Association for the Advancement of Behavior Therapy, New York.

Amrhein, P. C., Miller, W. R., Yahne, C. E., Palmer, M., & Fulcher, L. (2003). Client commitment language during motivational interviewing predicts drug use outcomes. *Journal of Consulting and Clinical Psychology, 71,* 862–878.

DiClemente, C. (2007). Mechanisms, determinants and processes of change in the modification of drinking behavior. *Alcoholism: Clinical and Experimental Research, 31*(S3), 13S–20S.

Drake, R. E., McHugo, G. L., Xie, H., Packard, J., & Helmsletter, B. (2006). Ten-year recovery outcomes for clients with severe mental illness. *Schizophrenia Bulletin, 32,* 464–473.

GA seeks statewide implementation of dual-diagnosis strategy. (2003). *Alcoholism and Drug Abuse Weekly, 15*(17), 1, 6, 7.

Gottlieb, N. H., Galavotti, C., McCuan, R. S., & McAllister, A. L. (1990). Specification of a social cognitive model predicting smoking cessation in a Mexican-American population: A prospective study. *Cognitive Therapy and Research, 14,* 529–542.

Kilpatrick, D. G., Edwards, C. N., & Seymour, A. E. (1992). Rape in America: A report to the nation. Arlington, VA: National Crime Victims Center.

Mayfield, D., McLeod, G., & Hall, p. (1974). The CAGE questionnaire: Validation of a new alcoholism instrument. *American Journal of Psychiatry, 131,* 1121–1123.

Miller, W. R., & Rollnick, S. (1991). Motivational interviewing: Preparing people to change addictive behavior. New York: The Guilford Press.

Minkoff, K., & Klein, C. A., (2004). Developing welcoming systems for individuals with co-occurring disorders: The role of comprehensive continuous integrated system of care model. *Journal of Dual Diagnosis, 1*(1), 65–89.

Morrissey, J. P., Ellis, A. R., Gatz, M., Amaro, H., Reed, B.G., Savage, A., et al. (2005). Outcomes for women with co-occurring disorders and trauma: Program and person-level effects. *Journal of Substance Abuse Treatment, 28*(2), 121–134.

Morrissey, J. P., Jackson, E. W., Ellis, A. R., Amaro, H., Brown, V. B., Najavits, L. M. (2005). Twelve-Month outcomes of trauma-informed interventions for women with co-occurring disorders. *Psychiatric Services, 56*(10), 1213–1222.

Mueser, K. T., Bellack, A. S., & Blanchard, J. J. (1992). Comorbidity of schizophrenia and substance abuse: Implications for treatment. *Journal of Consulting and Clinical Psychology, 60,* 845–856.

Mueser, K. T., Drake, R. E., Turner, W. C., & McGovern, M. P. (2006). Comorbid substance use disorders and psychiatric disorders. In W. R. Miller & K. M. Carroll (Eds.). *Rethinking substance abuse: What the science shows, and what we should do about it* (pp. 115–133). New York: Guilford Press.

Nace, E. P. (1990). Substance abuse and personality disorder. *Journal of Chemical Dependency Treatment, 3*(2), 183–198.

National Alliance for the Mentally Ill [NAMI]. (2003). Dual diagnosis and integrated treatment of mental illness and substance abuse disorder. Arlington, VA: Author.

Prochaska, J. O., & DiClemente, C. C. (1984). *Transtheoretical approach: Crossing traditional boundaries of therapy.* Homewood, IL: Dorsey Press.

Prochaska, J. O., DiClemente, C. C., & Norcross, J. C. (1992). In search of how people change—applications to addictive behaviors. *American Psychologist, 47*(9), 1102–1104.

Prochaska, J. O., & Norcross J. C. (2001). Stages of change. *Psychotherapy, 38*(4), 443–448.

Ridgely, M. S., Osher, F. C., & Talbott, J. (1987). *Chronic mentally ill young adults with substance abuse problems: Treatment and training issues*. Baltimore: University of Maryland, School of Medicine, Mental Health Policy Studies.

Rogers, C. R. (1946). Significant aspects of client-centered therapy. *American Psychologist, 1,* 415–422.

Sciacca, K. (1984). *Client disposition form.* Unpublished.

Sciacca, K. (1987a). New initiatives in the treatment of the chronic patient with alcohol substance use problems. TIE-Lines, The Information Exchange, *4*(3), 5–6.

Sciacca, K. (1987b). Preadmission Interview. Unpublished.

Sciacca, K. (1990–2008). MIDAA service manual: A step by step guide to program implementation and comprehensive services for dual/multiple disorders. New York: Sciacca Comprehensive Service Development for MIDAA.

Sciacca, K. (1991). An integrated treatment approach for severely mentally ill individuals with substance disorders. In K. Minkoff & R. Drake (Eds.), *New directions for mental health services, dual diagnosis of major mental illness and substance disorders.* San Francisco, CA: Jossey-Bass.

Sciacca, K. (1996). On co-occurring addictive and mental disorders: A brief history of the origins of dual diagnosis treatment and program development. *American Journal of Orthopsychiatry, 66*(3), 474–475.

Sciacca, K. (1997a). Cross-training yields continuity in dual-diagnosis programs. *Mental Health Weekly, 7*(21), 6.

Sciacca, K. (1997b). Removing barriers: Dual diagnosis treatment and motivational interviewing. *Professional Counselor, 12*(1), 41–46.

Sciacca, K. (1998a). Curriculum for MICAA and CAMI direct care providers: Mental illness, drug addiction and alcoholism MIDAA(R): Training, cross-training and program development. SAMHSA-CMHS Managed Care Initiative Co-Occurring Disorder Report: Co-occurring psychiatric and substance disorders in managed care systems: standards of care, practice guidelines, workforce competencies and training curricula. Rockville, MD: SAMHSA.

Sciacca, K. (1998b). Tennessee initiates state-wide dual diagnosis program development. *Alcoholism and Drug Abuse Weekly, 10*(7), 5.

Sciacca, K. (1999). D.C. reports progress with dual diagnosis integration initiative. *Alcoholism and Drug Abuse Weekly, 11*(41), 5.

Sciacca, K. (2000). Client behavior change up date form. Unpublished.

Sciacca, K. (2007). Dual diagnosis treatment and motivational interviewing for co-occurring disorders. *National Council Magazine, 2*, 22–23.

Sciacca, K., & Dobbins, K. R. (2001). Kentucky dual diagnosis residence yields remarkable outcome. *Mental Health Weekly, 11*(7), 5.

Sciacca, K., & Thompson, C. M. (1996). Program development and integrated treatment across systems for dual diagnosis: Mental illness, drug addiction and alcoholism, MIDAA. *The Journal of Mental Health Administration. 23*(3), 288–297.

Substance Abuse and Mental Health Services Administration. (2002). SAMHSA report to Congress on the prevention and treatment of co-occurring substance disorders and mental disorders. Rockville, MD: Author.

Substance Abuse and Mental Health Services Administration—Center for Mental Health Services. (1998). Managed care initiative co-occurring disorder panel report: Co-occurring psychiatric and substance disorders in managed care systems: Standards of care, practice guidelines, workforce competencies and training curricula. K. Minkoff, Chair, CMHS Panel on co-occurring disorders, Center for mental health policy and services research; Philadelphia. Rockville, MD: SAMHSA.

Sundram, C. J., Platt, I. L., & Cashen, J. A. (1986). The multiple dilemmas of the nultiply disabled: An approach to improving services for the mentally ill chemical abuser. Albany, NY: New York State Commission on Quality of Care for the Mentally Disabled.

Tessler, R. C., & Dennis, D. L. (1989). A synthesis of NIMH-funded research concerning persons who are homeless and mentally ill. Rockville, MD: National Institute of Mental Health, Division of Education and Service System Liaison.

Chapter **10**

Cranial Electrotherapy Stimulation in the Treatment of Addictions

Ray B. Smith, PhD

Cranial electrotherapy stimulation (CES) first caught this author's attention when it was found that it reversed symptoms of the drug abstinence syndrome better than any then current or present day medication and that it reversed back to normal what had until that time (1972) been thought of as permanent brain damage in addicted persons.

CES is the U.S. Food and Drug Administration's (FDA) name for what in most countries of the world is called electrosleep. It refers to a small electrical stimulation pulsed across the head for up to one hour a day for three weeks or less for FDA-accepted treatment claims of depression, anxiety, and insomnia. All these are symptoms of persons recovering from addictions, the drug abstinence syndrome.

While they vary, the stimulus parameters most encountered in the research literature are 100 pulses per second, biphasic, at 1.0 mAmp on a 20 percent duty cycle. The energy source is usually one nine volt battery. The electrode placement also varies, but most often electrodes are placed just behind the ears on the mastoid process or just below.

Because the vast majority of studies in the CES literature—more than 120 to date—have been either single or double blind, the treatment stimulation has been given below sensation threshold and the patient has felt no electrical stimulation during the treatment.

This author began early studies of CES in addicted persons when he was assigned by a U.S. National Institute of Mental Health (NIMH) grant to work as a research scientist in the District of Columbia's (Washington, DC) 600-bed

Rehabilitation Center for Alcoholics. Some two years later, Ray Gilmer, president of NeuroSystems, Inc., in Garland, Texas, arrived with a large, 15-pound device that promised to alleviate stress. At that time, the FDA did not have control over medical devices, so the safety and effectiveness of CES were not known. Nonetheless, there had been no problems found with its use in Europe and the then-Eastern Bloc nations since its development in 1953, and because we were to use it at subsensation stimulus thresholds, the hospital director determined that we should research it.

Perhaps fortunately, this author, who thought it was no less than the return of the Argon box, was not asked, but told to do the research. Subsequently 76 patients who had completed detoxification and had entered the rehabilitation program, many of whom were on daily doses of Lithium, Librium, Sinequan or other psychoactive medication, were entered into a research program in which one-half of them would receive stimulation while one-half received sham stimulation.

Since double-blinding devices had not become available at that time, the research was conducted in a single-blind manner, with not only the patient but the psychometrist and the statistician also blind. Only the nurse who gave the treatment knew who was getting treatment, and she was instructed to talk to the patient as little as possible. The instructions were simple. She would attach a moistened felt electrode pad on each side of the forehead just above the eyes and an electrode on each mastoid process with Velcro straps. After telling the patient to raise his hand when he felt something, she would slowly turn the current knob up until the patient raised his hand, then turn it back down until the patient lowered his hand, signifying that he no longer felt anything. At that point she would check her sheet of paper to see if the patient was to get actual treatment, in which case she would leave the current set at that level and go to the next patient. If the patient was to receive sham treatment, she would turn the stimulation off, down to but not including the final click of the on/off knob. The control knobs remained out of the line of vision of the patients.

This being a new treatment in the center, we didn't know what to measure but decided to use what the pharmaceutical companies were using to test their new psychoactive drugs, the Profile of Mood States (POMS), which presented the patient with 44 words or short phrases describing emotions, such as unhappy, confused, energetic, panicky, and so forth. The patient could mark the box labeled 0 for not at all, up to 4 for extremely, depending on his level of agreement with the word or phrase. From these items, five negative mood factors were statistically derived: tension/anxiety, depression/dejection, anger/hostility, fatigue/inertia, and confusion/bewilderment. A positive factor, describing a positive energy state but considered nonclinical, was also derived.

The score for the positive energy state was subtracted from the sum of the five negative factors to produce a total mood disturbance score.

Since we did not know how long the treatment would take to be effective, if at all, we had the patients complete the POMS every day following treatment. The center had different treatment and rehabilitation programs every hour, and it took the patients some 10 minutes to get to the next activity, so we decided to treat the patients with CES for 45 minutes each day, which would give the nurse 5 minutes to get the electrodes on and off the patients and still allow them to get to the next class. Because staff were not that readily available on weekends, the treatment was given Monday through Friday each week, for three weeks.

After the three weeks were over we found that the anxiety level in patients who received actual treatment was reduced 46 percent, while the depression was reduced 70 percent. And while we had not planned to study insomnia as such, it was later found that the prn Seconal medication requested each night by the patients who received actual treatment was reduced 64 percent during that three-week period, while remaining the same by the controls. The patients who were on sham treatment had actually gotten somewhat worse on all the dependent measures during the three-week period. Those results were significantly better that those obtained by even the most widely prescribed psychoactive medications today (Kirsch & Sapirstein, 1998).

The confusion/bewilderment (C/B) score was of interest to the present author who, as a physiological psychologist had an interest in the brain damage of addicted persons. He was intent on trying to find what exactly caused it and how to prevent it, because it was well known at that time that there was no cure. He was therefore quite surprised to discover that the C/B score, which correlated strongly with brain dysfunction scores on other tests, had also improved by 54 percent. The results were so surprising to our research team that we selected another 76 patients and repeated the study, which replicated almost exactly the results of the first, so we published the results (Smith & O'Neill, 1975).

We were curious how long the improvement would last, so we routinely had all patients who had been in the two studies complete the POMS every Friday morning following the studies. This went on for about six weeks when patient attrition—they were in a 90-day treatment program—forced us to discontinue the evaluation. What was found, however, was that patients who had received actual CES treatment continued to improve over the six weeks following treatment, while the scores of the sham-treated patients continued to deteriorate.

In the process we had yet another serendipitous finding: Patients who had received actual CES treatment stayed in treatment until their time was up,

while patients who had received sham treatment had a 50 percent greater likelihood of leaving the program early against medical advice (AMA).

At the center, psychologists often administered the Wechsler Adult Intelligence Scale (WAIS) to selected patients, and all patients entering the center were given the Army Beta intelligence test, a nonverbal test that could be given in a group setting.

Earlier the author had found a strong correlation between the C/B score on the POMS and the three so-called organic brain syndrome (OBS) scales on the WAIS. He also found the C/B score strongly correlated with the formboard test on the Army Beta, the one subtest among five that was significantly reduced in most alcoholic patients who arrived for treatment at our center.

Those subtest score reductions were thought to reflect the permanent brain damage that our patients were then thought to have. It seemed unlikely that if the C/B score on the POMS was so highly correlated with the OBS scale(s) on psychological tests that the C/B score should have been improving because of CES treatment. In subsequent research with CES it was decided to use those other mental tests also.

In 1977 we published a study in which the identical single-blind treatment protocol was used, but with the Army Beta test as the measure of choice. We found that the formboard subtest of the Army Beta test of those patients receiving actual CES treatment, but not the sham-treated controls, came back within normal limits when compared with the other four non-OBS scales. The improvement on that subscale was 86 percent (Smith & Day, 1977). Later, we were to test more than 300 patients in confirming this result (Smith, 1982). Later still, Schmidt, Capo, Frazier, and Boren (1984), in a double-blind study, repeated those findings, this time using the full WAIS as the dependent measure. For the first time, Schmidt and his group were studying patients with one or more addictions other than just alcohol alone. They found results in other addictions similar to those we had found in alcoholic patients.

Later still, in another double-blind study, Schmidt and his group replicated our finding that patients receiving actual CES treatment, compared with sham-treated patients, had reduced AMA attrition rates by 50 percent or more, and also reduced recidivism rates (patients returning for additional treatment) by 50 percent or more, while very effectively reducing their anxiety (Schmidt, Capo, & Boyd, 1986).

Other researchers would publish results of their treatment of alcoholic persons with CES, while others would research cocaine patients, marijuana users, smokers, and methadone users, among others. All found significant reductions in the stress involved in the drug abstinence syndrome, and those who checked for it also found reductions in AMA attrition rates and recidivism rates.

It is perhaps of interest that while many studies were of the relatively more expensive double-blind design, the results from those were almost exactly matched by the less expensive and time-consuming single-blind studies. Also, in the few studies that controlled for placebo effect, no such effect has ever been found from CES treatment in published studies to date.

MECHANISM OF ACTION

Earlier peer reviewers had difficulty with CES studies, because it was so new that few had heard about it. Some reviewed CES studies under the impression that they were studies using electroshock treatment. Others assumed that the small electrical current provided in CES stimulation could not effectively enter the brain, while yet others decided that even if by chance it could find its way past the cranium, the amount of stimulus was too small to have an effect on neurons in the brain.

Researchers over the years have found that the electric current from CES stimulation goes through every part of the brain. One of these researchers was a doctoral candidate studying the possibility of combining CES and biofeedback simultaneously, measuring the electroencephalograph (EEG) in the process (Shroeder, 1999). A second, a doctoral candidate using low resolution brain electromagnetic tomography (LORETA) imaging, found that CES induced changes in every one of 2,394 gray matter voxels measured (Kennerly, 2006). A study at the University of Wisconsin found that CES stimulated the emptying of presynaptic vesicles in the rabbit brain, then generated an increased number of vesicles in the presynaptic neuron as stimulation continued. These returned to a normal level following cessation of stimulation. These changes were measured by implanted devices placed in numerous areas of the brain, covering the majority of the cortex (Siegesmund, Sances, & Larson, 1967).

To learn the mechanism in which changes had been wrought in depression and anxiety scores, researchers began to measure neurotransmitter levels prior to and following CES treatment. Pozos, Strack, White, and Richardson (1971), at the University of Tennessee Medical School, deliberately Parkinsonized canine subjects by chemically altering their adrenergic/cholinergic balance. He then discovered that either L-dopa or CES could similarly bring the subjects back to a normal, non-Parkinsonian state in a few hours, while subjects put on normal rations of food and water required several days to return to a normal state. CES, he inferred from his results, acts as a rebalancing force in organisms whose normal neurotransmitter system is out of balance.

Gold and his cohorts later researched a similar system with patients withdrawing from methadone use. They theorized that heroin use had interrupted

the dopamine system as those neurons fired into the *locus cerelus*, and in the absence of methadone or heroin, the increased fire into the *locus cerelus* caused the withdrawal symptoms. In a double-blind study they gave either the dopamine blocker, Aldomet (alpha methly dopa), or CES to withdrawing patients. The physicians monitoring the patients' withdrawal could not determine which were on Aldomet and which were on CES until after the study, when the Aldomet patients suffered a rebound depression. They theorized that this was due to a chemical rebound reaction in the Aldomet patients, while CES was bringing the dopamine system back into a normal balance in those patients (Gold, Pottash, Sternback, Barbaban, & Annitto, 1982). Dougherty and Dafny (1989; Dougherty, Dong, Faillace, & Dafny, 1990), at the University of Texas, found similar results when withdrawing rats from morphine. Shealy et al. (1989) found altered serotonin and norepinephinre levels in the brain consequent to CES stimulation, as did Closson (1985), who also measured adrenocorticotropic hormone production.

Over the years, researchers began to organize the myriad studies of CES in meta-analyses. Harvard University researchers completed perhaps the first such study, combining CES studies of several different treatment populations. Bianco (1994) was the first to meta-analyze studies of CES in polysubstance abusers, in which he found a highly significant response to CES in those aggregated studies. Most recently, Smith (2007) has published a meta-analysis of 15 studies of the drug abstinence syndrome involving 535 research subjects, as well as 7 studies of cognitive dysfunction involving 558 polysubstance abuse subjects. He found an average improvement of 60 percent in the abstinence syndrome patients, which involved many substances of abuse, and 61 percent in the studies of cognitive dysfunction.

SAFETY

There was some reluctance among the first patients in our center to be treated with CES, since many of their relatives had earlier been treated with electroshock therapy in psychiatric institutions. From the first, and for several years afterward, we had to begin treatment by letting the patients hold the electrodes in their hands and then gradually turn up the current level themselves. When they felt the initial tingle we explained that this was all they would feel on their head during CES treatment, and if they were to be treated in a research project they would only feel the tingle for a second until the current was turned back down.

After completing research with more than a thousand patients and treating many thousands more, this author had encountered no significant negative side

effects from CES treatment. In our first research project, we found that three percent of the treated patients complained of headache. On further evaluation of the data, we found that a similar three percent of the sham-treated patients also complained of a headache. Looking into this still further, we found that all headaches occurred during those hours when the other patients were out in basketball practice. Nevertheless, some patients may have a slight headache if they turn their CES device up to a high stimulus intensity. Any such headache will go away when they turn the current down.

Earlier we also found that some patients who had been missing REM sleep for whatever reasons had the most vivid, intense dreams they had ever had, following their first few CES treatments. That sometimes alarmed patients who may have had relatives or friends who were schizophrenic or who had other mental problems. Once the clinician assured them that they were just making up for lost REM sleep and that this is normal, they were very pleased and sometimes were saddened when the intense dreams ceased, which they invariably did after a night or two.

The FDA was concerned about the safety of CES early on, so it gave a grant to the National Research Council's Division of Medical Sciences (National Research Council, 1976) to assess the possible safety problems from the use of CES. After researching it in some depth, the council reported back to the FDA that there could be no possibility or harm to a patient from this level of electrical stimulation.

Of more than 100 studies involving more than 4,000 patients that this chapter's author meta-analyzed, not one study reported a significant negative side effect from the treatment. Of course, for the most part the treatment was subsensation threshold. On the other hand, this author has had much clinical experience with teenage cocaine addicts whose habit was to turn their CES units up as high as they would go during treatment, then request new batteries the next day because the feeling the second day was less intense. This is contrasted with the many heroin addicts who did not wish to feel any of the electrical stimulation, so kept theirs turned down very low during treatment. In neither case were negative side effects seen.

We did have a couple of questions when CES first began to be used in addiction treatment. Would it cause seizures in patients known to have withdrawal seizures? Was CES itself addicting?

To avoid any possibility of triggering seizures in our patients, our clinical policy was to not treat patients with CES until they had been discharged from the detoxification unit and entered the main rehabilitation program. After a few years of this, we were told by the staff at Charity Hospital in Louisiana that they had always used CES specifically during detoxification in order to prevent

seizures and that this worked quite successfully. CES has also been used in other patients, such as closed head injured patients who were known seizure patients, without triggering seizures of any kind (Smith, Tiberi, & Marshall, 1994).

To discover whether or not CES might itself be habit forming or even addicting, 111 patients in a polysubstance abuse clinic in Santa Barbara, California, were given personalized units to take home with them upon discharge from the hospital. After one year they were called back in to the clinic and asked about their use of their CES device over the course of the year. Most of them reported using it only a few times for a few days when under unusual stress. None had used it during the past 30 days. They were obviously not addicted to CES, but they didn't want to give the units back either.

HOW TO USE CES

CES devices began in the United States as very large units that were considered for hospital or clinic use only. In the early 1980s they were miniaturized to about the size of a pack of cigarettes that could be worn in a shirt pocket. Much of the impetus for this was so that patients could have their own unit at home following treatment. We decided on this after a patient got on a Greyhound bus one day (our center was 40 miles from town) and arrived at our center, asking for the CES nurse. She was surprised to see him because he was a former patient who had been discharged several months before. He asked her if he could be put on the CES device for an hour. She complied with his request, but after 30 minutes he got up, removed the electrodes and said, "Thanks, nurse. That did it."

"Did what?" the nurse asked.

"I was on a dry drunk and that stopped it," he told her.

It was in this manner that we discovered that CES could successfully treat any recurring urge to use an addicting substance. Subsequently, we put a unit in each of our halfway houses so the men could get to them when they were experiencing the urge to use again. Finally, when the units were downsized even more, patients were often given their own device when leaving treatment, because by then we had also discovered that treatment with CES invariably reduced recidivism for additional treatment.

CES devices are prescription devices that can be ordered for a patient by any licensed health service provider, including most addiction treatment personnel such as physicians, nurses, psychotherapists, and even licensed massage therapists if they work with addicted persons. The CES device is usually used for 30

minutes to one hour on a daily basis for 10 to 15 days or until the symptoms being treated subside. Patients who are to leave with a CES device, or receive one on an outpatient basis, should be taught to use it daily for 30 minutes to one hour for two to three weeks until they feel it has had its maximum effect, then to use it again any time they feel they need it for stress reduction or to help with their sleep. They should also be told to wear it anytime they feel a withdrawal symptom arising, or a dry drunk, as some describe it, coming on.

ADDITIONAL READING

Kirsch self-publishes a very helpful book, the 2nd edition of which includes abstracts of 126 human and 29 animal studies. They are grouped under the condition studied, dependent measure used, and CES device used in the study. The book can best be found on the web (Kirsch, 2004). Earlier, Smith (1985) published possibly the largest review to date of all the CES studies in the U.S. literature up to that time, which included some studies that were not in his 2007 meta-analysis, with an explanation of why they were not included.

Kirsch's book tends to be updated as more studies come on stream, so it is usually more up-to-date than any other CES reference source.

SUMMARY

CES is a very potent addiction treatment device that can reduce or eliminate many of the symptoms of the drug abstinence syndrome more dramatically than any prescription pharmaceutical has been shown to do. Patients are more likely to complete a treatment program when CES is used, and they are less likely to come back for additional treatment following their discharge. No significant negative side effects have ever been found from its more than 45 years treatment history in the United States in which tens of thousands of patients suffering from almost every type of addiction have been treated.

REFERENCES

Bianco, F. Jr. (1994). The efficacy of cranial electrotherapy stimulation (CES) for the relief of anxiety and depression among polysubstance abusers in chemical dependency treatment. Unpublished doctoral dissertation, the University of Tulsa, Oklahoma.

Closson, W. J. (1985, May 31). *Transcutaneous electrical nerve stimulation and adrenocorticotropic hormone production*. Paper presented at the Polypeptide Meeting, George Washington University, Washington, DC.

Dougherty, P. M., & Dafny, N. (1989). Trans-cranial electrical stimulation attenuates the severity of naloxon-precipitated morphine withdrawal in rats. *Life Sciences, 44*(26), 2051–2056.

Dougherty, P. M., Dong, W. Q., Faillace, L. A., & Dafny, N. (1990). Trans-cranial electrical stimulation attenuates abrupt morphine withdrawal in rats assayed by remote computerized quantification of multiple motor behavior indices. *European Journal of Pharmacology, 175*(2), 187–195.

Gold, M. S., Pottash, A.L.C., Sternback, H., Barbaban, J., & Annitto, W. (1982, October). *Anti-withdrawal effects of alpha methyl dopa and cranial electrotherapy*. Paper presented at the Society for Neuroscience, 12th Annual Meeting, Minneapolis, Minnesota.

Kennerly, R. (2006). Changes in quantitative EEG and low resolution tomography following cranial electrotherapy stimulation. Unpublished doctoral dissertation, North Texas State University, Denton, TX.

Kirsch, D. (2004). *The science behind cranial electrotherapy stimulation* (2nd edition). Retrieved January 23, 2009, from http://www.electromedical productsinternational.com.

Kirsch, I., & Sapirstein, G. (1998). Listening to Prozac but hearing placebo: A meta-analysis of antidepressant medication. *Prevention and Treatment, 1*, 2A.

National Research Council. (1976). *FDA Contract 70–22, Task Order No. 20*. NTIS PB 241305, pp. 1–54.

Pozos, R. S., Strack, L. E., White, R. K., and Richardson, A. W. (1971) Electrosleep versus electroconvulsive therapy. In D. V. Reynolds & A. E. Sjorberg (Eds.), *Neuroelectric research* (pp. 221–225). Springfield, IL: Charles Thomas.

Schmidt, R., Capo, T., & Boyd, E. (1986). Cranial electrotherapy stimulation as a treatment for anxiety in chemically dependent persons. *Alcoholism: Clinical and Experimental Research, 10*(2), 158–160.

Schmidt, R., Capo, T., Frazier, H., & Boren, D. (1984). Cranial electrotherapy stimulation as a treatment for cognitive brain dysfunction in chemical dependence. *Journal of Clinical Psychiatry, 45*, 60–63.

Shealy, C. N., Cady, R. K., Wilkie, R. G., Cox, R., Liss, S., & Clossen, W. (1989). Depression: A diagnostic, neurochemical profile and therapy with cranial electrical stimulation (CES). *Journal of Neurological and Orthopaedic Medicine and Surgery, 10*(4), 319–321.

Shroeder, M. J. (1999). Acquisition and quantitative analyses of EEG during CES and concurrent use of CES and neurofeedback. Unpublished doctoral dissertation, the University of Texas at Austin.

Siegesmund, K. A., Sances, A., Jr., & Larson, S. J. (1967). The effects of electrical currents on synaptic vesicles in monkey cortex. In F. M. Wageneder & Schuy, S. (Eds.), *Electrotherapeutic sleep and electroanaesthesia* (International Congress Series No. 136, pp. 31–33). New York: Excerpta Medica Foundation.

Smith, R. B. (1982). Confirming evidence of an effective treatment for brain dysfunction in alcoholic patients. *Journal of Nervous and Mental Disease, 170*, 275–278.

Smith, R. B. (1985). Cranial electrotherapy stimulation. In J. B. Myklebust, J. F. Cusick, A. Sances, Jr., & S. J. Larson (Eds.), *Neural stimulation* (vol. 2). Boca Raton, FL: CRC Press.

Smith, R. B. (2007). Cranial electrotherapy stimulation; its first fifty years, plus three. Mustang, OK: Tate Publishing.

Smith, R. B., & Day, E. (1977). The effects of cerebral electrotherapy on short-term memory impairment in alcoholic patients. *International Journal of the Addictions, 12,* 575–582.

Smith, R. B., & O'Neill, L. (1975). Electrosleep in the management of alcoholism. *Biological Psychiatry, 10*(6), 675–680.

Smith, R. B., Tiberi, A., & Marshall, J. (1994). The use of cranial electrotherapy stimulation in the treatment of closed-head-injured patients. *Brain Injury, 8*(4), 357–361.

This page intentionally left blank

Chapter **11**

An Analysis of Service Providers' Treatment Model Choices for Cocaine-Using Clients: A Case Study

Maria Dinis, PhD, MSW

There are various models of alcohol and drug treatment to which service providers subscribe. These treatment models (e.g., moral/prevention, medical/disease, psychological, sociocultural, and integrative/biopsychosocial models) have been widely described in the literature and are often referred to as treatment philosophies or orientations (Institute of Medicine, 1990; Miller & Hester, 1989, 2003; Miller & Kurtz, 1994; Peterson, Nisenholz, & Robinson, 2003). In the Institute of Medicine (1990) report, the committee explained that "a model for treatment consists of a certain perspective on or orientation toward the etiology of alcohol problems that in turn specifies the preferred methods of intervention and suggests expected outcomes" (p. 51).

How do service providers select treatment models or approaches to treat their clients? In general, professionals may select treatment approaches based on factors such as their educational background, professional and experiential training, work setting, personal belief systems, age, and gender. For example, a social worker employed as a probation officer may or may not select the same treatment approach as a social worker working in a hospital setting. It is, therefore, important to understand the relation between the background characteristics of treatment providers and their treatment choices.

Scott (1982) described how organizational missions and goals, in addition to professional training and socialization, influence professionals' work structure and decision-making processes. Lipsky (1980), however, argued that organizations cannot control decision-making behavior because street-level bureaucrats (e.g., police officers, teachers, social workers, nurses, doctors, and

lawyers) implement public policies based on decisions they make, routines they establish, and devices they invent to cope with uncertainties and work pressures. Additionally, professionals' clinical decisions are influenced by personal beliefs that ascribe causal explanations to alcohol and drug problems (Lipsky, 1980; Morgenstern & McCrady, 1992; Rosenberg & Davis, 1994) and inform the selection of treatment approaches for different populations (Nunes-Dinis, 1996).

Previous studies have examined the influences on professionals' treatment model choices for people with alcohol problems (Nunes-Dinis, 1996). More specifically, professionals from diverse community agencies—for example, alcohol and drug, criminal justice, health care, social welfare, mental health, and religious—were interviewed about their treatment choices for people with alcohol problems. Study findings revealed a complex relationship between providers' beliefs and model choice for heavy alcohol users. Overall, respondents selected models that corresponded with their personal belief system, except when treatment was provided to pregnant women. That is, the medical model was considered the optimal treatment approach for pregnant women, and pregnancy status emerged as a better predictor of service model choice than the providers' professional training or work setting (Nunes-Dinis, 1996).

Although pregnancy status was an important factor across professional groups in their treatment model choice for heavy alcohol-using clients, it is unclear how other drug use would influence professionals' treatment choices in similar situations. Cocaine-using clients, for example, may benefit from the use of different treatment approaches by professionals because the stigmatization associated with drug versus alcohol use may vary (Chavkin, 1990; Murphy & Rosenbaum, 1999). In addition, the difference in physiological consequences of alcohol versus cocaine use on fetal development may also influence treatment choice. Studies have indicated that alcohol contains a teratogen agent that causes congenital malformations such as fetal alcohol syndrome, whereas cocaine exposure alone has not been shown to exhibit teratogenic consequences (Batshaw & Conlon, 1997; Berenson, Wilkinson, & Lopez, 1996; Chasnoff, 1988; Gómez, 1997; Inciardi, Surratt, & Saum, 1997; Levitt, 1998).

In chapter 14 in this volume Dinis, Merighi, and Anders provide a literature review describing models or therapeutic approaches, beliefs, and perceptions of providers when treating alcohol or drug-using clients. The focus of this case study is to describe professionals' treatment model choice for women (pregnant and nonpregnant) and men who misuse cocaine. Predictors of treatment model choice included professionals' beliefs about the causes of drug addiction, educational background, and work setting. The following questions were examined to test what demographic variables constitute the best predictors of

treatment model choice: (1) What demographic factors can be used to predict providers' selection of medical, sociocultural, or integrative treatment models for the treatment of cocaine misuse?; (2) Are there differences in treatment model choice for cocaine-using women (pregnant and nonpregnant) and men?; and (3) Do providers with similar personal beliefs, educational backgrounds, or work settings choose identical treatment models regardless of the gender and pregnancy status of their clients?

METHODS

Sample

This is a secondary data analysis of a provider study originally conducted during 1995 in a northern California county, the site of the Alcohol Research Group's Community Epidemiology Laboratory (CEL). The CEL county was selected based on a broad cross-section of population characteristics, including: ethnicity, rural and urban mix, socioeconomic status, and a variety of health and social services agencies. The overall methods and study design has been described in other reports (Room & Weisner, 1988; Weisner & Schmidt, 1995).

A two-stage probability sample approach of health and human service providers was representative of service organizations and staff in the county. This sample of agencies was stratified by the following service sectors: substance abuse treatment, criminal justice, mental health, health care, welfare, and religious organizations. This cross-sectional survey of providers included workers in health and human service organizations and solo practitioners in mental health treatment and health care systems. A listing of organizations and individuals in the county was developed by using telephone directory listings, snowballing of agencies' listings, and follow-up phone screening interviews with a provider from each organization. From a listing of 2,390 eligible organizations, 117 stratified agencies were selected randomly. A simple random sample of practitioners (i.e., physicians and psychotherapists) was drawn from a separate listing. Permission was obtained to interview providers in the 117 organizations, and a list was developed of all direct service professionals working at least half time. Individual providers were sampled proportionate to organization size. Line service providers were the units of analysis. Of the total 610 solo and agency providers, 73 percent were interviewed (N = 457). The response rate includes the nonresponse at both the organizational and individual levels of sampling.

Forty-two percent of the providers responding were male (n = 191) and 58 percent were female (n = 265). About two-thirds were married or living

with a partner. More than half of the providers were under 44 years of age. Approximately half of the sample completed graduate education at the master's or doctoral level, 22 percent were college graduates, and 28 percent had some college training.

Weights (ranging from 0.04–2.29) were calculated to adjust for nonresponse bias and for design effects due to stratification at the first stage of sampling and varying selection intervals at the second stage. Those weights were representative of the actual proportion of different kinds of providers working in the stratified service sectors.

Procedures

Trained interviewers who were not affiliated with any of the agencies administered a one-hour structured in-person survey usually in the workplace, but arrangements were made to interview providers in other locations when necessary. Informed consent was obtained and participation was completely voluntary.

Measurement

Our analysis focused on the kinds of treatment models providers would recommend for white women (pregnant and nonpregnant) and men who were using cocaine. Providers' drug addiction beliefs and sociodemographic characteristics were taken into account. Responding to case situations described in the survey, service providers recommended services options that were then classified into models using the criteria from the Institute of Medicine (1990) report. These models were categorized into program components and service types: medical, sociocultural, and integrative. Medical models are physician-driven and often employ medications such as antidepressants or beta-blockers. Sociocultural models operate in social settings like halfway houses and in self-help groups (e.g., Cocaine Anonymous). Integrative models are other models identified such as biopsychosocial, dual-diagnosis, or compulsory approach (some kind of legal sanction—anything from a fine to incarceration). The treatment models were classified according to the number of times providers selected them. Only three models (medical, sociocultural, and integrative) were used as outcomes (dependent) throughout the logistic regression analysis because providers primarily selected those models across the three groups: women (pregnant and nonpregnant) and men who were using cocaine.[1]

The independent variables associated with treatment model choice were the etiological beliefs of drug addiction, personal demographics, professional degree, and work settings. The etiological beliefs were: if service providers had

to choose, would they say that drug addiction was mostly a genetic problem—something a person is born with, or social, psychological, or multiple problems. The personal demographics of the service providers included: gender (male or female); age (under 35 years old, 35–44 years old, or 45-plus); and level of education (non-postgraduate [some college training and college graduates] and postgraduate [master's or doctoral level graduate education]). Professional degree was categorized as no advanced degree, criminal/social science/liberal arts/education, science/medical, counseling/therapy, social work, theology/divinity, and business/management/law. The work settings were alcohol/drug, criminal justice, mental health, health care, welfare, and religious.

Data Analysis

Chi-square tests were used to assess significant differences between model choice of service providers based on their etiological beliefs of drug addiction, type of professional degree, work settings, and sociodemographic characteristics. Weighted data were used.

Logistic regression analyses were used to predict providers' model choice using their etiological beliefs of drug addiction, type of professional degree, work settings, and sociodemographic characteristics of providers as independent variables. These independent variables were coded as 1 and 0; for example, gender was coded 1 for male and 0 for female. Other independent variables were coded with this same pattern: education (non-postgraduate vs. postgraduate); and etiology (genetic vs. all other beliefs, social vs. all other beliefs, and multiple vs. all other beliefs). The comparison group for all others for genetic belief included social, psychological, and multiple; for social belief, they consisted of genetic, psychological, and multiple; and for multiple beliefs, they contained genetic, social, and psychological beliefs. For independent variables with more than two attributes, the comparison group selected was: the category of counseling/therapy for the variable professional degree, alcohol/drug for settings, and 45-plus for age. The data analyses used SPSS (SPSS, 2006).

RESULTS

Table 11.1 compares demographic characteristics of respondents and their selection of models to treat three treatment groups. Women providers were more likely than men to select the sociocultural model for women clients misusing cocaine, and the same trend nearly approached significance ($p < .06$) for men misusing cocaine. Overall, age was not a significant predictor of provider's treatment model choices. Providers, ages of 35 to 44, were more likely to

Table 11.1
Demographic Characteristics by Service Model Choice for Cocaine-Using Clients (in percentages) (*N* = 457)

	Pregnant women			Nonpregnant women			Men		
Unweighted *N*	Medical (269)	Socio-cultural (27)	Integra-tive (134)	Medical (146)	Socio-cultural (133)	Integra-tive (134)	Medical (145)	Socio-cultural (123)	Integra-tive (130)
Gender									
Male	55	2*	34	30	22**	29	30	23	28
Female	60	6	30	30	34	31	35	31	30
Age									
< 35	63	3	28	32	32	26*	37	26	26
35–44	58	5	33	28	30	38	28	29	38
45+	57	5	31	32	28	26	35	28	24
Education									
Non-postgraduate	64**	2*	26*	31	26	31	37	23*	29
Postgraduate	52	7	37	29	33	29	29	34	30

Analyses are conducted on weighted data. Chi-square tests of significance were conducted within each model.
*$p < .05$; **$p < .01$.

choose the integrative model for women who were not pregnant. Education was important in model choice. Individuals with some college training and college graduates (non-postgraduate) selected the medical model for pregnant women, and nearly approached significance for men ($p < .08$), while those with master's or doctoral-level (postgraduate) education were less likely to select sociocultural and integrative models for pregnant women. Providers with postgraduate degrees also selected the sociocultural model for men misusing cocaine.

Table 11.2 examines the relationship between type of professional degree and providers' selection of treatment models. Regardless of the professional degree, the medical model was chosen for pregnant women. For other groups, the medical model was also recommended but not consistently chosen. For instance, those with professional degrees in counseling or therapy and science or medicine were less likely to select the medical model for nonpregnant women or men. Among those who chose the medical model, a larger proportion had professional degrees in business/management/law, criminal/social science/liberal arts/education, and theology/divinity.

Table 11.2
Type of Professional Degree by Provider's (Cocaine) Model Selection (in percentages) (*N* = 457)

	Pregnant women			Nonpregnant women			Men		
Unweighted *N*	Medical (269)	Socio-cultural (27)	Integra-tive (134)	Medical (146)	Socio-cultural (133)	Integra-tive (134)	Medical (145)	Socio-cultural (123)	Integra-tive (130)
Professional degree	59	4	30	30	36	25	39	30	25
No advanced degree (n = 44)									
Criminal/social science/liberal arts/education (n = 52)	71	—	23	41	26	24	43	26	24
Science/medical (n = 147)	50	5	32	14	37	33	18	35	30
Counseling/therapy (n = 67)	52	13	34	33	28	33	33	27	36
Social work (n = 24)	58	—	42	38	39	22	35	42	22
Theology/divinity (n = 32)	66	—	34	50	9	31	50	9	31
Business/management/law (n = 20)	80	5	10	35	25	10	38	25	10

Analyses are conducted on weighted data. Chi-square tests of significance were conducted within each model.
*p < .05; **p < .01; ***p < .001.

A higher proportion of providers adopted the medical model for pregnant women than for any other group and adopted the sociocultural model for nonpregnant women and men cocaine misusers. More providers selecting the medical model for all three client groups had professional degrees ranging from business to criminal justice to theology, even though a significant proportion had no advanced degree. Although not statistically significant, providers with scientific or medical, counseling or therapy, and social work educational backgrounds also selected integrative models for the three groups.

Table 11.3 shows the proportion of providers across work settings and their model choices. For all work settings, the medical model was by far the most frequently selected for pregnant women. Although the medical model was indi-

Table 11.3
Work Setting by Service Model Choice for Cocaine-Using Clients (in percentages) (*N* = 457)

	Pregnant women			Nonpregnant women			Men		
Unweighted *N*	Medical (269)	Socio-cultural (27)	Integra-tive (134)	Medical (146)	Socio-cultural (133)	Integra-tive (134)	Medical (145)	Socio-cultural (123)	Integra-tive (130)
Work settings Alcohol/drug	47	12	41	18	41	38	24	41	35
Criminal justice	78	4	15	42	33	18	42	33	18
Mental health	60	7	30	39	24	29	39	29	27
Health care	51	3	36	19	36	33	23	32	32
Welfare	63	5	31	42	17	34	44	17	34
Religious	59	2	26	39	16	24	41	11	24

Analyses are conducted on weighted data.

$^{*}p < .05$; $^{**}p < .01$.

cated for nonpregnant women and men cocaine misusers, a similar number of providers selected the sociocultural and integrative models—30 and 32 percent, respectively. In fact, in alcohol/drug and health care work settings, the sociocultural and integrative models were chosen more frequently than the medical model. The range of providers selecting the medical model for pregnant women was between 47 and 78 percent, whereas for the other two groups it was between 18 and 44 percent.

Clients' pregnancy status rather than a particular gender group influenced model choice of providers in various work settings. A higher proportion of providers selected the medical model, as compared to all other models, for pregnant women across the professional settings.

More providers selecting the medical model across the three groups worked in the criminal justice setting, followed by welfare, mental health, and religious settings. Providers adhering to the sociocultural model for nonpregnant women and men were primarily from alcohol/drug treatment settings, followed by health care and criminal justice settings.

Logistic regression analyses were conducted to determine significant predictors of model choice (medical vs. all others, sociocultural vs. all others, and integrative vs. all others), using etiology, profession, work setting, and demographics as independent variables (Table 11.4). Although not shown, providers reported that drug addiction is mostly a social problem (52%), followed by

Table 11.4
Adjusted Odds Ratios (95% CI) Predicting Model Choice for Women (Pregnant and Nonpregnant) and Men Cocaine Users (*N* = 457)

	Medical	Sociocultural	Integrative
Unweighted *N*			
Pregnant women	(269)	(27)	(134)
Etiology			
1. Genetic vs. all others	—	—	—
2. Social vs. all others	.60* (.36, .99)	—	1.81* (1.01, 3.22)
3. Multiple vs. all others	—	—	4.13** (1.67, 10.22)
Profession			
Theology	—	—	—
Demographics			
Gender (female)	1.76* (1.08, 2.87)	5.90* (1.37, 25.51)	.51** (.31, .87)
Age	—	—	—
Education (non-postgraduate)	—	.10* (.01, .68)	—
Nonpregnant women	(146)	(133)	(134)
Etiology			
1. Genetic vs. all others	—	—	—
2. Social vs. all others	—	2.06** (1.18, 3.59)	—
3. Multiple vs. all others	—	—	2.74* (1.14, 6.59)
Profession			
Theology	10.90** (1.92, 61.90)	.13* (.02, .79)	—
Demographics			
Gender (female)	—	2.46** (1.40, 4.33)	—
Age	—	—	2.10** (1.24, 3.58)
Education (non-postgraduate)	—	.43** (.23, .81)	—
Men	(145)	(123)	(130)
Etiology			
1. Genetic vs. all others	—	—	—
2. Social vs. all others	—	—	—
3. Multiple vs. all others	—	.30* (.10, .91)	3.31** (1.35, 8.10)
Profession			
Theology	6.57* (1.34, 32.17)	—	—
Demographics			
Gender (female)	1.88* (1.09, 3.24)	1.91* (1.09, 3.32)	—
Age	—	—	2.63*** (1.53, 4.53)
Education (non-postgraduate)	—	.36*** (.19, .67)	—

Analyses are conducted on weighted data. Each model also controlled for work setting and for all professional background types.
*$p < .05$; **$p < .01$; ***$p < .001$.

psychological (27%), genetic (12%), and multicausal (9%). Women providers were almost twice as likely to adopt the medical model for pregnant women and men cocaine users. When compared with counseling and therapy, those in the profession of theology were nearly 11 times more likely to select the medical model for nonpregnant women and over 6.5 times as likely to select the medical model for men.

Providers who believed drug addiction was a social problem were about half as likely to support a medical model for pregnant women but were twice as likely to select the sociocultural model for nonpregnant women and the integrative model for pregnant women. Women providers were nearly twice to six times more likely than men to select the sociocultural model for all three groups. Non-postgraduates were less than half as likely to select the sociocultural model for all groups. Providers in the clergy profession were 13 percent less likely to adopt the sociocultural model for nonpregnant women. It is important to take caution when interpreting results from the pregnant sociocultural category because there was inadequate statistical power due to small sample size in this category.

Providers who believed that drug addiction has multiple causes (i.e., those who alluded to more than one etiological cause of addiction) were nearly three to four times as likely to adopt an integrative model for all three groups and about a third less likely to select the sociocultural model for men. Women providers were half as likely to endorse the integrative model for pregnant women only. Providers, ages 35 to 44, were more than twice as likely to choose the integrative model for nonpregnant women and men.

DISCUSSION

In this study, the gender of providers was important in predicting their selection of treatment models. Individuals with postgraduate degrees in any field of study were more likely to choose the sociocultural and integrative models for pregnant women. Regardless of the work setting of the providers, the medical model was selected more for the pregnant women but less frequently for the other two groups.

The relationship between beliefs about etiology of drug addiction is imperfect in predicting model choice. The only significant match across all three groups was between providers who believed in multiple causes and their selection of the integrative model. This may have occurred because integrative models facilitate the combination of biological, psychological, and social factors in assessment and treatment of alcohol and drug problems (Brower, Blow, & Beresford, 1989; Institute of Medicine, 1990; Johnson, 2004; Peterson et al.,

2003). In this approach, there is recognition that problems are determined by multiple factors, involve different causes, and that one or a combination of the factors may determine individual outcomes. It may be speculated that providers may feel more comfortable with a combination of solutions offered by integrative models than by any one of them by itself. There were other congruent beliefs between social etiology and multiple etiologies and the selection of corresponding models for men and nonpregnant women. Providers who believed in a social or multiple etiologies for men and pregnant women were less likely to select medical or sociocultural models respectively. Although those beliefs are not necessarily congruent with the corresponding model, providers may be showing support for their selection of the integrative model for men and pregnant women. In the literature, however, providers are inconsistent in their beliefs about addiction and agree with conflicting perspectives ranging from the disease concept to psychoanalytic interpretations to moralistic attitudes (Kahle & White, 1991; Taxman & Bouffard, 2003; Taxman, Simpson, & Piquero, 2002; Wechsler & Rohman, 1982). This may explain why providers believe in various causes of drug addiction and do not necessarily adopt corresponding models of treatment for any of the groups.

Providers across work settings were not always consistent in the models they selected for the three groups. Work settings of providers were not an important predictor of model choice, but possessing a theology degree was significant. In the bivariate analysis, providers from various professional backgrounds selected the medical model. In the multivariate analysis, only providers from a theology professional background adopted the medical model for nonpregnant women and men misusing cocaine. With only 32 theologians in the sample, this finding probably represented a random result, given that we did many tests and the bivariate difference shown in Table 11.2 is not very dramatic.

Work settings were not related to beliefs about etiology and model choice. In the bivariate analysis (not shown), 47 percent of providers in the alcohol/drug settings believed that drug addiction is a social problem, but they selected the medical model for pregnant women. In fact, between 48 and 59 percent of providers in all work settings believed that drug addiction was a social problem, yet they selected the medical model for pregnant women. While only approximately 18 percent of providers believed that drug addiction was a multicausal problem, over one-third of the providers in alcohol/drug and health care settings selected the integrative model for nonpregnant women and men.

From the bivariate analysis, beliefs, professional degree, and work settings were important until we controlled for gender and education. In the multivariate analysis, gender and education significantly influenced model choice. Women providers selected the medical model for pregnant women and men, the socio-

cultural model for all three groups, and were less likely to select the integrative model for pregnant women. The extent of education seems to be more important than the type of professional education. Those with at least some college training and college graduates were less likely to select the sociocultural model for all three groups. That group may lack familiarity with the sociocultural model or perceive the use of professionals in medical and integrative models more effective for their clients than peer-run or self-help approaches (i.e., sociocultural). Also, respondents having either some college or only bachelor's-level training are perhaps in job positions that require following agency dictates rather than their own beliefs. For example, in some work settings such as criminal justice, they may have protocols that require first referring medically before suggesting other options. It is interesting to note, however, that as the level of providers' education increased to master's or doctoral-level training, the less likely they are to adopt the more professional-based medical model, but those with some college or bachelor's-level training do select the medical model.

It is unclear what variations in suggesting treatment for pregnant women may be attributed to pregnancy status or gender differences. It is not known if the perception of pregnancy as a medical condition influences providers to choose the medical model foremost. Further, it is unclear whether medical conditions of any type automatically determine a medical selection or if providers perceive a difference in treatment between men and women. Perhaps providers might also first choose a medical model for men with diabetes, for instance, before considering drug treatment issues. Providers may not recommend models of treatment based on the need of treatment but instead may first address medical conditions like pregnancy. In the bivariate analysis, similar proportions of providers selected across the three major treatment models for nonpregnant women and men.

In general, in this sample, providers selected the medical model to treat pregnant women while they chose similar proportions across all of the three models for the other two client groups. These findings suggest that providers primarily may not be selecting treatment approaches based on factors such as personal belief systems, educational or professional training, work setting, or demographic characteristics. Even though currently the medical condition of pregnancy dominates treatment choice for pregnant women, there are other cocaine-misusing clients where factors interplay in their selection of treatment options. Further research is needed to examine why providers have personal belief systems that are incongruent with their corresponding model choice. Perhaps providers select models that are incongruent with their beliefs because the current treatment approaches available restrict them. That is, providers may have particular beliefs and not a treatment model that corresponds to

their belief. For example, providers may believe that drug addiction is primarily caused by genetic factors and yet not necessarily equate that they would principally only want to treat the condition medically. Having a belief in a genetic predisposition for drug misuse does not automatically imply that providers will necessarily select a medical treatment option. Similarly, providers who believe in social or multiple causes of addiction may recognize the limitations of the corresponding models to ameliorate large-scale environmental social problems and other personal conditions (e.g., poverty, racism, class distinctions, and mental health issues) contributing to drug misuse. These findings indicate the complexity involved that providers must encounter when selecting treatment models for their clients because the interaction between beliefs, treatment models available, and what may work for a client at one time but not another are all factors that may be involved in the decision-making process of providers.

Acknowledgments

Preparation of this chapter was supported by a National Institute on Alcohol Abuse and Alcoholism (NIAAA) (R01-AA005595–11) award to the Alcohol Research Group in Berkeley, California and the California State University, Sacramento, Research and Creative Activity Award Program. The author wishes to acknowledge Constance Weisner, Lee Kaskutas, Lois Lowe, Joe Merighi, and Kelly Anders for helpful comments and Tammy Tam for statistical consultation.

NOTE

1. Fewer than eight percent of the providers adopted prevention or psychological models for the three groups, which resulted in a small sample size with cells less than five cases to include in the logistic regression analysis. Thus, percentages do not total 100 in the tables because providers also selected prevention and psychological models.

REFERENCES

Batshaw, M. L., & Conlon, C. J. (1997). Substance abuse: A preventable threat to development. In M. L. Batshaw (Ed.), *Children with disabilities* (pp. 143–162). Baltimore, MD: Paul H. Brookes.

Berenson, A. B., Wilkinson, G. S., & Lopez, L. A. (1996). Effects of prenatal care on neonates born to drug-using women. *Substance Use and Misuse, 31*(8), 1063–1076.

Brower, K. J., Blow, F. C., & Beresford, T. P. (1989). Treatment implications of chemical dependency models: An integrative approach. *Journal of Substance Abuse Treatment, 6*, 147–157.

Chasnoff, I. J. (1988). Drug use in pregnancy: Parameters of risk. *Pediatric Clinician of North America, 82,* 888–895.

Chavkin, W. (1990, April). Drug addiction and pregnancy: Policy crossroads. *American Journal of Public Health, 80*(4), 483–487.

Gómez, L. E. (1997). Misconceiving mothers: Legislators, prosecutors, and the politics of prenatal drug exposure. Philadelphia: Temple University Press.

Inciardi, J. A., Surratt, H. L., & Saum, C. A. (1997). *Cocaine-exposed infants: Social, legal, and public health issues* (vol. 5, Drugs, Health, and Social Policy Series). Thousand Oaks, CA: Sage Publications.

Institute of Medicine. (1990). *Broadening the base of treatment for alcohol problems.* Washington, DC: National Academy Press.

Johnson, J. L. (2004). *Fundamentals of substance abuse practice.* Belmont, CA: Brooks/Cole–Thompson Learning.

Kahle, D. B., & White, R. M. (1991). Attitudes toward alcoholism among psychologists and marriage, family and child counselors. *Journal of Studies on Alcohol, 52*(4), 321–324.

Levitt, P. (1998). Prenatal effects of drugs of abuse on brain development. *Drug and Alcohol Dependence, 51*(1/2), 109–125.

Lipsky, M. (1980). Street-level bureaucracy: Dilemmas of the individual in public services. New York: Russell Sage Foundation.

Miller, W. R., & Hester, R. K. (1989). Treating alcohol problems: Toward an informed eclecticism. In R. K. Hester & W. R. Miller (Eds.), *Handbook of alcoholism treatment approaches: Effective alternatives* (pp. 3–13). New York: Pergamon Press.

Miller, W. R., & Hester, R. K. (2003). Treating alcohol problems: Toward an informed eclecticism. In R. K. Hester & W. R. Miller (Eds.), *Handbook of alcoholism treatment approaches: Effective alternatives* (3rd ed., pp. 1–12). Boston: Allyn and Bacon.

Miller, W. R., & Kurtz, E. (1994). Models of alcoholism used in treatment: Contrasting AA and other perspectives with which it is often confused. *Journal of Studies on Alcohol, 55*(2), 159–166.

Morgenstern, J., & McCrady, B. S. (1992). Curative factors in alcohol and drug treatment: Behavioral and disease model perspectives. *British Journal of Addiction, 87,* 901–912.

Murphy, S., & Rosenbaum, M. (1999). *Pregnant women on drugs: Combating stereotypes and stigma.* New Brunswick, NJ: Rutgers University Press.

Nunes-Dinis, M. C. (1996). An analysis of model choice of service providers using Scott's and Lipsky's theoretical frameworks: Women (pregnant and nonpregnant) and men drinkers. Unpublished doctoral dissertation, University of California, Berkeley.

Peterson, J. V., Nisenholz, B., & Robinson, G. (2003). *A nation under the influence: America's addiction to alcohol.* Boston: Allyn and Bacon.

Room, R., & Weisner, C. (1988). *Studying community responses to alcohol problems: An interim report.* Presented at the 14th Annual Alcohol Epidemiology Symposium, Kettil Bruun Society, Berkeley, California, June 5–11.

Rosenberg, H., & Davis, L. A. (1994). Acceptance of moderate drinking by alcohol treatment services in the United States. *Journal of Studies on Alcohol, 55,* 167–172.

Scott, W. R. (1982). Managing professional work: Three models of control for health organizations. *Health Services Research, 17*(3), 213–240.

SPSS Inc. (2006). *SPSS for Windows base user's guide*. Chicago, IL: Author.

Taxman, F. S., & Bouffard, J. A. (2003). Substance abuse counselors' treatment philosophy and the content of treatment services provided to offenders in drug court programs. *Journal of Substance Abuse Treatment, 25,* 75–84.

Taxman, F. S., Simpson, S., & Piquero, N. (2002). Measuring and calibrating therapeutic integration in drug treatment programs. *Journal of Criminal Justice, 30*(3), 159–173.

Wechsler, H., & Rohman, M. (1982). Future caregivers' views on alcoholism treatment: A poor prognosis. *Journal of Studies on Alcohol, 43*(9), 939–955

Weisner, C., & Schmidt, L. (1995). The Community Epidemiology Laboratory: Studying alcohol problems in community and agency-based populations. *Addiction, 90*(3), 329–341.

This page intentionally left blank

Chapter **12**

Translating Efficacy to Effectiveness in Opioid Dependence Treatment

Van L. King, MD, Michael Kidorf, PhD, Kenneth B. Stoller, MD, Karin J. Neufeld, MD, MPH, Jessica Peirce, PhD, and Robert K. Brooner, PhD

Opioid dependence is a severe, persistent disorder often requiring long-term and at times intensive psychiatric treatment. Though opioid dependence in the United States has primarily involved heroin use, opioid users are increasingly turning to prescription opioid (e.g., oxycodone) misuse in recent years. To date, most approaches to opioid dependence treatment utilize single or sometimes two treatment approaches, often with only modest improvements in the patient's quality of life. In this review, we describe the development and implementation of Motivated Stepped Care (MSC), a community-based, adaptive, combination treatment model for chronic opioid dependence created through translational research that utilizes insights gained from medication development and behavioral experimental findings. MSC fully integrates medication therapy, behavioral reinforcement (also called contingency management) procedures, and counseling psychotherapy into an intervention that addresses numerous aspects of drug use disorders and results in improved outcomes. In particular, we demonstrate how MSC addresses poor treatment adherence, a problem in all medical interventions and a particularly acute problem with this treatment population. By focusing not only on the underlying neurochemistry of addiction with medications but also on altering the behaviors of patients through targeted behavioral reinforcement, we demonstrate how MSC increases the amount of treatment that is provided, thereby significantly improving treatment outcome.

First we outline the development of several efficacious interventions for the treatment of opiate dependence by discussing the development of medication

interventions, which include opioid medications (medications that act similarly to morphine and heroin) and opioid-blocking medications (medications that block the euphoric and pain-relieving effects of opioids). In this chapter, we use the word *efficacy* (efficacious) to describe treatment benefits to patients enrolled in research studies with well-defined subjects and controlled treatment conditions. *Effectiveness* refers to patient improvement demonstrated in nonresearch treatment settings that typically treat a less selected, real world patient population and so measure the utility of a treatment to appropriately trained community-based health care providers.

Much of the success of current treatment for chronic opioid dependence relies on medications developed in a translational manner by responding to naturalistic experiments using existing compounds. We use the term *translational* to describe the process that investigators use to develop novel research and clinical paradigms based on observations made by researchers in other areas of scientific inquiry. It not only refers to knowledge from basic scientific discoveries inspiring treatment applications (such as a new antibiotic developed on the basis of new information about a specific bacteria) but also to translating the results of efficacy studies into effective, everyday clinical practice (Woolf, 2008). For example, inspired by early morphine maintenance treatments (see later in this chapter), methadone maintenance was conceptualized in the 1960s as an adjunct to psychosocial treatment for opioid dependence. Because of its usefulness, other opioid and opioid-blocking medications (i.e., L-alpha-acetylmethadol, buprenorphine, and naltrexone) were developed in an attempt to improve on or offer alternatives to methadone maintenance treatment. The concept that chemical compounds could be targeted to affect a patient's desire to use opioid drugs was a key insight that translated into several extensive lines of investigation and subsequent medication treatments.

Next we discuss the translational development of behavioral interventions derived from learning theory (primarily operant conditioning) that have been used successfully in specialized behavioral treatments for drug dependence. Based on the idea that drug dependence is a highly specific, goal-oriented pattern of behaviors motivated by the expected positive consequences of drug use (Bigelow, Brooner, & Silverman, 1998; McHugh & Slavney, 1998), drug use behavior is understood as a consequence of the individual's decision to use drugs, and the result of weighing the positive and negative consequences of the behavior rather than a result of uncontrollable urges. This perspective recognizes the strength of drugs as powerful motivators in the lives of patients and can be used to help shape behaviors that compete with and ultimately supplant drug-dependent behavior. Contingency management treatment approaches are

an excellent example of how these more basic insights have been translated and adapted to create an array of behavioral treatments for substance dependence.

Medication and behavioral interventions are only partially effective, however. The next section underscores the importance of psychotherapy in combination treatments for substance use disorder. Unfortunately, even though most treatment experts agree that psychotherapy is an important ingredient in a comprehensive substance abuse treatment (APA, 2006), the effectiveness of psychotherapy is significantly limited by poor attendance. In fact, virtually all medical treatments are significantly limited by poor adherence, but this problem is especially problematic in substance use disorder treatment. Even relatively effective treatments such as methadone maintenance fall far short of a definitive treatment intervention, with many patients continuing to use illicit drugs on a regular basis and discontinuing treatment prematurely. One of the greatest challenges that accounts for these unsatisfactory results is poor patient adherence to the prescribed plan of care. How to improve patient adherence to maximize the effectiveness of psychotherapy and other treatment approaches is an ongoing challenge.

Finally, we describe how these complementary treatment approaches are incorporated into the MSC model. Utilizing research from disparate sources, we show how medication interventions and behavioral research findings dramatically improve treatment adherence rates in an opioid-dependent population. Targeted reinforcement of patient behavior is a key component in this model of care, creating a synergy that significantly improves treatment effectiveness. By focusing not only on offering care, but also on altering the behaviors of patients to ensure that the care is received, treatment outcomes are significantly improved. This unique model of care is the next advance in translating efficacious research findings into effective community-based treatment models.

MEDICATION TREATMENT TRANSLATIONS

Opiates (drugs derived from naturally occurring compounds that activate opiate receptors in the body) have been used for centuries as important medicinal compounds as well as for their euphorogenic, and therefore behaviorally reinforcing, properties. That is, many individuals like the subjective effects of these medications and will work to obtain and use them. Opioid drugs include all naturally occurring as well as synthetic drugs that activate opiate receptors. Chronic daily administration of opioids leads to tolerance (increasing doses of the drug must be taken to obtain the same effects) and physical dependence (routine use of the drug is required to prevent the onset of characteristic physi-

cal withdrawal symptoms). Even though opioid abusers initially use the drug for its euphorogenic effects, as tolerance develops, they increasingly use opioids to relieve the withdrawal symptoms consequent to the physical dependence (severe muscle aches, diarrhea, abdominal pain, vomiting, insomnia, malaise, goose flesh, and other symptoms) and get little pleasure from the drug use. Many seek treatment at this time, unable to tolerate the severe flulike symptoms of opioid withdrawal (so called cold turkey due to the presence of goose flesh and chills that accompany the syndrome) without medical intervention.

Treatment of opioid withdrawal symptoms has been approached in a variety of ways over the years, but the most difficult problems arise in those who develop the clinical syndrome of opioid dependence disorder (also called opioid addiction). This syndrome is characterized by a maladaptive pattern of drug use causing significant clinical impairment or distress (APA, 2006). Individuals typically exhibit physical tolerance and withdrawal symptoms if they stop opioid use, social problems such as failing to meet role obligations as a parent or head of household, and other symptoms consistent with a loss of control over drug use. These individuals frequently relapse to daily opioid use even after periods of detoxification and abstinence and in the face of serious social and legal sanctions. The strong psychological and behavioral aspects of opioid dependence are at least as important in the persistence of the drug use problems as the physical withdrawal symptoms. In this section we examine agonist, partial agonist, and antagonist medications that are used in the treatment of opioid dependence (Center for Substance Abuse Treatment, 2005; Stoller & Bigelow, 2006).

Opiate Receptor Agonists and Partial Agonists

Agonist medications activate the cellular receptors specific to the medication, causing a specific action as a result of the activation. Partial agonists can only activate the receptor to a degree that is less than maximal, so they have less powerful effects. Prior to the advent of agonist treatment, treatment for opiate dependence was hospital or residentially based and abstinence oriented, with patients expected to stop all drug use and follow the plans of the treatment providers after leaving the institution. Unfortunately, these approaches were often not effective, and physicians found themselves in the uncomfortable position of rationing or denying opiate prescriptions for opiate-dependent patients without acute medical needs for opiates but who either could not or would not discontinue opiate use. Rather than accept this at times adversarial role, and because they recognized the chronic and relapsing nature of opioid dependence, some physicians resorted to ongoing prescription of morphine (so-called mor-

phine maintenance) as a treatment for opiate dependence so that these patients would not resort to illegal means to acquire opiates. This approach was found to benefit some patients who could be maintained on relatively stable doses of morphine for extended periods of time. Subsequently, shortly before and around the year 1920, specialized morphine maintenance clinics were established in New York City, Shreveport, Louisiana, and other cities around the United States to improve access to maintenance treatment. These clinics were a rational response to a problem with no good solutions at the time.

However, in 1919, the Supreme Court upheld the U.S. Treasury Department's restrictive interpretation of the Harrison Narcotic Act, so all of these efforts were closed in the early 1920s. Opioid dependence came to be viewed as a criminal activity, and organized efforts to treat it with maintenance opiates were criminalized. Treatment for opioid dependence advanced little during the ensuing years, but in the early 1960s, mounting opinion from medical groups and the federal government favored trials of opioid maintenance treatment to address an escalating national problem with heroin abuse. Vincent Dole, MD, a researcher in metabolism at the Rockefeller University, received a grant in 1962 to investigate the feasibility of opioid maintenance treatment. He recruited Marie Nyswander, MD, a psychiatrist and author specializing in addictions treatment, to collaborate with him to develop an opioid agonist treatment for heroin dependence that would complement psychotherapy and other rehabilitative treatment.

Using observations from the early morphine maintenance projects, they took a more rigorous and scientific approach, recruiting volunteer opioid-dependent subjects for inpatient studies to determine which opioid agonist medication would be most suitable for this purpose. Of the several medications in the trials, methadone had the most desirable chemical properties. It was well absorbed orally and had a long action in the body such that once-daily dosing was adequate to treat opiate withdrawal symptoms. Sufficient doses of methadone (typically in the range of 50–150 mg daily) allowed the patient substantial control over opioid cravings and withdrawal symptoms and blunted the effects of illicit opioids by reducing the capacity to experience desired opioid drug effects (APA, 2006). Simply put, if patients don't receive the euphoric effects of opioid use, they reduce their use dramatically. The results were extremely positive. Patients maintained on individualized maintenance doses of methadone were much more likely to remain in programs of rehabilitation, acquiring employment and eventually ceasing all heroin use. These results were in stark contrast to the poor results obtained from detoxification and psychotherapy treatments available at the time (Dole, Nyswander, & Kreek, 1966). Their program of methadone maintenance treatment was introduced in New York City in 1965.

Methadone maintenance is now the most researched treatment for chronic opioid dependence, with overall better results than treatments not utilizing opioid agonist or partial agonist medications (Mattick, Breen, Kimber, & Davoli, 2003). However, there are shortcomings to methadone maintenance treatment such as daily dosing and restriction to certified narcotic treatment programs, so other medications with different profiles of activity have been developed over the years as treatment options to complement the effectiveness of methadone maintenance treatment. The first of these opioid agonist medications was LAAM (L-alpha-acetyl-methadol), another synthetic opioid that, like methadone, was developed in Germany during World War II. It was introduced in the United States in 1993 as a longer-acting agent that was taken only every two to three days. This quality of the medication reduced some of the burden of methadone treatment when patients would otherwise be required to report to the treatment clinic every day. Buprenorphine, a partial opioid agonist, also has a longer action in the body than methadone and a safer risk profile in overdose. It was approved in 2000 as a Schedule 3 compound that could be prescribed by certified physicians for office-based treatment of opioid dependence, potentially expanding the capacity of opioid dependence treatment providers into entirely new treatment venues not restricted to certified clinics. Investigators and clinicians recognized the importance of the opioid agonist activity for the acceptability and the utility of agonist and partial agonist treatments. They provide rapid symptomatic relief of opioid craving and physical withdrawal symptoms that is often of primary concern to patients entering treatment, thus making these compounds mainstays for chronic opioid dependence treatment.

Opiate Receptor Antagonists

A completely different approach to medication treatment involves blocking the opiate receptor with an antagonist compound such that individuals are unable to experience opiate agonist effects. Ideally, an antagonist would cause no drug effects itself but would simply prevent the effects of opioids that were taken after the antagonist. The compound cyclazocine was originally investigated as an opioid antagonist for possible use in substance abuse treatment, but it had unpleasant adverse effects that significantly limited its utility (Resnick, Schuyten-Resnick, & Washton, 1979). However, investigators recognized that two subsets of patients could be identified based on their response to cyclazocine. One group refused to take the medication and responded better to agonist treatment. These patients appeared to use opiates as a form of self-medication to relieve symptoms of emotional problems and chronic stress. The group that was more likely to respond positively to cyclazocine did not have the same lev-

els of emotional problems, and opiate use was largely linked to environmental and conditioning factors. The assumption was that more patients in this latter group would agree to take an antagonist if it were better tolerated. These facts, combined with a growing heroin abuse problem, led to a federal government mandate in 1971 to synthesize and develop opioid antagonist compounds that might be more acceptable for use in substance abuse treatment (Julius, 1976). Naltrexone was chosen for this initiative, and the federal government funded the entire medication development effort through the point of approval by the Food and Drug Administration. For the most part, naltrexone was a success: good oral bioavailability, affordable, with few side effects, and very effective in preventing opioid-induced euphoria and physical dependence.

However, naltrexone suffers from one of the same major problems as cyclazocine: patient nonadherence. Most patients will not continue taking the medication though it will reliably prevent development of opioid physical dependence (Fals-Stewart & O'Farrell, 2003). It is much less effective in substance abuse treatment compared to agonist medication. Similar to cyclazocine, naltrexone tends to be more helpful for patients who are less psychologically impaired and more highly socially rehabilitated with other compelling motivation to take the medication and remain abstinent from opioid use (e.g., professionals at risk for losing their professional licensure). In this case, application of basic science knowledge was translated into a rational and efficacious treatment, but with limited effectiveness. The motivation of the patient to take the opioid agonist (e.g., heroin) may overpower the motivation to take the antagonist unless there are significant supports in place to ensure that the patient continues to ingest the antagonist medication (for a contemporary perspective see Sullivan, Comer, & Nunes, 2006).

Unfortunately, the poor effectiveness of naltrexone as a treatment for opioid dependence is not an exception among substance abuse treatments. In addition, though there are many fewer problems with adherence to opioid agonists compared to adherence to antagonists, simply taking the medication usually does not eliminate heroin use. All of these medications are only partially effective in treating chronic opioid dependence and have little, if any, effect on nonopioid abused substances. This is because individual patient characteristics, environment, and social settings contribute greatly to continued drug use. Many patients exhibit co-occurring substance use disorders (e.g., alcohol, cocaine) and other psychiatric and medical disorders that also influence continued drug use. Without a supportive context for the medication treatment, the drug dependence will successfully compete with more prosocial behaviors and the patient will benefit only partially, remaining at risk for many serious problems of continued substance abuse: crime, communicable diseases, social isolation, and demoralization.

BEHAVIORAL TREATMENT TRANSLATIONS

Behavioral approaches to substance abuse treatment are based on a large body of basic experimental research on operant conditioning in animals and humans. *Operant* refers to behaviors of an organism that change (or operate on) the surrounding environment, and operant conditioning is based on the understanding that the consequences of a behavior predict whether the behavior will occur under the same conditions at a future time (Stitzer, Petry, & Silverman, 2006). According to this perspective, substance use disorders are learned behaviors supported by the often powerful and biologically reinforcing effects of drugs of abuse. Many of these behavioral insights were first identified in animal research studies, where a variety of animal species demonstrate that they self-administer many of the same drugs that humans abuse. Human and animal studies also show that manipulating the consequences of behavior can increase or decrease drug self-administration. Therefore, behavioral reinforcers (consequences of a behavior that *increase* the likelihood of that behavior in the future) and punishments (consequences of a behavior that *decrease* the likelihood of that behavior in the future) are logical means to effect behavior change in treatments for substance abuse.

Translating these basic behavioral insights, *contingency management* procedures use behavioral reinforcement to promote a desirable behavior change (e.g., submitting drug-negative urine samples) by use of motivational interventions. These interventions use positive reinforcers (e.g., monetary vouchers), negative reinforcers (e.g., removal of medication time restrictions such that patients can receive methadone dosing at more desirable times), or punishments (e.g., methadone dose taper) in varied and creative ways to motivate drug abstinence or other important treatment goals. Many research studies have demonstrated the utility of contingency management approaches (Stitzer et al., 2006).

Behavioral reinforcers can be conceptualized on a continuum from high specificity to the type of treatment offered (e.g., methadone take home doses in methadone maintenance treatment) to very low specificity to the type of treatment offered (e.g., availability of recreational activities with a family member contingent on following the treatment plan). For example, many studies have shown that patients will reduce drug use if provision of negative urine samples is tied to availability of methadone. These reinforcers include methadone take-home doses such that patients do not have to attend the clinic daily for medication ingestion (Brooner, Kidorf, King, & Stoller, 1998; Calsyn & Saxon, 1999; Chuatape, Silverman, & Stitzer, 1999; Kidorf & Stitzer, 1996), availability of continued methadone treatment services (Calsyn & Saxon, 1987; Kidorf & Stitzer, 1993), and availability of higher daily methadone doses (Stitzer, Bickel, Bigelow, & Liebson, 1986). Use of the inherent reinforcing effects of agonist

medications is a very therapeutic and useful aspect of methadone or buprenorphine treatment that can easily be integrated into the plan of care.

Other behavioral reinforcers that are less specific to the type of treatment can also be effective. Some strategies allow individuals to work and earn money based on negative drug testing. This can occur in the setting of an employee health program, where patients must resolve drug or alcohol use problems to stay employed (Lawental, McLellan, Grissom, Brill, & O'Brien, 1996), but has also been used in specialized programs where access to work on a daily basis is allowed based on drug testing several times per week (Silverman et al., 2002). However, this latter method requires close collaboration with an employer or provision of workplace opportunities, which is unusual in treatment settings. Strategies that use cash or monetary vouchers as reinforcers for drug abstinence are more flexible and less clinic specific because the vouchers can be exchanged for a wide variety of goods and services, and higher reinforcer value is more likely to improve the response to the intervention (Preston, Umbricht, & Epstein, 2000; Silverman, Chutuape, Bigelow, & Stitzer, 1999). However, some criticize the monetary voucher reinforcement approach because of concern that the voucher reinforcement is temporary and that patients will resume drug use after the voucher motivation is removed (Ledgerwood & Petry, 2006; Piotrowski et al., 1999). The cost of the voucher reinforcement procedure is also cited as an impediment to this type of contingency management, though low cost incentives (e.g., approximately $100–$200 per patient over a 12-week period) have been shown to decrease drug use and improve treatment adherence (Peirce et al., 2006; Petry, Martin, & Simcic, 2005). Even so, these drawbacks currently limit the implementation of this approach in the community setting.

Even though highly structured behavioral treatments can be effective in reducing substance use, they are not commonly used as stand-alone treatments in community-based treatment programs because the effects of the treatments don't tend to continue once the behavioral treatment is discontinued. Like medication treatments, they are only partially effective because other psychological, social, and environment factors play key roles in the patient's overall progress. Other, complementary approaches are needed to develop a comprehensive substance abuse treatment model that addresses all areas of rehabilitation.

PSYCHOTHERAPY AND THE PROBLEM OF POOR TREATMENT ADHERENCE

Psychotherapy Treatments for Substance Abuse

Group and individual psychotherapies form the core of most substance abuse treatment interventions. Cognitive-behavioral therapies, motivational

enhancement therapy (MET), psychodynamic and interpersonal therapies, family-couples therapies, group therapies, and Twelve-Steporiented approaches have demonstrated benefits in the treatment of substance use disorder (APA, 2006). Whether these therapies are delivered in individual or group formats, patients learn how to reduce and eventually stop abusing substances by changing maladaptive behaviors, gaining necessary insights, and receiving important emotional support. These therapies also offer the opportunity for therapists to aid the patient in achieving other important goals such as medical care, legal referral, or treatment for other psychiatric problems. All have in common the interpersonal nature of the treatment and the focus on rational behavior change to promote rehabilitation, and they address the social and environmental aspects of the patient in a manner that is different from and complementary to medication or behavioral treatments. Though several studies have examined the differential efficacy of these various treatments (e.g., Project MATCH, 1997), no specific psychotherapy has been shown to be more efficacious overall than any other approach. On the other hand, most treatment experts agree that psychotherapy of some type is an essential component in a comprehensive plan of rehabilitation.

But these treatments are plagued with poor adherence. Careful reading of many studies of psychotherapy for substance abuse reveals fair to poor adherence to scheduled psychosocial counseling sessions, with substantial rates of treatment attrition (Crits-Christoph & Siqueland, 1996). This at least in part explains the lack of positive findings or poor treatment response reported in the studies. For example, adherence to scheduled counseling sessions can be abysmally low in standard methadone maintenance clinics, often falling below 50 percent attendance for minimal levels of counseling service (Ball & Ross, 1991). Two studies on the use of specialized psychotherapies in methadone maintenance patients also highlight the poor rates of attendance to these types of treatment (Rounsaville, Glazer, Wilber, Weissman, & Kleber, 1983; Woody, McLellan, Luborsky, & O'Brien, 1995) as well as substantial rates of attrition (e.g., 46%–62% in the Rounsaville et al., 1983 study). The National Institute on Drug Abuse Collaborative Cocaine Treatment Study (Crits-Christoph, Siqueland, Blaine, Frank, Luborsky, & Onken, et al., 1999) also reported poor attendance to scheduled counseling sessions (<50%) as well as significant treatment attrition (approximately 70% did not complete the six-month treatment episode). Many studies do not report adherence data, so one cannot be certain if the poor outcomes were due to a lack of treatment delivery due to nonadherence rather than a lack of treatment efficacy.

Comparing Treatments for Substance Abuse versus Other Chronic Medical Conditions

Poor adherence is a common and pervasive problem in the treatment of all chronic medical conditions. A recent World Health Organization report identified this treatment parameter as one of the largest problems affecting treatment effectiveness (Sabate, 2003). A reasonable conclusion from this report is that the best way to improve outcome is to ensure that the prescribed amount and intensity of treatment is actually delivered to patients.

Patient adherence to medication treatment regimens is an excellent example of this phenomenon. Adherence problems are common with most prescribed medications including antihypertensives (Burnier, Santschi, Favrat, & Brunner, 2003), antidepressants (Delgado, 2000; Thompson, Peveler, Stephenson, & McKendrick, 2000), asthma medications (Rand & Wise, 1994), diabetes management regimens (Rubin, 2005), as well as medications to aid in substance abuse treatment such as disulfiram (Liebson, Tommasello, & Bigelow, 1978) and naltrexone (Fals-Stewart & O'Farrell, 2003; Volpicelli et al., 1997). Rates of adherence to medication regimens range from 30 percent to 80 percent depending on the medication and the complexity of the medication regimens, with adherence tending to decrease rapidly over time.

Figure 12.1 shows the rates of adherence to scheduled treatment for chronic medical problems in developed and developing countries, and compares them to substance abuse counseling attendance in 12 communities in the United States (Kerr, McGlynn, Adams, Keesey, & Asch, 2004; Sabate, 2003). Even though developed countries are more likely to have efficacious treatments and the patients to have more resources to access these treatments, substance abuse counseling adherence falls between the adherence rates for treatment of chronic medical problems in developing versus developed countries. This fact significantly limits the effectiveness of even the most promising treatment. Furthermore, if one component of a multiple intervention treatment is poorly attended (such as psychotherapy) or not regularly ingested (such as medication) the entire treatment plan is jeopardized. A simple calculation tells us if a group of hypothetical patients is only 50 percent adherent to counseling and only 50 percent adherent to additional medication treatments (generous adherence given the results of many studies), very few patients will receive the most common type of prescribed combined treatment. Essentially, though a two-component treatment intervention is planned, the majority of patients will receive either one or no treatment intervention.

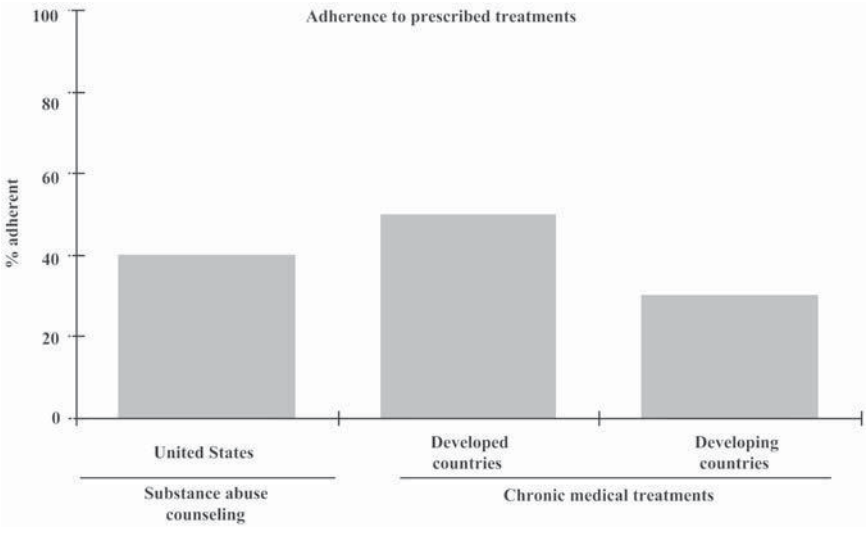

FIGURE 12.1. Adherence to Prescribed Treatments. Rates of adherence to scheduled treatment for chronic medical conditions in developed and developing countries compared to adherence to substance abuse counseling attendance in 12 communities in the United States.

Another perspective is even more concerning. In certain instances, poor adherence to treatment can be detrimental to the patient (Moos, 2005). Perhaps the easiest example to cite involves antiretroviral therapy for HIV infection. Poor adherence can result in development of resistant strains of the virus, shortening the patient's lifespan by reducing availability of effective antiviral agents. In another example, if patients miss antidepressant medication doses they can develop uncomfortable medication discontinuation effects that can be mistaken for worsening depression. Patients may then be treated with higher-than-required doses or needlessly switched to other, potentially more hazardous agents. In psychotherapy treatments, lack of effectiveness may result simply from poor attendance, yet an otherwise efficacious treatment may be abandoned. From a psychological perspective, patients may become demoralized because of the mistaken belief that they are not able to benefit from treatment. From a societal perspective, only low intensity treatments may be offered because higher intensity treatments are no more effective than low intensity treatments. Unfortunately, the primary difficulty may be poor adherence to the prescribed treatment, and few treatments work well if not taken as prescribed.

Therefore, specific efforts to enhance adherence are needed to avoid these types of poor outcomes. We next show how contingency management can help avoid this pitfall in the example of psychotherapy treatments.

Using Contingency Management to Enhance Treatment Adherence

Given the evidence that psychotherapies can be very effective in reducing substance abuse symptoms, organizing systems of care to increase patient attendance to these therapies could be an important strategy to improve treatment effectiveness. Table 12.1 lists several studies that demonstrate how clinic-based behavioral reinforcement positively affects counseling attendance in methadone maintenance treatment. A wide variety of contingencies can be used to reinforce targeted behaviors (in this instance, counseling adherence). These incentives range from methadone take-home doses (Iguchi et al., 1996; Kidorf, Stitzer, Brooner, & Goldberg, 1994), to chances at winning a prize in a lottery drawing (Petry et al., 2005), to vouchers redeemable for goods and services (Jones, Haug, Silverman, Stitzer, & Svikis, 2001), to cash (Svikis, Lee, Haug, & Stitzer, 1997), and to an integrated set of behavioral reinforcers (Brooner et al., 2004). Some of these studies demonstrate dramatic improvements in counseling attendance and are associated with overall improvement in treatment outcome, demonstrating the benefits of integrating contingency management approaches with other forms of treatment.

Community-based behavioral reinforcement can also be very effective in motivating treatment attendance. Perhaps one of the most common types is the drug court model where offenders are mandated to attend treatment services to avoid incarceration. Individuals in these programs can respond as well or better than patients who are not legally mandated to treatment (Kelly, Finney, & Moos, 2005; Lawental et al., 1996; Miller, 1975). Clearly, this approach relies on the threat of punishment to motivate the patient, but a broader approach would utilize positive reinforcement as well. In the community reinforcement approach (CRA; Azrin et al., 1994; Higgins et al., 1993; Hunt & Azrin, 1973; Smith, Meyers, & Delaney, 1998), family members or other individuals in the community who are important to the patient provide contingent reinforcement (e.g., room and board, access to employment, shared recreational activities, etc.) for important treatment goals. The reinforcers used in CRA are limited only by the extent of the patient's integration into his community and the ability of the treatment staff to engage these individuals in the treatment process. Strongly positive findings highlight the concept that relevant environmental and prosocial motivators can be excellent reinforcers of substance abuse treatment and

Table 12.1
Effect of Behavioral Contingencies on Counseling Attendance

Study	Reinforcer	Counseling attendance	
		With reinforcer	Without reinforcer
Kidorf et al., 1994	Methadone take-home dose	75%	7%
Iguchi et al., 1996	Methadone take-home dose	60%	< 1%
Svikis et al., 1997	$10 cash incentive	46%	31%
Jones et al., 2001	Escalating voucher incentive	74%	59%
Brooner et al., 2004	MSC positive and negative contingencies	83%	44%
Petry et al., 2005	Lottery prize drawing	55%	25%

eventual drug abstinence. This approach also taps an important treatment resource often missing in opioid dependence treatment and treatment research: family and community supports for substance abuse treatment and drug abstinence (Kidorf et al., 2005).

Agonist therapy, psychotherapy, and contingency management are all efficacious therapeutic interventions. However, each approach by itself is only partially effective, with many patients continuing to suffer with significant symptoms, and often with rapid return of substance abuse symptoms if the interventions are discontinued. The next step in the evolution of treatment translation utilizes each of the aforementioned approaches in an integrated model of care, expanding on previous research models. In this translation, contingency management is used to reinforce additional aspects of treatment other than just attendance and drug abstinence, including more permanent and personally meaningful behavioral change (e.g., developing friendships with drug-free individuals and acquiring employment). This long-term perspective is a key ingredient in the successful treatment of chronic disorders such as severe opioid dependence.

TRANSLATING EFFICACY TO EFFECTIVENESS: MOTIVATED STEPPED CARE (MSC)

The MSC model combines and thoroughly integrates medication, psychotherapy, and behavioral reinforcement (contingency management) into a

comprehensive treatment service delivery system for severe opioid dependent patients. Using an adaptive, stepped care approach (Murphy, Lynch, Oslin, McKay, & TenHave, 2007; Sobell & Sobell, 2000), MSC addresses the long-term requirements of chronic substance dependent patients in an efficient and cost-effective manner by matching the intensity of the service to the severity of the problem in a dynamic fashion over time using behavioral reinforcement to motivate counseling adherence and treatment plan progress. In this way, only as much treatment as is needed to improve and stabilize functioning is prescribed, and then intensity of care is reduced to minimize intrusiveness and cost. Figure 12.2 is a schematic diagram of the most current MSC model. All patients begin at Step 2, receiving 0.5 to 1.5 hours of weekly drug abuse counseling combined with weekly urine monitoring and individualized methadone maintenance dosing. For those patients who respond well to this step of care for several months (no drug use, steady progress on the treatment plan), a reduction to Step 1 care is appropriate (0.5 hours of counseling monthly and twice monthly urine testing), though patients can attend more counseling if desired.

For patients who do not respond well to Step 2 (i.e., continued drug use, treatment nonadherence), advancement to Step 3 is required. Step 3 patients receive an additional two hours per week of intensified group-based counseling treatment in an effort to curb ongoing drug use and make progress in the plan of rehabilitation. If patients do not respond to Step 3 care within two to four weeks, they are advanced to an intensive outpatient level of care, Step 4. Here patients attend up to nine hours of counseling per week including individual counseling, stress management therapy group, relapse control therapy group, cognitive-behavioral therapy group, coping skills therapy group, and a community support therapy group.

Behavioral reinforcement is active at all steps of care but becomes increasingly important at higher steps, and access to methadone medication treatment is one of the most powerful behavioral reinforcers available in the clinic. For example, not only are patients expected to attend the clinic on all scheduled days for medication, all scheduled counseling must be attended to avoid advancement to higher steps of care. If patients do not attend scheduled counseling sessions, they are subject to increasingly later restrictions on available methadone medication dosing times (however, they can be medicated earlier if they attend a scheduled session). For example, a patient who has an unexcused absence from counseling one day will have his earliest methadone medication dosing time advanced from 12 p. m. to 4 p. m. This time restriction for dosing is then reversed after one week of adherence, but would advance by one hour for each day of missed counseling. Take-home metha-

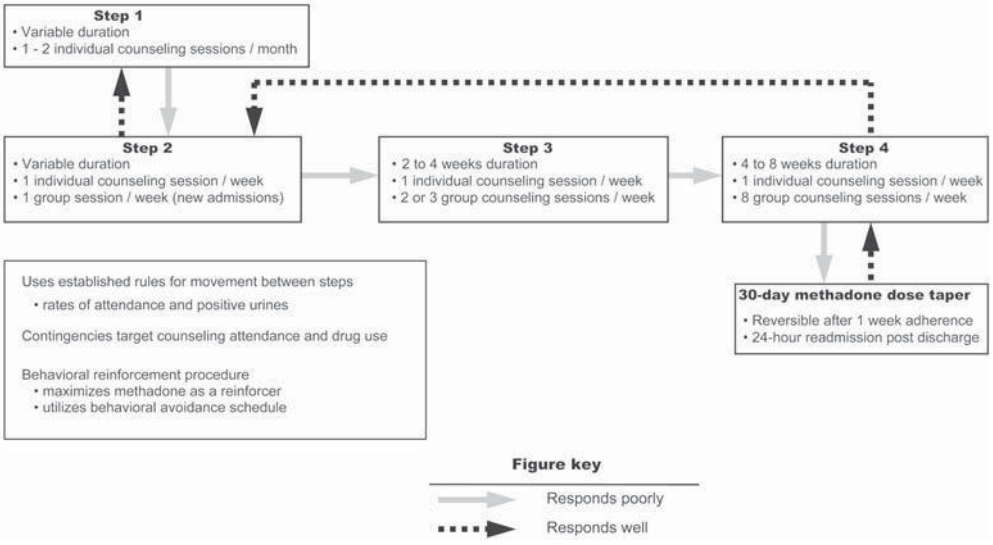

FIGURE 12.2. Motivated Stepped Care. Patients begin at Step 2 and move to Step 3 for two to four weeks after missing any counseling sessions and/or submitting drug-positive urine specimens for two consecutive weeks. They return to Step 2 after attending all scheduled counseling and testing negative for drug use for two consecutive weeks. Patients in Step 2 that attend all scheduled counseling and submit drug-negative urine specimens for six-months may reduce their intensity of care to Step 1. Patients who do not adhere to the schedule of treatment in Step 3 are advanced to Step 4 and must attend all scheduled counseling and provide negative urine specimens for four consecutive weeks within an eight-week period to return to Step 2. If they do not accomplish this goal, they begin a 30-day methadone dose taper in preparation for discharge from the clinic. However, the taper can be stopped by attending all counseling sessions for one week and providing a drug-negative urine specimen. Step 4 patients who complete a methadone dose taper are discharged but are guaranteed rapid readmission, restarting Step 4 with the goal of adherence to the updated treatment plan.

done doses are removed for nonadherence but are returned when adequate adherence to the treatment plan is accomplished. Ultimately, if patients do not adhere to the plan of care, they are subject to a methadone taper in preparation for discharge and referral to alternate treatment. However, this taper can be reversed after one week of adherence to the entire treatment plan, and patients begin Step 4 again with the aim of four weeks of consecutive adherence. Once this is accomplished, the patient is transferred to Step 2 with continuing care aimed at solidifying gains accomplished during the Step 4 episode.

Behavioral Contingencies Motivate Counseling Attendance and Reduced Drug Use

Brooner et al. (2004) reported on a trial of MSC versus a noncontingent, standard methadone maintenance treatment control. The MSC condition was basically conducted as described in the preceding section (we made some minor changes in groups and step movement over the years), while, as seen in Figure 12.3,

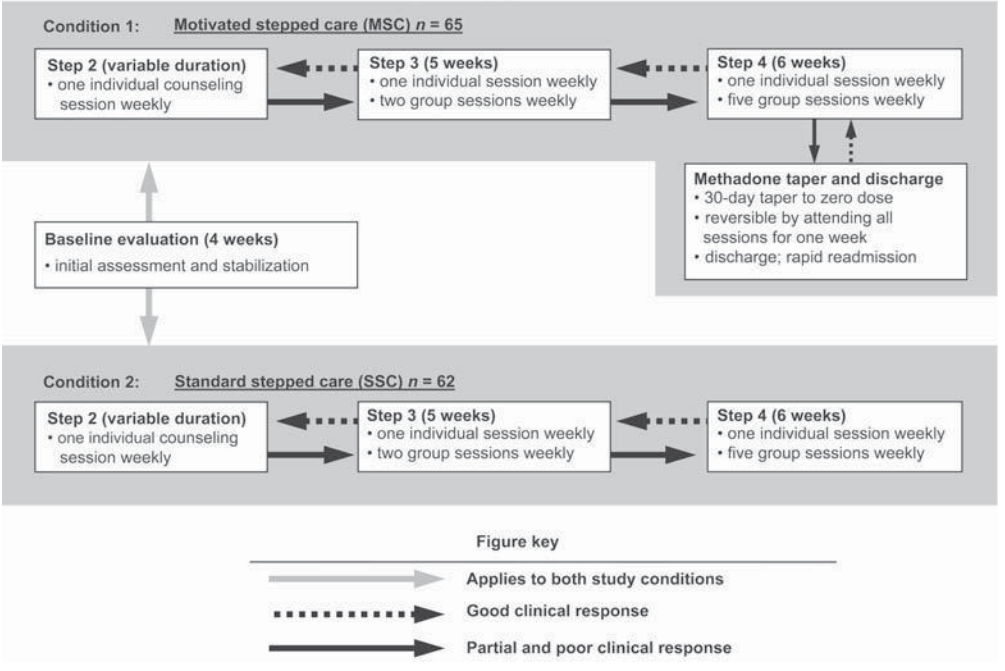

FIGURE 12.3. Randomized Treatment Conditions. MSC and SSC subjects begin randomized treatment in Step 2 following the baseline evaluation (Brooner et al., 2004). All subjects move to Step 3 after submitting drug-positive urine specimens or missing counseling sessions in any two out of three weeks. They return to Step 2 after attending all counseling and testing negative for drug use in weeks four and five. Subjects in Step 3 that miss counseling sessions or submit drug-positive urine samples in weeks four or five move to Step 4. They return to Step 3 after attending all counseling and testing negative for drug use in weeks five and six. Subjects in the MSC condition that demonstrate a partial and poor response to Step 4 (i.e., are nonadherent to counseling and submit drug-positive urine specimens in week five and six) begin a 30-day methadone dose taper in preparation for discharge. However, the taper is reversible and subjects move back to Step 3 after producing two consecutive weeks of counseling attendance and drug-negative urine specimens. Step 4 patients who complete a methadone dose taper are discharged but are guaranteed rapid readmission. Subjects in the SSC condition stay in Step 4 until they satisfy criteria to reduce intensity to Step 3, or until they satisfy the 90-day therapeutic rescue procedure that transfers them to the MSC condition.

patients in the standard stepped care (SSC) condition were encouraged to attend the same counseling sessions, but no contingencies were placed on the methadone medication treatment if they did not adhere to the treatment plan. During the first 90 days of the study, the MSC group attended significantly more counseling sessions (MSC 83% versus SSC 44%, $p < .001$). Figure 12.4 illustrates how behavioral reinforcement in the MSC model shifted the counseling attendance rates dramatically over the study period, with many more patients attending more than 50 percent of scheduled counseling in the MSC versus SSC condition. Not surprisingly, there were nonsignificant differences in overall drug use (including opioids, cocaine, alcohol, and benzodiazepines) over the first 90 days because there was limited time for the MSC patients to be exposed to the higher intensities of care (MSC 49% drug-positive urine specimens versus SSC 54%, $p = ns$).

At the end of the first 90 days of the study, patients assigned to the SSC condition that were doing poorly in treatment were therapeutically transferred to the routine MSC condition. In this subset of patients, significant changes in counseling attendance *and* urine drug tests were seen over the subsequent 90 day study period (see Figures 12.5 and 12.6). In these figures, the first 90 days of treatment (SSC) is compared to the second 90-day period (MSC).

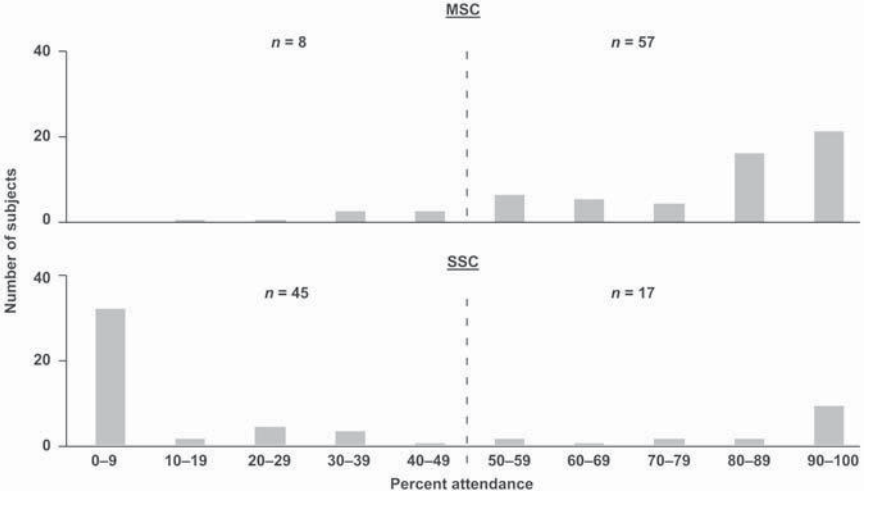

FIGURE 12.4. Group Comparisons of Counseling Attendance. Behavioral reinforcement in the MSC condition shifted the counseling attendance rates dramatically over the 90-day study period, with many more patients attending more than 50 percent of scheduled counseling in the MSC versus SSC condition.

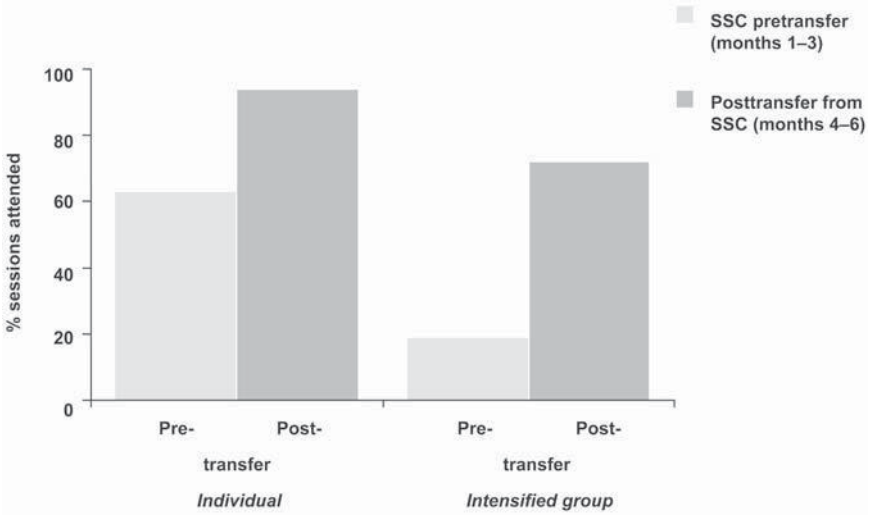

FIGURE 12.5. Within-Subject Crossover Comparison Counseling Attendance. At the end of the first 90 days of the study, patients assigned to the SSC condition that were doing poorly in treatment were therapeutically transferred to the routine MSC condition. In these figures, the first 90 days of treatment (SSC) is compared to the second 90-day period (MSC). Significant changes in rates of counseling attendance were seen over the subsequent 90-day study period (Individual sessions: pretransfer = 63%, SD = .35 vs. posttransfer = 94%, SD = .16; $t(35) = 5.48$, $p < .001$. Intensified group sessions: pretransfer = 19%, SD = .25 vs. posttransfer = 72%, SD = .27, $t(33) = 8.19$, $p < .001$.).

Marked increases in counseling attendance were correlated with significant reductions in drug use after behavioral reinforcement was added to the SSC condition. Another important finding in this study was that six-month retention was good in the MSC condition (70%) and comparable to the SSC condition and to six-month retention in other community programs even though this treatment was significantly more demanding than standard methadone maintenance treatment.

In a separate study, Brooner et al. (2007) used a four group design to compare MSC with and without voucher reinforcement for drug abstinence with a control, noncontingent counseling condition, also with and without voucher reinforcement for drug abstinence. Again, the findings were remarkably positive in this six-month study. Use of MSC plus voucher reinforcement demon-

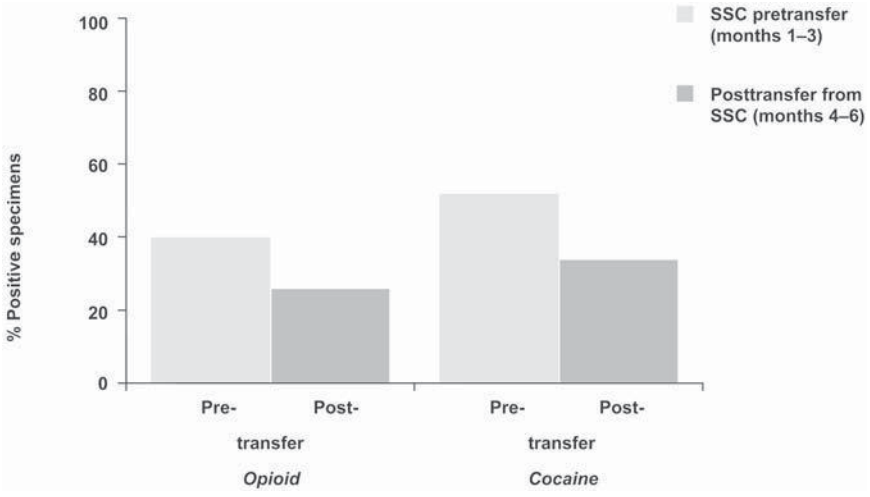

FIGURE 12.6. Within-Subject Crossover Comparison Urinalysis Results. At the end of the first 90 days of the study, patients assigned to the SSC condition that were doing poorly in treatment were therapeutically transferred to the routine, MSC condition. In these figures, the first 90 days of treatment (SSC) is compared to the second 90-day period (MSC). Significant changes in rates of drug use were seen over the subsequent 90-day study period (Opioid use: pretransfer = 40%, *SD* = .35 vs. posttransfer = 26%, *SD* = .29; $t(35) = 3.65$, $p = .001$. Cocaine use: pretransfer = 52%, *SD* = .36 vs. posttransfer = 40%, *SD* = .34; $t(35) = 2.54$, $p = .016$).

strated the best drug use outcomes (60% drug-negative urine specimens), the MSC-only and voucher-only conditions were next best (49% and 50% drug-negative urine specimens, respectively), and the noncontingent condition was significantly worse than all the contingent conditions (29% drug-negative urine specimens). Counseling attendance was excellent in the contingent counseling conditions. This study also showed that voucher-based positive reinforcement was associated with improved six-month retention versus the nonvoucher experimental conditions (74% versus 59%, respectively). Similar to the previous study of MSC (Brooner et al., 2004), MSC demonstrated powerful treatment effects.

MSC Initiates and Reinforces a Wide Spectrum of Prorehabilitation Behavior Change

Behavioral reinforcement with methadone in MSC is associated with improved outcomes across several important substance abuse treatment

goals: employment, social support, as well as the primary goals of adherence to scheduled counseling and accompanying reductions in substance use. Table 12.2 shows results from several studies utilizing the MSC treatment model. The first two studies in Table 12.2 were discussed earlier in this chapter. Given these results, it is no surprise that patients with persistent marijuana use problems also benefited from behavioral reinforcement (Kidorf, Neufeld, King, Clark, & Brooner, 2007). In this study, MSC acted as an avoidance schedule for patients with lower frequency marijuana use (i.e., they stopped to avoid

Table 12.2
Motivated Stepped Care Studies

Article	Targeted behaviors	Findings
Brooner et al., 2004 "Behavioral Contingencies Improve Counseling Attendance in an Adaptive Treatment Model"	Counseling attendance; drug use	MSC associated with increased counseling attendance and decreased drug use
Brooner et al., 2007 "Comparing Adaptive Stepped Care and Monetary Voucher Interventions in the Treatment of Opioid Dependent Outpatients"	Drug use; counseling attendance	MSC and voucher reinforcement associated with decreased drug use; MSC also associated with increased counseling attendance
Kidorf et al., 2007 "A Stepped Care Approach for Reducing Cannabis Use in Opioid Dependent Outpatients"	Marijuana use	MSC associated with decreased marijuana use in chronic marijuana users
King et al., 1999 "ADHD and Treatment Outcome in Opioid Abusers Entering Treatment"	Drug use; counseling attendance	No difference in treatment outcomes when patients with history of ADHD vs. no ADHD treated with MSC
King et al., 2006 "A 12-Month Controlled Trial of Methadone Medical Maintenance Integrated into an Adaptive Treatment Model"	Counseling attendance; drug use; responsible handling of methadone take-home doses	Highly stable patients respond as well to medical maintenance as they did to standard care using MSC
Kidorf et al., 1998 "Increasing Employment of Methadone Maintenance Outpatients: An Intensive Behavioral Intervention"	Employment acquisition	Patients unemployed > 1 year required to obtain 20+ hrs/wk verified employment; 75% started work within 60 days

(continued)

Table 12.2 (continued)

Article	Targeted behaviors	Findings
Kidorf et al., 2004 "Combining Stepped-Care Approaches with Behavioral Reinforcement to Motivate Employment in Opioid-Dependent Outpatients"	Employment acquisition	93% of able-bodied patients in methadone maintenance clinic currently working 15+ hrs/wk. Work associated with improved substance abuse treatment outcomes
Kidorf et al., 1997 "Motivating Methadone Patients to Include Drug-Free Significant Others in Treatment: A Behavioral Intervention"	Acquire drug-free significant other (community support person) to attend conjoint weekly therapy group and participate in weekly social activity	85% of 75 patients obtained significant other (SO) and patients who identified SO attended 77% of scheduled sessions
Kidorf et al., 2005 "Involving Significant Others in the Care of Opioid-Dependent Patients Receiving Methadone"	Acquire drug-free significant other (community support person) to attend conjoint weekly therapy group and participate in weekly social activity	93% of 59 patients obtained SO and 78% achieved at least 4 weeks drug abstinence during the intervention

more intensified counseling treatment) and as a platform for delivering higher intensity treatment for patients who were dependent on marijuana. Examining a different population, the strong behavioral reinforcement element of MSC may have been responsible for the comparable and very positive outcomes when comparing patients with a history of attention deficit hyperactivity disorder (ADHD) versus no history of these behavioral problems (King, Brooner, Kidorf, & Mirsky, 1999). Rates of intensified treatment as well as treatment retention were comparable between the ADHD and non-ADHD groups. The fifth study, examining methadone medical maintenance, demonstrated how the MSC approach serves as an excellent platform to expand the range of treatment intensity for highly stable patients who desire long-term substance abuse treatment (King et al., 2006). This expanded service delivery system matched appropriate levels of substance abuse monitoring and treatment with the needs

of highly stable patients who occasionally experienced increases in substance use disorder symptoms. Finally, MSC behavioral reinforcement motivated unemployed patients to seek and begin employment (Kidorf, Hollander, King, & Brooner, 1998; Kidorf, Neufeld, & Brooner, 2004) and socially alienated and isolated individuals to form initial community supports for drug abstinence (Kidorf, Brooner, & King, 1997; Kidorf et al., 2005). Targeting specific behaviors with behavioral reinforcement in an integrated care model initiates important behavioral changes and, with the help of the community support intervention, begins to integrate the patient into the larger society that then provides additional reinforcement for drug abstinence. This may be most apparent when key community reinforcers that are provided by family and other social supports (e.g., employers and spiritual organizations) move the patient's rehabilitation out of the clinic and into his emerging life as a healthier and more productive person. It is apparent from this range of studies that the MSC model can motivate chronic opioid dependent patients to attain many important short-term and long-term treatment goals.

RESEARCH IMPLICATIONS AND OTHER POTENTIAL APPLICATIONS

These findings have important research implications, especially for studies involving clinical populations. Given the importance of adherence behavior in successful treatment outcomes, this variable should be assessed not only as a primary overall outcome measure, but also for any discrete elements of the treatment (e.g., medication versus psychotherapy adherence). In this way, adherence to separate treatment elements can be evaluated, and considerations for types of motivational interventions can be made for elements that exhibit poor adherence. For maximum treatment effectiveness, service delivery systems that maximize adherence to the plan of care are essential. As in the case of naltrexone treatment for opioid dependence, this step in the process of treatment innovation can be just as important as the original findings of efficacy.

This general model of care, including the use of behavioral reinforcement that is available in the clinic or in the community, could be transposed to many different clinical situations with appropriate modifications. The parallel is perhaps most apparent for other chronic disorders that have significant behavioral aspects that influence the onset or course of the illness. For many patients, these disorders are primarily caused and exacerbated by lifestyle behaviors. For example, behavioral reinforcement could be used in a structured treatment program to motivate desirable eating and exercise behaviors in patients with diabetes or hypertension. Starting with simple behavioral prescriptions that

are reinforced and supported by family involvement, many patients would not only take medication more regularly but also improve their own health maintenance. Clearly, this approach would demand more time and effort on the part of care providers, but tackling disorders that are caused by continuing detrimental lifestyle choices with medications and brief, occasional, office–based individual counseling are unlikely to fully treat these problems because these approaches are only partially effective. Integrating contingency management into our routine treatments could have dramatic positive effects, helping to optimize the effectiveness of our present and future medical treatments. To do this, we need to shift our focus from just the primary treatments to include and emphasize new delivery systems that specifically address patient behaviors with the goal of increasing treatment adherence. Translating efficacious research-based treatments into more effective, community-based clinical services could have an enormous impact on long-term patient health.

CONCLUSION

Scientific understanding of the molecular and genetic basis of neurological and psychiatric disorders is progressing at a rapid pace, and along with these new understandings comes the promise of powerful new therapies. However, one important and overarching aspect of therapeutics has lagged behind in our scientific advancements: patient behavior as the means by which these scientific advancements can be translated into meaningful changes in the lives of patients. Patient behavior can support and enhance the new molecular and genetic-based therapies, or it can drastically limit their effectiveness. These advancements are on our doorstep but will ultimately disappoint when patients continue to be poorly adherent to prescribed treatment regimens. One can easily appreciate how the promises of new genetic discoveries and medication therapies will be substantially reduced by treatment nonadherence. As simple a task as taking a medication once daily is frequently not accomplished by patients.

Though adherence problems are common in all areas of medical treatment, they are even more pernicious in psychiatric settings, where patients are more likely to have behavioral disorders that either interfere with the treatment process or are themselves the focus of treatment. Therefore, the best opportunity to transform efficacious treatments into more effective treatments is to improve adherence. The extensive literature on behavioral reinforcement clearly demonstrates how this principle can be used to greatly enhance treatment outcomes, yet these approaches are rarely used in routine treatment settings. To get the most out of powerful and sophisticated new treatments, we must use service delivery systems of equal power to ensure that the treatment is received. Without significant improvements in this aspect of care, we will develop mounting num-

bers of treatments found to be efficacious in well-controlled studies, but with continued problems of limited effectiveness in real-world settings.

In this chapter we examined how translational insights were used in the development of medication and behavioral therapies for opioid dependence. However, these approaches by themselves are only partially effective. Psychotherapy is another important treatment approach but is plagued with the problem of poor adherence. Using all three treatment strategies in an integrated fashion is most effective. However, successful implementation depends on all treatment components working synergistically, because if patients do not adequately adhere to one component, then the entire treatment model is jeopardized. The final strategy for effective treatment integration expands the use of behavioral reinforcement by directly addressing adherence behavior in the overall treatment plan.

The MSC model of treatment for severe opioid dependent patients was developed specifically using these key understandings. MSC combines and translates efficacious interventions into a more effective, integrated model of care that recognizes and addresses the very common problems associated with poor treatment adherence. Contingent access to methadone motivates the patient to attend counseling sessions. It can also motivate other prorecovery target behaviors such as job seeking and involving community supports in treatment, thus allowing systematic exposure to other elements of treatment that might not otherwise occur without the behavioral reinforcement intervention. As expected, patients demonstrate remarkably improved outcomes when adherence is improved. The key translational insights of opioid agonist therapy and behavioral reinforcement enable the treatment providers to engage the patient more completely in the rehabilitation process and obtain treatment outcomes that exceed any of the results of the individual treatments used alone. MSC is the next developmental advance in the translational application of efficacious interventions for substance use disorder, and it also serves as a potential model for approaching other chronic medical disorders that have significant behavioral components.

REFERENCES

American Psychiatric Association. (2006). Practice guideline for the treatment of patients with substance use disorders, second edition. *American Journal of Psychiatry, 163*(Suppl.), 5.

Azrin, N. H., McMahon, P. T., Donohue, B., Besalel, V. A., Lapinski, K. J., Kogan, E. S., et al. (1994). Behavior therapy for drug abuse: A controlled treatment outcome study. *Behaviour Research and Therapy, 32*(8), 857–866.

Ball, J. C., & Ross, A. (1991). *The effectiveness of methadone maintenance treatment*. New York: Springer-Verlag.

Bigelow, G. E., Brooner, R. K., & Silverman, K. (1998). Competing motivations: Drug reinforcement vs. non-drug reinforcement. *Journal of Psychopharmacology, 123,* 8–14.

Brooner, R. K., Kidorf, M. S., King, V. L., & Stoller, K. (1998). Preliminary evidence of good treatment response in antisocial drug abusers. *Drug and Alcohol Dependence, 49,* 249–260.

Brooner, R. K., Kidorf, M., King, V. L., Stoller, K. B., Neufeld, K. J., & Kolodner, K. (2007). Comparing adaptive stepped care and monetary voucher interventions in the treatment of opioid dependent outpatients. *Drug and Alcohol Dependence, 88S,* S14–S23.

Brooner, R. K., Kidorf, M., King, V. L., Stoller, K. B., Peirce, J. M., Bigelow, G. E., et al. (2004). Behavioral contingencies improve counseling attendance in an adaptive treatment model. *Journal of Substance Abuse Treatment, 27,* 223–232.

Burnier, M., Santschi, V., Favrat, B., & Brunner, H. R. (2003). Monitoring compliance in resistant hypertension: An important step in patient management. *Journal of Hypertension, 21* (Suppl. 2), S37–S42.

Calsyn, D. A., & Saxon, A. J. (1987). A system for uniform application of contingencies for illicit drug use. *Journal of Substance Abuse Treatment, 4,* 41–47.

Calsyn, D. A., & Saxon, A. J. (1999). An innovative approach to reducing cannabis use in a subset of methadone maintenance clients. *Drug and Alcohol Dependence, 53,* 167–169.

Center for Substance Abuse Treatment. (2005). *Medication-assisted treatment for opioid addiction in opioid treatment programs.* Treatment Improvement Protocol (TIP) Series 43. DHHS Publication no. (SMA) 05-4048. Rockville, MD: Substance Abuse and Mental Health Services Administration.

Chuatape, M. A., Silverman, K., & Stitzer, M. L. (1999). Use of methadone take-home contingencies with persistent opiate and cocaine abusers. *Journal of Substance Abuse Treatment, 16,* 23–30.

Crits-Christoph, P. , & Siqueland, L. (1996). Psychosocial treatment for drug abuse. *Archives of General Psychiatry, 53,* 749–756.

Crits-Christoph, P. , Siqueland, L., Blaine, J., Frank, A., Luborsky, L., Onken, L. S., et al. (1999). Psychosocial treatments for cocaine dependence. *Archives of General Psychiatry, 56,* 493–502.

Delgado, P. L. (2000). Approaches to the enhancement of patient adherence to antidepressant medication treatment. *Journal of Clinical Psychiatry, 61*(Suppl. 2), 6–9.

Dole, V. P., Nyswander, M. E., & Kreek, M. J. (1966). Narcotic blockade. *Archives of Internal Medicine, 118,* 304–309.

Fals-Stewart, W., & O'Farrell, T. J. (2003). Behavioral family counseling and naltrexone compliance for male opioid-dependent patients. *Journal of Consulting and Clinical Psychology, 71*(3), 432–442.

Higgins, S. T., Budney, A. J., Bickel, W. K., Hughes, J. R., Foeg, F., & Badger, G. (1993). Achieving cocaine abstinence with a behavioral approach. *American Journal of Psychiatry, 150,* 763–769.

Hunt, G. M., & Azrin, N. H. (1973). A community reinforcement approach to alcoholism. *Behaviour Research and Therapy, 11,* 91–104.

Iguchi, M. Y., Lamb, R. J., Belding, M. A., Platt, J. J., Husband, S. D., & Morral, A. R. (1996). Contingent reinforcement of group participation versus abstinence in a methadone maintenance program. *Experimental and Clinical Psychopharmacology, 4*(3), 315–321.

Jones, H. E., Haug, N., Silverman, K., Stitzer, M., & Svikis, D. (2001). The effectiveness of incentives in enhancing treatment attendance and drug abstinence in methadone-maintained pregnant women. *Drug and Alcohol Dependence, 61,* 297–306.

Julius, D. (1976). NIDAS's naltrexone research program. In D. Julius & p. Renault (Eds.), *NIDA research monograph series 9: Narcotic antagonists: Naltrexone progress report* (pp. 5–11). Rockville, MD: U.S. Dept. of Health, Education, and Welfare, Alcohol, Drug Abuse and Mental Health Administration.

Kelly, J. F., Finney, J. W., & Moos, R. (2005). Substance use disorder patients who are mandated to treatment: Characteristics, treatment process, and 1- and 5-year outcomes. *Journal of Substance Abuse Treatment, 28,* 213–223.

Kerr, E. A., McGlynn, E. A., Adams, J., Keesey, J., & Asch, S. M. (2004). Profiling the quality of care in twelve communities: Results from the CQI study. *Health Affairs, 23,* 247–256.

Kidorf, M., Brooner, R. K., & King, V. L. (1997). Motivating methadone patients to include drug-free significant others in treatment: A behavioral intervention. *Journal of Substance Abuse Treatment, 14,* 23–28.

Kidorf, M., Hollander, J. R., King, V. L., & Brooner, R. K. (1998). Increasing employment of methadone maintenance outpatients: An intensive behavioral intervention. *Drug and Alcohol Dependence, 50,* 73–80.

Kidorf, M. S., King, V. L., Neufeld, K., Stoller, K. B., Peirce, J., & Brooner, R. K. (2005). Involving significant others in the care of opioid-dependent patients receiving methadone. *Journal of Substance Abuse Treatment, 29,* 19–27.

Kidorf, M. S., Neufeld, K., & Brooner, R. K. (2004). Combining stepped-care approaches with behavioral reinforcement to motivate employment in opioid-dependent outpatients. *Substance Use and Misuse, 39,* 2215–2238.

Kidorf, M. S., Neufeld, K. J., King, V. L., Clark, M., & Brooner, R. K. (2007). A stepped care approach for reducing cannabis use in opioid dependent outpatients. *Journal of Substance Abuse Treatment, 32,* 341–347.

Kidorf, M. S., & Stitzer, M. L. (1993). Contingent access to methadone maintenance treatment: Effects on cocaine use of mixed opiate-cocaine abusers. *Experimental and Clinical Psychopharmacology, 1,* 200–206.

Kidorf, M. S., & Stitzer, M. L. (1996). Contingent use of take-homes and split dosing to reduce illicit drug use of methadone patients. *Behavior Therapy, 27,* 41–51.

Kidorf, M. S., Stitzer, M. L., Brooner, R. K., & Goldberg, J. (1994). Contingent methadone take-home doses reinforce adjunct therapy attendance of methadone maintenance patients. *Drug and Alcohol Dependence, 36,* 221–226.

King, V. L., Brooner, R. K., Kidorf, M. S., & Mirsky, A. F. (1999). ADHD and treatment outcome in opioid abusers entering treatment. *Journal of Nervous and Mental Disease 187,* 487–495.

King, V. L., Stoller, K. B., Kidorf, M. S., Kolodner, K., Schwartz, R., & Brooner, R. K. (2006). A 12-month controlled trial of methadone medical maintenance integrated into an adaptive treatment model. *Journal of Substance Abuse Treatment, 31,* 385–393.

Lawental, E., McLellan, A. T., Grissom, G. R., Brill, p. , & O'Brien, C. (1996). Coerced treatment for substance abuse problems detected through workplace urine surveillance: Is it effective? *Journal of Substance Abuse, 8*(1), 115–128.

Ledgerwood, D. M., & Petry, N. M. (2006). Does contingency management affect motivation to change substance use? *Drug and Alcohol Dependence, 83,* 65–72.

Liebson, I. A., Tommasello, A., & Bigelow, G. E. (1978). A behavioral treatment of alcoholic methadone patients. *Annals of Internal Medicine, 89,* 342–344.

Mattick, R. P., Breen, C., Kimber, J., & Davoli, M. (2003). *Methadone maintenance therapy versus no opioid replacement therapy for opioid dependence (Cochrane Review).* In: The Cochrane Library, Issue 1 Oxford: Update Software.

McHugh, p. R., & Slavney, P. R. (1998). *The perspectives of psychiatry* (2nd ed.). Baltimore, MD: The Johns Hopkins University Press.

Miller, p. M. (1975). A behavioral intervention program for chronic public drunkenness offenders. *Archives of General Psychiatry, 32,* 915–918.

Moos, R. H. (2005). Iatrogenic effects of psychosocial interventions for substance use disorders: Prevalence, predictors, prevention. *Addiction, 100,* 595–604.

Murphy, S. A., Lynch, K. G., Oslin, D., McKay, J. R., TenHave, T. (2007). Developing adaptive treatment strategies in substance abuse research. *Drug and Alcohol Dependence, 88* (Suppl. 2), S24-30.

Peirce J. M., Petry N. M., Stitzer M. L., Blaine J., Kellogg S., Satterfield F., et al. (2006). Effects of lower-cost incentives on stimulant abstinence in methadone maintenance treatment: A National Drug Abuse Treatment Clinical Trials Network study. *Archives of General Psychiatry, 63,* 201–208.

Petry, N. M., Martin, B., & Simcic, F. (2005). Prize reinforcement contingency management for cocaine dependence: Integration with group therapy in a methadone clinic. *Journal of Consulting and Clinical Psychology, 73,* 354–359.

Piotrowski, N. A., Tusel, D. J., Sees, K. L., Reilly, p. M., Banys, p. , Meek, p. , et al. (1999). Contingency contracting with monetary reinforcers for abstinence from multiple drugs in a methadone program. *Experimental and Clinical Psychopharmacology, 7,* 399–411.

Preston, K. L., Umbricht, A., & Epstein, D. H. (2000). Methadone dose increase and abstinence reinforcement for treatment of continued heroin use during methadone maintenance. *Archives of General Psychiatry, 57,* 395–404.

Project MATCH Research Group. (1997). Matching alcoholism treatments to client heterogeneity: Project MATCH posttreatment drinking outcomes. *Journal of Studies on Alcohol, 58,* 7–29.

Rand, C. S., & Wise, R. A. (1994). Measuring adherence to asthma medication regimens. *American Journal of Respiratory and Critical Care Medicine, 149,* S69–S76.

Resnick, R. B., Schuyten-Resnick, E., & Washton, A. M. (1979). Narcotic antagonists in the treatment of opioid dependence: Review and commentary. *Comprehensive Psychiatry, 20,* 116–125.

Rounsaville, B. J., Glazer, W., Wilber, C. H., Weissman, M. M., & Kleber, H. D. (1983). Short-term interpersonal psychotherapy in methadone-maintained opiate addicts. *Archives of General Psychiatry, 40,* 629–636.

Rubin, R. R. (2005). Adherence to pharmacologic therapy in patients with type 2 diabetes mellitus. *American Journal of Medicine, 118,* 27S–34S.

Sabate, E. (2003). *Adherence to long-term therapies: Evidence for action.* World Health Organization, Geneva, Switzerland.

Silverman, K., Chutuape, M. A., Bigelow, G. E., & Stitzer, M. L. (1999). Voucher-based reinforcement of cocaine abstinence in treatment-resistant methadone patients: Effects of reinforcement magnitude. *Psychopharmacology, 146,* 128–138.

Silverman, K., Svikis, D., Wong, C. J., Hampton, J., Stitzer, M. L., & Bigelow, G. E. (2002). A reinforcement-based therapeutic workplace for the treatment of drug abuse: Three-year abstinence outcomes. *Experimental and Clinical Psychopharmacology, 10,* 228–240.

Smith, J. E., Meyers, R. J., & Delaney, H. D. (1998). The community reinforcement approach with homeless alcohol-dependent individuals. *Journal of Consulting and Clinical Psychology, 66,* 541–548.

Sobell, M. B., & Sobell, L. C. (2000). Stepped care as a heuristic approach to the treatment of alcohol problems. *Journal of Consulting and Clinical Psychology, 68,* 573–579.

Stitzer, M. L., Bickel, W. K., Bigelow, G. E., & Liebson, I. A. (1986). Effect of methadone dose contingencies on urinalysis test results of polydrug-abusing methadone-maintenance patients. *Drug and Alcohol Dependence, 18,* 341–348.

Stitzer, M. L., Petry, N., & Silverman, K. (2006). Contingency management therapies. In E. C. Strain & M. L. Stitzer (Eds.), *The treatment of opioid dependence* (pp. 151–177). Baltimore, MD: The Johns Hopkins University Press.

Stoller, K. B., & Bigelow, G. E. (2006). Introduction and historical overview. In E. C. Strain & M. L. Stitzer (Eds.), *The treatment of opioid dependence* (pp. 3–17). Baltimore, MD: The Johns Hopkins University Press.

Sullivan, M. A., Comer, S. D., & Nunes, E. V. (2006). Pharmacology and clinical use of naltrexone. In E. C. Strain & M. L. Stitzer (Eds.), *The treatment of opioid dependence* (pp. 295–320). Baltimore, MD: The Johns Hopkins University Press.

Svikis, D., Lee, J. H., Haug, N., & Stitzer, M. L. (1997). Attendance incentives for outpatient treatment: Effects in methadone-and nonmethadone-maintained pregnant drug dependent women. *Drug and Alcohol Dependence, 48,* 33–41.

Thompson, C., Peveler, R. C., Stephenson, D., & McKendrick, J. (2000). Compliance with antidepressant medication in the treatment of major depressive disorder in

primary care: A randomized comparison of fluoxetine and a tricyclic antidepressant. *American Journal of Psychiatry, 157,* 338–343.

Volpicelli, J. R., Rhines, K. C., Rhines, J. S., Volpicelli, L. A., Alterman, A. I., & O'Brien, C. P. (1997). Naltrexone and alcohol dependence. *Archives of General Psychiatry, 54,* 737–742.

Woody, G. E., McLellan, A.T., Luborsky, L., & O'Brien, C. P. (1995). Psychotherapy in community methadone programs: A validation study. *American Journal of Psychiatry, 152,* 1302–1308.

Woolf, S. H. (2008). The meaning of translational research and why it matters. *Journal of the American Medical Association, 299,* 211–213.

Chapter **13**

Shamanistic Harm-Reduction Practices[1]

Michael Winkelman, MPH, PhD

Drug use and addiction are obviously driven by biological dynamics associated with the alteration of consciousness, but conventional addiction treatment approaches have, in general, not directly engaged this modification of consciousness as a normal and intrinsic part of human nature and healing processes. Nonetheless, a number of investigators have noted relationships among substance dependence, altered states of consciousness (ASC), ritual, and spirituality (e.g., Heggenhougen, 1997; Jilek, 1994; McPeake, Kennedy, & Gordon, 1991; Metzner, 1994; Miller, 1998; Rioux, 1996; Smith, 2000; Winkelman, 2001a, 2003). Siegal (1990) and Winkelman (2000) provide models that illustrate how addictions reflect an innate drive to seek ASC. McPeake et al. (1991) suggest that addictions be viewed in the context of a universal human desire to enter ASC, and they propose that natural modifications of consciousness be used to reduce recidivism. Mandell (1980) conceptualized this common basis for addiction and ASC in a psychobiological model of "transcendent states" in which both endogenous and exogenous mechanisms of altering consciousness share a common biological substrate in the serotonergic system. Commonalities in addictive and spiritual ASC reflect an underlying biologically based integrative mode of consciousness (Winkelman, 2000). Metzner (1994), however, notes that addictions constitute a contracted state of consciousness, in contrast to the expansive states of consciousness of mysticism.

The biological basis for ASC and our addictive tendencies is manifested as a universal of human societies—shamanistic healers who lead community ritual practices for achieving spiritual experiences, ASC, and healing. These

practices originated in the shaman, who leads dramatic all-night rituals involving drumming, singing, clapping, dancing, myth-telling, and visionary experiences. Shamanism involved the most central communal activity found worldwide in hunter-gatherer and other technologically simple societies. While shamanic practices changed as a function of sociocultural evolution, the same biological potentials underlying shamanism remain in more complex societies, reflecting the persistence of ASC-based healing practices. Features of the original forms of shamanism persisted across sociocultural evolution (e.g., the development of agriculture, political integration) in communal use of ASC in healing rituals (Winkelman, 1992, 2000). The innate psychology involved in communal ritual healing processes evolved to meet psychobiological needs for personal and social integration and provided experiences of unity and transcendence. While shamanistic practices evolved to meet humans' psychobiological needs (Winkelman & Baker, 2008), such practices are virtually absent from the mainstream spiritual practices of contemporary urban societies, where instead epidemics of drug addiction rage.

Shamanistic practices are extensions of a genetically based primate ritual system that evolved for the coordination of the individual with society and the production of psychological integration. Shamanism's roots are found in primate social bonding rituals that were elaborated across human evolution to enhance group unification, psychosocial bonding, and therapeutic responses (Winkelman, 2004a, 2008). Shamanic processes engage some of our innate cognitive modules, integrating these specialized systems to overcome a fragmented consciousness and contributing to the evolution of modern symbolic cognition (Winkelman, 2002). These ancient spiritual practices constitute natural healing processes, an evolved psychology providing mechanisms for integration of consciousness and emotional well-being. A shamanic approach to harm reduction recognizes that humans' innate drive to alter consciousness underlies both addictive behaviors and ritual practices for inducing spiritual transcendence. Shamanic harm-reduction approaches provide ritual methods for inducing safe ASC experiences, using them to integrate the psyche in healing processes.

ALTERED STATES OF CONSCIOUSNESS THERAPY

McPeake et al. (1991) suggested that relapse prevention and recovery could be enhanced by teaching recovering individuals to experience nonsubstance-induced ASC that can help instill a positive sense of self and provide alternatives to the drug-induced state. Shamanic ASC can meet this drive to alter consciousness in constructive ways, providing a prevention strategy, adjuncts

to primary treatment modalities, and specialized alternative approaches for addressing recidivism. Shamanic practices provide far more powerful tools for altering consciousness than found at typical Alcoholics Anonymous meetings—where consumption of tobacco and coffee serve as mechanisms for altering consciousness. Shamanism provides more direct techniques for the alteration of consciousness and production of spiritual experiences, which are considered fundamental to recovery in Alcoholics Anonymous, where step 11 recommends meditation and step 12 refers to the needed change in consciousness as a "spiritual awakening."

These conventional approaches, however, have failed to consider the role of ASC in human nature or drug dependence. This in part reflects cultural perceptions and biases regarding ASC, a puritanical attitude that rejects ASC in general. "Despite the rich literature on ASCs in psychology and spiritual writing, and despite the fact that AA clearly points its members toward an ASC, reviewing the published literature on rehabilitation treatment or surveying current rehabilitation treatment programming reveals a paucity of writing or treatment programming that clearly integrates ASCs with recovery" (McPeake et al., 1991, p. 78). McPeake et al. propose an ASC therapy (ASCT) approach to substance abuse as a natural complement to AA's Twelve-Step recovery program. ASC can enhance recovery by inducing "positive mental attitudes, goal directedness, certain types of cognition or self-talk, certain types of imagery, as well as consciousness of a power greater than self" (McPeake et al., 1991, p. 78). An ASCT approach can meet innate human needs to alter consciousness and, furthermore, provide tools for personal exploration and transpersonal growth. Such an approach can help address substance abusers' genetic or other predispositions to seek altered states, giving them safe and controllable experiences of ASC and providing insight that can promote growth beyond their developmentally fixated stage of drug dependence. Shamanic rituals can provide a direct mechanism for inducing the transformation of consciousness and spiritual awakening considered essential to recovery in AA programs.

An ASCT program should provide both patient and staff education, as well as a variety of opportunities for nondrug-induced alterations of consciousness. These would include: physical activities, including extensive exercise, which could induce the opiate-mediated, so-called runners high; activities in nature, which can provide opportunities for natural high experiences; relaxation training based on progressive relaxation; yogic techniques and the use of vivid imagery; cognitive therapies in art and other forms of aesthetic experience; and meditation and prayer, including Eastern meditative techniques, mantras, biofeedback, dance, music, and other natural methods of inducing ASC (see McPeake et al., 1991). While there is still a lack of double-blind clinical studies

evaluating such effects, McPeake et al. point to the abundant anecdotal information indicating the efficacy of these approaches. Where such programs have been provided, the clients strongly endorse these ASC treatment principals; therapists already utilize many of these modalities as supportive aspects of therapy; and the ASCT approach helps many people recontextualize the spiritual and religious aspect of the Twelve-Step program in ways that are easier to understand and incorporate into their lives.

This chapter elaborates on these ASCT bases for a harm-reduction approach involving the potentials of shamanism by: (1) providing a brief description of shamanic practices, ASC, and healing principles; (2) characterizing the common psychobiology of shamanic and drug ASC; and (3) summarizing clinical studies of shamanistic practices—drumming circles and Transcendental Meditation—used as adjunctive therapies for addiction. Shamanism provides harm-reduction approaches in: (1) precluding addictive impulses with natural methods for altering consciousness that can address the basic psychobiological needs that drive the dynamics of addiction and (2) approaches for reducing recidivism through powerful therapeutic tools for accessing deep levels of the unconscious to discover the dynamics of one's addiction. Chapter 20 in this volume addresses another shamanic technique, the use of sacred plants that induce dramatic transformations of consciousness and personal experiences that produce changes in identity necessary for ending one's career as an addict.

SHAMANISM IN CROSS-CULTURAL PERSPECTIVE

The term *shaman* is used in a variety of ways; it reflects something empirical, a cross-culturally valid concept representing similar religious practitioner found in premodern foraging and simple horticultural and pastoral societies worldwide (Winkelman, 1992, 2000). Eliade (1964) championed a cross-cultural concept of the shaman as someone who entered ecstasy to interact with the spirits on behalf of the community. Eliade characterized the shamanic performance as an unparalleled activity in the lives of the members of the community. The shamanic ritual is typically a nocturnal event for the entire local community; they congregate around a fire with the shaman, who dances while drumming, rattling, or clapping, exhorting the spirits through ancient songs and chants. The shaman would exhort evil spirits to cease their afflictions or exorcise them with assistance from their spirit allies. The shaman may describe these encounters or act out his struggles with the spirits, the behaviors of the animal powers he has summoned, or his journey through the spirit realms.

While shamans were generally male, females might also practice prior to or following their reproductive years. Key to becoming a shaman was a spe-

cial relationship with the spirit world that resulted from the shaman's ability to enter into special ecstatic ASC. These ASC might occur spontaneously, or be produced by fasting and isolation; the effects of drumming, singing, chanting, and dancing; or ingestion of emetics, tobacco, and other psychoactive substances. Shamans spend hours dancing, drumming, and chanting to deliberately induce ASC that provide entry into the spirit world and contact with supernatural powers. While in ASC, they are able to diagnose the causes of disease, determine the reasons why the hunters have not found game, to find friends and enemies, and to divine the future.

A central characteristic of the shaman's ASC is a soul journey or magical flight, a spirit world encounter in which one experiences his soul or spirit traveling to spirit worlds. These shamanistic soul journeys or out-of-body experiences are based in several innate capacities, including body-representation, imagery, and self and other representation processes (Hunt, 1995; Winkelman, 2000). The body-referenced aspect of soul flight (e.g., the out-of-body experience) is a cultural universal that derives from biologically based self-representation structures that reflect an innate system of self-conceptual structures. Humans know the world through their bodies and the action of their bodies on the world; the body becomes by extension the metaphoric system for all human knowledge. Thus out-of-body experiences constitute a symbolic evolution superseding the limitations of body-fixated egocentric perspectives and knowledge.

A significant feature of the soul journey is a disembodied sense of self, seeing one's body from the perspective of the other. Hunt analyzes these experiences in terms of the innate capacity to see one's own self from the perspective of others, which provides the capacity to inform one's own behavior with the perspectives of others. Hunt discusses these experiences as involving presentational symbolism, an imagistic modality that is different from our representational (word) symbolism. These experiences reflect symbolic structures of the nonverbal mind and provide connections with self, emotions, and repressed memories. These shamanic encounters with spirits engage an alternate understanding of the self and social others, manifested in identities linked to animals and animal spirits. These capacities reflect innate structures of humans' evolved psychology, combining the innate system for animal representation with the innate features of the human cognitive architecture used in individual and group identity psychodynamics. These animal-based representations provide tools for individuation and social identity by modeling individual qualities and shared social characteristics.

The spirits involved in the shaman's activities are typically acquired during a vision quest that is part of the shaman's training and development. During

this quest, an initiate is expected to encounter a spirit ally who will teach him a particular power that he will develop during his life. Older shamans may supervise and guide the initiates during their vision quests, but typically the shaman encounters the spirit powers alone in the wilderness. These initiatory experiences typically involve the initiate's experience of his personal death, followed by a subsequent rebirth. This often occurs in conjunction with visions in which the shaman experiences being torn apart by animals and then put back together by the animals and spirit beings. The spirits reassemble the shaman into a stronger and more knowledgeable person with powers to enter the invisible worlds. Because of the shaman's personal experience of death and knowledge of the ways to control spirits, that the shaman is called on to find lost souls and to guide the souls of the deceased to their afterlife destination.

Although the view of the world takes different forms in different cultures, shamans' cosmologies share a view of the world as consisting of at least three principal realms or levels. Humans occupy the middle world, while spirits and other supernatural beings are found in both this world and in the upper and lower worlds. These different realms are connected by a horizontal axis mundi, a world axis, often depicted as a tree or hollow mountain that connected the levels. The beings found in these different realms have different powers, which they use to keep the universe functioning and in balance. The animal and plant spirits that the shaman acquires provide him with numerous powers and abilities, including the potential to transform into animals (his spirit allies). Shamans can learn many things from these beings, such as how to travel along the paths that connect the worlds and how to acquire and use the powers of the spirit beings. This knowledge allows them to journey throughout the universe in quest of the knowledge they need to help their communities. These shamanic views of the interconnectedness of nature reflect contemporary cognitive science theories of meaning (see Hubbard, 2002).

Biological Bases of Shamanic Universals

Winkelman (2000, 2002, 2004a) illustrates how central features associated with shamanic practices around the world involve underlying biological systems in:

- Community bonding rituals that extend mammalian attachment processes, eliciting the opioid-attachment mechanisms that united groups and helped assure well-being through enhanced access to resources and support;
- Singing, chanting, dancing, and drumming, manifestations of ancient mammalian, primate, and hominid emotional communication and social integration systems;

ASC, particularly a soul flight, which reflects a higher level of integration of consciousness produced by the separation of self-reference from the body;
Spirits, particularly animal powers, that provided systems of individuation and self-expression that facilitate psychological development and social identity formation;
Divination and prophecy that integrated diverse forms of unconscious information into symbolic visionary experiences (see Winkelman & Peek, 2004); and
Healing rituals that managed a variety of endogenous healing processes, including effects on serotonergic and opioid neurotransmitter systems (also see Winkelman & Baker, 2008).

The worldwide distribution of shamanism derives from human nature and aspects of humans' evolved psychology, particularly the activation of biological processes such as those involved in ASC, as well as innate modules involved in our capacities for inferring the thoughts and desires of others and reasoning about the qualities of the natural world and animal species (Winkelman, 2000, 2002, 2004a; Winkelman & Baker, 2008). Many universal features of shamanism are directly related to utilization of some of these basic evolved features of the innate modules of the human brain. For example, what is called the natural history module, a component of our mental hardware that enables us to understand animal species and their differences, is exploited by shamanism. Shamans take this innate capacity for understanding animals and extends it to produce more complex representations of the self and others. This is manifested in terms of understanding one's own potentials as animal powers and allies, and in totemism, in which animal species provide systems of representation for social groups (e.g., bear clan). Shamans' roles in managing aspects of self and social identity through animal concepts are exemplified in the special relationships shamans have with animal powers and the transformation of identity provided by animal familiars. Shamanic ritual activities engage these innate cognitive processes related to unconscious aspects of self and identity to facilitate social and psychological integration. Shamanic rituals and concepts provided adaptive strategies and technologies for integrating a variety of capacities—biological, social, and cognitive—that contributed to human evolution, adaptation and survival.

THE PSYCHOBIOLOGY OF SHAMANIC ASC

Understanding how shamanistic activities can provide a harm-reduction strategy requires recognition of the fundamental similarities in altered states produced by a variety of processes, ritual and pharmacological. Shamanic ASC

are typically predisposed by fasting, sexual abstinence, and isolation and are induced through singing, chanting, drumming, and dancing. This leads to a phase of collapse and apparent unconsciousness during the shaman's experiential engagement with the spirit world. These ASC central to shamanic training and practice are induced by a variety of procedures that have the same basic overall physiological dynamics—activation of the autonomic nervous system (sympathetic excitation) until exhaustion and collapse (parasympathetic dominance) induces the body's relaxation response (Winkelman, 2000).

ASC also stimulate coherent theta brain waves (three to six cycles per second) that induce synchronization of the brain. These brain wave patterns are produced by discharge activity in the limbic system (the emotional or paleomammalian brain) that activates serotonin linkages with lower brain structures (Mandell, 1980). These discharges generate powerful synchronous brain waves that propagate upward into the frontal cortex, where they replace the normal fast and desynchronized brain wave activity with the coherent slow wave discharges in the alpha and theta range (Mandell, 1980; see Winkelman, 2000, for review). Overall effects of shamanic ASC integrate brain information from the emotional and behavioral brains into the frontal areas of the brain.

The common physiological mechanisms that Mandell (1980) proposes underlie diverse ASC (drug and nondrug) are exemplified in the effects of meditation (Walton & Levitsky, 1994) and psychointegrators (hallucinogens) (Winkelman, 2007a). This high voltage slow wave electroencephalograph (EEG) activity (alpha, theta, and delta, especially three to six cycles per second) is produced by hypersynchronous discharges across the hippocampal-septal-reticular-raphe circuit linking our emotional limbic brain with the so-called reptilian behavioral brain. These lower brain discharge activities spread to the frontal lobes, producing pleasurable sensations and feelings of well-being. These patterns contribute to healing by reducing stress hormone levels and increasing serotonin levels, which play central roles in integration of emotional and motivational processes and the synthesis of information across the functional levels of the brain. ASC integrate information from the lower emotional and behavioral preverbal brain structures into the language-mediated activities of the frontal cortex. Consequently ASC are often experienced as providing enlightenment, a sense of unity, and feelings of connection and personal integration.

Physiological changes associated with ASC facilitate healing and psychological and physiological well-being through: physiological relaxation; facilitating self-regulation of physiological processes; reducing tension, anxiety, and phobic reactions; manipulating psychosomatic effects; accessing unconscious information in visual symbolism and analogical representations; inducing inter-

hemispheric fusion and synchronization; and facilitating cognitive-emotional integration and social bonding and affiliation (Winkelman, 2000).

Social Opioid Mechanisms in Shamanic Practice

While most of the psychobiological research on shamanic ASC has focused on their interactions with the serotonergic system, there is also ample evidence that these practices constructively engage the endogenous opioid mechanisms as well. Shamanistic rituals involve practices that enhance social integration, strengthen group identity, and promote interpersonal and social bonding at physiological and psychosocial levels. A literature review (Frecska & Kulcsar, 1989) indicates that community activities elicit the opioid-attachment mechanisms, with ritual eliciting our innate drives for affiliation and evoking opioid release. Ceremonial opioid release is based in emotionally charged cultural symbols that are cross-conditioned with endocrine systems during early socialization, linking the body's physiological responses with cultural symbol systems. This association of physiology and symbols provides a basis for ritual elicitation of the opioid system through shamanic rituals. Opiate receptor locations and functions in the brain illuminate the adaptive functions of ritual ASC. The opiate receptors are most highly concentrated in the limbic system and mid-brain structures primarily responsible for attachment, motivational and affective behaviors, and our reward centers. It is in this region of the brain that we find the most intensive foci of ASC theta wave activity and the densest concentrations of opioid receptors in the brain. Winkelman (1997, 2000) reviews the physiological aspects of many different shamanic ASC practices, illustrating that they have inherent therapeutic effects related to elicitation of our endogenous opioids. Physical mechanisms for opioid elicitation in shamanistic healing include: extensive drumming, dancing, and clapping; repetitive physical activity; temperature extremes (e.g., sweat lodges); stressors such as fasting, flagellation, and self-inflicted wounds; and nighttime activities when endogenous opioids are naturally highest (see Prince, 1982 and Winkelman 1997, 2000, for reviews).

SOUL LOSS AND SPIRITUALITY IN SHAMANISTIC THERAPEUTIC INTERVENTIONS

Shamanic cultures consider a number of spiritual causes of illness. One cause involves soul loss, which often occurs when an individual experiences a great fright or commits a transgression that causes the soul to leave the body. Diseases may also be caused by a foreign object that has intruded into the body.

Such objects often take the form of magical darts, which may be sent into the body by a malevolent individual (such as a sorcerer) desiring to harm or kill the person. In the ecstatic state, the shaman can see into the body of a sick person, find the disease-causing object, and then remove it.

A major paradigm of shamanic healing is soul recovery, involving reintegration of aspects of the self. Soul loss reflects dissociation from the self, as well as a lack of individual connection with broader social identity. Shamanic conceptualizations of soul loss involve a view similar to AA concepts of addiction as resulting from a lack of spirituality. Spirituality can be seen as reflecting collective dimensions of the individual connectedness with the social group. Soul loss reflects this lack of individual connection with broader social and cultural identity. Since fundamental aspects of shamanic practice are concerned with establishing a connectedness with animal spirits and personal souls and allies, such practices can provide a means for addressing the collective soul loss and lack of spirituality. Spiritual perceptions have a psychobiological basis, reflecting basic structures of the brain and consciousness (Winkelman, 2004b). The spiritual experiences induced by shamanic practices make it a potentially important resource in addressing both psychological integration and incorporation of the spiritual aspects of the self, providing a new sense of identity.

Shamanic rituals heal through recovery of lost aspects of one's soul and by obtaining allies and powers. Power animals and guardian spirits are conceptualized as metaphors that represent different kinds of healing energies (Rioux, 1996), providing an expansion of the Twelve-Step concept of a Higher Power and its healing effects. A number of therapists have illustrated how to introduce shamanic techniques into work with addict populations that are treatment resistant and high recidivist (e.g., see Eshowsky, 1993, 1998, 1999; Rioux, 1996; Winkelman, 2004c). Shamanic practices provide tools for assessing the spiritual dimensions of healing and direct mechanisms for experiencing spiritual contact. Shamanistic practices provide access to the deep structure of the unconscious, where engagement with the spirit world involves access to aspects of the self. These visionary experiences involve the brain's primary processing capacities, symbolic structures of the nonverbal mind and representations of self, emotions, and others (Winkelman, 2000). These experiences provide information from the deep unconscious that helps to meet therapeutic needs for self-awareness, emotional insight, and psychological integration, thereby enhancing therapeutic processes (Rioux, 1996).

The role of spirituality in recovery from substance abuse may reflect a protective factor provided by religious involvement and spiritual engagement (Miller, 1998). Spiritual practices may have role in the healing of addiction by providing a sense of meaning to life often found lacking among substance abus-

ers. Shamanism is the original form of spirituality, providing a direct spiritual awakening, a means for transcendence of personal consciousness, and consequently a mechanism for overcoming addiction.

Rioux (1996) illustrates that shamanic techniques can play a role in a holistic addiction counseling approach that integrates spiritual perspectives into a biopsychosocial approach to healing. Shamanism has exceptional techniques for engaging one in a focus on inner realities to produce self wholeness. Shamanic relations with animal spirits and personal allies can strengthen identity and character formation. Shamanic ASC can help clients achieve wholeness, transforming consciousness by providing a link between inner and outer realities, and enabling inner world perspectives to operate on the outer world to produce harmony and wholeness. Important aspects of the shamanic ASC include the focusing of intention on one's purpose and spending time in relaxed conditions that provide restful healing. ASC practices also free one from ego-bound emotions and provide a balance of conflicting internal energies. Work through dreaming and shamanic journeying can help achieve the sense of wholeness, countering the sense of self-loss at the basis of addictive dynamics. Spiritual relations and experiences can enhance self-esteem by providing connectedness beyond the egoistic self.

Drumming as a Shamanistic Harm-Reduction Strategy

A universal feature of shamanism is percussion, particularly drumming. Researchers (e.g., Blackett & Payne, 2005; Eshowsky, 1993, 1998, 1999; Winkelman, 2003, 2004c) report beneficial effects on addictions from various drumming activities. Winkelman reviews how drumming facilitates management of addictive tendencies through physiological, psychophysiological, psychological, and social effects. Blackett and Payne note therapeutic effects of drumming that have far-reaching implications for a harm-reduction approach. These effects include: reduction of stress; fostering of emotional processing; enhanced immune system responses; enhanced social cohesion; enhanced communication; transcendental spiritual experiences and other beneficial alterations of consciousness; and facilitation of concentration, memory, and other cognitive processes.

Drumming has many effects indicated by clinical studies (see Friedman, 2000, for reviews). Drumming induces the signature brainwaves of ASC, eliciting synchronous responses in the theta and alpha range (see Winkelman, 2000, for reviews). These slow wave discharges synchronize the frontal areas of the brain with ascending waves that integrate nonverbal information from lower brain structures. Enhanced connection with the lower brain integrates its infor-

mation into the frontal cortex, addressing psychodynamic needs of addicts for self-awareness and insight. Effects of drumming on the limbic (emotional) brain often enhance awareness of preconscious material, particularly repressed emotions. Drumming can contribute to an integration of the self and an enhanced sense of connectedness with others, reducing the self-centeredness, isolation, and alienation characteristic of addicts. Among physiological effects of drumming is an increased hypnotic susceptibility, which contributes to relaxation, as well as pleasurable experiences. Other physiological changes associated with ASC-induced healing in general include: facilitating self-regulation through psychosomatic effects that reduce tension, anxiety, and phobic reactions; providing access to unconscious and preconscious material through visual imagery and dreamlike mentation; increasing cognitive integration through inducing interhemispheric fusion and synchronization of the frontal cortex; and integrating information across the brain's neuraxis, contributing to integration of behavioral, emotional, and cognitive dynamics (Winkelman, 2003).

Drumming groups also address dynamics of addiction and support recovery through enhancing social support networks that are particularly relevant for addicts whose social isolation and lack of support are significant in the relapse. Drumming groups enhance a natural sense of connection among participants and emotional bonds important for personal transformation of the addict. Drumming circles also can produce psychobiological effects, as community rituals can evoke physiological responses in the opioid attachment mechanisms (see Frecska & Kulcsar, 1989). Reports from people in rehabilitation programs using drumming suggest drumming induces relaxation responses. The positive effects of group drumming experiences on recovery attested to by people in substance abuse rehabilitation programs provide a compelling rationale for its utilization (see Winkelman, 2003, 2004c). Drumming can provide harm-reduction and therapeutic responses through: induction of relaxation; release of emotional trauma; reintegration of self; and addressing the addict's self-centeredness, isolation, and alienation by creating a sense of connectedness with self and others.

Physiological and Psychosocial Effects of Drumming

Drumming produces physiological, psychological, and social stimulations that enhance recovery processes. Drumming enhances hypnotic susceptibility, increases relaxation, and induces shamanic experiences (Maurer, Kumar, Woodside, & Pekala, 1997). Drumming and other rhythmic auditory stimulation impose a driving pattern on the brain, particularly in the theta and alpha ranges (see Winkelman, 1997, for review). The enhanced theta and alpha wave

entrainment produced by drumming typifies general physiological effects of ASC, (Mandell, 1980; Winkelman, 1997, 2000), including meditation (Walton & Levitsky, 1994). Drumming induces an enhanced awareness of preconscious dynamics, a release of emotional trauma, and reintegration of self. Drumming provides a secular approach to accessing Higher Power yet can apply spiritual perspectives to the psychological and emotional dynamics of addiction. Drumming circles have important roles as complementary addiction therapy, particularly for repeated relapse and where other counseling modalities have failed.

A variety of benefits have been noted from community drumming practices:

Relaxation
Stress-reduction
Enhancement of your brain's pleasure centers
Reestablishment of balance in opioid and serotonergic systems
Alteration of consciousness and meditation
Experiences of transcendence
Greater awareness of your unconscious powers
Connection with your emotions
Reduction of self-centeredness, alienation, and isolation
Emotional insight
Personal and psychological integration
Enhanced self-esteem
Reinforcement and empowerment for personal change
Healing and release of emotional trauma
Forgiveness and release of guilt
Tapping your visionary potential
Obtainment of spiritual guidance
Production of spiritual awareness, experiences, and contact
Increased self-awareness and understanding
Connection with others
Community healing and bonding
Reprogramming the self with spiritual assistance
Making spiritual amends
Spiritual awakening and self-assessment
Spiritual transcendence

TRANSCENDENTAL MEDITATION AS ADDICTION TREATMENT AND HARM REDUCTION

The effectiveness of shamanistic ASC in harm reduction is illustrated in clinical assessments of Transcendental Meditation (TM) (see Gelderloos,

Walton, Orme-Johnson, & Alexander, 1991; O'Connell & Alexander, 1994). A meta-analysis (Alexander, Robinson, & Rainforth, 1994) of 19 studies utilizing TM for the treatment of drug addiction reported an effect size of between .5 (medium) and .8 (large). TM had substantially larger effect sizes than: relaxation, peer pressure resistance, DUI intervention, and combined drug prevention programs. Comparisons with controls illustrate that TM treatment effects are not due to nonspecific aspects (e.g., attention, expectancy, social support, motivation). Alexander et al. (1994) suggest TM's effectiveness in addressing substance abuse derives from effects on physiological through social levels, intervening in a range of causal and contributory factors.

Effects of meditation on addiction have physiological foundations that address anxiety, tension, and stress and imbalances in neurotransmitter systems produced by drugs (Alexander, et. al., 1994). TM reduces stress and physiological arousal, increases serotonin levels, reestablishing psychophysiological equilibrium and changing autonomic responses to stressful stimuli. Walton and Levitsky's (1994) neuroendocrine model of the mechanisms of meditation addresses its effects at the physiological level involving depletion of serotonin by chronic substance abuse. The ability of TM to reduce stress and enhance serotonin functioning addresses the physiological level of addictive behavior and the cycle of addiction—the depletion of serotonin by chronic substance abuse and the interference that it produces in homeostasis and stress responses. Meditation provides a natural process for producing relief from stress, producing physically relaxed states reflected in low levels of autonomic arousal and enhanced EEG coherence. Major areas of the brain at which meditation can intervene in the cycle of drug dependence include the "components of the central nervous system responsible for maintaining homeostasis . . . the hypothalamus, the hippocampus, the locus coeruleus and the raphe nuclei. These areas . . . interface with the endocrine system principally through the hypothalamic-pituitary-adrenocortical (HPA) axis, the sympatico-adrenomedullary axis, and the parasympathetic nervous system" (Walton & Levitsky, 1994, p. 95). Effects of TM in reducing addictive behaviors results from actions on the *locus coeruleus* (LC), producing an increased availability of serotonin that exercises an inhibitory action on the LC similar to sedatives. The effects of meditation in increasing serotonin levels has the consequence of reducing cortisol levels and the release of corticotropin-releasing factor. This results in a reduction of stimulation of anger and fear centers of the limbic brain and their effects on the hypothalamic pituitary adrenal (HPA) axis that contributes to chronic stress. "[B]ecause of serotonin's roles at multiple sites involved in successful interaction with the environment, its decreased availability may be one of the most important neurochemical components in the production of tendencies toward

addiction" (Walton & Levitsky, 1994, p. 110). TM-induced increases in serotonin availability mirror Mandell's (1980) model of serotonergic mechanisms for ASC (transcendent states) in general and suggests their generic role in providing biological mechanisms through which ASC are able to interrupt drug dependence. The overall physiological dynamics of meditation ASC are the same as those associated with other ASC (Mandell, 1980; Winkelman, 2000), suggesting that the effectiveness of meditation in drug rehabilitation would also be obtained from other kinds of naturally induced ASC.

TM addresses psychological factors contributing to addictive tendencies, including low self-esteem, negative affect, impulsiveness and rebelliousness, external locus of control, inadequate coping skills, and distorted and overlearned or stereotypic cognitive response patterns (Alexander, et al., 1994, p. 16). Alexander et al. suggest Vedic psychology provides perspectives from which to appreciate how meditation can provide relief for addicts and substance abusers. These include: producing restful alertness, enhanced mental functioning, pleasant affective experiences, and a sense of intimacy with all of life. Meditation is "a natural way to achieve the experiences substance users are looking for: relief from distress, increased self esteem, enhancement of well-being and self-efficacy, and a sense of personal power and meaning in life" (Gelderloos et al., 1991, p. 317). Meditation counters negative emotions associated with addiction (depression, anxiety, anger), helping to resolve the emotional and interpersonal difficulties that trigger relapse. Meditative practices enhance awareness of deeper levels of the mind and the unconscious, promoting self-actualization, which has an important role in producing spiritual experiences.

CONCLUSIONS

Development of shamanic approaches to harm reduction is strongly justified—even demanded—in light of the consequences of widespread drug addiction and recidivism and limited success rates of current treatment modalities. Shamanistic practices can be applied to recurrent problems encountered in substance abuse rehabilitation to facilitate recovery, reduce recidivism and the harm experienced by addicts, and can be employed proactively to reduce the drive to experience ASC through the harmful avenues of drug addiction.

Alexander et al.'s (1994) review of the roles of TM in addressing addictions suggests that shamanistic approaches in general can address addiction at multiple levels: the physiological aspects of addiction; the cognitive, emotional and psychodynamic aspects; the spiritual dimension associated with core values and a dependence on a Higher Power; and the social environment, particularly the proximal social relations affecting substance abuse. Research shows that TM

provides an effective antidote to the addict's poor coping abilities and problems with interpersonal stressors and mental anxiety. Alexander et al. (1994) expand on the perspective that addictive behaviors derive from spiritual problems, suggesting that so-called loss of soul contributes to addictive behaviors. This concept of soul may be viewed as a subtle aspect of one's existence and personal substance involving connectedness with others. Alexander et al. suggest that spiritual wellbeing derives from healthy functioning at all levels of the individuals—"body, mind, spirit, social interaction and environment" (Alexander et al., 1994, p. 18). The shamanic healing practices are renowned for such effects across the multiple dimension of being.

Winkelman (2001a, 2001b, 2003, 2004c; also see Winkelman and Roberts 2007) outlines a variety of ways in which the shamanic paradigm and ASC can be applied to substance abuse rehabilitation. Drumming circles and other shamanic ASC activities can address multiple needs of addicted populations through:

The physiological effects that induce natural ASC characterized by enhanced theta wave entrainment, a natural method for altering consciousness that addresses the psychobiological needs for pleasure, balance, and integration that drive addictive behaviors;

Inducing the relaxation response and restoring balance in the neurotransmitter systems disturbed by drug abuse;

Meeting psychodynamic needs for self-awareness, insight, emotional healing, and psychological integration;

The support group that can meet social needs for an intimate community for affiliation, interpersonal support, and connectedness with others and interpersonal support;

The psychological and emotional effects of the shamanic journey that meets psychodynamic needs for spiritual contact and personal relationships with a power beyond ourselves; and

opportunities for emotional insight, self-awareness, psychological integration, and therapeutic processes acquired through accessing deep levels of the unconscious.

An evolutionary perspective on substance abuse suggests an understanding in which humans evolved addictive potentials for adaptive reasons that may be explained by the dopamine hypothesis, which postulates their role in producing feelings of well-being. Since ASC reflect underlying psychobiological structures and innate needs, when societies fail to provide legitimate procedures for accessing these conditions, they are sought through other means. Because contemporary Indo-European societies lack mainstream legitimate institution-

alized procedures for accessing ASC, they tend to be sought and utilized in deleterious and self-destructive patterns—alcoholism, tobacco abuse, and illicit substance dependence

The shamanic approach supports the harm-reduction principles of alternatives to abstinence with natural and nonaddictive ways of meeting human needs to experience transcendent states of consciousness. It engages the client's desires for transformations of consciousness and constructive avenues of change with powerful tools that access deep levels of the self and unconscious. Shamanic procedures have the potential to help the client access deep levels of personal history that have contributed to addictive behaviors. They also provide visionary experiences that reveal the problematic aspects of substance abuse and help envision new goals and alternate futures instead of waiting until drug problems lead to hitting the bottom.

NOTE

1. This chapter includes material adapted from previous publications (Winkelman, 2001a, 2003, 2004b; Winkelman & Baker, 2008).

REFERENCES

Alexander, C., Robinson, P. , & Rainforth, M. (1994). Treating and preventing alcohol, nicotine, and drug abuse through transcendental meditation: A review and statistical meta-analysis. *Alcoholism Treatment Quarterly, 11*(1/2), 13–87.

Blackett, P. , & Payne, H. (2005). Health rhythms: A preliminary inquiry into group-drumming as experienced by participants on a structured day services programme for substance misusers. *Drugs: Education, Prevention and Policy, 12*(6), 477–491.

Eliade, M. (1964). *Shamanism archaic techniques of ecstasy*. New York: Pantheon Books.

Eshowsky, M. (1993). Practicing shamanism in a community health center. *Shamanism, 5*(4), 4–9.

Eshowsky, M. (1998). Community shamanism: Youth, violence, and healing. *Shamanism, 11*(1), 3–9.

Eshowsky, M. (1999). Behind these walls where spirit dwells. *Shamanism, 12*(1), 9–15.

Frecska, E., & Kulcsar, Z. (1989). Social bonding in the modulation of the physiology of ritual trance. *Ethos, 17*(1), 70–87.

Friedman, R. (2000). *The healing power of the drum*. Reno, NV: White Cliffs Media.

Gelderloos, P. , Walton, K., Orme-Johnson, D., & Alexander, C. (1991). Effectiveness of the Transcendental Meditation program in preventing and treating substance misuse: A review. *International Journal of the Addictions, 26*, 293–325.

Heggenhougen, C. (1997). *Reaching new highs: Alternative therapies for drug addicts*. Northvale, NJ: Jason Aronson.

Hubbard, T. (2002). Some correspondences and similarities of shamanism and cognitive science: Interconnectedness, extension of meaning and attribution of mental states. *Anthropology of Consciousness, 13*(2), 26–45.

Hunt, H. (1995). *On the nature of consciousness*. New Haven, CT: Yale University Press.

Jilek, W. (1994). Traditional healing in the prevention and treatment of alcohol and drug abuse. *Transcultural Psychiatric Research Review, 31,* 219–258.

Mandell, A. (1980). Toward a psychobiology of transcendence: God in the brain. In J. Davidson & R. Davidson (Eds.), *The psychobiology of consciousness* (pp. 379–464). New York: Plenum.

Maurer, R. L., Kumar, V. K., Woodside, L., & Pekala, R. J. (1997). Phenomenological experience in response to monotonous drumming and hypnotisability. *American Journal of Clinical Hypnosis, 40,* 129–145.

McPeake, J., Kennedy, B., & Gordon, S. (1991). Altered states of consciousness therapy: A missing component in alcohol and drug rehabilitation treatment. *Journal of Substance Abuse Treatment, 8,* 75–82.

Metzner, R. (1994). Addiction and transcendence as altered states of consciousness. *The Journal of Transpersonal Psychology, 26*(1), 1–17.

Miller, W. (1998). Researching the spiritual dimension of alcohol and other drug problems. *Addiction, 93,* 979–990.

O'Connell, D., & Alexander, C. (Eds.). (1994). *Self-recovery: Treating addictions using Transcendental Meditation and Maharishi Ayur-Veda*. New York: Hayworth Press.

Prince, R. (1982). The endorphins: A review for psychological anthropologists. *Ethos, 10*(4), 299–302.

Rioux, D. (1996). Shamanic healing techniques: Toward holistic addiction counseling. *Alcoholism Treatment Quarterly, 14*(1), 59–69

Siegel, R. (1990). Intoxication: Life in pursuit of artificial paradise. New York: Dutton.

Smith, D. (2000). Shamanism and addiction: The mask of therapeutic containment. *Spirit Talk, 11,* 8–12.

Walton, K., & Levitsky, D. (1994). A neuroendocrine mechanism for the reduction of drug use and addictions by transcendental meditation. In D. O'Connell & C. Alexander (Eds.), *Self-recovery: Treating addictions using transcendental meditation and Maharishi Ayur-Veda* (pp. 13–88). New York: Hayworth Press.

Winkelman, M. (1992). *Shamans, priests and witches, a cross-cultural study of magico-religious practitioners*. Anthropological Research Papers (No. 44). Tempe, AZ: Arizona State University.

Winkelman, M. (1997). Altered states of consciousness and religious behavior. In S. Glazier (Ed.), *Anthropology of religion: A handbook of method and theory* (pp. 393–428). Westport, CT: Greenwood.

Winkelman, M. (2000). *Shamanism the neural ecology of consciousness and healing.* Westport, CT: Bergin and Garvey.

Winkelman, M. (2001a). Alternative and traditional medicine approaches for substance abuse programs: A shamanic perspective. *International Journal of Drug Policy, 12,* 337–351.

Winkelman, M. (2001b). Psychointegrators: Multidisciplinary perspectives on the therapeutic effects of hallucinogens. *Complementary Health Practice Review, 6*(3), 219–237.

Winkelman, M. (2002). Shamanism and cognitive evolution. *Cambridge Archaeological Journal, 12*(1), 71–101.

Winkelman, M. (2003). Complementary therapy for addiction: "Drumming out drugs." *American Journal of Public Health, 93*(4), 647–651.

Winkelman, M. (2004a). Shamanism as the original neurotheology. *Zygon Journal of Religion and Science, 39*(1), 193–217.

Winkelman, M. (2004b). Spirits as human nature and the fundamental structures of consciousness. In J. Houran (Ed.), *From shaman to scientist essays on humanity's search for spirits* (pp. 59–96). Lanham, MD: Scarecrow Press.

Winkelman, M. (2004c). Spirituality and the healing of addictions: A shamanic drumming approach. In L. L. Barnes & S. S. Sered (Eds.), *Religion and healing in America* (pp. 455–470). New York: Oxford University Press.

Winkelman, M. (2007a). Therapeutic bases of psychedelic medicines: Psychointegrative effects. In *Psychedelic medicine: New evidence for hallucinogenic substances as treatments,* (vol. 1, pp. 1–19). Westport, CT: Praeger/Greenwood Publishers.

Winkelman, M. (2007b). Shamanic guidelines for psychedelic medicines . In M. Winkelman & T. Roberts (Eds.), *Psychedelic medicine: New evidence for hallucinogenic substances as treatments,* vol. 2, pp. 143–167. Westport, CT: Praeger/Greenwood Publishers.

Winkelman, M. (2008). Cross-cultural and biogenetic perspectives on the origins of shamanism. In D. Whitley & K. Hays-Gilpin (Eds.), *Belief in the Past Theoretical Approaches to the Archaeology of religion* (pp. 43–66). Walnut Creek, CA: Left Coast Press.

Winkelman, M. & Baker, J. (2008). *Supernatural as natural.* Upper Saddle River, NJ: Prentice-Hall.

Winkelman, M., & Peek, P. (2004). Introduction: Divination and healing processes In M. Winkelman & P. Peek (Eds.), *Divination and healing: Potent vision* (pp. 3–28). Tucson: University of Arizona Press.

Winkelman, M., & Roberts, T. (Eds.). (2007). *Psychedelic medicine: New evidence for hallucinogenic substances as treatments* (vols. 1 & 2). Westport, CT: Praeger/Greenwood Publishers.

This page intentionally left blank

Part III

PRACTICES

This page intentionally left blank

Chapter **14**

Therapeutic Approaches, Beliefs, and Perceptions of Providers When Treating Alcohol- or Drug-Using Clients: Selecting Best Practices

Maria Dinis, PhD, MSW, Joseph R. Merighi, PhD, MSW, and Kelly Anders, MAL

How do providers select therapeutic approaches when they treat clients with alcohol or drug problems? When clinicians make treatment decisions, they may utilize empirically validated interventions or models, have knowledge of specific treatment outcomes, or incorporate their own belief systems to inform their treatment choice. At the same time, they may be limited to services available in their geographic region or by requirements imposed by insurers or funders. The interplay of treatment approaches, outcomes, provider beliefs, availability of services, and funding is central to a provider's selection of a best practice approach when treating people with substance abuse problems.

PRINCIPLES OF ADDICTION TREATMENT

The 13 principles of effective addiction treatment set forth by the National Institute on Drug Abuse (1999) specify a road map for providers to use when working with chemically addicted persons. These principles are: (1) having providers match treatment settings, interventions, and services to the needs of their clients because there is not one single treatment that is suitable for everyone; (2) having treatment available when individuals are ready to receive it; (3) having treatment address the range of needs of an individual beyond his drug use; (4) having the treatment and service plan assessed and modified as the individual's needs change; (5) having individuals remain in treatment for an appropriate amount of time (usually up to three months) to achieve treatment effectiveness; (6) having individual or group counseling to assist

with motivation concerns, develop skills and problem-solving techniques to resist using drugs, and use other behavioral therapies to improve functioning with personal, family, and community relationships; (7) having individuals use medications to reduce craving or treat mental health disorders in combination with other counseling and behavioral therapies; (8) having individuals with co-occurring substance and mental health disorders assessed and treated for both conditions; (9) having medical detoxification as only the first stage to address acute physical symptoms such as withdrawal prior to clients participating in a longer term addiction treatment program; (10) having involuntary treatment coerced by the family, employer, or criminal justice system, which can be effective because sanctions can increase the rates of entering and staying in treatment as well as drug treatment intervention success; (11) having individuals monitored during drug treatment (e.g., urinalysis) to detect their current drug use so that treatment plans can be adjusted accordingly and clients can be provided with important feedback; (12) having individuals assessed in treatment programs for many types of infectious diseases (e.g., HIV/AIDS, hepatitis B and C, and tuberculosis) and provide counseling to assist clients with behavior modification of high-risk behaviors and assist those already infected to manage their own illness; and (13) having multiple treatments available for persons who relapse, and participating in self-help programs during and after treatment to maintain abstinence and rebuild personal functioning.

Whether providers take account of the aforementioned principles—which are considered the gold standard of effective addiction treatment as set forth by the National Institute on Drug Abuse (1999)—when they make their selection of treatment approaches and services for chemically addicted clients is not easy to discern. Providers selecting best practices to treat alcohol or drug-using clients may depend on a combination of factors related to treatment approaches, belief systems, and availability of services. In other words, the selection of best practices is likely to encompass the cumulative knowledge providers have of treatment models and outcomes; their beliefs of what treatment approach works best; and the availability of services in the client's geographic region.

MODELS OF ALCOHOL/DRUG TREATMENT

Alcohol or drug treatment models to which service providers subscribe or to which they refer clients (e.g., moral/prevention, medical/disease, psychological, sociocultural, and integrative/biopsychosocial) are widely described in the literature and are often referred to as a treatment philosophy or orientation (Institute of Medicine, 1990; Miller & Hester, 1989, 2003; Peterson, Nisenholz, & Robinson, 2003). These models embrace a range of services (e.g.,

medical, counseling, psychiatric, or social services) and methods (e.g., pharmacotherapy, psychotherapy, or self-help groups) to achieve the overall treatment goal of harm reduction or abstinence from alcohol or drug use. Treatment models contain a perspective or orientation toward the etiology of alcohol or drug problems, and thus specify the method of intervention (Institute of Medicine, 1990). While the term *treatment approach* is used to refer to a kind of model, treatment approaches also encompass treatment systems that may utilize various models. With respect to definitions, treatment systems are also called treatment settings because they describe the living arrangement or institutional milieu in which treatment takes place (e.g., inpatient, outpatient, hospital, prison, residential facility, day treatment center, and halfway house) (Institute of Medicine, 1990; Musto, 2002; White, 1998). The term *medical* refers to approaches that are physician driven and "pertaining to medicine or to the treatment of diseases" (Saunders, 1988, p. 992). In contrast, the term *disease* is taken to mean a lack of individual-level ability to control drinking, which eventually causes physical, emotional, or social harm (Alexander, 1987; Jellinek, 1960).

Given that alcohol and drug use may be seen as a crime, illness, way of life, or deviant behavior (Siegler & Osmond, 1968), professionals across disciplines and in different work settings may have differing views on alcohol misuse and treatment. Their views may also be applied differently depending on the population that is misusing alcohol. For example, pregnant women who misuse alcohol and drugs have been offered drug treatment and, in some cases, have been prosecuted criminally (i.e., charged with child neglect and/or losing custody of their children) (Chavkin, 1990). However, these sanctions have not applied to fathers. Part of this confusion may be related to the disagreement among obstetricians and their staff about the use of alcohol or drugs during pregnancy. Some providers believe that pregnant women should abstain from alcohol during pregnancy while others promote moderate drinking; others screen pregnant women, not as a way to prevent future alcohol/drug-related problems, but to establish the need for substance abuse treatment (Kennedy, Finkelstein, Hutchins, & Mahoney, 2004; Morse, Greshan, & Hutchins, 1997; Roche & Richard, 1991).

Because service providers maintain different attitudes about optimal choices for substance use treatment, professionals across disciplines may also subscribe to different models when addressing these problems. Professionals such as physicians, ministers, psychotherapists, and police officers may use diverse definitions and criteria for alcohol addiction. The guidelines physicians use to determine whether alcoholism is a disease are not necessarily the same guidelines that ministers use to determine if alcoholism is a sin, or psychotherapists

use to define it as a neurotic problem. Within different disciplines or professions, there are different theories that define alcohol addiction. There is no obvious way to compare these theories, however, because they are different across and within disciplinary lines.

While models are not theories, they can be used to classify theories (Nunes-Dinis, 1996; Siegler & Osmond, 1968; Siegler, Osmond, & Newell, 1968). In general, models are constructed from the theoretical dimensions (i.e., diagnosis, etiology, prognosis, treatment, etc.) of various professional disciplines (Institute of Medicine, 1990; Siegler & Osmond, 1968), and they can be used to inform the treatment of substance use problems. Although earlier writers focused exclusively on alcohol, the current conceptualization of overlapping alcohol and drug problems has resulted in the use of these models for alcohol and drugs interchangeably (Hubbard, 1990).

Treatment models can be classified under five major categories or perspectives: moral/prevention, medical/disease, psychological, sociocultural, and integrative/biopsychosocial (Miller & Hester, 2003; Nunes-Dinis, 1996; Peterson et al., 2003). Although there are contemporary prevention models premised on moral, ethical, and value-based ideologies of health to prevent alcohol problems, the broad public health model emphasizes the interaction of factors (e.g., individual, environment, and agent-based) and encompasses primary (prevention), secondary (early detection), and tertiary (treatment) intervention and preventive approaches (Bukoski, 1991; Institute of Medicine, 1989; Johnson, 2004; Leukefeld & Bukoski, 1991; Miller & Hester, 2003; Wallack, 1984).

The terms *medical* or *disease model* are used synonymously in the literature (Miller & Hester, 2003; Miller & Kurtz, 1994; Peterson et al., 2003). The medical model, which is physician driven, is perhaps the dominant model among providers to treat those with alcohol and drug problems (Brower, Blow, & Beresford, 1989; Keller, 1990; Leonelli, 1993; Miller & Hester, 2003). In accord with this model, medical treatments (e.g., detoxification as the standard first stage regimen) and pharmacotherapy (e.g., disulfiram for alcohol users, naltrexone for drug users, and tricyclic antidepressants for alcohol and/or drug users) are often used to reduce craving, change behavior, and diminish anxiety and depression (McNeece & DiNitto, 2005; Torrens, Fonseca, Mateu, & Farre, 2005).

Psychological models, and especially psychotherapeutic methods, are nonpharmacological approaches utilized in alcohol and drug treatment. These treatment approaches, which are under the auspices of mental health professionals, focus on individual or family change and consist of long-term psychotherapy (individual, group, or combination) (Institute of Medicine, 1990; Kissin, 1977; Lindstrom, 1992; Miller & Hester, 2003). Psychological models may include

such approaches as motivational, social learning (behavioral model), stages of changes (transtheoretical model), family therapy, and brief interventions (DiClemente, 2003; Johnson, 2004; McNeece & DiNitto, 2005; Miller & Hester, 2003; Peterson et al., 2003; Prochaska & DiClemente, 1992).

In the sociocultural models, the treatment foci are on both the person and the social and physical environments, employing a variety of techniques such as environmental and social restructuring (e.g., changing the person's drinking behavior by creating new social relationships), changing the social environment by providing alternative living arrangements (such as a halfway house), or involvement with self-help groups (like Alcoholic Anonymous [AA] or other mutual self-help groups) (Institute of Medicine, 1990; Lindstrom, 1992; McNeece & DiNitto, 2005; Miller & Hester, 2003; Peterson et al., 2003).

Finally, integrative/biopsychosocial models strive to address alcohol/drug problems on various levels, that is, physiological, psychological, or sociological. An example is dual diagnosis, which integrates the self-medication and medical (disease) models to treat both depression and chemical dependency (Brower et al., 1989). The Minnesota model also has been seen by proponents as integrating alcohol/drug problems at these various levels (Anderson, 1981; Johnson, 2004; Laundergan, 1982; Peterson et al., 2003; Spicer, 1994). Curiously, this approach is regarded as limiting because it includes everything in the diagnosis and treatment at all levels, which then may be excessive (Oakley & Ksir, 1999; Peterson et al., 2003).

Summary of Model Elements

A summary of key model elements, including strengths and weaknesses of each, is included (Table 14.1). In examining the relative advantages and disadvantages of a given approach toward substance abuse treatment, the evolutionary and profession-driven manner in which treatment models have arisen is highlighted. One model's weakness will be addressed strongly in a second model, but in so doing the latter model suffers by taking a less thorough tact on a key treatment component of the former. For instance, a strength of the psychological models lies in their focus on the resolution of individual personality conflicts, a dimension addressed inadequately by sociocultural models, which instead stress social and physical environments (at the expense of handling individual psychological issues thoroughly). Similarly, a major weakness of the psychological models is their tendency to blame the victim; this consideration becomes a strength with the moral and sociocultural models, in which users are held responsible for solutions (but are not blamed for having the problem in the first place). In addition, sociocultural models have a tendency to blame

Table 14.1
Summary of Model Elements

Model	Strength	Weakness	Treatment goal	Type of staff
Moral/prevention	User is responsible	Punitive	Increase willpower, promote moderate drinking	Clergy, law enforcement, public health workers
Medical/disease	Blame the disease not the person	Focus on the biological	Abstinence, methadone, maintenance, and medications	Physicians, nurses, counselors
Psychological	Resolving personality conflicts	Blaming the victim	Improve psychological health and self-control through learning new behaviors	Mental health professionals (psychologists and therapists)
Sociocultural	Focus on individual, family, social, and physical environment	Blaming others and not taking responsibility for solutions	Improve individual and social functioning by changing coping responses of users and environments	Recovery persons, counselors, social workers
Integrative/biopsychosocial	Addresses biological, psychological, and social factors of the client	Lack of resources	Assist users with personality restructuring and change social conditions	Physicians, nurses, psychotherapists, social workers, counselors, recovery persons

others for the problems, while psychological models address problems by striving to resolve some personality-related issues. Finally, although both sociocultural and integrative models recognize the contribution of social and physical environments in developing and sustaining alcohol/drug problems, political and economic constraints generally result in a situation in which accountability for solution-finding still sits at the individual level. In fact, a lack of resources (a good example being the lack of treatment slots for pregnant women with children) is a key weakness of integrative models; and under these circumstances, holding alcohol/drug users responsible for solutions seems quite untenable. In effect, these treatment models at best can be used to treat only some individuals and families, even though their elasticity ranges from simply treating the individual to addressing the social and physical environments as well. It is also

possible that, should a combination of models be used simultaneously in treatment programs, the strengths and weaknesses of the models could be in fundamental conflict with or cancel each other out so much that very little would be left to utilize, and mixed treatment messages could potentially be puzzling for both clients and providers. For example, holding users responsible in the moral model is in fundamental conflict with the disease model's focus on causes outside an individual's control (Nunes-Dinis, 1996).

WHAT MODELS DO SERVICE PROVIDERS CHOOSE?

Models of alcohol and drug addiction, and their treatment, have been constructed from theoretical dimensions of various professional disciplines, but in practice the use of a given treatment does not necessarily follow a provider's professional orientation. The relationship between treatment model application and provider discipline is an understudied area. Generally, studies instead present professionals' views on the disease concept and opinions about their primary treatment goal; few have considered the providers' choice of alcohol and drug treatment models or approaches. However, there are many studies that examine staff or provider beliefs about addiction treatment and their subsequent selection of treatment models and approaches.

Staff or Provider Beliefs about Addiction Treatment

While there are significant limitations with regard to previous studies' validity (Nunes-Dinis, 1996), scholars generally agree that beliefs congruent with the medical/disease model or with assumptions that hold alcoholics as responsible for their disease are common among a variety of medical and mental health professions (Kahle & White, 1991; Mackinem & Higgins, 2007; Miller & Frances, 1986; Ogborne, Rush, & Ekdahl, 1986; Palm, 2006; Schwartz & Taylor, 1989; Weisner, 1981).

In terms of providers' views toward the goal of treatment, almost without exception, the medical and mental health professionals support complete abstinence (Kahle & White, 1991; Lee, 1983; Miller & Frances, 1986; Schwartz & Taylor, 1989). Endorsement is not quite as universal among alcohol treatment agencies (outpatient, inpatient or mixed, halfway houses, and prevention/education), although abstinence as a treatment goal was endorsed by over three-quarters of the respondents in another study (Rosenberg & Davis, 1994). Turning to differences based on whether an agency offers services only for alcoholism or offers more general social services, Weisner (1981) found that providers in alcohol-specific agencies endorse abstinence, while those in non-

alcohol-specific agencies were more likely to recognize and treat the underlying disorders (psychological and sociological) that providers in those agencies believed cause the alcohol problem. However, changes in the nature of alcohol treatment service delivery in both public and private sectors (with many agencies now delivering combined alcohol and drug treatment services) may affect contemporary providers' views of treatment goals. In addition, there has been a decrease in public sector programs, which may further modify the treatment goals across agencies.

Previous studies, using a variety of sample frames, have found some differences in choice of treatment approach according to providers' particular profession (Cutler & Storm, 1973; Knox, 1973; Lee, 1983; Miller & Frances, 1986; Morgenstern & McCrady, 1992; Rosenberg & Davis, 1994; Weisner, 1981). For instance, Lee (1983) found that psychologists were more likely than psychiatrists and paraprofessionals to support the behavioral model, while psychiatrists (also Miller & Frances, 1986) and paraprofessionals supported the disease model; doctoral-level experts were more likely to choose both behavioral and disease models (as advocated by AA) (Morgenstern & McCrady, 1992); and AA was the preferred approach among staff at alcohol treatment programs (Rosenberg & Davis, 1994). Humphreys, Noke, and Moos (1996) noted that staff members in recovery did not necessarily endorse the disease and psychosocial (learned behavior) models but an eclectic approach to treat substance abuse problems.

These studies carry with them a number of limitations. Some do not identify the treatment model or do not control for variables that may explain model choice; others have low response rates or are otherwise difficult to generalize; and most fail to explain why some modalities are used more often than others. However, without controlling for professional training or background, beliefs, as well as other variables such as number of years in practice or type of work setting, it is difficult to ascertain the reasons for, or the meaning of, these findings. For example, psychologists may support the behavioral model and controlled drinking more than psychiatrists do because they work more with outpatient rather than residential or hospital clients. Alcohol counselors may support the disease model and abstinence because most are themselves recovering persons in AA and strongly endorse the AA approach. Non-alcohol-specific agencies may adhere to a variety of models but give preference to the psychological, reflecting their professional background training in a way that is not as evident in alcohol-specific agencies (which may not have resources to train their staff on other models).

Further, Knox's (1973) findings in the Veterans Administration (VA) regarding the dominance of AA in that system are important, but it is difficult

to generalize from social workers working in the VA to others in a different work setting. Knox's (1973) study neither explains the effect of working for VA nor does it compare the results with social workers working in other settings. It is probable that the alternatives available to social workers working with alcoholics in the VA are quite different than the options other social workers have when working in a different setting (e.g., probation, jail, mental health agencies, clinics, or community organizations).

More recently, Willengring et al. (2004) reporting on the beliefs about evidence-based practices among providers (physicians, social workers, psychiatric staff, nurses, and addiction counselors) in the VA program, noted that 93 percent of the respondents in that system routinely recommend integrated treatment of psychiatric disorders and cognitive behavioral relapse prevention whereas over half (55%) do not recommend verbal confrontation. A third of these VA providers also did not recommend disulfiram to treat alcohol dependence, nor did they know whether to recommend a methadone dose of equal to or greater than 60 mg/day. It is relevant to note that more than 85 percent of the programs were outpatient and continuing care, with 64 percent of the patients receiving self-help groups, less than 30 percent participating in psychotherapy, and 73 percent receiving urine toxicology screening. The main barriers perceived by the VA providers to implement evidence-based practices in addiction treatment were lack of administrative support (50%), insufficient staff time (49%), and lack of skills or knowledge (38%).

As a final example from previous studies, the finding that self-help groups are used more frequently among psychiatrists (Miller & Frances, 1986) may reflect psychiatrists' belief that such groups are very effective, or it may simply be a pragmatic approach to serving a poor population who cannot afford other types of treatment. Of note in that study is the finding that about three-quarters of the staff respondents indicated that life events and practical experiences were the major reasons they became interested in alcohol and drug dependency, demonstrating potential confounders that make it difficult to explain modality or concept choice among providers in these studies.

More recent studies have also examined counselor treatment philosophies. Thombs and Osborn (2001) found that client-directed counselors who thought the causes of addiction were unique to each individual client were more likely to be white and have more advanced professional certification and educational degrees compared to counselors who endorsed a mix of psychological and disease factors involved in causing addiction. The study conducted by Moyers and Miller (1993) on the philosophical perspectives of alcoholism counselors found that therapists in recovery were more likely to support the disease model of addiction and those with higher educational levels adhered

to a belief of multiple causes for addiction. Taxman, Simpson, and Piquero (2002) examined the relationship between perspectives on the causes of drug abuse and endorsing a particular treatment technique. They found that the social learning perspective was supported the most, followed by social control and labeling perspectives; however, their interventions were not necessarily consistent with these approaches. Instead, the counselors endorsed sociological theories of social control, social learning, and strain for treatment interventions. Then, Taxman and Bouffard (2003) conducted another study on the perceptions of the causes and interventions by counselors on services delivered to offenders in drug court programs. Although counselors support a number of different causes of substance abuse (disease model, cognitive behavioral deficits, psychopathic personality traits and antisocial values, and social learning and social control issues), they tend to attribute the cause of substance abuse within the individual personality or their experiences compared to societal-level influences or even moral failings. While the counselors support a number of different causes of substance abuse, they do not endorse a single treatment approach. Instead, they adhered to the use of eclectic models (cognitive-behavioral strategies, education and aftercare, environmental safety [psychological and physical] and self-exploration, and self-help or peer support therapeutic community) that they believe were effective to treat clients. These results show that counselors do not have a preference for the types of components they believe are effective, and the knowledge and beliefs they have are not necessarily translated into their choices for treatments. Throughout the literature, we have often found that a provider's beliefs and subsequent choice of treatment model are often inconsistent—they believe in one perspective of causation for addiction and yet select other type of approach to treat the addiction (Kahle & White, 1991; Knox, 1969, 1971, 1973; Taxman & Bouffard, 2003; Taxman et al., 2002; Wechsler & Rohman, 1982). Consequently, mixed messages are likely to be delivered to clients between the belief systems providers hold and their subsequent selection of eclectic evidence-based treatment approaches, which may affect long-term success of different treatment interventions. The lack of knowledge about selecting every evidence-based practice approach may translate into delivering very little, especially in short treatment sessions with limited focus and brief discussions on topics. This may result in treatment programs lacking clearly defined goals and objectives and the need to focus on skill development for counselors (Taxman & Bouffard, 2003).

There is a need to understand provider's treatment philosophies to gauge how they adopt and use evidence-based practices to deliver services. A series of studies has shown providers select treatment approaches differently across settings, job categories, and levels of education (Ball et al., 2006; Forman, Bovasso,

& Woody, 2001). Forman et al. (2001) surveyed staff members (i.e., counselors, physicians, nurses, social workers, case managers, and assistants) on their beliefs about addiction treatment. The 18-item survey was on beliefs about addiction treatment modalities. The staff was from the Delaware Valley Clinical Trial Network with treatment settings in Pennsylvania, New Jersey, and Delaware. These treatment settings had been in operation at least 10 years within the network. The following were the types of treatment settings and their distribution in the study: hospital-based inpatient detoxification and rehabilitation programs (10%); non-hospital-based detoxification and rehabilitation centers (16%); outpatient and intensive outpatient drug-free clinics (40%); methadone maintenance clinics (10%); and freestanding recovery houses (24%). The primary demographic characteristics of the staff were: education level (master's level [30%], bachelor's level [17%], and MD/PhD level [11%]); major job category (42% were counselors/therapists); and experience (28% of the staff had 11 or more years of work experience in the addiction field). Gender of the staff was not reported.

Although only 57 percent of the 317 staff completed the survey, more than 75 percent of the respondents endorsed items such as innovation (new approaches should be used more . . .), Twelve-Step/traditional treatment (addiction is really a disease, Twelve-Step programs and recovery houses should be used more . . .), and spirituality (spirituality should be emphasized more . . .) (Forman et al., 2001). There were differences of opinion found between staff members for various policies: (1) the policy to discharge noncompliant patients (36% in favor versus 40% in disagreement); (2) the use of medications (38% supported increased use versus 12%, and 46% who were specifically unsure about the role of naltrexone); (3) more staff (40%) opposed than supported (33%) using methadone maintenance; and (4) over half of the staff (52%) supported the increased use of psychiatric medications, while 20 percent did not. Nearly half of the staff (46%) believed that confrontational techniques should be used more often with clients, which is a strategy not recommended by the research community. This support was found among those with the least formal education. Those working in methadone maintenance clinics (89%) supported this approach as compared to only 20 percent of the staff working in drug-free clinics. Regardless of treatment setting, registered nurses and case managers were the most likely to disagree with increasing the use of methadone maintenance, and they were also the staff most involved with operating these clinics; physicians, in comparison, were in favor of increasing the use of methadone maintenance (Forman et al., 2001).

In another study, Ball et al. (2006) surveyed 66 clinicians from 11 community treatment programs who were mainly white (77%), female (65%),

highly educated (52% had a master's degree, and another 18% had a bachelor's degree); half of them had certification/licensure in drug/alcohol and 21 percent in social work, and they had an average of 7.1 years of counseling experience. In the clinician and supervisor survey, the clinicians rated the following theoretical orientations or techniques from *most* to *least* used in their practice: (1) relapse prevention/cognitive-behavioral; (2) Twelve-Step/disease concept; (3) Rogerian/client-centered; (4) reality therapy; (5) motivational interviewing; (6) psychodynamic/interpersonal; and (7) gestalt/experiential. But when they rated a client they had seen in the last six months, clinicians scored highest using the motivational enhancement therapy/motivational interviewing (MET/MI) approach. Clinician beliefs differed across gender and education. Female clinicians were less likely than males to view effective counseling as advice, guidance, suggestions, and reaching resistant clients. More males than females adhered to a cognitive-behavioral/relapse prevention approach. Those with a master's degree were more likely than others with lower levels of education to select a psychodynamic orientation and were less likely to adhere to the Twelve-Step/disease concept. As it might be expected, clinicians who self-identified to be in recovery themselves, compared to all others, reported a stronger allegiance to the Twelve-Step/disease concept and endorsed the importance of the abstinence goal. The counselors with a master's level of education selected more psychodynamic approaches in treating their clients, and recovering counselors incorporated more of the Twelve-Step principles.

In another study, Knudsen, Ducharme, Roman, and Link (2005) also found that counselors without a master's degree and those who adhered to a Twelve-Step treatment philosophy were less likely to accept and perceive the effectiveness of the buprenorphine medication to treat opiate addiction. A similar finding was noted by McGovern, Fox, Xie, and Drake (2004) that clinicians who endorsed a Twelve-Step model were less likely to adopt addiction medications. Similarly, Thomas, Wallack, Lee, McCarty, and Swift (2003) found that only 15 percent of physicians and 5 percent of nonphysicians report prescribing or recommending naltrexone medication often to treat alcoholism even though they scored high in the importance medications plays on the role of treating alcohol abuse. Those clinicians who recommend naltrexone believed the medication is effective while others do not. The barriers to adopt naltrexone that clinicians reported were: high cost, lack of knowledge about the medication, and insufficient evidence on its effectiveness. Clinicians who endorsed a Twelve-Step model were less likely to adopt addiction medications.

Additionally, disseminating empirically supported therapies through manual-guided approaches that emphasize best practice guidelines for treatment programs to utilize is regarded as being crucial to address the changes in health

care policy because of the need to improve standardized treatment procedures, cost-effectiveness, and outcome (Ball et al., 2006; Morgenstern, Morgan, McCrady, Keller, & Carroll, 2001). Distributing manual-guided therapies to providers is an important step to bridge the gap in the technology transfer process between researchers and practitioners; however, the infiltration of treatment manuals into standard clinical practice remains sparse because clinicians feel that structured manuals restrict their ability to respond to the individual needs of their clients (Ball et al., 2006). The gap between research and practice is considered wider in the addiction field than others because of the differences between the training, professional identification, and treatment philosophy or orientation of the researchers and practitioners. This gap may influence the success of one approach over another to transfer the technology and knowledge between researchers and practitioners. Matching types of training to types of clinicians may be a necessary procedure to ensure the success of a particular evidence-based intervention to use with alcohol/drug clients (Ball et al., 2006; Morgenstern et al., 2001).

Finally, Forman et al. (2002) surveyed 10 community-based addiction treatment organizations from the National Drug Abuse Clinical Trials Network, whose staff participated in an educational session about research practices and human subject protections. Pre- and postsession surveys were completed by 119 respondents: 13 clinical supervisors, 52 counselors, 15 case managers, 18 administrators, and 21 others, that is, psychiatry ($n = 1$), nursing ($n = 4$), social work ($n = 7$), clerical ($n = 6$), and other unidentified categories ($n = 3$). Prior to the educational session, 44 percent of the participants believed that clients could participate in clinical trials without understanding what would happen during the course of the study, 30 percent agreed that clients should be required to stay in a study even if they were doing poorly, and 55 percent believed clinicians had a right to transfer clients that were doing poorly in a clinical trial. After the educational session, 76 percent of the staff believed that clients participating in clinical trials had to be completely informed but almost one-quarter of them did not believe the right of informed consent would be protected in clinical trials. More than half (52%) of the staff believed that patients doing poorly could transfer out of a study. Seventy percent of the staff believed they had a right to transfer clients out of clinical trials. Even after the educational session, one-fourth of the staff believed that paying clients to participate in clinical trials could undermine their recovery, and only 29 percent of the staff agreed that clients' chances for recovery were improved by participating in a clinical trial. This study suggests that providers do not adequately recognize human subject protections built into clinical trials, the importance of informed consent, or even the relevance of their clients participating in a

clinical trial to improve their recovery—another gap that may widen and place a strain between researchers and practitioners. Similarly, Knudsen, Ducharme, Roman, and Link (2007), using data from the National Institute on Drug Abuse's Clinical Trials Network, reported a higher counselor turnover intention when they perceived that their job demands had increased due to research being conducted in community-based treatment organizations. This relationship was significantly lower if the treatment counselors perceived the research activities to result in improvements for clients and the organization.

PUBLIC TREATMENT AVAILABILITY

Regardless of how providers select treatment models/approaches or even the interplay of their belief systems and subsequent choices for treatment options, the availability of public treatment programs has been affected by a recognition of alcohol/drug abuse being considered a public health and safety problem as well as the changes in the management of health care during the 1990s (McLellan, Carise, & Kleber, 2003). By 2001, 55 percent of all clients referred to substance abuse treatment were from the criminal justice system, while the welfare system referred an additional 10 percent, and mental health and health clinics referred between 10 and 15 percent more individuals (*Drug Abuse Services Information System*, 2001). Referrals for clients who were coerced from these systems required professionals with extensive experience and a variety of medical and social service options generally not available in many of the treatment centers (Belenko, 1998; McLellan et al., 2003; Woolis, 1998). In the case of the managed health care system, by 2002, less than 10 percent of all treatment programs were residential or inpatient hospital, 12 percent were methadone maintenance programs, and 78 percent were abstinence-oriented outpatient programs (*National Survey of Substance Abuse Treatment Services*, 2001).

McLellan et al. (2003) interviewed the directors and staff from a representative sample of 175 alcohol/drug treatment programs. They found that less than half of the programs had a full-time physician or nurse on staff, and social workers or psychologists were rare in the treatment programs. Abstinence-oriented group counseling was the most common approach used by the staff to treat persons with alcohol/drug problems. Admission intake procedures ranged from two to four hours to collect data and fulfill the reporting requirements of managed care organizations and those of city, state, and federal agencies; these paperwork procedures were neither useful for clinical decision-making nor for program planning. Very few programs had a clinical information system to conduct or monitor care.

In recent years, there has been a development of effective medications and various forms of individual therapies that require trained clinical personnel and information management systems currently not available in most public treatment programs (McLellan et al., 2003; McLellan, O'Brien, Lewis, & Kleber, 2000; O'Brien, 1996). However, the pay is too low to attract high-level professional staff, staff turnover is high, closure and administrative reorganization of treatment programs is common, there is a lack of insurance or Medicaid reimbursement, and the technical infrastructure of programs is not funded adequately in order to operate clinics. Financial and technical investment is necessary to improve the regulating, monitoring, and credentialing of the public treatment system as well as provide incentives to raise the quality of those programs and attract excellent personnel (Appel, Ellison, Jansky, & Oldak, 2004; McLellan et al., 2003; Powell, 2006).

CONCLUSION

In sum, having treatment models or best-practices approaches for providers to select or treatment services for them to refer clients is only relevant if the programs exist in the first place. As McLellan et al. (2003) reported, 15 percent of the public facilities either closed or had stopped providing substance abuse treatment, and another 29 percent had been reorganized under a different administrative structure (i.e., mental health or another agency). This implies that what national treatment experts may consider state-of-the-art treatment will be scarce for the majority of public clients and only accessible to people who can afford to pay privately. The treatment-client community as a whole may not adequately benefit from the evidence-based knowledge professionals are currently developing or the gains made in substance abuse treatment strategies, including medications, until the treatment infrastructure is supported and the public demands quality of treatment care in the same way that it recognized that alcohol/drug abuse was associated with public health and safety problems. Funding public treatment programs sufficiently is a necessary step to address current infrastructure weaknesses and support our own acknowledgment that public health and safety problems are important concerns in society that need our attention and vital resources to maintain and utilize evidence-based practices in the treatment of addictions.

REFERENCES

Alexander, B. K. (1987). The disease and adaptive models of addiction: A framework evaluation. *Journal of Drug Issues, 17*(1), 47–66.

Anderson, D. J. (1981). *Perspectives on treatment: The Minnesota experience*. Center City, MN: Hazelden Foundation.

Appel, P. W., Ellison, A. A., Jansky, H. L., & Oldak, R. (2004). Barriers to enrollment in drug abuse treatment and suggestions for reducing them: Opinions of drug injecting street outreach clients and other system stakeholders. *American Journal of Drug and Alcohol Abuse, 30*(1), 129–153.

Ball, S., Bachrach, K., DeCarlo, J., Farentinos, C., Keen, M., McSherry, T., et al. (2006). Characteristics, beliefs and practices of community clinicians trained to provide manual-guided therapy for substance abusers. *Journal of Substance Abuse Treatment, 23*, 309–318.

Belenko, S. (1998). *Behind bars: Substance abuse and America's prison population*. New York: National Center for Addiction and Substance Abuse at Columbia University.

Brower, K. J., Blow, F. C., & Beresford, T. P. (1989). Treatment implications of chemical dependency models: An integrative approach. *Journal of Substance Abuse Treatment, 6*, 147–157.

Bukoski, W. J. (1991). A framework for drug abuse prevention research. In C. G. Leukefeld & W. J. Bukoski (Eds.), *Drug abuse prevention intervention research: Methodological issues* (DHHS Publication No. ADM 91-1761, NIDA Research Monograph 107, pp. 7–28). Rockville, MD: National Institute on Drug Abuse.

Chavkin, W. (1990). Drug addiction and pregnancy: Policy crossroads. *American Journal of Public Health, 80*(4), 483–487.

Cutler, R., & Storm, T. (1973). *Drinking practices in three British Columbia cities. III. Professional survey*. Vancouver, British Columbia, Canada: The Alcoholism Foundation of British Columbia.

DiClemente, C. (2003). Addiction and change: How addictions develop and addicted people recover. New York: Guilford Press.

Drug abuse services information system. (2001). Rockville, MD: Substance Abuse and Mental Health Services Administration.

Forman, R. F., Bovasso, G., & Woody, G. (2001). Staff beliefs about addiction treatment. *Journal of Substance Abuse Treatment, 21*, 1–9.

Forman, R. F., Bovasso, G., Woody, G., McNicholas, L., Clark, C., Royer-Malvestuto, C., et al. (2002). Staff beliefs about drug abuse clinical trials. *Journal of Substance Abuse Treatment, 23*, 55–60.

Hubbard, R. L. (1990). Treating combined alcohol and drug abuse in community-based programs. In M. Galanter (Ed.), *Recent developments in alcoholism: Vol. 8. Combined alcohol and other drug dependence* (pp. 273–284). New York: Plenum Press.

Humphreys, K., Noke, J. M., & Moos, R. H. (1996). Recovering substance abuse staff members' beliefs about addiction. *Journal of Substance Abuse Treatment, 13*(1), 75–78.

Institute of Medicine. (1989). *Prevention and treatment of alcohol problems: Research opportunities*. Washington, DC: National Academy Press.

Institute of Medicine. (1990). *Broadening the base of treatment for alcohol problems*. Washington, DC: National Academy Press.

Jellinek, E. M. (1960). *The disease concept of alcoholism*. New Brunswick, NJ: College and University Press and Hillhouse Press.

Johnson, J. L. (2004). *Fundamentals of substance abuse practice*. Belmont, CA: Brooks/Cole–Thompson Learning.

Kahle, D. B., & White, R. M. (1991). Attitudes toward alcoholism among psychologists and marriage, family and child counselors. *Journal of Studies on Alcohol, 52*(4), 321–324.

Keller, M. (1990). *Models of alcoholism: From days of old to nowadays* (Pamphlet series). New Brunswick, NJ: Center of Alcohol Studies, Rutgers University.

Kennedy, C., Finkelstein, N., Hutchins, E., Mahoney, J. (2004). Improving screening for alcohol use during pregnancy: The Massachusetts ASAP program. *Maternal and Child Health Journal, 8*(3), 137–147.

Kissin, B. (1977). Theory and practice in the treatment of alcoholism. In B. Kissin & H. Begleiter (Eds.), *The biology of alcoholism: Vol. 5. Treatment and rehabilitation of the chronic alcoholic* (pp. 1–51). New York: Plenum Press.

Knox, W. J. (1969). Attitudes of psychologists toward alcoholism. *Journal of Clinical Psychology, 25*(4), 446–450.

Knox, W. J. (1971). Attitudes of psychiatrists and psychologists toward alcoholism. *American Journal of Psychiatry, 127*(12), 1675–1679.

Knox, W. J. (1973). Attitudes of social workers and other professional groups toward alcoholism. *Quarterly Journal of Studies on Alcohol, 34,* 1270–1278.

Knudsen, H. K., Ducharme, L. J., Roman, P. M., & Link, T. (2005). Buprenorphine diffusion: The attitudes of substance abuse treatment counselors. *Journal of Substance Abuse Treatment, 29,* 95–106.

Knudsen, H. K., Ducharme, L. J., Roman, P. M., & Link, T. (2007). Research participation and turnover: An exploratory analysis of substance abuse counselors. *Journal of Substance Abuse Treatment, 33,* 211–217.

Laundergan, J. C. (1982). *Easy does it: Alcoholism treatment outcomes, Hazelden and the Minnesota model.* Minneapolis, MN: Hazelden Foundation.

Lee, H. B. (1983). The attitudes of professionals, paraprofessionals and nonprofessionals toward alcoholism and its treatment. Unpublished doctoral dissertation, University of South Dakota.

Leonelli, B. T. (1993). Provider expectations and alcoholism treatment: A clinical perspective. *Alcoholism Treatment Quarterly, 10*(1/2), 209–215.

Leukefeld, C. G., & Bukoski, W. J. (1991). An introduction to drug abuse prevention intervention research: Methodological issues. In C. G. Leukefeld & W. J. Bukoski (Eds.), *Drug abuse prevention intervention research: Methodological issues* (DHHS Publication No. ADM 91-1761, NIDA Research Monograph 107, pp. 1–6). Rockville, MD: National Institute on Drug Abuse.

Lindstrom, L. (1992). *Managing alcoholism: Matching clients to treatments*. New York: Oxford University Press.

Mackinem, M. B., & Higgins, P. (2007). Tell me about the test: The construction of truth and lies in drug court. *Journal of Contemporary Ethnography, 36,* 223–251.

McGovern, M. P., Fox, T. S., Xie, H., & Drake, R. E. (2004). A survey of clinical practices and readiness to adopt evidence-based practices: Dissemination research in an addiction treatment system. *Journal of Substance Abuse Treatment, 26,* 305–312.

McLellan, A. T., Carise, D., & Kleber, H. D. (2003). Can the national addiction treatment infrastructure support the public's demand for quality care? *Journal of Substance Abuse Treatment, 25,* 117–121.

McLellan, A. T., O'Brien, C. P., Lewis, D. L., & Kleber, H. D. (2000). Drug addiction as a chronic medical illness: Implications for treatment, insurance and evaluation. *Journal of the American Medical Association, 284,* 1689–1695.

McNeece, C. A., & DiNitto, D. M. (2005). *Chemical dependency: A systems approach* (3rd ed.). Boston: Allyn and Bacon.

Miller, S. I., & Frances, R. (1986). Psychiatrists and the treatment of addictions: Perceptions and practices. *American Journal of Drug Alcohol Abuse, 12*(3), 187–197.

Miller, W. R., & Hester, R. K. (1989). Treating alcohol problems: Toward an informed eclecticism. In R. K. Hester & W. R. Miller (Eds.), *Handbook of alcoholism treatment approaches: Effective alternatives* (pp. 3–13). New York: Pergamon Press.

Miller, W. R., & Hester, R. K. (2003). Treating alcohol problems: Toward an informed eclecticism. In R. K. Hester & W. R. Miller (Eds.), *Handbook of alcoholism treatment approaches: Effective alternatives* (3rd ed., pp. 1–12). Boston: Allyn and Bacon.

Miller, W. R., & Kurtz, E. (1994). Models of alcoholism used in treatment: Contrasting AA and other perspectives with which it is often confused. *Journal of Studies on Alcohol, 55*(2), 159–166.

Morgenstern, J., & McCrady, B. S. (1992). Curative factors in alcohol and drug treatment: Behavioral and disease model perspectives. *British Journal of Addiction, 87,* 901–912.

Morgenstern, J., Morgan, T. J., McCrady, B. S., Keller, D. S., & Carroll, K. M. (2001). Manual-guided cognitive-behavioral therapy training: A promising method for disseminating empirically supported substance abuse treatments to the practice community. *Journal of Addictive Behaviors, 15,* 83–88.

Morse, B., Greshan, S., Hutchins, E. (1997). *Screening for substance abuse during pregnancy: Improving care, improving health.* Arlington, VA: National Center for Education in Maternal and Child Health.

Moyers, T. B., & Miller, W. R. (1993). Therapists' conceptualizations of alcoholism: Measurement and implications for treatment decisions. *Psychology of Addictive Behaviors, 7*(4), 238–245.

Musto, D. F. (2002). Recovery from alcoholism. In D. R. Musto (Ed.), *Drugs in America: A documentary history* (pp. 158–159). New York: New York University Press.

National Institute on Drug Abuse. (1999). *Principles of drug addiction treatment: A research-based guide* (NIH Publication No. 00-4180). Bethesda, MD: Author.

National survey of substance abuse treatment services. (2001). Rockville, MD: Substance Abuse and Mental Health Services Administration.

Nunes-Dinis, M. C. (1996). An analysis of model choice of service providers using Scott's and Lipsky's theoretical frameworks: Women (pregnant and non-pregnant) and men drinkers. Unpublished doctoral dissertation, University of California, Berkeley.

Oakley, R., & Ksir, C. (1999). *Drugs, society and human behavior.* Boston: McGraw-Hill.

O'Brien, C. P. (1996). Recent developments in the pharmacotherapy of substance abuse. *Journal of Consulting and Clinical Psychology, 64,* 677–686.

Ogborne, A. C., Rush, B., & Ekdahl, A. (1986). The views of health and social service professionals regarding alcohol problems and their treatment. *The International Journal of the Addictions, 21*(9/10), 1051–1058.

Palm, J. (2006). Priorities in Swedish alcohol and drug treatment: Policies, staff views, and competing logics. *Contemporary Drug Problems, 33*(3), 367–398.

Peterson, J. V., Nisenholz, B., & Robinson, G. (2003). *A nation under the influence: America's addiction to alcohol.* Boston: Allyn and Bacon.

Powell, D. J. (2006). It's time for a national approach on staff development: The substance abuse field needs to harness existing training resources to prepare for a major personnel crisis. *Behavioral Healthcare, 26*(3), 42–45.

Prochaska, J., & DiClemente, C. (1992). Stages of change in the modification of problem behaviors. In M. Hersen, R. M. Eisler, & P. M. Miller (Eds.), *Progress in behavior modification* (pp. 184–214). Sycamore, IL: Sycamore Press.

Roche, A. M., & Richard, G. P. (1991). Doctors willingness to intervene in patient's drug and alcohol problems. *Social Science and Medicine, 33,* 1053–1061.

Rosenberg, H., & Davis, L. A. (1994). Acceptance of moderate drinking by alcohol treatment services in the United States. *Journal of Studies on Alcohol, 55,* 167–172.

Saunders, W. B. (1988). *Dorland's illustrated medical dictionary* (27th ed.). Philadelphia: PA: W. B. Saunders Company.

Schwartz, L. S., & Taylor, J. R. (1989). Attitudes of mental health professionals toward alcoholism recognition and treatment. *American Journal of Drug Alcohol Abuse, 15*(3), 321–337.

Siegler, M., & Osmond, H. (1968). Models of drug addiction. *The International Journal of the Addictions, 3*(1), 3–24.

Siegler, M., Osmond, H., & Newell, S. (1968). Models of alcoholism. *Quarterly Journal of Studies on Alcohol, 29*(3), 571–591.

Spicer, J. (1994). The Minnesota model: The evolution of the multidisciplinary approaches to addiction recovery. Center City, MN: Hazelden Foundation.

Taxman, F. S., & Bouffard, J. A. (2003). Substance abuse counselors' treatment philosophy and the content of treatment services provided to offenders in drug court programs. *Journal of Substance Abuse Treatment, 25,* 75–84.

Taxman, F. S., Simpson, S., & Piquero, N. (2002). Measuring and calibrating therapeutic integration in drug treatment programs. *Journal of Criminal Justice, 30*(3), 159–173.

Thomas, C. P., Wallack, S. S., Lee, S., McCarty, D., & Swift, R. (2003). Research to practice: Adoption of naltrexone in alcoholism treatment. *Journal of Substance Abuse Treatment, 24,* 1–11.

Thombs, D. L., & Osborn, C. L. (2001). Cluster analytic study of clinical orientations among chemical dependency counselors. *Journal of Counseling and Development, 79*, 450–458.

Torrens, M., Fonseca, F., Mateu, G., & Farre, M. (2005). Review: Efficacy of antidepressants in substance use disorders with and without comorbid depression. A systematic review and meta-analysis. *Drug and Alcohol Dependence, 78*, 1–22.

Wallack, L. (1984). Practical issues, ethical concerns and future directions in the prevention of alcohol-related problems. *Journal of Primary Prevention, 4*(4), 199–224.

Wechsler, H., & Rohman, M. (1982). Future caregivers' views on alcoholism treatment: A poor prognosis. *Journal of Studies on Alcohol, 43*(9), 939–955.

Weisner, C. (1981). Community response to alcohol-related problems: A study of treatment providers' perceptions. Berkeley, CA: Alcohol Research Group.

White, W. L. (1998). *Slaying the dragon: The history of addiction treatment and recovery in America*. Bloomington, IL: Chestnut Health Systems/Lighthouse Institute.

Willengring, M. L., Kivlahan, D., Kenny, M., Grillo, M., Hagedorn, H., & Postier, A. (2004). Beliefs about evidence-based practices in addiction treatment: A survey of Veterans Administration program leaders. *Journal of Substance Abuse Treatment, 26*, 79–85.

Woolis, D. D. (1998). Family works: Substance abuse treatment and welfare reform. *Public Welfare, 56*(1), 24–32.

Chapter **15**

Brief Telephone-Based Interventions for Clients with Alcohol and Other Drug Abuse Problems

Thomas Beers, PhD, Melinda Hohman, PhD, Katharine Slack, MSW, Daniela Sisneros, MSW, and Susan I. Woodruff, PhD

Alcohol and other drug (AOD) abuse and dependency continue to be major problems in the United States as they contribute to elevated health risks related to cardiovascular disease, stroke, pancreatitis, cirrhosis, and various types of cancer (Li, Hewitt, & Grant, 2007). Other problems associated with AOD abuse and dependency include violence, family dysfunction, poverty, poor work performance, and criminal behavior (Compton, Thomas, Stinson, & Grant, 2007).

AOD abuse and dependency are often combined together when studying problems, issues, and rates but are usually defined as distinct categories for intervention or treatment purposes. The *Diagnostic and Statistical Manual-IV-TR* defines AOD abuse as "maladaptive patterns of substance use [or alcohol use] leading to clinically significant impairment or distress . . . it is manifested by one or more of the following symptoms: (1) recurrent use [or drinking] resulting in failure to fulfill major role obligations at work, school, or home; (2) Recurrent use [or drinking] in situations in which it is physically hazardous; (3) Recurrent substance- [or alcohol] related legal problems; or (4) Continued substance [or alcohol use] despite persistent or recurrent social or inter-personal problems or exacerbated by the effects of alcohol" (American Psychiatric Association [APA], 2000, pp. 198-199, 214). AOD dependence is similar to abuse except that that the definition adds tolerance, withdrawal effects, and drinking (or using drugs) in larger amounts or over a longer time than was intended (APA, 2000). There are some who question these as distinct categories and propose that alcohol use disorders fall more along a continuum,

where the various criteria could fall in different places than those proposed by the *DSM-IV-TR* (Helzer, Bucholz, & Gossop, 2007; Saha, Stinson, & Grant, 2007). Also, quantifying the amount ingested at one sitting may be relevant for diagnosis. For instance, alcohol abuse has been defined as a pattern of drinking that brings the blood alcohol level (BAL) to .08 or above, which is typically five drinks for men and four for women (Li et al., 2007; Saha et al., 2007).

In 2001–2002, the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) (N = 43,093) found that 65 percent of those surveyed over the age of 18 drank alcohol; alcohol abuse (meeting *DSM-IV* diagnostic criteria) over the past year was 6.93 percent of males and 2.55 percent of females, and drug abuse was 2.0 percent in males and .8 percent in females. Lifetime rates of AOD abuse were even higher, with 24.6 percent of males and 11.5 percent of females abusing alcohol and 10.6 percent of males and 5.2 percent of females abusing drugs (Compton et al., 2007; Grant et al., 2004).

In the NSEARC study, only 8.1 percent of the sample who were AOD abusers had ever obtained any kind of treatment. When they did seek treatment, it was more likely to be from a private physician or some other health care provider (Compton et al., 2007). Because of this and other prior research, it was felt that screening patients in emergency departments and primary care clinics while they wait for care may be a way to identify those who are high-risk drinkers or drug users in what may be a teachable moment and to provide a brief intervention to reduce AOD use and concomitant problems (Gentilello et al., 1988; Solberg, Maciosek, & Edwards, 2008). These formal screenings and intervention projects, or SBIRT (Screening, brief intervention, and referral to treatment), were funded in 2003 in 6 states by the Substance Abuse and Mental Health Administration (SAMSHA), with current contracts with 10 states and 12 university-based sites. As of August 2007, over 536,000 screenings had been performed (SBIRT, 2008).

SCREENING AND BRIEF INTERVENTIONS

SBIRT is defined as,"a comprehensive, integrated, public health approach to the delivery of early intervention and treatment services for persons with substance use disorders and those at risk" (Babor, McRee, Kassebaum, Grimaldi, & Bray, 2007, p. 7). The brief screening and intervention follows four steps. First, health educators, physicians, or other health professionals raise the issue of alcohol and drug use and ask permission from patients to discuss these topics with them. Next, patients are screened using the Alcohol, Smoking, and Substance Involvement Screening Test or ASSIST (WHO ASSIST Working Group, 2002) and the results of their scores are fed back to the patient. Third,

using motivational techniques (Miller & Rollnick, 2002), patients are asked what they make of those scores, how ready they are to change their AOD use, and to explore the benefits and concerns of this use. Finally, patients are asked to negotiate a goal or determine their next step regarding reduction of AOD use. If the screening has found them to be in a no- or low-risk category, patients are encouraged to maintain this level. Those who are at risk in their use are given a brief intervention on site and advised to cut down. Those who are at high risk (AOD abusers) are given a referral for free, outpatient counseling, which is described later in this chapter. Those who are at severe risk (AOD dependent) are referred for traditional outpatient or residential AOD counseling (Academic ED SBIRT Research Collaborative, 2007; Babor et al., 2007). The whole procedure takes about 5–15 minutes (Bertholet, Daeppen, Wietlisbach, Fleming, & Burnand, 2005).

CASBIRT

The California Screening, Brief Intervention, and Referral to Treatment (CASBIRT) program is one of several national SBIRT demonstration projects funded by the Substance Abuse and Mental Health Services Agency (SAMHSA). CASBIRT was established in 2002 and funded until 2006. It was refunded in June 2007, and is currently administered by the California Department of Alcohol and Drug Programs (DADP), managed by the county of San Diego Alcohol and Drug Services Department, and implemented by San Diego State University School of Social Work's Center for Alcohol and Other Drug Studies and Services. As of March 2008, over 20,000 screenings have been attempted in 12 hospital/clinic sites. Screenings were completed for approximately 65 percent of the patients (15,000), with most incomplete screenings due to the patient being too ill or injured to be fully interviewed. The current screening sites include emergency, trauma, burn departments, and a primary clinic. The screenings and brief interventions are provided by 15 health educators and three supervisors, all of whom are bilingual in Spanish. Health educators use the ASSIST to quickly determine the level of AOD use and risk category (i.e., no/low risk, at risk, high risk, or severe risk). Approximately 600 patients (4%) fell into the high-risk category and were referred to the Brief Treatment Program.

THE CASBIRT BRIEF TREATMENT PROGRAM

One of the core elements of CASBIRT is the brief treatment (BT) program. This program provides assessment and psychoeducational services to nonde-

pendent substance users who have been identified as being at *high risk* for future substance use problems during the screening process. Using a harm-reduction model, the goal of BT is to provide cognitive-behavioral skills and strategies to assist these clients in reducing the risk in their use of substances. This program was developed as there are few programs in the community that provide services for those who fall into the abuse category of AOD use. The program starts when the health educators (HEs) screen individuals as being at high risk for subsequent substance use problems. Those individuals categorized as high risk according to the ASSIST measure are encouraged by the HE to begin the BT program. The full BT program consists of three initial assessment/motivational sessions followed by psychoeducational sessions that cover up to six additional topics. The topics covered in BT are based on those used in previous research studies evaluating short-term cognitive-behavioral approaches, and include: (1) functional analysis; (2) dealing with urges to use; (3) refusal skills; (4) alternate pleasurable activities and support systems; (5) problem-solving and relapse prevention; and (6) termination and discharge assessment (Barry, 1999; Carroll, 1998; Monti, Abrams, & Cooney, 1989; Sampl & Kadden, 2001).

In the first session of BT, the HE meets face to face with the client and conducts a structured interview using the ASSIST to identify the client's risk level for future substance use problems. For those clients scoring in the high-risk level, the HE conducts an immediate Brief Intervention session. This consists of the HE providing feedback on the client's risk level, giving advice regarding the physician's recommended levels of drug and alcohol use, and asking for a commitment to reduce substance use to these levels. In addition the HE assesses the client's motivation to change and offers a referral for the client to speak with a counselor about continuing in the BT Program.

If the client accepts the referral, the HE arranges an in-person or telephone follow-up appointment with a counselor. At this first session, the counselor describes the BT Program, assesses the client's concerns and motivation to change, and explores the client's interest in setting up an orientation/intake appointment to continue in the program. This appointment and all subsequent sessions of BT may be conducted in-person or by phone depending on clients' preferences or needs.

At the orientation/intake appointment, the counselor discusses the client's concerns, describes the BT program, reviews the program consent form, and answers any questions the client may have. All BT counselors have specific training in the principles and techniques of motivational interviewing (MI) (Miller & Rollnick, 2002) and use this approach in all interactions with clients. At this point the counselor asks the client if he is ready to sign the consent that constitutes the intake to the BT program. If the session is conducted over

the phone, the counselor requests the client's permission to address the legal requirements of the program and record a verbal consent (the counselor will send the client a copy of the *Informed Consent*, *Audio Consent*, and a packet of session handouts). After the consent is completed, the client and counselor begin the assessment process with a brief review of the client's current pattern and history of substance use. Finally the counselor and client set initial goals for participation in the program using the *Personal Goal Worksheet* (Miller & Rollnick, 2002).

The third session of BT is the extended evaluation. During this session the counselor and client review the impact that substance use has had on major areas of the client's life. To obtain and record this information the counselor conducts a structured interview using the Addiction Severity Index Lite (ASI-L) (McLellan, Luborsky, O'Brien, & Woody, 1980) with the client. The counselor and client assess high-risk situations for substance misuse by completing the *Challenging Situations Tool*. At the completion of the assessment the counselor provides feedback to the client as to her diagnosis according to *DSM IV-TR* (APA, 2000), reviews and updates the *Personal Goal Worksheet*, and discusses the next steps in the process.

Subsequent involvement in the BT program depends on the client's diagnosis. Counselors give feedback to clients who meet *DSM IV-TR* criteria for substance dependence that the physician's advice is for them to abstain. Since the BT program is designed to be used with nondependent substance users, the counselor offers to refer the client to more intensive community services to assist him in achieving abstinence. Dependent clients who decline referrals to more intensive treatment and are not in need of medically managed care (e.g. detoxification, methadone maintenance) may continue in the BT program as long as the BT clinical director and counseling staff determine them to be appropriate for and profiting from services. Those dependent clients who are in need of medically managed services will be told that the BT program is not able to accommodate their needs, will be discharged from BT, and will be given referrals for the appropriate level of care.

Clients who meet *DSM IV-TR* criteria for co-occurring psychiatric disorders are encouraged to seek treatment for these conditions and will be given referrals for psychological/psychiatric treatment. Clients with co-occurring disorders may continue in BT if the BT clinical director and counseling staff determine that their psychiatric condition is stable and will not interfere with participation in the BT program. Those clients whose psychiatric conditions are judged by BT clinical director and counseling staff to be likely to interfere with the program will be discharged from BT and given referrals for community resources for treatment of both psychiatric and substance use disorders.

Counselors give clients who meet diagnostic criteria for substance abuse feedback on their diagnosis and start them on the aforementioned topics covered in BT. It is important to make clear that counselors will continue to assess clients' appropriateness for participation in BT throughout the program. Whenever the clinical director and counseling staff determine that a client is no longer appropriate for BT services, the counselor will discharge the client from BT and refer her to those community agencies that can best address the client's needs.

As previously mentioned, there are six additional topics covered in BT that constitute the cognitive-behavioral portion of the program. These topics consist of the functional analysis of the client's use of substances and four skill training topics along with a final treatment review/termination session. Each topic is designed to be covered in a single 45–60-minute session but may be continued to additional sessions if circumstances require. The counselors follow a manualized format for each session, which, while not a script, does present a recommended sequence of issues to be covered for each topic. The content and format of these sessions were drawn from previous work on brief interventions with substance use disordered patients (Carroll, 1998; Monti et al., 1989; Sampl & Kadden, 2001). The counselors incorporate the principles of MI in their work with clients and utilize MI techniques and interventions throughout the BT program.

In the functional analysis session, the counselor describes the importance of understanding the role that substances play in the client's life. The counselor and client then use the Functional Analysis Worksheet (Jaffe, Brown, Korner, & Witte, 1988) to analyze a recent episode of the client's use of substances. In this process, they review the areas of: triggers for use, thoughts and expectations regarding use, patterns of use, and positive and negative consequences of use. The counselor points out how future sessions will focus on acquiring skills to address each of the areas in the functional analysis process and assigns homework for the client to complete between sessions. The homework for this topic consists of the client completing an additional functional analysis worksheet for another incident of substance use.

The next four skill training topics cover the areas of: coping with urges and impulses to use, learning refusal skills, acquiring alternate pleasurable activities/improving support systems, and learning problem-solving (D'Zurrilla & Goldfried, 1971) and relapse prevention techniques. During each session the counselor reviews the client's goals for use, his current usage of substances, and the level of motivation to reach his goals. For each topic the counselor first presents information regarding the importance of the skill in helping the client to reduce his risks in the use of substances. Next the counselor and client

practice the skill in the session and discuss any challenging situations the client anticipates for the next week. Finally the counselor assigns homework for the client to complete between sessions.

The final topic of BT is the termination/discharge assessment session. During this meeting, the counselor and client establish the client's current level of substance use, review her goals for reducing risks and assess what progress she has made while going through the program. The counselor and client then discuss next steps for the client to take in achieving/maintaining any gains she has made and what referrals may be helpful. Finally the counselor asks the client for feedback on what she found helpful in the BT program and any suggestions she might have for improvement.

BRIEF TELEPHONE-BASED COUNSELING

Offering counseling services by telephone is a promising new option for treatment. Despite the brief history of this approach, recent research has shown promising results for telephone counseling. Telephone therapy has evolved from primarily being used to provide crisis intervention or hotline services, to being paired with traditional face-to-face counseling, to today being offered as a stand-alone treatment (Reese, Conoley, & Brossart, 2006). Telephone services are now widely utilized in providing a number of health services and health behavior interventions (McBride & Rimer, 1999). The CASBIRT BT program offers sessions via phone to enable maximum patient access to brief intervention services. Telephone counseling services reach many patients who otherwise would be unable to attend an in-person office appointment.

Reese, Conoley, and Brossart (2002) found that over 82 percent of clients reported improvement on their presenting problem based on participation in telephone counseling. They also reported strong levels of satisfaction with telephone therapy. A later study by the same authors (2006) noted that more than half (58%) of the participants who had received both telephone and traditional face-to-face therapy preferred telephone counseling; 96 percent would be willing to receive counseling services by phone again, compared to 63.1 percent who would be willing to seek face-to-face counseling.

Research highlights several advantages of telephone counseling services. Clients report convenience, accessibility, control, and lack of inhibition as appealing aspects of telephone counseling (Reese et al., 2006). Clients appreciate the convenience of receiving counseling at home by phone, as well as being able to receive services the day they initiate contact with a providing agency; services are easily accessible. Clients have a part in controlling the process of the session, and some clients feel that receiving counseling services by phone

provides more privacy, and as a result they feel less inhibited in talking about sensitive issues.

As indicated previously, the CASBIRT BT program primarily utilizes MI and cognitive-behavioral therapy, offering services by telephone or in person. Much of the research on telephone counseling has incorporated MI or motivational enhancement techniques (Mermelstein, Hedeker, & Wong, 2003; Tedeschi, Zhu, Anderson, Cummins, & Ribner, 2005). One study examined both the use of MI and service delivery by telephone. Study results suggest that an adaptation of MI administered through telephone sessions may significantly reduce drinking for alcohol abuse or dependent male primary care patients (Brown, Saunders, Bobula, Mundt, & Koch, 2007).

Expressing empathy is one of four main principles of MI (Miller & Rollnick, 2002). MI is empathic in that the counselor listens to the client's feelings without judgment, criticism, or blame. The delivery of counseling services by telephone, as previously stated, may enhance empathy and rapport-building within the client-counselor relationship due to an absence of potential visual biases (Reese et al., 2002). The absence of visual biases in telephone services may also improve the potential for collaboration to occur, another major component of MI.

CASBIRT counselors' experiences with telephone counseling mirror much of the research to date. Phone services appear to reach those who otherwise would not be reached by traditional face-to-face counseling. Additionally, as the research suggests, many clients initially question the benefits of telephone counseling. Once clients have completed a session or two by phone, however, they appear to appreciate the convenience, privacy, and anonymity of telephone counseling. The majority of our clients (90%), when given the choice of meeting in person or by telephone, choose the latter.

It is also important to note that Reese et al. (2002) found no difference in the amount of therapeutic bonding or social influence, the most consistent predictor of therapeutic outcomes, in telephone counseling sessions versus face-to-face services. CASBIRT counselors also commented that the level of anonymity offered by phone sessions appeared to, in some cases, decrease the impact of differences such as age, gender, culture, or ethnicity on the rapport built in the client-counselor relationship. The therapists have also found that clients, whether in person or by telephone, tend to expect that the counseling will be traditional, confrontational, and abstinence-oriented in approach, and are often surprised to experience the MI approach with harm-reduction goals.

A challenge of telephone counseling is actually born of one of the advantages—lack of inhibition. As a result of clients feeling more privacy, and in turn less inhibition, a counselor may actually need to be more conscientious about

defining the boundaries of the therapeutic relationship. There are virtually no outside controls or cues to inform the counselor-client relationship. There is no office with a receptionist that the client visits once a week for an appointment—a client could be anywhere, doing anything, when a counselor calls for an appointment. The unique circumstances of delivering counseling services by telephone may call for stricter monitoring of transference and counter-transference issues because of the mode of service delivery. Further research could explore whether these aspects of telephone counseling would in fact create any major challenges to service delivery.

Telephone counseling also requires adjustment on the part of the therapist. Few clinicians receive any training on conducting counseling services by telephone. The telephone offers an entirely different therapeutic medium, and adjustments have been made in the training and practice of CASBIRT clinicians. For this project, the therapist training includes role-playing the various sessions sitting back-to-back with a "client" to practice the intervention and adapt it to this type of setting. Many of the visual cues that guide the clinician in building rapport and trust with a client are lost when counseling is done via the telephone. For instance, when a client is silent, the therapist may have difficulty interpreting what it means (sad? crying? upset? asleep?) and use more probing questions than would normally occur. Additionally, when conducting a telephone session, CASBIRT therapists tend to ask permission more often when transitioning from one topic to the next or probing a client's thoughts.

Many clients are accustomed to the face-to-face counseling model and are therefore reluctant to try telephone counseling. Reese et al. (2006) found that before receiving telephone counseling, fewer than half (44.3%) of the study's participants thought it would be helpful. CASBIRT counselors observe this in their clients as well. As previously mentioned, many clients initially question the benefits of telephone counseling but are quick to adjust to and often prefer this as a service delivery method.

CULTURAL NEEDS AND IMPLICATIONS

There is no one best or most effective way to counsel minority groups; however, certain considerations should be taken into account. According to Torres-Rivera, Wilbur, Phan, Maddux, and Roberts-Wilbur (2004), cultural aspects specific to minority groups are often overlooked by conventional Euro-American substance abuse counseling approaches, which suppose problems are a result of individual intrapsychic disorganization or dysfunction. Instead, a culturally specific counseling approach that allows for substance abuse problems to be explained from a multidimensional perspective should be applied

when working with minority groups. This section will mainly focus on working effectively with Latinos in a clinical setting, as they are the largest minority group served by CASBIRT in San Diego County.

Counselors engaging in a therapeutic relationship with Latinos should have knowledge about interpersonal etiquette specific to Latino cultural value orientations. When engaging with Latinos, it is appropriate to address them in a kind and respectful manner. Individual differences exist among Latinos; however, according to Santiago-Rivera, Arredondo, and Gallardo-Cooper (2002), the following value orientations are most common in Latino interpersonal relationships and should be applied when engaging with Latinos: *personalismo, respeto, dignidad, simpatía, confianza* and *cariño*. Showing *personalismo* is a demonstration to the client that he is more important than time or what is occurring at the moment. *Respeto* is one of the most important value orientations in the Latino culture and has several meanings. It is demonstrated by being compassionate towards the client's situation. *Respeto* also mean respecting authorities, family, and tradition. Counselors should also be aware that Latinos believe in *dignidad,* meaning that everyone is worthy and should be respected. *Simpatía* refers to someone who is considered a pleasant person. Latinos are attracted to individuals who are relaxed, friendly, and entertaining. *Confianza* refers to trust and closeness in a relationship. At first most Latinos are reserved; however, once trust has been established Latinos are known to form close bonds with those around them. Latinos also need the restrictions of confidentiality to be explained to them. Some Latinos may hold back on sharing information in fear that they might be reported to immigration authorities. Lastly, counselors working with Latinos should consider being *cariñosos,* as it is greatly admired in the Latino culture. *Cariño* in the Latino culture is expressed through verbal and nonverbal endearment messages, such as, greeting a Latino with a hug (Santiago-Rivera et al., 2002).

Most important when counseling Latinos is matching client and counselor characteristics to help ease and bring cohesion to the therapeutic relationship. Matching can be based on language, race, or culture, but language matching is critical because language barriers are often the reason why Latinos do not seek counseling or health services (Santiago-Rivera et al., 2002). Not conducting a counseling session in Spanish when necessary could result in an inconsistent assessment of the Latino client's feelings. Counselors should also determine the need for a translator by conducting an acculturation level assessment of the client. Counselors working with specific Latinos should be bilingual, with a strong grasp of the Spanish language, since many Latinos communicate with proverbs, metaphors, regionalism, and sayings particular to their culture

(Santiago-Rivera et al., 2002). All of the CASBIRT health educators are bilingual in Spanish and several of the BT staff are bilingual.

Several other counseling considerations to have when working with Latinos include assessing their spirituality and religious influence. Because of the strong belief in fatalism in the Latino culture, it is has been suggested by Sue and Sue (2008) that counselors not change this belief but rather comprehend it. Assessing for acculturation levels is also recommended when working with Latinos, since many Latinos hold on to their traditional beliefs while others assimilate to the main culture. Counselors working with Latinos should specifically assess for cultural and societal contributions to the problem. External factors such as poverty, lack of shelter, unemployment, English language deficiency, racism, lack of familiarity, interactions with bureaucracies, and fear of deportation can be great stressors that are contributing to the problem (Sue & Sue, 2008).

Brief telephone counseling has been shown to be promising when working with minority groups such as Latinos. According to Williams and Douds (2002), phone therapy allows for client control and anonymity, in addition to resolving geographic obstacles. Having to come into an office for substance abuse counseling can be shameful, whereas counseling over the telephone gives clients power to end the conversation if they feel or become uncomfortable. Another advantage of telephone counseling is that clients are able remain anonymous and control for the negative consequences of their self-disclosure (Williams & Douds, 2002). This is true for Latinos and other ethnic groups residing in the U.S. illegally, who may avoid services available to them for fear of possible deportation. Telephone counseling also resolves geographical obstacles to services for clients who lack mobility or who live far from where the counseling services are being offered.

Significant efforts have been made to improve culturally sensitive counseling, yet most counseling values and practices continue to be tailored to serve a portion of the population. Bilingual CASBIRT counselors have found that telephone counseling does not take away from their ability to apply their knowledge about the interpersonal etiquette specific to Latinos and that it even has additional value with certain Latino groups. Counselors have also observed an increase in the rate at which *confianza* develops between counselor and client. They note that the level of anonymity offered by phone sessions, in some cases, decreases the impact of differences such as age, gender, culture, or ethnicity on the rapport or *confianza* built in the client-counselor relationship. Overall, the CASBIRT experience has been promising when applying telephone counseling to Latinos; however, further research is still needed to fully explore the impact of telephone counseling on the counseling relationship.

INITIAL FINDINGS

CASBIRT data indicate that about 4 percent (n = 600) of the patients screened to date fell into the high-risk category and were encouraged by the HE to accept a call from a BT counselor. Half (n = 300) of these individuals agreed to have the BT counselor call them to set up an initial counseling appointment conducted either face-to-face or over the phone. BT counselors were able to contact 34 percent (approximately 100) of the 300 potential clients; the remaining 66 percent could not be conducted because of an incorrect/disconnected or nonworking phone number, or they did not return the BT counselor phone messages.

Fifty-two patients received at least one BT session. Two-thirds of these patients were male. The average age was 41, with a range from 20 to 65 years. About 40 percent of these patients were Hispanic/Latino; 16 percent of the patients had sessions delivered in Spanish. The mean number of sessions (i.e., dose) delivered was 2.76 sessions (SD = 2.8). Ninety percent of sessions were conducted over the telephone.

Analysis of 23 patients with pre-BT and post-BT effectiveness measures showed that daily alcohol consumption decreased significantly over time (t = 2.11, p <.05), and that confidence in reducing alcohol/drug use increased significantly over time (t = –2.005, p < .05). Linear regression analysis showed that a higher dose of sessions tended to be related to positive changes in confidence, accounting for about 10 percent of the variance; changes in daily consumption, however, was not related to dose. Other measures of effectiveness (e.g., frequency of alcohol consumption, importance of changing one's alcohol/drug use) showed changes in the desired direction but did not reach statistical significance due in part to the small sample size.

SUMMARY AND CONCLUSIONS

SBIRT programs were developed to provide a continuum of care for individuals varying in their levels of alcohol and drug use severity. Opportunistic screenings, brief on-the-spot interventions, and referrals to more intensive treatment are conducted in nontraditional settings, such as emergency departments while patients wait for care (SBIRT, 2008; Solberg et al., 2008). The brief treatment element is offered to those patients whose screening results indicate they are at high risk for AOD.

The CASBIRT BT program was developed to offer short-term, harm-reduction treatment services for high-risk AOD abusers who typically would not be treated by community-based services. BT counselors utilize MI principles and

cognitive-behavioral approaches to assist clients in reducing their use of substances. To minimize barriers to access, the program offers no-cost services, bilingual counselors, flexible scheduling, and phone or in-person sessions.

Telephone-based counseling was implemented as an alternative way to engage reluctant clients and to provide a resource for those who have other psychosocial issues, such as lack of transportation or difficult work hours. This too has proven to be a challenge as we have described. However, once clients get past their initial concerns about telephone-based counseling, they engage with the counselor and are willing to participate. Interestingly, Latino clients have responded well to the telephone-based services. BT counselors have had to amend their counseling styles by paying attention to cues such as silence and probing more often to determine clients' thoughts and feelings.

Despite the efforts to overcome the obstacles to access, treatment counselors have encountered both logistic and motivational problems engaging clients in brief treatment services. As mentioned in the "Initial Findings" section of this chapter, counselors were unable to contact a large percentage of referred clients who did not have a permanent address or access to a phone. This may be an unavoidable consequence of the relatively low socioeconomic status of a great deal of the clients targeted by the program. Many other clients that counselors did contact felt that their problems were not that bad and that they could modify their AOD use without treatment. To some extent this reluctance to participate in services may reflect the reality that many abuse-level clients have not yet thought much about these issues, are in an early stage of readiness of change (precontemplation), and are not prepared to initiate reductions in use. In this case the counselors focused on raising the clients' concerns about their use, reinforcing the clients' independent efforts to change, and leaving the door open for utilizing BT services in the future. Other reasons for clients' reluctance to participate in services may be related to their concerns about common societal attitudes regarding problematic AOD use. Among these is the cultural understanding of alcoholism/addiction as a disease, widespread confusion over the issues of abuse versus dependency, and commonly held beliefs that treatment is limited to abstinence-oriented goals for those who are dependent. As a result, abusers may not see themselves as needing help to achieve harm-reduction goals. From a public health perspective, this may mean public education programs need to teach that treatment is not just for alcoholics/addicts with the goal of abstaining from AOD use and attending self-help programs. Treatment needs to be more broadly conceptualized as including getting help to cut down to lower-risk drinking or use patterns.

Our preliminary experience indicates that telephone counseling is a viable treatment alternative to traditional face-to-face counseling. Research reveals

telephone service delivery as having a number of positive effects and results similar to in-person counseling (Brown et al., 2007; McBride & Rimer, 1999; Reese et al., 2002), and our limited data suggest positive changes in AOD behavior as a result of the BT. Because the approach is relatively new, there is much left to be discovered about telephone-delivered services. Future research could continue to explore the advantages and challenges of telephone counseling and formally evaluate the effects of the approach.

REFERENCES

Academic ED SBIRT Research Collaborative. (2007). An evidence-based alcohol screening, brief intervention and referral to treatment (SBIRT) curriculum for emergency department (ED) providers improves skills and utilization. In R. Saitz & M. Galanter (Eds.), *Alcohol/drug screening and brief intervention: Advances in evidence-based practice* (pp. 79–92). Birmingham, NY: Haworth Medical Press.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Babor, T. F., McRee, B. G., Kassebaum, p. A., Grimaldi, p. L., & Bray, J. (2007). Screening, brief intervention, and referral to treatment (SBIRT): Toward a public health approach to the management of substance abuse. In R. Saitz & M. Galanter (Eds.), *Alcohol/drug screening and brief intervention: Advances in evidence-based practice* (pp. 7–30). Birmingham, NY: Haworth Medical Press.

Barry, K. L. (1999). *Brief intervention and brief therapies for substance abuse* (TIP 34, DHHS Publication No. [SMA] 99–3353). Rockville, MD: Substance Abuse and Mental Health Services Administration, Center for Substance Abuse Treatment.

Bertholet, N., Daeppen, J. B., Wietlisbach, V., Fleming, M., & Burnand, B. (2005). Reduction of alcohol consumption by brief intervention in primary care: Systematic review and meta-analysis. *Archives of Internal Medicine, 165,* 986–995.

Brown, R. L., Saunders, L. A., Bobula, J. A., Mundt, M. P., & Koch, P. E. (2007). Randomized-controlled trial of a telephone and mail intervention for alcohol use disorders: Three-month drinking outcomes. *Alcoholism Clinical and Experimental Research, 31,* 1372–1379.

Carroll, K. M. (1998). *A cognitive-behavioral approach: Treating cocaine addiction* (NIH Publication Number 98–44308). Bethesda, MD: National Institute on Drug Abuse.

Compton, W. M., Thomas, Y. F., Stinson, F. S., & Grant, B. F. (2007). Prevalence, correlates, disability, and comorbidity of DSM-IV drug abuse and dependence in the United States. *Archives of General Psychiatry, 64,* 566–576.

D'Zurrilla, T. J., & Goldfried, M. R. (1971). Problem-solving and behavior modification. *Journal of Abnormal Psychology, 78*(1), 107–126.

Gentilello, L. M., Duggan P. , Drummond, D., Tonnesen, A., Degner, E. E., Fischer, R. P., et al. (1988). Major injury as a unique opportunity to initiate treatment in the alcoholic. *American Journal of Surgery, 156,* 558–561.

Grant, B. F., Dawson, D. A., Stinson, F. S., Chou, S. P., Dufour, M. C., & Pickering, R. P. (2004). The 12-month prevalence and trends in DSM-IV alcohol abuse and dependence: United States, 1991–1992 and 2001–2002. *Drug and Alcohol Dependence, 74,* 223–234.

Helzer, J. E., Bucholz, K. K., & Gossop, M. (2007). A dimensional option for the diagnosis of substance dependence in the DSM-V. *International Journal of Methods in Psychiatric Research, 16*(1), S24–S33.

Jaffe, A., Brown, J., Korner, p. , & Witte, G. (1988). *Relapse prevention for the treatment of problem drinking: A manual for therapists and patients*. New Haven, CT: University of Connecticut Health Center.

Li, T-K., Hewitt, B. G., & Grant, B. F. (2007). Is there a future for quantifying drinking in the diagnosis, treatment, and prevention of alcohol use disorders? *Alcohol and Alcoholism, 41*(4), 57–63.

McBride, C. M., & Rimer, B. K. (1999). Using the telephone to improve health behavior and health service delivery. *Patient Education & Counseling, 37,* 3–18.

McLellan, A. T., Luborsky, L., O'Brien, P. , & Woody, G. E. (1980). An improved diagnostic instrument for substance abuse patients: The Addiction Severity Index. *Journal of Nervous and Mental Diseases, 168,* 26–33.

Mermelstein, R., Hedeker, D., & Wong, S. C. (2003). Extended telephone counseling for smoking cessation: Does content matter? *Journal of Consulting and Clinical Psychology, 71,* 565–574.

Miller, W. R., & Rollnick, S. (2002). Motivational interviewing: Preparing people to change addictive behavior (2nd ed.). New York: Guilford.

Monti, p. M., Abrams, D. B., & Cooney, N. L. (1989). Treating alcohol dependence: A coping skills training guide in the treatment of alcoholism. New York: Guilford.

Reese, R., Conoley, C., & Brossart, D. (2002). Effectiveness of telephone counseling: A field-based investigation. *Journal of Counseling Psychology, 49,* 233–242.

Reese, R., Conoley, C., & Brossart, D. (2006). The attractiveness of telephone counseling: An empirical investigation of client perceptions. *Journal of Counseling and Development, 84,* 54–60.

Saha, T. D., Stinson, F. S., & Grant, B. F. (2007). The role of alcohol consumption in future classifications of alcohol use disorders. *Drug and Alcohol Dependence, 89,* 82–92.

Sampl, S., & Kadden, R. (2001). *Motivational enhancement therapy and cognitive behavioral therapy for adolescent cannabis users:5 Sessions*. Rockville, MD: Substance Abuse and Mental Health Services Administration.

Santiago-Rivera, A. L., Arredondo, p. , & Gallardo-Cooper, M. (2002). *Counseling Latinos and la familia: A practical guide.* Thousand Oaks, CA: Sage Publications.

Screening, brief intervention, and referral to treatment. (2008). Retrieved April 7, 2008, from http://www.sbirt.samhsa.gov/index.htm.

Solberg, L. I., Maciosek, M. V., & Edwards, N. M. (2008). Primary care intervention to reduce alcohol misuse: Ranking its health impact and cost effectiveness. *American Journal of Preventative Medicine, 34*(2), 143–152.

Sue, D. W., & Sue, D. (2008). *Counseling the culturally diverse: Theory and practice*. Hoboken, NY: John Wiley & Sons.

Tedeschi, G. J., Zhu, S. H., Anderson, C. M., Cummins, S. H. & Ribner, N. G. (2005). Putting it on the line: Telephone counseling for adolescent smokers. *Journal of Counseling and Development, 83*, 416–424.

Torres-Rivera, E., Wilbur, M. P., Phan, L. T., Maddux, C. D., & Roberts-Wilbur, J. (2004). Counseling Latinos with substance abuse problems. *Journal of Addictions and Offender Counseling, 25*, 26–42.

WHO ASSIST Working Group. (2002). The Alcohol, Smoking, and Substance Involvement Screening Test (ASSIST): Development, reliability, and feasibility. *Addiction, 97*, 1183–1194.

Williams, T., & Douds, J. (2002). The unique contributions of telephone therapy. In D. Lester (Ed.), *Crisis intervention and counseling by telephone* (pp. 57–63). Springfield, IL: Charles C. Thomas.

Chapter **16**

Screening and Brief Intervention for Alcoholics

Ramadugu Shashikumar, MD

SCREENING

Screening is the procedure that identifies a disease or disorder in its early or presymptomatic stages and provides efficient identification of those users of alcohol who need a more comprehensive diagnostic evaluation and treatment planning. By screening for hazardous and harmful drinkers among regular alcohol consumers we can identify high-risk alcohol users. (Pal, Jena, and Yadav, 2004). Hazardous drinking is a pattern of alcohol consumption that increases the risk of harmful consequences for the user or others; such drinkers are of public health significance despite the absence of any current disorder in the individual user. *Harmful use* refers to alcohol consumption that results in physical, mental health, and social health consequences (Babor, Campbel, Room, & Saunders, 1994; World Health Organization [WHO], 1993).

Social environmental influences, ignorance of knowledge of drinking limits, and the risk associated with alcohol consumption are known to contribute to problem drinking (hazardous and harmful). We should be concerned about these people on two counts. First, that although not all problem drinkers become dependent, the pathway to dependence is through problem drinking. Second, many of them are amenable to change and betterment. So screening these people offers an opportunity to take preventive measures (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001).

Screening is also important because many people who are at risk have or who are already experiencing alcohol-related problems do not seek treatment (Grant, 1997; Sobell & Sobell, 1993).

Alcohol use disorder represents more of a spectrum of illnesses, and that morbidity and mortality needs to be assessed on a continuum rather than as a dichotomous entity. There is high prevalence of harmful and dangerous drinking among otherwise asymptomatic patients in primary care settings, and a considerable burden of the disease is borne by them (Odd, 2003).

QUESTIONNAIRES

Screening can be done by using self-reporting questionnaires and measurement of biomarkers for alcohol. The former are easier to apply in a large population and several such are available. These are rapid, inexpensive, and often appealing for application in general populations. Some of the commonly used scales are the CAGE questionnaire, Michigan Alcoholism Screening Test (MAST), Alcohol Use Disorders Identification Test (AUDIT), AUDIT Consumption Studies (AUDIT-C), and Lubeck Alcohol Dependence and Abuse Screening Test (LAST) (Pal et al., 2004).

CAGE

CAGE, a mnemonic based on the first letter of a key word from each of the four items it measures is a rapid Screening-instrument. A score of two out of four indicates problem drinking (Ewing, 1984). See Questionnaire 1 in the Appendix.

MAST

MAST is a 25-item test (see Questionnaire 2 in the Appendix). It was originally developed for screening general population (Selzer, 1971). However, both CAGE and MAST have drawbacks in that they depend heavily on possibly unreliable self-reporting, they detect lifetime use rather than current use, they focus on dependent or nondependent use of alcohol, and they do not give quantity and frequency data (Schuckit & Irwin, 1988).

LAST

LAST was developed in a general hospital sample by combining CAGE and MAST; the questionnaire consists of seven dichotomous items (two from CAGE and five from MAST) and is scored without weightings, with a cut-off score of 2 (see Questionnaire 3 in the Appendix). However, it is more suitable in a hospital setting rather than in general population (Rumpf, Hapke, Dawedeit, Meyer, & John, 1998; Rumpf, Hapke, Meyer, & John, 2002).

AUDIT-C

The AUDIT-C includes the first three items of the original AUDIT instrument (see Questionnaire 4 in the Appendix). (Bush, Kivlahan, McDonell, Fihn, & Bradley, 1998). It is faster but is not more sensitive than AUDIT in detecting at-risk drinking (Rumpf et al., 2002).

AUDIT

AUDIT was developed by WHO as a simple method of screening for excessive drinking and to assist in brief assessment (see Questionnaire 5 in the Appendix). The AUDIT differs from other self-report screening tests in that it was based on data collected from a large multinational sample and used an explicit conceptual statistical rationale for item selection. It emphasizes identification of hazardous drinking rather than long-term dependence and adverse drinking consequences (Babor et al., 2001). Studies have shown that it is more culturally appropriate than CAGE or MAST and that it reflects the extent of alcohol involvement along a broad continuum of severity (Fleming, Barry, & MacDonald, 1991; Hays, Merz, & Nicholas, 1995; Sinclair, Mcree, & Babor, 1992). Advantages of using AUDIT are that it has been validated for use in primary health care patients. It is the only screening test that identifies hazardous and harmful alcohol use, as well as dependence. Other advantages include it being rapid, brief, and flexible. AUDIT has high internal consistency and high test-retest reliability (r = 0.86). These qualities have been established in several language versions and across several nations. There are three domains of AUDIT, namely, hazardous use of alcohol brought out by the first three questions, dependence symptoms brought out by the fourth to sixth questions, and harmful use of alcohol brought out by the last four questions (Babor, 2001) (Babor et al., 2001).

BOX 1

Domains and Item Content of the AUDIT

Domains	Question Number	Item Content
Hazardous alcohol use	1	Frequency of drinking
	2	Typical quantity
	3	Frequency of heavy drinking
Dependence symptoms	4	Impaired control over drinking
	5	Increased salience of drinking

Harmful alcohol use	6	Morning drinking
	7	Guilt after drinking
	8	Blackouts
	9	Alcohol-related injuries
	10	Others concerned about drinking

Source: Babor, T. F., Higgins-Biddle, J. C., Saunders,J. B., & Monteiro, M. G. (2001). WHO, Department of Mental Health and Substance Abuse. AUDIT—The Alcohol Use Disorders Identification Test: Guidelines for use in primary care. WHO/MSD/MSB/01.6a. Geneva, Switzerland.

Total scores of 8 or more are recommended as indicators of hazardous and harmful alcohol use, as well as possible dependence. A cut-off score of 10 will provide greater specificity at the cost of sensitivity. The purpose of AUDIT is to give primary emphasis to screening, not case-finding. The validity status revealed that 100 percent of alcoholics had a score of 10 or higher and only 0.5 percent of nondrinkers had a score of 8 or more. Overall the Cronbach's alpha was 0.80, suggesting a high internal consistency (Duane & John, 2002).

Having identified problem drinkers, what do you do with them? General practitioners in primary health care have generally been reluctant to screen for problem drinkers and, when any are identified, these practitioners are more reluctant to do something about it. Reasons generally put forth are that it would require too much time, that they have inadequate training to deal with such things, that they fear antagonizing patients over a sensitive personal issue, or that alcohol is altogether not a matter that needs to be addressed in primary health care (Babor et al., 2001) and that alcoholics do not respond to primary care interventions (Aira, Kauhanen, Larivaara, & Rautio, 2003; Heather, 1996; Rush, Powell, Crowe, & Ellis, 1995). Another reason could be that alcohol screening and interventions are laborious and time-consuming activities that have a small yield (Aalto & Seppä, 2004).

BIOMARKERS

Screening questionnaires, especially self-reporting types, when used in a population study often need to be complimented by use of a biological marker for identification of problem drinkers. This would facilitate early detection and intervention. Alcohol biomarkers include tests that tell us about the acute/chronic alcohol consumption (state/trait markers). They help improve knowledge of drinking patterns in both individuals and populations and are valuable

tools for the objective evaluation of intervention programs (Montalto & Bean, 2003).

There are several substances that can be evaluated in an alcoholic individual to ascertain an alcohol drinker's pattern of drinking and his/her abstinent status. Such a substance should be amenable to be tested in a large number of people. The test should be easily available, be cost-effective, and most importantly be useful as a tool in the assessment of the brief intervention program. More research is needed to determine the value of markers along with or without questionnaires to help us arrive at clearer clinical algorithms and have well-defined outcome variables. Several biomarkers have been available and have been used in different kinds of contexts and settings. One of the most traditional biological markers was alanine transaminase, aspartate transaminase, and mean corpuscular volume (MCV). The former two are susceptible to change by several other substances like drugs and diseases of the liver, and the latter is much influenced by age (Gotz, Klaus, Jorg, Daniela, & Karl, 1999; Karen, 2004) and its sensitivity is too low (24%) to allow use in a general population (Reynaud et al., 2000). Over the years, therefore, in a yearning for a biological marker that has both high specificity and sensitivity, several substances have been evaluated. Some of these are fatty acid ethyl esters (FAEE), sialic acid (SA), serum, activity of "N-acetyl β hexosaminidase" (HEX), Leptin, sialysation of apolipoprotein J (SIJ), carbohydrate deficit transferrin (CDT), and gamma glutamyl transferase (GGT).

FAEE

FAEE level in human hair is influenced by quantity and frequency of alcohol intake. The cut-off mark to distinguish heavy drinking/alcoholism from social/moderate drinking is 0.4 ng/mg of hair (Wurst et al., 2004). Though FAEE has high sensitivity and specificity (100% and 90%, respectively) (Karen, 2004), use is restricted by its limited availability and has not been much researched yet (Minna, Maya, Nissen, Anetie, & Swolainen, 2007). FAEE is also measured in serum. FAEE is a nonoxidative esterification product of alcohol and ethanol. It is detectable even after 24 hours of alcohol consumption and even with as little ethanol as 100 mg/L, which by other tests for alcohol would have tested negative. Thus it may evolve as both a short- and long-term marker of ethanol ingestion. (Michael, 1997).

HEX

HEXN-acetyl β hexosaminidase is a lysosomal enzyme. HEX has a sensitivity of 94 percent and specificity of 91 percent, which makes it more effective than CDT (Stowe, Stowel, Barrett, & Robinson, 1997). However, its

normalization on abstinence takes place faster than GGT, making it difficult to ascertain long-term alcohol abuse (Markowski, Ferens-Sieczowska, Zwierz, & Wojtulewska, 2003).

Leptin

Leptin is a cytokine-type peptide recently implicated as a putative state marker of alcohol use (Wurst et al., 2003). Leptin levels are increased by even a moderate level of alcohol intake (15–30 g/day) (Roth et al., 2003). However, another study has shown that there is only a minor influence on leptin levels of acute alcohol intoxication in alcohol-detoxified patients (Wurst et al., 2003).

Sialic Acid

Sialic acid, because of its clear potential as a highly specific marker for alcohol use, is being directly measured in plasma rather than as part of a complex protein chain. Sialic acids are the acetyl derivatives of muraminic acids that occur as nonreducing terminal residues of glycoproteins/gylcolipids in biological fluids and membranes. It has similar sensitivity and specificity to CDT for measuring alcohol consumption. It takes longer than GGT or CDT to decrease in abstinent patients. It is not affected by nonalcoholic liver damage. However, its widespread availability is still restricted. It is likely to be a useful test in the future (Karen, 2004; Romppanen et al., 2002).

SIJ

Plasma sialic acid index of apolipoprotein J is another substance that can be used to detect alcohol levels. Apolipoprotein J is a glycoprotein found in lipoproteins that are responsible for transporting fats in the blood. Sialic acid content of these proteins is reduced after alcohol intake. Sialic acid content is four times more in apolipoprotein J than in transferrin, it is therefore easier to measure changes in it caused by heavy alcohol intake. It has a sensitivity of 90 to 92 percent. It is valid when alcohol intake exceeds 60 g/day and is markedly diminished in chronic alcoholics (Ghosh, Hale, & Lakshman, 2001).

CDT

Carbohydrate deficit transferrin is a collective term referring to isoforms of transferrin from which sialic acid residues have been removed. Alcohol intake knocks off that sialic acid, and the concentration of CDT increases (Das, Nayak, & Vasudevan, 2003). Some of the advantages of using CDT as an alco-

hol state marker are its high specificity, the fact that it is not affected by liver disease, and its low incidence of false positives (Stibler, 1991).

However, its main disadvantages are lack of wide availability, high cost (TIP 45 SAMHSA, 2006), and the fact that some people with high levels of transferring may have elevated CDT even without alcohol intake (Karen, 2004). Some primary care studies using CDT have shown low sensitivity (Salaspuro, 1999) and may even have false negatives (Karen, 2004). It may therefore not be suitable for screening for subjects with alcohol abuse in the general population (Golka & Wiese, 2004).

GGT

Gamma glutamyl transferase is a biliary canalicular enzyme that is induced by alcohol and serum levels rise in response to acute hepatocellular damage (Rosalki, 1984). Because during abstinence in a case of a heavy drinker GGT levels fall gradually and return to normal levels only after six to eight weeks, it provides a useful confirmation that alcohol was the cause of the cellular damage (Das et al., 2003). It is a useful screening substance even though it is also elevated easily due to nonalcoholic liver damage (Gotz, Klaus, Jorg, Daniela, & Karl, 1999). GGT is a measure of cumulative alcohol use (Golka & Wiese, 2004). It is more influenced by drinking intensity than frequency (Anton, Stout, Roberts, & Allen, 1998). It has also been found that those with high GGT levels have more vulnerability to alcohol, a characteristic that appears to be stable with time (Daeppen, Schoenfeld-Smith, Smith, & Schuckit, 1999).

Sensitivity of GGT in different studies ranges from 40 to 70 percent (Mihas & Tavassoli, 1992; Reynaud et al., 2000; TIP 45 SAMHSA, 2006) and specificity is from 40 to 80 percent ([Reynaud et al., 2000; TIP 45 SAMHSA, 2006], that is, the ability to not misidentify or confirm alcohol use to other disorder). It does correlate with alcohol intake but requires more than 60 g/day intake. GGT is helpful as a motivational enhancer in patients with a high degree of denial to show them both evidence of drinking and liver damage. Reducing levels with abstinence provides motivation in remaining abstinent.

Most biological markers are influenced by duration of abstinence, age, and alcohol consumption (Das et al., 2003; Gotz, Klaus, Jorg, Daniela, & Karl, 1999). GGT has the advantage of being more sensitive in those who are recently abstinent (≤ 4 days). Age has no effect on CDT and GGT but on MCV with increasing values with age (Gotz, Klaus, Jorg, Daniela, & Karl, 1999).

Thus we see that none of the biological markers are clearly optimal to play a role as an alcohol intake marker in the general population. However, in view

of its easy availability, low cost, and greater sensitivity (which is an essential characteristic in screening tests and also a tool to monitor abstinence states), GGT is an ideal screening biological marker (Hoaskema & de Bock, 1993). When it is combined with CDT, its usefulness in identifying problem drinkers is enhanced greatly.

BRIEF INTERVENTION

But what are the interventions that are available and known to be effective in primary health care, and how can they be delivered? Three different kinds of intervention are generally available for implementation among the alcoholic population. They are alcohol education, simple advice, and brief counseling.

BOX 2

What Interventions to Be Given

Risk Level	Intervention	AUDIT Score
Zone I	Alcohol education	0–7
Zone II	Simple advice	8–15
Zone III	Simple advice plus brief counseling and continued monitoring	16–19
Zone IV	Referral to specialist for diagnostic evaluation and treatment	20–40

Source: Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., Monteiro, M. G.(2001). WHO, Department of Mental Health and Substance Abuse. AUDIT—The Alcohol Use Disorders Identification Test: Guidelines for use in primary care. WHO/MSD/MSB/01.6a. Geneva, Switzerland.

Alcohol education is appropriate for those procuring a score below 8 on AUDIT. Though no intervention as such is required, it contributes to the general awareness of alcohol risk in the community (Saunders, Kypri, Walters, Laforge, & Larimer, 2004).

Simple advice is for those who score between 8 and 15 on AUDIT. Such individuals require advice to avoid drinking to intoxication. It entails giving them the feedback that their drinking patterns are risky for their health and social outcomes in future. They are provided information on specific risks of continued drinking above recommended guidelines. Then the most important part of simple advice is for the patient to establish a goal to change drinking behavior and give advice on drinking limits (Saunders et al., 2004).

However, some in this group are to be given advice to stop drinking rather than on low-risk drinking. These are those with a prior history of alcohol/drug dependence, prior or current mental illness, women who are pregnant, and those whose medical conditions require complete abstinence of alcohol (Saunders et al., 2004).

Brief counseling is appropriate for those who score 16–19 on AUDIT. It differs from simple advice in that it is a systematic, focused process that relies on rapid assessment, quick engagement of the patient, and immediate implementation of change strategies. Its goal is to provide tools to the patient to change basic attitudes and handle a variety of underlying problems. It involves giving information booklets. The patient goes through stages of precontemplation, contemplation, preparation, action, and maintenance. An important component of brief intervention at all stages is giving encouragement (Saunders et al., 2004). Brief intervention for alcoholics encompasses both simple advice and counseling. Those scoring above 20 on AUDIT should ideally be referred to a specialist treatment center.

Who provides this brief intervention? Several studies have shown that when this is implemented in a clinic at all levels of primary health care system or even by a nurse, it is effective (Aalto et al., 2001; Drummond, Thom, Brown, Edwards, & Mullan, 1990; Israel et al., 1996; Kaariainen, Pekka, Pauli, & Seppa, 2001; Latt & Saunders, 2002; McIntosh, Leigh, Baldwin, & Marmulak, 1991; Ylva, Anders, & Aberg, 1998).

Even when it is applied as a 5–60-minute motivational counseling and alcohol education over one to four scheduled sessions to problem drinkers, it would still help a large number of excessive drinkers to cut down consumption to safer levels (Grant, 1997). Controlled studies at primary health care settings have shown up to 20 percent reduction in alcohol consumption by problem drinkers (Latt & Saunders, 2002; Sobell & Sobell, 1993), whereas some have found up to 50 percent having significant reduction in drinking (Aalto et al., 2001). Odd Nilssen, in a nine-year old follow-up study of the effect of brief intervention in at-risk alcohol drinkers, found that after one year there was a 50 percent reduction in alcohol intake in the intervention groups and a 20 percent increase in the control group. The impact of brief intervention appears to be long-lasting. At nine years follow-up, at-risk drinkers disclosed GGT values close to that of the background population (Odd, 2003). Grossberg, Brown, and Fleming also reported reduction in high-risk drinking behaviors and consequences in a four-year study (Grossberg, Brown, & Fleming, 2004).

Saunders et al. reported that using AUDIT for screening and providing very brief advice by a general practitioner (GP) when appropriate found 30 percent reduction in hazardous drinking (Saunders, Aasland, Babor, de la Fuente, &

Grant, 1993). Fleming et al.'s 48-month study found direct evidence that brief physician advice is associated with sustained reductions in alcohol use, health care interventions, motor vehicle accidents , and associated costs (McIntosh et al., 1991).

Aalto et al. found that when a GP discussed the amount of alcohol consumed and harm caused by alcohol with patients, even when such discussions lasted for less than four minutes, this was not only acceptable but also useful (Aalto & Seppä, 2004). Similar findings are suggested by Kaariainen et al. (2001).

As a policy matter also, brief interventions are very useful, even if approximately 1 percent of patients who have required counseling make lasting changes. A brief intervention is cost-effective, and if approximately 10 percent change, much resources of health care will be saved (Andréason, Hjalmarsson, & Rehnman, 2000). A meta-analytical review of studies comprising brief interventions with either controlled or extended treatment conditions found little difference between brief and extended treatment conditions. This review also suggested that because evidence regarding brief interventions comes from different types of investigations with different samples, generalization should be restricted to the populations, treatment characteristics, and contents represented in those studies (Moyer, Finney, Swearingen, Carolyn, & Vergun, 2002).

Bein, Miller, and Tonigan. also reported in a meta-analytical review of 32 controlled studies that brief intervention is as effective as more intensive treatments (Bein et al., 1993). Several other studies have also found that those receiving brief intervention did better than those not receiving it at 6 and 12 month periods, while some have found that it might serve as an initial treatment for severely dependent patients seeking extended treatment (Bein et al., 1993; Moyer et al., 2002).

While some studies have showed that since the public health impact is enormous, further research is required on specific issues, because cases of harmful use of alcohol can be effectively attended by well-designed intervention strategies, which are feasible even as employee assistance programs (Kahan, Wilson, & Becker, 1995; Wilk, Jensen, & Havighurst, 1997).

Despite all this evidence, it is generally seen that brief intervention is not widely implemented and attempts to integrate it in regular practice have not been successful, probably because of lack of interest in prevention methods themselves or ineffective marketing strategies Andréason et al., 2000). Whitlock, Polen, Green, Orleans, and Klein also suggested that future research should focus on implementation strategies to facilitate adoption of means of identifying and providing intervention for problem drinkers in routine health care (Whitlock, Polen, Green, Orleans, & Klein, 2004). Roche et al. proposed

that perhaps we have the right vehicle but the wrong driver and that until closer scrutiny is made of this key prevention area it will continue to fail to achieve optimum results (Roche & Freeman, 2004).

CONCLUSION

To conclude, we may say that brief intervention, when provided after appropriate screening procedures, is very effective. This is true even whether it is provided by the primary physician, the health worker, or the nurse. It would go a long way in reducing the burden of ill health on the individual and the society at large at a very minimal cost to the health care system.

APPENDIX

1. CAGE Questionnaire

The CAGE consists of the following four items:

1. Have you ever felt you ought to cut down on your drinking?
2. Have people annoyed you by criticizing your drinking?
3. Have you ever felt bad or guilty about your drinking?
4. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover?

Source: Ewing, J. A. (1984). Detecting Alcoholism: The CAGE Questionnaire. The *Journal of the American Medical Association,* 252, 1905–1907.

2. Questionnaire: Michigan Alcohol Screening Test (MAST)

Sr No		Yes	No
1.	Do you feel you are a normal drinker? (By normal we mean you drink less than or as much as most other people.)		
2.	Have you ever wakened the morning after drinking the night before and found that you could not remember a part of the evening?		
3.	Does your wife, husband, a parent, or other near relative ever worry or complain about your drinking?		
4.	Can you stop drinking without a struggle after one or two drinks?		
5.	Do you ever feel guilty about your drinking?		
6.	Do friends or relative think you are a normal drinker?		
7.	Are you able to stop drinking when you want to?		
8.	Have you ever attended a meeting of Alcoholics Anonymous?		
9.	Have you ever gotten into physical fights when drinking?		
10.	Has drinking ever created a problem between you and your wife, husband, a parent, or other near relative?		

11. Has your wife, husband, a parent, or other near relative ever gone to anyone for help about your drinking?
12. Have you ever lost friends or a partner because of your drinking?
13. Have you ever gotten into trouble at work because of your drinking?
14. Have you ever lost a job because of drinking?
15. Have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking?
16 Do you drink before noon fairly often?
17. Have you ever been told you have liver trouble? Cirrhosis?
18. After heavy drinking have you ever had delirium tremens (DTs) or severe shaking, or heard voices or seen things that weren't really there?
19. Have you ever gone to anyone for help about your drinking?
20. Have you ever been in a hospital because of your drinking?
21. Have you ever been a patient in a psychiatric hospital or on a psychiatric ward of a general hospital where drinking was part of the problem that resulted in hospitalization?
22. Have you ever been seen at a psychiatric or mental health clinic or gone to any doctor, social worker, or clergyman for help with any emotional problem, where drinking was part of the problem?
23. Have you ever been arrested for drunken driving, driving while intoxicated, or driving under the influence of alcoholic beverages?
24. Have you ever been arrested, even for a few hours, because of other drunken behavior?

Source: Selzer, M. L. (1971). The Michigan Alcoholism Screening Test: The quest for a new diagnostic instrument. *American Journal of Psychiatry,* 127, 1653–1658.

3. LAST Questionnaire

1. Are you able to stop drinking when you want to?
2. Have you ever felt you ought to cut down on your drinking?
3. Have people annoyed you by criticizing your drinking?
4. Does any near relative or close friend worry or complain about your drinking?

Source: Hans-Jurgen Rumpf, Ulfert Hapke, Andreas Hill, and Ulrich John. Development of a Screening Questionnaire for the General Hospital and General Practices. *Alcoholism: Clinical and Experimental Research*. 1997: 21; 894–898. Blackwell Publishing.

4. Questionnaire: AUDIT-C

1. How often do you have a drink containing alcohol?

Never	Monthly or less	2–4 times a month	2–3 times a week	4 or more times a week

2. How may drinks containing alcohol do you have on a typical day when you are drinking?

1 or 2	3 or 4	5 or 6	7 or 9	10 or more

3. How often do you have 6 or more drinks on one occasion?

Never	Monthly or less	2–4 times a month	2–3 times a week	4 or more times a week

Source: Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., Monteiro, M. G.(2001). WHO, Department of Mental Health and Substance Abuse. AUDIT—The Alcohol Use Disorders Identification Test: Guidelines for use in primary care. WHO/MSD/MSB/01.6a. Geneva, Switzerland.

5. Questionnaire: AUDIT (Alcohol Use Disorders Identification Test Screening Instrument)

Please circle the answer that is correct for you

1. How often do you have a drink containing alcohol?

Never	Monthly or less	2–4 times a month	2–3 times a week	4 or more times a week

2. How may drinks containing alcohol do you have on a typical day when you are drinking?

1 or 2	3 or 4	5 or 6	7 or 9	10 or more

3. How often do you have 6 or more drinks on one occasion?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

4. How often during the last year have you found it difficult to get the thought of alcohol out of your mind?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

5. How often during the last year have you found that you were not able to stop drinking once you had started?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

6. How often during the last year have you been unable to remember what happened the night before because you had been drinking?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

7. How often during the last year have you needed a first drink in morning to get yourself going after a heavy drinking session ?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

8. How often during the last year have you had a feeling of guilt or remorse after drinking?

Never	Less than monthly	Monthly	Weekly	Daily or almost daily

9. Have you or someone else been injured as a result of your drinking?

No	Yes, but not in the last year	Yes, during the last year

10. Has a relative or friend, or a doctor or other health worker been concerned about your drinking or suggested you cut down?

No	Yes, but not in the last year	Yes, during the last year

Source: Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., Monteiro, M. G. (2001). WHO, Department of Mental Health and Substance Abuse. AUDIT-The Alcohol Use Disorders Identification Test: Guidelines for use in primary care. WHO/MSD/MSB/01.6a. Geneva, Switzerland.

REFERENCES

Aalto, L. M., & Seppä, K. (2004). Usefulness, length and content of alcohol-related discussions in primary health care: The exit poll survey. *Alcohol and Alcoholism, 39*(6), 532–535.

Aalto, M., Seppä, K., Mattila, P. , Mustonen, H., Ruuth, K., Hyvrinen, H., et al. (2001). Brief intervention for male heavy drinkers in routine general practice: A three-year randomized controlled study. *Alcohol and Alcoholism, 36*(3), 224–30.

Aira, M., Kauhanen, J., Larivaara, P. , & Rautio, P. (2003). Factors influencing inquiry about patients' alcohol consumption by primary health care physicians: Qualitative semi-structured interview study. *Family Practice, 20,* 270–275.

Andréason, S., Hjalmarsson, K., & Rehnman, C. (2000). Implementation and dissemination of methods for prevention of alcohol problems in primary health care: A feasibility study. *Alcohol and Alcoholism, 35*(5), 525–530.

Anton, R. F., Stout, R. L., Roberts, J. S., & Allen, J. P. (1998). The effect of drinking intensity and frequency on serum carbohydrate-deficient transferrin and gamma-glutamyl transferase levels in outpatient alcoholics. *Alcoholism: Clinical and Experimental Research, 22,* 1456–1462.

Babor, T. F., Campbel, L. R., Room, R., & Saunders, J. (Eds.). (1994). *Lexicon of alcohol and drug terms.* Geneva: World Health Organization.

Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., & Monteiro, M. G. (2001). WHO, Department of Mental Health and Substance Abuse. AUDIT—The Alcohol

Use Disorders Identification Test: Guidelines for use in primary care (2nd ed.). Geneva: World Health Organization.

Bein, T., Miller, W. R., & Tonigan, J. S. (1993). Brief interventions for alcohol problems. A review. *Addiction, 88*, 315–336.

Bush, K., Kivlahan, D. R., McDonell, M. B., Fihn, S. D., & Bradley, K. A.(1998). The AUDIT Alcohol Consumption Questions (AUDIT-C). An effective brief screening test for problem drinking. *Archives of Internal Medicine, 158*, 1789–1795.

Daeppen, J. B., Schoenfeld-Smith, K., Smith, T. L., & Schuckit, M. A. (1999). Characteristics of alcohol dependent subjects with very elevated levels of gamma-glutamyl transferase (GGT). *Journal of Studies on Alcohol and Drugs, 60*(5), 589–594.

Das, S. K., Nayak, P. D., & Vasudevan, M. (2003). Biochemical markers for alcohol consumption. *Indian Journal of Clinical Biochemistry, 18*(2), 111–118.

Drummond, D. C., Thom, B., Brown, C., Edwards, G., & Mullan, M. J. (1990). Specialist versus general practitioner treatment of problem drinkers. *Lancet, 336*, 915–918.

Duane, F. R., & John, P. A. (2002). The Alcohol Use Disorders Identification Test (AUDIT): A review of recent research. *Alcoholism: Clinical and Experimental Research, 26*(2), 272–279.

Ewing, J. A. (1984). Detecting alcoholism: The CAGE questionnaire. *Journal of the American Medical Association, 252*, 1905–1907.

Fleming, M. F., Barry, K. L., & MacDonald, R. (1991). The alcohol use disorders identification test (AUDIT) in a college sample. *International Journal of the Addictions, 26*, 1173–1185.

Ghosh, P. , Hale, E. A., & Lakshman, M. R. (2001). Plasma Sialic acid index of apolipoprotein J (SIJ): A new alcohol intake marker. *Alcohol, 25*(3), 173–179.

Golka, K., & Wiese, A. (2004). Carbohydrate-deficient transferrin (CDT) a biomarker for long-term alcohol consumption. *Journal of Toxicological Environmental Health. Part B: Critical Reviews, 7*(4), 319–337.

Gotz, M., Klaus, A., Jorg, M., Daniela, S., & Karl, M. (1999). Influence of age, alcohol consumption and abstinence on the sensitivity of carbohydrate-deficient transferrin, gamma-glutamyl transferase and mean corpuscular volume. *Alcohol and Alcoholism, 34*(5), 760–766.

Grant, B. F. (1997). Barriers to alcoholism treatment: Reasons for not seeking treatment in a general population sample. *Journal of Studies on Alcohol, 58*, 365–371.

Grossberg, P. M., Brown, D. D., & Fleming, M. F. (2004). Brief physician advice for high-risk drinking among young adults. *Annals of Family Medicine, 2*(5), 474–480.

Hays, R. D., Merz, J. F., & Nicholas, R. (1995). Response burden, reliability and validity of the CAGE, short MAST, and AUDIT alcohol-screening measures. *Behavioural Research Methods, Instruments and Computers, 27*, 277–280.

Heather, N. (1996). The public health and brief interventions for excessive alcohol consumption: The British experience. *Addictive Behaviours, 21*, 857–868.

Hoaskema, H. L., & de Bock, G. H. (1993). The value of laboratory tests for the screening and recognition of alcohol abuse in primary care patients. *Journal of Family Practice, 37*, 268–273.

Israel, Y., Hollander, O., Sanchez-Craig, M., Booker, S., Miller, V., Gingerich, R., et al. (1996). Screening for problem drinking and counseling by the primary care physician-nurse team. *Alcoholism: Clinical and Experimental Research, 29*, 1443–1450.

Kaariainen, J., Pekka, S., Pauli, P. , & Seppa, K. (2001). Opinions on alcohol-related issues among professionals in primary occupational, and specialized health care. *Alcohol and Alcoholism, 36*(2), 141–146.

Kahan, M., Wilson, L., & Becker, L. (1995). Effectiveness of physician-based interventions with problem drinkers: A review. *Canadian Medical Association Journal, 152*(6), 851–859.

Karen, P. (2004). Biomarkers for alcohol use and abuse: A summary. *Alcohol Research and Health* (Winter). Retrieved June 2, 2007, from http://findarticles.com/p/articles/mi_m0CXH/is_1_28/ai_n14709591

Latt, N., & Saunders, J. B. (2002). Alcohol misuse and dependence. Assessment and management. *Australian Family Physician, 31*(12), 1079–1085.

Markowski, T., Ferens-Sieczowska, M., Zwierz, K., & Wojtulewska, A. (2003). The activity of N-acetyl-beta-hexosamimidase and gamma-glutamyl transferase in the serum of alcohol dependent people hospitalized after a long lasting drinking period. *Psychiatria Polska, 37*(3), 495–502.

McIntosh, M. C., Leigh, G., Baldwin, N. J., & Marmulak, J. (1991). Reducing alcohol consumption. Comparing three brief methods in family practice. *Canadian Family Physician, 43*, 1959–1962, 1965–1967.

Michael, L. (1997). Fatty acid ethyl esters short term and long term serum markers of ethanol intake. *Clinical Chemistry, 43* (8, B), 1527–1534.

Mihas, A. A., & Tavassoli, M. (1992). Laboratory markers of ethanol intake and abuse: A critical appraisal. *American Journal of Medical Sciences, 303*, 415–428.

Miller, N. S., Kipnis, S. S. (2006). *Detoxification and substance abuse treatment.* Treatment Improvement Protocol (TIP) Series 45. Center for Substance Abuse Treatment. DHHS Publication No. (SMA) 06-4131. Rockville, MD: Substance Abuse and Mental Health Services Administration.

Minna, H., Maya, L., Nissen, K., Anetie, T., & Swolainen, M. J. (2007). Biochemical markers in alcoholism. *Clinical Chemistry and Laboratory Medicine, 45*(8), 953–961.

Montalto, N. J., & Bean, P. (2003). Use of contemporary biomarkers in the detection of chronic alcohol use. *Medical Science Monitor, 9*(12), 285–290.

Moyer, A., Finney, J., Swearingen, W., Carolyn, E., & Vergun, P. (2002). Brief interventions for alcohol problems: A meta-analytical review of controlled investigations in treatment-seeking and non treatment seeking populations. *Addiction, 97*(3), 279–292.

Odd Nilssen (2003). Long term follow up of brief intervention in at risk alcohol drinkers: A 9 year follow-up study. *Alcohol and Alcoholism, 38*(5), 421–426.

Pal, H. R., Jena, R., & Yadav, D. (2004). Validation of the Alcohol Use Disorders Identification Test (AUDIT) in urban community outreach and de-addiction center samples in north India. *Journal of Studies on Alcohol and Drugs, 65*(6), 794–800.

Reynaud, M., Schellenberg, F., Loisequx-Meunier, M. N., Schwan, R., Maradeix, B., Planche, F., et al. (2000). Objective diagnosis of alcohol abuse: Compared values of carbohydrate-deficient transferrin (CDT), gamma-glutamyl transferase (GGT), and mean corpuscular volume (MCV). *Alcoholism: Clinical and Experimental Research, 24*(9), 1414–9.

Roche, A. M., & Freeman, T. (2004). Brief interventions: Good in theory but weak in practice. *Drug Alcohol Rev, 23*(1), 11–18.

Romppanen, J., Punnonen, K., Anttila, P., Jakobsson, T., Blake, J., & Niemela, O. (2002). Serum sialic acid as a marker of alcohol consumption: Effect of liver disease and heavy drinking. *Alcoholism: Clinical and Experimental Research, 26*(8), 1234–1238.

Rosalki, S. (1984). Identifying the alcoholic. In S. Rosalki (Ed.), *Clinical biochemistry of alcoholism* (pp. 65–92). Edinburgh: Churchill Livingstone.

Roth, M. J., Baerm D. J., Albert, P. S., Castonguay, T. W., Dorgan, J. F., Dawsey, S. M., et al. (2003). Relationship between serum leptin levels and alcohol consumption in a controlled feeding and alcohol ingestion study. *Journal of National Cancer Institute, 95*(22), 1772–1775.

Rumpf, H. J., Hapke, U., Dawedeit, A., Meyer, C., & John, U. (1998). Triggering and maintenance factors of remitting from alcohol dependence without formal help. *European Addiction Research, 4,* 209–210.

Rumpf, H. J., Hapke, U., Meyer, C., & John, U. (2002). Screening for alcohol use disorders and At-risk drinking in the general population: Psychometric performance of three questionnaires. *Alcohol and Alcoholism, 37*(3), 261–268.

Rush, B. R., Powell, L. Y., Crowe, T. G., & Ellis, K. (1995). Early interventions for alcohol use: Family physicians' motivations and perceived barriers. *Canadian Medical Association Journal, 152,* 863–869.

Salaspuro, M. (1999). Carbohydrate-deficient transferrin as compared to other markers of alcoholism: A systematic review. *Alcohol, 19*(3), 261–271.

Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993). Development of the Alcohol Use Disorders Identification Tests (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption—II. *Addiction, 88,* 791–804.

Saunders, J. B., Kypri, K., Walters., S. T., Laforge, R. G., & Larimer, M. E. (2004). Approaches to brief intervention for hazardous drinking in young people. *Alcoholism: Clinical and Experimental Research, 28*(2), 322–329.

Schuckit, M. A., & Irwin, M. (1988). Diagnosis of alcoholism. *Medical Clinics of North America, 72*(5), 1133–1153.

Selzer, M. L. (1971). The Michigan Alcoholism Screening Test: The quest for a new diagnostic instrument. *American Journal of Psychiatry, 127*(12), 89–94.

Sinclair, M., Mcree, B., & Babor, T. F. (1992). *Evaluation of the reliability of AUDIT*. Unpublished report, University of Connecticut School of Medicine, Alcohol Research Center.

Sobell, M. B., & Sobell, L. C. (1993). *Problem drinkers: Guided self change treatment*. New York: Guilford Press.

Stibler, H. (1991) Carbohydrate—deficient transferrin in serum: A new marker of potentially harmful alcohol consumption reviewed. *Clinical Chemistry, 37,* 2029–2037.

Stowe, I. L., Stowel, A., Barrett, N., & Robinson, G. (1997). Comparison of serum and hexosaminadase isoenzyme B activity with serum carbohydrate deficit transferrin and other markers of alcohol abuse. *Alcohol and Alcoholism, 32*(6), 703–714.

Whitlock, E. P., Polen, M. R., Green, C. A., Orleans, T., & Klein, J. (2004). Behavioral counseling interventions in primary care to reduce risky/harmful alcohol use by adults: A summary of the evidence for the U. S. Preventive Services Task Force. *Annals of Internal Medicine, 140*(7), 164.

Wilk, A., Jensen, N., & Havighurst, T. (1997). Meta-analysis of randomized control trials addressing brief interventions in heavy alcohol drinkers. *Journal of General Internal Medicine, 12,* 274–283.

World Health Organization. (1993). The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research. Geneva: Author.

Wurst, F. M., Alexson, S., Wolfersdorf, M., Bechtel, G., Forster, S., Alling, C., et al. (2004). Concentration of fatty acid ethyl esters in hair of alcoholics: Comparison to other biological state markers and self reported-ethanol intake. *Alcohol, 39*(1), 33–38.

Wurst, F. M., Bechtel, G., Forster, S., Wolfersdorf, M., Huber, P., Scholer, A., et al. (2003). Leptin levels of alcohol abstainers and detoxification patients are not different. *Alcohol and Alcoholism, 38*(4), 364–368.

Ylva, T., Anders, R., & Aberg, H. (1998). Excessive drinking—brief intervention by a primary health care nurse. A randomized control study. *Scandinavian Journal of Primary Health Care, 16,* 188–192.

Chapter **17**

Service Profiles: Provider Choices for Treatment of Alcohol- and Cocaine-Using Clients

Maria Dinis, PhD, MSW, Lyndsay Ammon, MPH,
and Kelly Anders, MAL

A variety of treatment services, including medical, psychological, peer-based, and social services, are available to providers treating alcohol and drug users. These are being offered either alone or in combination with another service. Examples of these services include the following: physician-supervised, hospital-based programs, including pharmacological approaches; nonmedical residential programs such as halfway houses; mental health programs, including individual or group psychotherapy facilitated by counselors, psychologists, social workers, or religious-based clerics; Twelve-Step programs such as Alcoholics Anonymous or Narcotics Anonymous; and the criminal justice or penal system (Gerstein & Harwood, 1990; Institute of Medicine, 1990; Musto, 2002; White, 1998).

This chapter explores whether provider treatment recommendations are related to a disciplinary perspective that varies by provider's work setting and demographic characteristics. Work settings, for example, include alcohol and drug, criminal justice, mental health, hospital and medical, social welfare, and religious. Demographic characteristics of the providers are gender, age, and education level. Specifically, this chapter investigates providers' decisions regarding their treatment choices because these choices may reflect how a client type (e.g., men, pregnant women, and nonpregnant women) is matched to a particular treatment, especially considering complicating factors (such as pregnancy) that may restrict options, and the interplay between the provider work setting and demographics influencing the selection of one treatment method over another.

Earlier work concluded that the "medical" or "disease" model, terms used interchangeably in the literature, is probably the most dominant approach

among specialized treatment providers for treating people with alcohol and drug problems (Brower, Blow, & Beresford, 1989; Institute of Medicine, 1990; Keller, 1990; Leonelli, 1993). The dominant treatment approach of nonspecialized treatment providers is not known for certain. Preliminary research suggests a complex relationship between provider beliefs and model choice for heavy alcohol users (Nunes-Dinis, 1996). When a probability sample of professionals from diverse community agencies (e.g., alcohol and drug, criminal justice, health care, social welfare, mental health, and religious) were asked to select treatment models, respondents tended to answer according to their personal beliefs (e.g., alcohol is a genetic, social, or multiple-cause problem). The only exception was treatment for pregnant women, for whom the medical model was generally considered the optimal treatment approach. Pregnancy status emerged as a better predictor of service model choice than provider professional training or work setting (Nunes-Dinis, 1996).

Both academic researchers and public policymakers seek to better understand what influences professionals' treatment choices for their clients. Currently the variability of the decisions they make in the selection of treatment services is not clearly understood (Fischhoff, 1992; Mitchell, Dyer, & Peay, 2006). For example, factors influencing the delivery of methadone maintenance are reported to vary according to the physician's professional orientation, geographical location, and experience as well as the patient's characteristics such as pregnancy, employment status, appearance, and social support (Mitchell et al., 2006). In addition, the medical specialty of physicians, their practice setting, and their age, gender, race, and social class are known to influence their clinical decision making in the delivery of medical care (Clark, Potter, & McKinlay, 1991; McKinlay, Potter, & Feldman, 1996). The gender, education, and age of providers have also been studied with therapists in relation to client utilization of health services (Crane, Wood, Law, & Schaalje, 2004); to predict the therapeutic alliance between client and therapist in alcohol treatment (Connors et al., 2000); to estimate variations in outcomes attributed to therapists in managed care (Wampold & Brown, 2005); to understand physicians' clinical decision-making processes (Clark et al., 1991), and to give alcohol-related treatment advice to patients by primary care providers (Burman et al., 2004). Understanding the variability in the decisions made by treatment providers may inform policymakers and administrators who are planning and funding alcohol and drug treatment programs (Simpson, 2002; Trentacoste, Holtgrave, Collins, & Abdul-Quader, 2004); enhance the knowledge of ramifications for treatment retention and outcome (Bell, 2000; Mitchell et al., 2006; Strang & Sheridan, 2001); emphasize the need for training treatment providers (Mitchell et al., 2006; Simpson,

2002); and promote technology transfer between researchers and practitioners (Forman, Bovasso, & Woody, 2001; Simpson, 2002).

Several researchers have pointed out the importance of understanding the variation in the decision making of providers choosing either one or a combination of services and why they use particular services with specific populations (Clark et al., 1991; Fischhoff, 1992; McKinlay et al., 1996; Mitchell et al., 2006; Simpson, 2002; Strang & Sheridan, 2001). For instance, does pregnancy status play a role in treatment selection; and does the choice vary depending on provider's own work setting and demographic characteristics? If so, what are the actual treatment choices being made? For example, professionals working in a hospital setting may tend to recommend only inpatient hospital treatment for their clients rather than referring them either to halfway houses, which cost a fraction (13%–17%) of inpatient care and about 50 percent of outpatient hospital-based treatment, or to no-cost self-help groups (Borkman, Kaskutas, Room, Bryan, & Barrows, 1998; Kaskutas, Witbrodt, & French, 2004). Conversely, those working in alcohol/drug treatment programs may recommend a broader range of services that they believe would result in better treatment outcomes for some clients, such as pregnant women (Hankin, McCaul, & Heussner, 2000; Howell & Chasnoff, 1999; Weisdorf, Parran, Graham, & Snyder, 1999).

To further study the question of provider treatment choice, we used Delany et al. (Delany, Broome, Flynn, & Fletcher, 2001) concept of treatment service delivery profiles derived from the Drug Abuse Treatment Outcome Study. This line of research considered the range and type of services offered (i.e., minimal versus comprehensive and inpatient versus outpatient). Our approach is further guided by earlier work that has examined organizational factors such as the academic training of treatment staff, and the types and amount of treatment services (medical, psychological, family, legal, educational, vocational, and financial services) offered to clients who use alcohol or drugs (D'Aunno & Vaughn, 1995; Etheridge, Craddock, Dunteman, & Hubbard, 1995; Etheridge, Hubbard, Anderson, Craddock, & Flynn, 1997; Friedmann, Alexander, & D'Aunno, 1999). This chapter addresses the services that providers would recommend for clients, asking respondents not to be constrained by the services offered in their own work settings. We first determined the dominant treatment recommendations and then used those to develop service profiles for treating alcohol- and cocaine-using men and for pregnant and nonpregnant women. Next, we tested whether provider work setting and demographics are related to their service profile treatment choices and whether some providers are more likely than others to select a single service rather than a combination of services.

METHOD

Sample and Procedure

This is a secondary data analysis of a provider study conducted during 1995 in a Northern California County, the site of the Alcohol Research Group's Community Epidemiology Laboratory (CEL). The CEL county was selected based on a broad cross-section of population characteristics including ethnicity, rural and urban mix, and socioeconomic status. The overall methods and study design have been described in other reports (Room & Weisner, 1988; Weisner, Matzger, Tam, & Schmidt, 2002; Weisner & Schmidt, 1995).

In the provider study, a two-stage probability sample of health and human service providers was drawn in order to be representative of service organizations and staff in the county. This sample of agencies was stratified by the following service sectors: substance abuse treatment, criminal justice, mental health, health care, welfare, and religious organizations. A listing of organizations and individuals in the county was developed by using telephone directory listings, snowballing of agencies' listings, and follow-up phone screening interviews with a provider from each organization. From a listing of 2,390 eligible organizations, 117 stratified agencies by service sectors were randomly selected. A simple random sample of physicians and psychotherapists was drawn from a separate listing. Permission was obtained to interview providers in the 117 organizations and a list was developed of all direct service professionals working at least half time. Individual providers were sampled proportionate to the size of the organization. Service providers were the units of analysis.

Of the total 610 solo and agency providers, 73 percent were interviewed (N = 457). The response rate was calculated by multiplying the number of eligible participating agencies (66/75) by the number of eligible respondents (457/549) who completed the interview (see Nunes-Dinis, 1996). Fifty-eight percent of the providers responding were female (n = 265). About two-thirds were married or living with a partner. More than half of the providers were under 45 years of age. Approximately half of the sample completed graduate education at the master's or doctoral level, 17 percent were college graduates, and 33 percent had less than a college degree.

Weights (ranging from .04 to 2.29) were calculated to adjust for nonresponse bias and for design effects due to stratification at the first stage of sampling and varying selection intervals at the second stage. Those weights were representative of the actual proportion of different kinds of providers working in the stratified service sectors (Gassman & Weisner, 2005; Nunes-Dinis, 1996).

Measures

Six hypothetical vignettes were presented to service providers in order to obtain their treatment recommendation for each distinct group: (1) a heavy-drinking pregnant woman, (2) a cocaine-using pregnant woman, (3) a heavy-drinking nonpregnant woman, (4) a cocaine-using nonpregnant woman, (5) a heavy-drinking man, and (6) a cocaine-using man. Each vignette instructed the provider to assume the client was white, single, 30 years of age, and had no money constraints (see Table 17.1 for example of a vignette). A heavy drinker was defined as someone who drinks five or more drinks in one sitting at least once a week, and a cocaine-using person was defined as someone who uses cocaine regularly. For each scenario, providers were given a list of nine possible treatment services: (1) hospital-based alcohol and drug program; (2) Twelve-Step program (Alcoholics Anonymous or Cocaine Anonymous); (3) one-on-

Table 17.1
Example of an Alcohol Vignette

These next questions ask for your treatment recommendation for people with different kinds of problems. First, please think about a pregnant woman who is a heavy drinker—by that I mean she drinks five or more drinks at a sitting at least once a week. Assume she's white, 30 years old, and single. If money were no object, what kind of service would you recommend to help her stop drinking? You'll find the possible answers to this on page 13 (of your booklet).

CODE ALL MENTIONED, WITHOUT PROBING FOR ANY OTHERS.

[ ] Would not recommend any service

(1) Medications like Antabuse or antidepressants to help her stop drinking

(2) Treatment in a hospital-based alcohol program under the direct supervision of a physician

(3) Prevention programs such as health education or community intervention

(4) Some kind of legal sanction—anything from a fine to incarceration

(SPECIFY: _____)

(5) Alcoholics Anonymous or a Twelve-Step program

(6) A stay in a halfway house or some other nonmedical residential program

(7) One-on-one or group psychotherapy

(8) Counseling by a minister, priest, or clergyman

Some other kind of service:

(9) (SPECIFY: _____)

(10) (SPECIFY: _____)

one or group psychotherapy; (4) counseling by a minister, priest, or clergyman; (5) nonmedical residential program (halfway house); (6) medications, such as Antabuse, antidepressants, or beta-blockers; (7) legal sanctions, such as anything from a fine to incarceration; (8) prevention or educational health or community intervention programs; or (9) specify any other kind of service. Providers were asked which treatment service, either only or in combination with other services, they would recommend to treat clients in each vignette.

Initial data analyses of service providers' treatment recommendations were performed by type of substance used (alcohol or cocaine), gender, and pregnancy status (pregnant or nonpregnant). These analyses showed similar service recommendation patterns by substance type and gender but not by pregnancy status. Based on these findings, analyses were then conducted by collapsing alcohol and cocaine users into one substance use variable and creating a dichotomous client grouping variable comprising (1) pregnant women and (2) men and nonpregnant women.

Dependent Variable (Service Profiles)

The most frequently reported services for pregnant women versus men and nonpregnant women were analyzed via reliability tests. Initially, an alpha value of .50 or higher was used to determine what services would be included in the categorization of each service profile. To maximize the total number of providers in each service profile, we included every service they selected. We categorized the four major services selected by providers (i.e., hospital-based alcohol and drug program, Twelve-Step program, psychotherapy, and halfway house) for the two client groups as either (1) recommended alone or (2) recommended alone or in combination with any other services. There were two types of dependent variables used as outcomes throughout the analysis for all client groups—the first included only one service, and the second included the service alone *or* in combination with any other services.

Independent Variables (Demographic Characteristics)

The demographic variables in this study include gender, education (less than a college degree, college degree, or postgraduate degree), age (less than 45 years or 45 years and over) and work setting (alcohol/drug, criminal justice, mental health, health care, welfare, and religious).

Data Analysis

Pearson's chi-square test of association was conducted on all categorical data. We report *p*-values. The chi-square test was conducted *within* each service

profile (and not across service profiles) to meet the mutually exclusive conditions required of categorical variables. The percentages shown in the tables are greater than 100 because providers could select multiple responses (or more than one service profile).

Logistic regression analyses were conducted to discern the best predictors for each provider service profile choice. The service profiles selected by providers were coded to distinguish whether the service was recommended: (1) alone or in combination with any other services versus not at all and (2) as one service profile only versus not at all. The independent variables were coded as 1 and 0. For example, gender was coded 1 for male and 0 for female. The independent variable age was coded using this same pattern (45+ vs. <45). For the independent variables with more than two categories, the comparison group "alcohol/drug" was selected for work setting variable and "less than a college degree" for the education variable. Alcohol/drug was selected as the comparison group because providers in this setting work directly in delivering alcohol and drug treatment services and are regarded as professionals with substance misuse treatment expertise. Providers in other work settings, however, may or may not provide direct alcohol/drug treatment services to their clients other than serving as referral sources. The data analyses were conducted with SPSS Version 6.0 (SPSS, 1996).

RESULTS

Service Profile Recommendations

Table 17.2A shows the top four service recommendations for treating alcohol- and cocaine-using pregnant women, and men and nonpregnant women by provider work setting, gender, age, and education. These four service profiles include: hospital-based alcohol and drug program, Twelve-Step program, one-on-one or group psychotherapy, and nonmedical residential program (halfway house), either alone or in combination with other services.

The majority of providers selected hospital-based treatment for pregnant women (88%) and about one-third recommended a Twelve-Step program. This relationship differed for treating the men and nonpregnant women, with over 60 percent of the providers selecting Twelve-Step programs and over half recommending hospital-based services. A similar proportion of providers (22%–30%) chose psychotherapy or halfway house services to treat both client groups.

Differences in characteristics of providers were found when they chose Twelve-Step for either pregnant women or men and nonpregnant women. Nearly half of the providers in the alcohol and drug setting recommended

Table 17.2A
Top Four Service Profiles Recommended by Providers across Their Work Setting and Demographics for Alcohol/Cocaine-Using Women (Pregnant and Nonpregnant) and Men Clients: Percentage Choosing That Service Alone or in Combination with any Other Services (*N* = 457)

		Hospital-based		Twelve-Step		Psychotherapy		Halfway house	
	n =	Pregnant women 392	Men and nonpregnant women 251	Pregnant women 144	Men and nonpregnant women 273	Pregnant women 97	Men and nonpregnant women 127	Pregnant women 105	Men and nonpregnant women 132
% of providers overall who picked services		88	56	32	61	22	29	24	30
Setting	n =	ns	ns	$p < .05$	$p < .001$	ns	ns	ns	ns
Alcohol/drug	93	88	53	47	65	24	24	41	53
Criminal justice	74	90	50	16	61	20	21	12	15
Mental health	89	87	66	29	52	21	29	25	34
Hospital/medical	77	88	48	40	76	27	39	16	27
Social welfare	69	95	61	27	61	20	34	25	27
Religious	43	80	57	28	44	18	30	13	20
Gender		ns	$p < .05$	$p < .05$	$p < .05$	ns	ns	ns	$p < .05$
Female	237	90	59	37	69	22	33	21	31
Male	218	84	48	26	58	25	33	16	22

Age		ns	ns	ns	p < .05	ns	ns	ns	ns
<45	241	90	55	36	69	24	35	19	30
45+	216	85	54	28	59	22	30	19	24
Education		ns	ns	ns	ns	Ns	ns	ns	ns
<College degree	151	89	55	32	66	17	29	20	24
College degree	78	88	52	27	71	23	39	12	23
Postgraduate degree	228	87	55	35	60	27	33	23	31

Analyses are conducted on weighted data. Percentages do not equal 100 because providers may have selected more than one service profile. Chi-square tests of significance were conducted within each service profile. ns = not significant.

Twelve-Step for pregnant women while less than one-fifth in the criminal justice setting selected this profile ($p < .05$). In addition, proportionately more female compared to male providers recommended Twelve-Step to a pregnant woman ($p < .05$). There were no differences in provider age and education in recommending Twelve-Step programs to pregnant women.

However, providers in a hospital or medical setting were most likely to recommend Twelve-Step programs for men and nonpregnant women, while those in a religious setting were least likely to select Twelve-Step for this population ($p < .001$). Furthermore, female and younger providers were more likely to choose Twelve-Step to treat men and nonpregnant women ($p < .05$). The only remaining significant differences in recommended service profiles were found with more female providers selecting hospital-based and halfway house services ($p < .05$).

One-Service Profile Recommendations

Table 17.2B presents comparisons of providers who selected only one service to treat alcohol- and cocaine-using pregnant women, men, and nonpregnant women. Nearly half of the service providers recommended only hospital-based treatment for pregnant women. Of those providers, over half in the criminal justice, mental health care, and social welfare settings chose only hospital-based treatment, while approximately one-third did so in the alcohol/drug, hospital/medical, or religious settings ($p < .01$). There were no other significant differences in selecting hospital-based treatment for pregnant women. Few providers recommended any other one-service profile for pregnant women.

There were few reports of providers selecting only one type of service to treat men and nonpregnant women. Less than one-fifth of the providers recommended hospital-based or Twelve-Step treatments alone for men and nonpregnant women.

Service Profile Alternatives

In an effort to further understand how providers select services for their clients, we next considered which of the dominant service profiles providers selected if they did not recommend one of the four major profiles. For example, what service did a provider choose who had not selected the Twelve-step profile for men and nonpregnant women? Table 17.3 presents the service profile alternatives to hospital-based, Twelve-step, psychotherapy, and halfway house treatments alone or in combination with any other services chosen by providers for treating alcohol- and cocaine-using pregnant women, men, and nonpregnant women. Few providers did not select the hospital-based service profile

Table 17.2B
Top Four Service Profiles Recommended by Providers across Their Work Setting and Demographics for Alcohol/Cocaine-Using Women (Pregnant and Nonpregnant) and Men Clients: Percentage Choosing One-Service Profile Only (*N* = 457)

		Hospital-based only		1Twelve-Step only		Psychotherapy only		Halfway house only	
		Pregnant women	Men and nonpregnant women	Pregnant women	Men and nonpregnant women	Pregnant women	Men and nonpregnant women	Pregnant women	Men and nonpregnant women
	n =	202	83	3	62	2	6	8	16
% of providers overall who picked services		45	19	<1	14	<1	1	2	4
Setting	n =	p < .01	ns	ns	ns	ns	ns	ns	ns
Alcohol/drug	93	35	6	—	12	—	—	6	12
Criminal justice	74	63	23	—	25	2	—	—	2
Mental health	89	51	30	—	8	—	—	2	2
Hospital/medical	77	36	7	—	21	—	2	2	2
Social welfare	69	51	25	—	12	—	3	2	—
Religious	43	37	22	2	96	—	2	—	4
Gender		ns	ns	ns	ns	ns	ns	ns	ns
Female	237	47	18	—	18	—	2	3	3
Male	218	39	17	1	12	1	2	1	2
Age		ns	ns	ns	ns	ns	ns	ns	ns
<45	241	47	15	—	17	<1	1	2	3
45+	216	40	20	<1	14	--	3	2	2

(*continued*)

Table 17.2B
(continued)

		Hospital-based only		1Twelve-Step only		Psychotherapy only		Halfway house only	
		Pregnant women	Men and nonpregnant women	Pregnant women	Men and nonpregnant women	Pregnant women	Men and nonpregnant women	Pregnant women	Men and nonpregnant women
	n =	202	83	3	62	2	6	8	16
Education		ns	ns	ns	ns	ns	ns	ns	ns
<College degree	151	39	16	1	18	1	1	1	2
College degree	78	58	17	—	12	—	—	—	1
Postgraduate degree	228	40	18	—	16	—	3	3	3

Analyses are conducted on weighted data. Percentages do not equal 100 because providers may have selected more than one service profile. Chi-square tests of significance were conducted within each service profile. ns = not significant. Analyses were not conducted in the shaded areas due to small numbers within these comparisons.

Table 17.3
Providers' Alternatives to Hospital-Based, Twelve-Step, Psychotherapy, or Halfway House Services Alone or in Combination with any Other Services for Alcohol/Cocaine-Using Women (Pregnant and Nonpregnant) and Men (*N* = 457), in Percentages

	No hospital-based		No Twelve-Step		No psychotherapy		No halfway house	
Services	Pregnant women	Men and nonpregnant women	Pregnant women	Men and nonpregnant women	Pregnant women	Men and nonpregnant women	Pregnant women	Men and nonpregnant women
n =	53	194	301	172	348	318	340	313
Hospital-based	N/A	N/A	88	68	88	54	91	53
Twelve-Step	33	75	N/A	N/A	18	54	25	59
Psychotherapy	25	32	6	13	N/A	N/A	16	24
Halfway house	38	25	10	17	12	17	N/A	N/A

Analyses are conducted on weighted data. Percentages do not equal 100 because providers may have selected more than one service profile.

for pregnant women (n = 53). Among those few, at least one-quarter recommended psychotherapy, Twelve-step, or a halfway house treatments. Although 301 providers did not select Twelve-step programs for pregnant women, 88 percent reported they would choose hospital-based treatment. A similar trend was found for those providers who would not recommend sending a pregnant woman to psychotherapy or a halfway house.

Approximately 70 percent of the providers did not select psychotherapy, while 68 percent did not choose a halfway house for men and nonpregnant women. Over half of those providers thus recommended Twelve-step and hospital-based treatment.

Service Profile Predictors

Logistic regression analyses were conducted to determine multivariate predictors of service profile choice (Tables 17.4A and 17.4B). Of the 202 providers who recommended only the hospital-based program for pregnant women compared to those in the alcohol and drug setting, providers in the criminal justice (odds ratio [OR] = 3.12, $p < .01$) and mental health (OR = 1.90, $p < .05$) settings were more likely to select this service. Additionally, male compared to female providers had 0.60 times the odds of recommending only the hospital-based service. Compared to those with less than a college degree, providers with a college degree were more likely to choose hospital-based services alone, and postgraduates were more likely to select both hospital-based and psychotherapy services. Providers who work in the criminal justice (OR = 0.29, $p < .01$), mental health (OR = 0.52, $p < .05$) and social welfare settings (OR = .41, $p < .05$) compared to those in an alcohol and drug setting were less likely to recommend Twelve-step alone or in conjunction with other services. A similar trend was seen for recommending halfway houses for pregnant women.

For men and nonpregnant women, providers in criminal justice, mental health, social welfare, and religious settings were more likely to select hospital-based treatment alone compared to providers in an alcohol and drug setting (OR = 7.68, $p < .001$; OR = 5.30, $p < .01$; OR = 6.36, $p < .001$; OR = 5.27, $p < .01$; respectively). No other significant differences arose for recommending only hospital-based treatment. Twelve-step alone was more likely to have been recommended by providers in the criminal justice system (OR = 3.03, $p < .05$) and the hospital/medical field (OR = 2.55, $p < .05$) compared to those in the alcohol and drug setting. Psychotherapy services were twice as likely to be selected by postgraduates compared to those with less than a college degree. Finally, providers in the alcohol and drug setting were more likely to choose

Table 17.4A
Logistic Regression Analyses Predicting Service Profiles Recommended by Providers across Their Work Settings and Demographics for Treating Alcohol/Cocaine-Using Pregnant Women Clients (*N* = 457)

	Hospital-based		Twelve-Step		Psychotherapy		Halfway house	
	Alone or with other services	Only	Alone or with other services	Only	Alone or with other services	Only	Alone or with other services	Only
Pregnant women n =	392	202	144	3	97	2	105	8
Setting (vs. alcohol/drug)								
Criminal justice		3.12**	.29**				.27**	
Mental health		1.90*	.52*				.46*	
Hospital/medical							.33**	
Social welfare			.41*				.38*	
Religious							.25**	
Gender								
Male (vs. female)		.60*						
Age								
45+ (vs. <45)								
Education								
College degree (vs. <college)		1.81*						
Postgraduate degree (vs. <college)	2.39*				2.76***			

Analyses are conducted on weighted data. Percentages do not equal 100 because providers may have selected more than one service profile. Analyses were not conducted in the shaded areas due to small numbers within these comparisons.

*$p < .05$; **$p < .01$; ***$p < .001$.

halfway house treatment either alone or in conjunction with other services compared to any other professional setting. Age was also a factor in selecting halfway house services. Providers 45 years or older had 0.64 times the odds of recommending halfway house services alone or with other services compared to those younger than 45.

Table 17.4B
Logistic Regression Analyses Predicting Service Profiles Recommended by Providers across Their Work Settings and Demographics for Treating Alcohol/Cocaine-Using Men and Nonpregnant Women Clients (*N* = 457)

	Hospital-based		Twelve-Step		Psycho-therapy		Halfway house	
	Alone or with other services	Only	Alone or with other services	Only	Alone or with other services	Only	Alone or with other services	Only
Men and nonpregnant women *n* =	251	83	273	62	127	6	132	16
Setting (vs. alcohol/drug)								
Criminal justice		7.68***		3.03*			.16***	
Mental health		5.30**					.37**	
Hospital/medical				2.55*			.28***	
Social welfare		6.36***					.25***	
Religious		5.27**	.40*				.19***	
Gender								
Male (vs. female)								
Age								
45+ (vs. <45)							.64*	
Education								
College degree (vs. <college)								
Postgraduate degree (vs. <college)					2.08**			

Analyses are conducted on weighted data. Percentages do not equal 100 because providers may have selected more than one service profile. Analyses were not conducted in the shaded areas due to small numbers within these comparisons.

*$p < .05$; **$p < .01$; ***$p < .001$.

DISCUSSION

We found that providers selected four dominant services to treat alcohol- and cocaine-using women (pregnant and nonpregnant) and men: hospital-based, Twelve-step, psychotherapy, and halfway house treatment. For pregnant women, providers selected primarily hospital-based treatment followed by Twelve-step programs, whereas for men and nonpregnant women this service profile preference was reversed. In contrast, selection of psychotherapy and halfway house services did not appear to be associated with the client group.

Service Profile Selection: Are There Differences across Provider Work Setting?

There is evidence that providers in alcohol and drug treatment settings have a different decision-making framework than other providers when selecting service profiles for pregnant and nonpregnant clients. For example, criminal justice and mental health providers were two to eight times more likely than those working in alcohol and drug programs to select hospital-based treatment as the only service profile for all client groups. There may be several reasons for this outcome. For instance, prior referral agreements may be in place; providers may perceive that hospital-based treatment represents a best practice or more effective treatment modality; or providers may assume that a hospital environment provides a more intensive level of patient care. The recommendation of hospital-based treatment for pregnant women may also be related to a variety of health concerns and legal issues. These issues may include the availability of prenatal care in the hospital setting, a decreased likelihood of legal intervention for the pregnant mother, and potential liability issues for the providers who fear medical complications coupled with potential negative birth outcomes (Finkelstein, 1993, 1994; Weisdorf et al., 1999).

Although 70 percent of the providers did not recommend Twelve-step at all for pregnant women, criminal justice and hospital/medical providers in non-alcohol/drug treatment settings were more likely than those working in the alcohol/drug treatment sector to select Twelve-step-only services for men and nonpregnant women. These providers may not think that formal treatment contributes greatly to the resolution of client alcohol or drug problems. These results are consistent with arguments made by Weisner et al. (Weisner, McCarty, & Schmidt 1999) regarding the disconnection between expectations of referral sources, like the criminal justice system and the workplace, versus those of treatment programs. Providers who are only in the treatment system may have a better appreciation for the limits of hospital-based treatment,

especially pertaining to provision of wraparound services that would address client's criminal behavior or absenteeism—the very issues that are paramount to criminal justice providers. Treatment providers also recognize the need for ongoing social support following a hospital-based treatment episode and thus tend to also recommend Twelve-step programs for all client groups.

In addition, several groups of providers were less likely than alcohol/drug treatment staff to recommend Twelve-step for men and nonpregnant women (religious setting) or for pregnant women (criminal justice, mental health, and social welfare). This may reflect a perception of Twelve-Step as giving too much authority, control, and responsibility to the client. This is consistent with our companion finding that criminal justice and hospital/medical providers are more likely (than treatment providers) to recommend Twelve-Step-only for nonpregnant clients. Since Twelve-Step services are free, these providers may be considering the following as relevant when pregnancy is not an issue: financial barriers to receive health services (Friedmann et al., 1999), insurance problems (Friedmann, D'Aunno, Jin, & Alexander, 2000; Greenfield et al., 2004), and growing evidence that client participation in self-help groups increases their abstinence rates from drugs and alcohol (Bond, Kaskutas, & Weisner, 2003; Humphreys et al., 2004; Humphreys & Moos, 2001).

One final revealing finding in terms of work setting difference between treatment system workers and those in other settings is the apparent reluctance of nontreatment staff to recommend halfway house services for all client groups. This may be a result of philosophical and professional differences. The providers working in the alcohol/drug setting, especially staff in halfway houses, are often individuals in recovery themselves who may be influenced by the principle of Twelve-Step programs that recovering alcohol/drug users are the best persons to help those with alcohol/drug addiction issues (Borkman et al., 1998). The medicalization and professionalization of substance abuse treatment does not favor the selection of peer-based halfway house treatment because medical intervention and licensing requirements transfer authority "from experiential knowledge to clinical training" (Borkman et al., 1998, p. 12). Providers may have selected halfway house services less often because they viewed these services as suitable only for some of their clients. For example, halfway house programs tend to serve low-income and minority populations (Borkman et al., 1998), so providers may select these programs less often for their middle-class clients with insurance. In addition, providers may be concerned that alcohol clients with a higher education level than peers or the staff may not view their authority or experiential knowledge with legitimacy (Borkman et al., 1998).

While outside of the alcohol/drug setting there was this lack of popularity of halfway house treatment, 12 percent of those within the treatment sector

selected halfway house as the only service profile for men and nonpregnant women. About one-quarter of the providers in our sample recommended halfway house services for pregnant women, a finding similar to Grella's (1999) study that found a higher proportion of pregnant women in women-only residential drug treatment programs and that service providers were more likely to refer pregnant than pregnant women to residential drug treatment. The preference for residential treatment of some sort was also reported in Howell and Chasnoff (1999). Taken together, these studies indicate a perceived need to remove pregnant substance-using women from their social and physical environment. More research is needed to further judge the apparent reluctance to recommend halfway houses.

Do Provider Demographics Predict Service Profile Choice?

We found that the demographic characteristics of providers were important in predicting their selection of service profiles. In the bivariate analysis, there were small but significant demographic differences of 10 percent more female than male providers selecting hospital-based, Twelve-Step, and halfway house services. In the multivariate analysis, male providers were less likely to recommend only the hospital-based service to treat pregnant women. The influence of provider gender has been studied and found to be the most important variable in relation to medical ethical decision making (Wertz, 1994). Wertz reported that female geneticists were less authoritative, more perceptive, and more compliant with patient requests than male geneticists. Provider gender has also been examined in regard to client satisfaction with community mental health services (Vetter, 2003) and how male and female service providers prioritize ethical dilemmas differently when selecting vocational rehabilitation services for persons with disabilities (Baker, 1996). As client demographic characteristics have often been used to understand and explain the potential reasons for their alcohol and drug treatment outcomes (Craddock, Rounds-Bryant, Flynn, & Hubbard, 1997; Simpson, 2003, 2004; Simpson, Joe, & Brown, 1997), provider gender also seems to influence the selection of alcohol and drug treatment services.

Similarly, provider age and level of education were significant in predicting their selection of service profiles. Providers 45 years of age or older were less likely to select halfway house services, and individuals with higher education were more likely to recommend hospital-based and psychotherapy services. Older providers and those with college degrees may be in higher-level management positions that require following agency dictates. For example, in some work settings such as the criminal justice, protocols may require providers to

refer clients medically before suggesting other service options. Ogborne et al. (Ogborne, Braun, & Schmidt 1998, 2001) have argued that research is needed on the characteristics of service providers (e.g., education) so that such information is available for the increased demands associated with establishing standards of care and accountability, having baseline data on treatment providers to monitor changes, and planning agency certification and professional development opportunities. Our research findings suggest that provider characteristics have intrinsic value as well, in that they predict service recommendations.

Limitations

The study limitations include: (1) limited generalizability; (2) a hypothetical whites-only client group; (3) categorization of the service profiles; and (4) age of the data set. This study is difficult to generalize because it examines a single U.S. county. However, the alcohol and drug problems and treatment policy of this county are similar to others throughout the United States; it has been used for the CEL studies and the U.S. site for the World Health Organization's Study of Community Response to Alcohol (Greenfield & Weisner, 1995; Weisner, Delucchi, Matzger, & Schmidt, 2003; Weisner & Schmidt, 1995). Issues of generalization have been previously addressed by testing measures and conducting comparisons with other national databases such as the National Alcohol Survey, the National Drug and Alcohol Treatment Utilization Survey/ Uniform Facility Data Survey, and the National Household Survey on Drug Abuse (see Greenfield & Weisner, 1995; Weisner et al., 2003).

The hypothetical treatment groups in this study were composed solely of white clients, which limits the generalizability of this hypothetical client. In future studies, hypothetical vignettes should include ethnically diverse clients in order to examine the potential effect of ethnicity on provider profile selection. This study was limited to vignettes about one racial group because the sample size (i.e., 457 providers across 10 strata was too small to incorporate additional vignettes).

Although the services recommended by providers were grouped according to the number of times they selected that service, this categorization may constitute a faulty classification scheme of the service profiles that may raise construct validity issues. Treatment in a hospital-based alcohol and drug program or some other nonmedical residential program also includes components of other services such as individual or group psychotherapy. The implications of incorrectly classifying profiles are serious because providers may recommend one type of service and consider it to be inclusive of all treatment components. For example, providers may not have selected psychotherapy as frequently as

other services because they consider that service already included in either hospital-based or nonmedical residential treatment programs.

Finally, the provider study data were collected more than 10 years ago. Since then, there have been changes in the treatment system and in the development and testing of new therapeutic approaches that may influence how providers would now recommend services. For instance, the current dominant setting for alcohol and drug treatment (more than 85%) is outpatient (McLellan, McKay, Forman, Cacciola, & Kemp, 2005; Substance Abuse and Mental Health Services Administration, 2002; Weisner et al., 2000). Furthermore, the number of uninsured individuals continues to climb (Fong, 2003; Wu, Kouzis, & Schlenger, 2003), and for the privately insured, alcohol and drug treatment under managed care reforms has become less intensive with the use of more group, episodic, and brief therapies (Oggins, 2003). Treatment therapies (Twelve-Step facilitation, cognitive-behavioral, and motivational enhancement), including medications such as naltrexone and acamprosate, have also been tested in clinical trials showing similar reductions in alcohol use from baseline to follow-up (Fiellin, Reid, & O'Connor, 2000). This may give providers treatment options to choose depending on what is locally available. Another therapeutic approach, the strengths perspective, which therapists utilize to empower clients in learning how to identify and build on their internal and external capacities to manage social problems, is used and has been recommended by social workers since the late 1990s to treat clients in addiction treatment (Saleebey, 1992, 2002; van Wormer & Davis, 2003). These changes may have an effect on provider selection of treatment services.

Implications

In general, providers in this study primarily selected four service profiles to treat all client groups. Clearly, one service was not considered adequate or custom-built for providers to treat clients. For those few providers recommending only one service, in consideration of the complex nature of many client alcohol and drug problems, we may need to develop training opportunities in treatment facilities and professional schools that emphasize the advantages of providing multiple services. Even though some providers may only offer referrals to clients for treatment services, their knowledge of viable and multiple treatment options is an important educational training goal in augmenting the service delivery alternatives recommended for clients. Further research is needed to examine why some providers select services within and outside of their own work setting. This will enhance our understanding of professionals' use and development of coordination of services across disciplines, including issues of

turf battles. Additionally, it is important to examine the role that client type or provider demographic characteristics plays in the selection of alcohol and drug treatment services. Such considerations may lead to the provision of appropriate gender-specific treatment services and to the development of curriculum opportunities and career planning advisement in professional schools as well as in treatment training programs.

A combination of survey and qualitative (e.g., in-depth interviews) research is needed to uncover provider motivations and intentions in treatment decision making, and to elucidate the processes by which past professional and personal experiences play a role in the variety of services selected to effectively treat alcohol/drug-using clients. To better understand the reasons why providers selected specific services for different client groups, future research should illuminate some of the questions currently inside the black box of treatment.

Acknowledgments

The authors wish to acknowledge Lee Kaskutas for support and mentorship on this project, and Connie Weisner, Lois Lowe, Enoch Haga, and Joseph Merighi for helpful comments on earlier drafts of this chapter. Preparation of this chapter was supported by National Institute on Alcohol Abuse and Alcoholism (NIAAA) (R01-AA005595-11) and California State University, Sacramento, Research and Creative Activity Award Program. Chapter presented at the Kettil Bruun Society, Social and Epidemiological Research on Alcohol: 31st Annual Symposium, May 30–June 3, 2005, Riverside, California.

REFERENCES

Baker, C. (1996). *The influence of gender on decision-making responses to ethical dilemmas encountered in transition planning.* Unpublished doctoral dissertation, Southern Illinois University at Carbondale, Carbondale, IL.

Bell, J. (2000). Quality improvement for methadone maintenance treatment. *Substance Use and Misuse, 35,* 1735–1756.

Bond, J., Kaskutas, L. A., & Weisner, C. (2003). The persistent influence of social networks and Alcoholics Anonymous on abstinence. *Journal of Studies on Alcohol, 64,* 579–588.

Borkman, T. J., Kaskutas, L. A., Room, J., Bryan, K., & Barrows, D. (1998). An historical and developmental analysis of social model programs. *Journal of Substance Abuse Treatment, 15*(1), 7–17.

Brower, K. J., Blow, F. C., & Beresford, T. P. (1989). Treatment implications of chemical dependency models: An integrative approach. *Journal of Substance Abuse Treatment, 6*(3), 147–157.

Burman, M. L., Kivlahan, D., Buchbinder, M., Broglio, K., Zhou, X. H., Merrill, J. O., et al. (2004). Alcohol-related advice for Veterans Affairs primary care patients: Who gets it? Who gives it? *Journal of Studies on Alcohol, 65*, 621–630,

Clark, J. A., Potter, D. A., & McKinlay, J. B. (1991). Bringing social structure back into clinical decision making. *Social Science and Medicine, 42*(8), 853–866.

Connors, G. J., DiClemente, C. C., Dermen, K. H., Kadden, R., Carroll, K. M., & Frone, M. R. (2000). Predicting the therapeutic alliance in alcoholism treatment. *Journal of Studies on Alcohol, 61*, 139–149.

Craddock, S. G., Rounds-Bryant, J. L., Flynn, P. M., & Hubbard, R. L. (1997). Characteristics and pretreatment behaviors of clients entering drug abuse treatment: 1969–1993. *American Journal of Drug and Alcohol Abuse, 23*(1), 43–60.

Crane, D. R., Wood, N. D., Law, D. D., & Schaalje, B. (2004). The relationship between therapist characteristics and decreased medical utilization: An exploratory study. *Contemporary Family Therapy, 26*(1), 61–69.

D'Aunno, T. A., & Vaughn, T. E. (1995). An organizational analysis of service patterns in outpatient treatment units. *Journal of Substance Abuse, 7*, 27–42.

Delany, p. J., Broome, K. M., Flynn, P. M., & Fletcher, B. W. (2001). Treatment service patterns and organizational structures: An analysis of programs in DATOS-A. *Journal of Adolescent Research, 16*(6), 590–607.

Etheridge, R. M., Craddock, S. G., Dunteman, G. H., & Hubbard, R. L. (1995). Client services in two national studies of community-based drug abuse treatment programs. *Journal of Substance Abuse, 7*, 9–25.

Etheridge, R. M., Hubbard, R. L., Anderson, J., Craddock, S. G., & Flynn, P. (1997). Treatment structure and program services in the Drug Abuse Treatment Outcome Study (DATOS). *Psychology of Addictive Behaviors, 11*, 244–260.

Fiellin, D. A., Reid, M. C., & O'Connor, P. G. (2000). New therapies for alcohol problems: Application to primary care. *The American Journal of Medicine, 108*, 227–237.

Finkelstein, N. (1993). Treatment programming for alcohol and drug-dependent pregnant women. *The International Journal of the Addictions, 28*, 1275–1309.

Finkelstein, N. (1994). Treatment issues for alcohol- and drug-dependent pregnant and parenting women. *Health and Social Work, 19*(1), 7–15.

Fischhoff, B. (1992). Decisions about alcohol: Prevention, intervention and policy. *Alcohol Health and Research World, 16*(4), 257–265.

Fong T. (2003, October 6). Up in arms-for now: Rise in uninsured draws immediate reaction. (The Week in Healthcare). *Modern Healthcare*, 1–2.

Forman, R. F., Bovasso, G., & Woody, G. (2001). Staff beliefs about addiction treatment. *Journal of Substance Abuse Treatment, 21*, 1–9.

Friedmann, P. D., Alexander, J. A., & D'Aunno, T. A. (1999). Organizational correlates of access to primary care and mental health services in drug abuse treatment units. *Journal of Substance Abuse Treatment, 16*(1), 71–80.

Friedmann, P. D., D'Aunno, T. A., Jin, L., & Alexander, J. A. (2000). Medical and psychological services in drug abuse treatment: Do stronger linkages promote client utilization? *Health Services Research, 35*(2), 443–465.

Gassman, R. A., & Weisner, C. (2005). Community providers' views of alcohol problems and drug problems. *Journal of Social Work Practice in the Addictions, 5*(4), 101–115.

Gerstein, D., & Harwood, H. (Eds.). (1990). *Treating drug problems* (vol. 1). Washington DC: National Academy Press.

Greenfield, L., Burgdorf, K., Chen, X., Porowski, A., Roberts, T., & Herrell, J. (2004). Effectiveness of long-term residential substance abuse treatment for women: Findings from three national studies. *The American Journal of Drug and Alcohol Abuse, 30*(3), 537–550.

Greenfield, T. K., & Weisner, C. (1995). Drinking problems and self-reported criminal behavior, arrests, and convictions: 1990 U.S. alcohol and 1989 county surveys. *Addiction, 90,* 361–373.

Grella, C. E. (1999). Women in residential drug treatment: Differences by program type and pregnancy. *Journal of Health Care for the Poor and Underserved, 10*(2), 216–229.

Hankin, J., McCaul, M. E., & Heussner, J. (2000). Pregnant, alcohol-abusing women. *Alcoholism: Clinical and Experimental Research, 24*(8), 1276–1286.

Howell, E. M., & Chasnoff, I. J. (1999). Perinatal substance abuse treatment: Findings from focus groups with clients and providers. *Journal of Substance Abuse Treatment, 17*(1–2), 139–148.

Humphreys, K., & Moos, R. H. (2001). Can encouraging substance abuse inpatients to participate in self-help groups reduce the demand for outpatient aftercare? A quasi-experimental study. *Alcoholism: Clinical and Experimental Research, 25,* 711–716.

Humphreys, K., Wing, S., McCarty, D., Chappel, J., Gallant, L., Haberle, B., et al. (2004). Self-help organizations for alcohol and drug problems: Toward evidence-based practice and policy. *Journal of Substance Abuse Treatment, 26,* 151–158.

Institute of Medicine. (1990). *Broadening the base of treatment for alcohol problems.* Washington, DC: National Academy Press.

Kaskutas, L. A., Witbrodt, J., & French, M. (2004). Outcomes and costs of day hospital treatment and nonmedical day treatment for chemical dependency. *Journal of Studies on Alcohol, 65,* 371–382.

Keller, M. (1990). *Models of alcoholism: From days of old to nowadays* (pamphlet series). New Brunswick, NJ: Center of Alcohol Studies, Rutgers University.

Leonelli, B. T. (1993). Provider expectations and alcoholism treatment: A clinical perspective. *Alcoholism Treatment Quarterly, 10*(1/2), 209–215.

McKinlay, J. B., Potter, D. A., & Feldman, H. A. (1996). Non-medical influences on medical decision-making. *Social Science and Medicine, 42*(5), 769–776.

McLellan, A. T., McKay, J. R., Forman, R., Cacciola, J., & Kemp, J. (2005). Reconsidering the evaluation of addiction treatment: From retrospective follow-up to concurrent recovery monitoring. *Addiction, 100*(4), 447–458.

Mitchell, T. B., Dyer, K. R., & Peay, E. R. (2006). Patient and physician characteristics in relation to decision making in methadone maintenance treatment. *Substance Use and Misuse, 41,* 393–404.

Musto, D. F. (2002). Recovery from alcoholism. In D. F. Musto (Ed.), *Drugs in America: A documentary history* (pp. 158–159). New York: New York University Press.

Nunes-Dinis, M. C. (1996). An analysis of model choice of service providers using Scott's and Lipsky's theoretical frameworks: Women (pregnant and non-pregnant) and men drinkers. Unpublished doctoral dissertation, University of California, Berkeley.

Ogborne, A. C., Braun, K., & Schmidt, G. (1998). Working in addictions treatment services: Some views of a sample of service providers in Ontario. *Substance Use and Misuse, 33*(12), 2425–2440.

Ogborne, A. C., Braun, K., & Schmidt, G. (2001). Who works in addictions treatment services? Some results from an Ontario survey. *Substance Use and Abuse, 36*(13), 1821–1837.

Oggins, J. (2003). Changes in health insurance and payment for substance use treatment. *The American Journal of Drug and Alcohol Abuse, 29*(1), 55–74.

Room, R., & Weisner, C. (1988). *Studying community responses to alcohol problems: An interim report.* Presented at the 14th Annual Alcohol Epidemiology Symposium, Kettil Bruun Society, Berkeley, California, June 5–11.

Saleebey, D. (1992). The strengths perspective in social work practice. New York: Longman.

Saleebey, D. (2002). Introduction: Power in the people. In D. Saleebey (Ed.), *The strengths perspective in social work practice* (3rd ed.). Boston: Allyn & Bacon.

Simpson, D. D. (2002). A conceptual framework for transferring research to practice. *Journal of Substance Abuse Treatment, 22,* 171–182.

Simpson, D. D. (2003). Introduction to 5-year followup treatment outcome studies [editorial]. *Journal of Substance Abuse Treatment, 25,* 123–124.

Simpson, D. D. (2004). A conceptual framework for drug treatment process and outcomes. *Journal of Substance Abuse Treatment, 27,* 99–121.

Simpson, D. D., Joe, G. W., & Brown, B. S. (1997). Treatment retention and follow-up outcomes in the drug abuse treatment outcome study (DATOS). *Psychology of Addictive Behaviors, 11*(4), 294–307.

SPSS Inc. (1996). SPSS for Windows: Base system user's guide, release 6.0. Chicago, IL: Author.

Strang, J., & Sheridan, J. (2001). Methadone prescribing to opiate addicts by private doctors: Comparison with NHS practice in south east England. *Addiction, 96,* 567–576.

Substance Abuse and Mental Health Services Administration. (2002). *National Survey of Substance Abuse Treatment (NSSAT): Data for 2000 and 2001* (DHHS Publication No. SMA 98-3176). Washington, DC: U.S. Government Printing Office.

Trentacoste, N. D., Holtgrave, D. R., Collins, C., & Abdul-Quader, A. (2004). Disseminating effective behavioral interventions for HIV prevention: A cost analysis of a risk-reduction intervention for drug users. *Journal of Public Health Management and Practice, 10*(2), 130–139.

Van Wormer, K., & Davis, D. R. (2003). *Addiction treatment: A strengths perspective*. Pacific Grove, CA: Brooks/Cole–Thomson Learning.

Vetter, H. M. (2003). Effects of ethnicity and consumer-provider ethnic match on adult client satisfaction with a community health center. Unpublished doctoral dissertation, University of La Verne, La Verne, CA.

Wampold, B. E., & Brown, G. S. (2005). Estimating variability in outcomes attributable to therapists: A naturalistic study of outcomes in managed care. *Journal of Consulting and Clinical Psychology, 73*, 914–923.

Weisdorf, T., Parran, T. V., Graham, A., & Snyder, C. (1999). Comparison of pregnancy-specific interventions to a traditional treatment program for cocaine-addicted pregnant women. *Journal of Substance Abuse Treatment, 16*(1), 39–45.

Weisner, C., Delucchi, K., Matzger, H., & Schmidt, L. (2003). The role of community services and informal support on five-year drinking trajectories of alcohol dependent and problem drinkers. *Journal of Studies on Alcohol, 64*, 862–873.

Weisner, C., Matzger, H., Tam, T., & Schmidt, L. (2002). Who goes to alcohol and drug treatment? Understanding utilization within the context of insurance. *Journal of Studies on Alcohol, 63*, 673–682.

Weisner, C., McCarty, D., & Schmidt, L. (1999). New directions in alcohol and drug treatment under managed care. *The American Journal of Managed Care, 5*, SP57-SP69.

Weisner, C., Mertens, J., Parthasarathy, S., Moore, C., Hunkeler, E., Hut, T. W., et al. (2000). The outcome and cost of alcohol and drug treatment in an HMO: Day hospital versus traditional outpatient regimens. *Health Services Research, 35*(4), 791–812.

Weisner, C., & Schmidt, L. (1995). The Community Epidemiology Laboratory: Studying alcohol problems in community and agency-based populations. *Addiction, 90*(3): 329–341.

Wertz, D. C. (1994). Provider gender and moral reasoning: The politics of an "ethics of care." *Journal of Genetic Counseling, 3*(2), 95–112.

White, W. L. (1998). *Slaying the dragon: The history of addiction treatment and recovery in America*. Bloomington, IL: Chestnut Health Systems/Lighthouse Institute.

Wu, L-T., Kouzis, A. C., & Schlenger, W. E. (2003). Substance use, dependence, and service utilization among the US uninsured nonelderly population. *American Journal of Public Health, 93*(12), 2079–2085.

Chapter **18**

An Innovative Approach to Prison-Based Substance Use Treatment: The Implementation of Inmate Peer Mentor Programs

Jana Cook, MD, PhD, Scott E. McClure, PhD,
Igor Koutsenok, MD, MS, and Scot Lord, CATC, FACT

SUBSTANCE USE AND INCARCERATION

The United States currently maintains the largest prison population in the world. Over the past 30 years there has been a continuous increase in the number of inmates in U.S. state and federal prisons. The 2006 Bureau of Justice Statistics figures illustrate that nearly 1.6 million inmates occupy our nation's federal and state prisons. California leads the nation's population totals with 175,512 inmates, rivaled only by Texas, which hosts 172,116 inmates. During 2006, the prison population grew at an alarming rate of 2.8 percent from 2005, making it the fastest rate of growth in the past five years.

The relationship between substance use and criminal incarceration is an undeniable factor in the nation's prison crisis. The 2003 Arrestee Drug Abuse Monitoring Program (ADAM) report identified that nearly 74 percent of all male and female offenders tested positive for either drugs or alcohol. Due to the high prevalence of substance use disorders among criminal offenders, rigorous efforts to develop effective treatment interventions have occurred. Significant advances in addiction treatment methods and research have lead to substantial progress in the identification and implementation of evidenced-based addiction treatment practices, which are essential for the continued growth and improvement of prison-based addiction treatment (Friedmann, Taxman, & Henderson, 2007).

PRISON-BASED THERAPEUTIC COMMUNITY TREATMENT

The therapeutic community (TC) is identified as a highly effective evidence-based treatment model for incarcerated individuals with substance use disorders (Pearson & Lipton, 1999; Welsh, 2007). The TC is defined as a long-term, highly structured, hierarchical, intensive behavioral modification treatment model. TCs are drug-free, mostly residential treatment environments that foster increased levels of personal and social responsibilities, primarily through peer influence. The TC model utilizes a variety of group processes to help individuals learn and assimilate prosocial norms, values, skills, and behaviors. The TC comprises a mix of professional staff and peers, with the primary agent of change being the norms and rules of the community. The treatment process is focused on overlapping areas of personal development that include: behavioral modification; emotional/psychological development; intellectual and spiritual growth; vocational/educational growth; life coping skills; and biomedical management if needed.

Meta-analytic reviews have identified the TC as an effective model to reduce recidivism rates for incarcerated individuals with substance use histories (Aos, Miller, & Drake, 2006; Lipton, Pearson, Cleland, & Yee, 2002). The addition of postrelease continuing care has been shown to further reduce recidivism rates compared to untreated control groups and in-prison-treatment-only groups with no continuation of care (Knight, Simpson, & Hiller, 1999; Martin, Butzin, Saum, & Inciardi, 1999; Wexler, Melnick, Lowe, & Peters, 1999). While the majority of the literature highlights the necessity of aftercare following in-prison TC treatment for successful reductions in recidivism and reduced relapse potential (Inciardi, Martin, & Butzin, 2004; Knight et al. 1999; Martin et al., 1999; Messina, Burdon, Hagopian, & Prendergast, 2004; Wexler et al., 1999), there is evidence that prison-based TC treatment can effectively reduce recidivism rates independent of continuation of care postrelease (Prendergast, Hall, Wexler, Melnick, & Cao, 2004). Most recently, Welsh (2007) examined the two-year postrelease treatment outcomes across five Texas prison-based TC programs and identified that TC treatment significantly reduced rearrest and reincarceration rates compared to treatment control groups that consisted of a mix of psychoeducation, peer-led ancillary support groups, and drug and alcohol education programs. Most importantly, these results were maintained without continuation of care postrelease and may lend support to the fundamental TC principle that the power of the community is the agent of change.

CHALLENGES WITH PRISON-BASED TC TREATMENT PROGRAMS

Clearly the TC is an effective treatment model for incarcerated individuals with substance use disorders; what is less clear are the discrepancies in treatment effectiveness found across treatment programs. One potential explanation for differences in treatment outcomes may be an inconsistency of treatment programming, which often leads to a modification of the evidence-based model. The rapid increase of TC implementation in corrections-based treatment has lead to deviations from the original model to meet the specific needs of the institutions providing treatment. These needs are affected by the diversity of the population being treated, mixing the general inmate population with the treatment population, limited resources for treatment implementation resulting in efforts to reduce treatment costs, and institutional demands such as overcrowding and limited availability of qualified staff for both corrections and treatment. Unfortunately, these modifications usually result in less intensive programming schedules, curriculum-driven treatment, and staff that is not adequately trained to deliver services as demanded by the evidence-based model. These modifications to the original evidence-based model can place limitations on the ability to effectively utilize essential TC components, which ultimately eliminate the core principle of the community as the agent of change, the critical component that drives the TC model (Saum et al., 2007).

Modifications to the evidence-based model hinder the ability of researchers to identify the effective components of TC treatment. Simultaneously, modifications that have not been adequately tested to demonstrate effective treatment may result in reduced quality of treatment. Other dilemmas for effective TC treatment implementation include obstacles such as frequent staff turnover, often influenced by salaries that are not adequate for the cost of living in some regions, as well as burnout due to individuals working with a demanding population and a lack of adequate training and experience. Farabee et al. (1999) identified the recruitment and training of treatment staff as one of the major common barriers to developing effective correctional treatment. This theory has persisted for nearly a decade and was recently revisited in the February 2007 Inspector General's Report (Cate, 2007), which identified the implementation of treatment as a current failure of our system rather than a lack of effective treatment models.

The University of California Los Angeles (UCLA) noted in its evaluation report of in-prison treatment programs that providers in rural areas have difficulties locating and hiring staff with previous training and experience in the

therapeutic community treatment model (Prendergast, 2002). Limited staffing pools and high turnover rates among the substance abuse treatment counselors have led to problems in maintaining quality and continuity in treatment services. The importance of variables related to effective treatment counselors seems to be underestimated and is insufficiently represented in the literature (Wormith et al., 2007).

In a typical TC, clients and staff make up the community, creating a social environment in which peers and staff represent role models of successful personal change and serve as guides to the recovery process. In order to stimulate an effective treatment process, it is crucial to address the treatment delivery system. One solution to the maintenance and retention of qualified staff in areas with limited applicant pools of qualified treatment staff is to hire and train individuals who were successful participants in prison TC treatment. Unfortunately, the limited acceptability of hiring individuals in recovery and ex-felons makes the situation even more difficult. Although the original TC model supports the use of recovering addicts as staff (DeLeon, 2000), it is very difficult and often impossible to hire ex-offenders, primarily because of the internal policies of many correctional institutions.

DEVELOPMENT OF A PEER MENTOR–FACILITATED TC

An alternative and promising strategy for effective treatment delivery in a difficult-to-staff correctional environment is to recruit and train inmates with long prison sentences as peer counselors and mentors. This strategy would eliminate staff turnover issues and allow the TC to operate as a 24-hour continuous treatment environment as the original model intended. Furthermore, well-trained recovering inmates are likely to be more capable of establishing credibility and demonstrating understanding compared to hired treatment staff with little or no background in substance use treatment. In addition, long-sentenced inmates are active participants in both the TC and prison in general, which bring back a model of treatment more like the original TCs that were entirely peer driven (DeLeon, 2000).

The successful implementation of inmates as peer mentors has already taken place at the Amity prison-based TC project, established in 1990 at Richard J. Donovan (RJD) state prison in San Diego. Specially trained and supervised lifer mentors ensured 24-hour support of the TC culture and environment beyond the working hours of treatment staff (Prendergast et al., 2004), thus enhancing the power of community.

In October 2006, the California Men's Colony (CMC) prison in San Luis Obispo, faced with staff recruitment and retention difficulties, decided to uti-

lize long-term sentenced inmates as peer mentors to lead their prison-based TC program. Prior to this decision, several attempts to utilize contracted treatment providers from within the state as well as one out-of-state treatment provider had failed. This protracted struggle inspired the correctional counselor III (George Freitas) to use the human resources available, inmates with long prison terms. Mr. Freitas recognized that there was a large pool of participants in need of treatment services as well as a valuable resource of peer-mentor inmates with long-term sentences who had previous experience in TC treatment. Some of these participants had a history of active involvement in prison-based TC treatment that stretched back over 15 years, and their experience with the TC model was unquestionable. Mr. Freitas approached the California Department of Corrections and Rehabilitation (CDCR) with a proposal to begin the implementation of a new type of program, a program in which peer mentors would provide substance abuse treatment under close correctional and clinical supervision. CDCR accepted the proposal, and the CMC Substance Abuse Treatment Program "Our House" became the first custody-based TC program within CDCR entirely run by peer mentors, independent from any outside treatment providers.

TRAINING THE PEER-MENTOR FACILITATORS

Upon program approval, the Office of Substance Abuse Programs (OSAP) requested that the mentors receive the most comprehensive and highest level training possible. The University of California San Diego (UCSD) Center for Criminality and Addiction Research, Training, and Application (CCARTA) has long held the responsibility of training all California state in-prison treatment staff in substance abuse treatment and was therefore recruited to train the peer mentors. UCSD provides a Work Force Development (WFD) training series that features 155 hours of comprehensive substance abuse treatment curriculum, including a 30-hour cross training in which treatment and custody professionals strive to improve their working relations through team-building and problem-solving exercises. The training is fully compatible with the instruction given to outside providers and is designed to fulfill the educational requirements of the nationally accredited Forensic Addictions Correctional Treatment (FACT) certification program.

UCSD CCARTA delivered the full curriculum directly to the peer mentors on-site at CMC over the course of five months, which began in February 2007, and finished in June 2007. In addition to UCSD training materials, the peer mentors were provided with publications from the Substance Abuse and Mental Health Services Administration's (SAMHSA) Treatment

Improvement Protocol (TIP) and Technical Assistance Publication (TAP) series of books. The inmates were noticeably well-studied and enthusiastic to learn new material. Following each training session, the trainees completed a written test to ensure they met the highest standards of competency. The cross training proved to be a particular highlight of the series as correctional officers and peer mentors participated together in the 30-hour training. This provided a unique experience in which correctional officers and inmate peer-mentor trainees worked cooperatively to learn how to support one another in the common goal of collaboratively producing positive treatment outcomes.

Following completion of the Work Force Development training, the Orange County Department of Education (OCDE) joined the CMC Our House TC program to help supervise and manage program logistics such as providing materials needed to run the community, curriculum arrangement, and liaison services between custody and peer mentors. OCDE staff is composed of qualified professionals in the delivery of alternative education for youth and adults who have an established working relationship with CDCR. Their staff also attended CCARTA WFD trainings to enhance their understanding of substance use treatment in correctional settings but did not stand in as replacement counselors in an effort to allow the community to remain peer driven. In other words, the OCDE plays an important role in supporting the community's structural and educational needs but does not provide clinicians or counselors.

THE OUR HOUSE TC TREATMENT PROGRAM

The Our House treatment program is an inmate peer-mentor-facilitated multiphase treatment continuum grounded in the TC treatment approach. It adheres to the TC model by holding daily process groups, diverse subject matter curriculum groups, community celebrations and ceremonies, and community meetings designed to recognize and address positive and negative behaviors displayed by participants.

The curriculum includes information on the development of employment skills (i.e., job search resources, resume writing, interviewing skills); life skills (i.e., decision making, effective communication, building healthy relationships); and education on relapse prevention strategies, appropriate parenting techniques, introduction to computer literacy and information technology, as well as functional financial strategies such as budgeting and credit restoration.

It is important to mention that the phase progression process (or lack of such) for each participant is based entirely on behavioral indicators as identified by the community. This process is more in line with the original TC model, rather than phase transition based on time enrolled in the program, which is

unfortunately the common determinant for phase progression in most of the California prison-based TCs. Overall, the program is designed to facilitate comprehensive behavioral modification toward prosocial norms, a core function of any TC program.

The first phase of the Our House TC program is orientation. It is facilitated by two of the most seasoned TC peer mentors, each with more than 15 years of experience in the model. During the orientation phase, participants learn the basic principles of a TC, how it operates, and how they can benefit from being a part of it. Participants are also instructed in what behaviors are deemed appropriate and inappropriate within the community as well as the range of reinforcements for prosocial behaviors and the consequences of negative actions. During this phase, participants are informed of the community expectation of sharing personal experiences as a method to enhance personal growth.

Following completion of the orientation phase, participants progress to the second phase of treatment, called "breaking barriers." During this phase, peer mentors guide participants toward an increased understanding of the group process, their personal process, emotional maturity, and coping strategies for counterproductive emotional states with the aid of the Our House program breaking barriers group. For some participants, this is their first exposure to the group process, so the peer mentor's role is to safely guide them toward an understanding of this process as well as train them in enhanced communication skills and healthy boundaries.

The third treatment phase, alternatives to violence, requires an increased expectation for TC participants to demonstrate critical analysis of their behavior and increased motivation for change. During this phase, peer mentors progressively challenge participants' behaviors to create a safe and supportive environment within a culturally diverse group. This is particularly important because prison settings in California actively participate in racial segregation and inmate participants often have difficulty with emotional expression. A blend of nondirective and interactive forums guided by the alternatives to violence and life skills curriculum, process groups that utilize a "here and now" focus for the exploration of emotions and related behaviors, are used to achieve these goals.

The final in-prison treatment phase, prerelease, is focused on preparation for parole. It addresses such subjects as continuing education, employment skills, and financial literacy. In order to minimize postrelease-related stress, peer mentors address the following issues with the participants: (1) Is the participant ready for transition? (2) Is the participant prepared to get a job and maintain it? (3) Does the participant have a safe support system (including continued care) in place? Each participant develops a personal postrelease plan, which is discussed and scrutinized within the community. The maturity and

supportive structure of the community during this phase offers participants a safe environment to talk about the pressing issues and anxieties associated with the transition to parole.

During the prerelease phase, participants are given the option to attend the Truck Driving Academy (TDA). The TDA is a widely accepted and successful truck driving program equipped with state-of-the-art simulators, software, and projectors. The TDA offers quality instruction and curriculum and provides participants with the opportunity to pass a written truck driving exam. This truck driving course is currently the only one of its kind in California, and completion provides each graduate with a new employable skill upon parole.

CONCLUSION

In times of organizational change within prison-based substance abuse treatment programs, the ability to provide cost-effective, professional, and evidence-based treatment is essential. The recent development and implementation of the peer-mentor-facilitated Our House TC program may present a solution to some of the barriers that limit the success of prison-based addiction treatment.

The concept of the mentor has ancient origins as described in the classic Greek epic poem Homer's *Odyssey* when Odysseus left his young son in the care of a companion named Mentor. Today, peer mentors remain a valid and irreplaceable component of addiction treatment. Mentoring is the process by which an older (or more experienced) person counsels and guides a younger (or less experienced) person in the needed skill set. The mentor serves as a protector, coach, and counselor.

Through critical self-examination and strategic planning, the peer-mentor-facilitated Our House TC treatment pilot project will continue to evolve. Since its establishment in October 2006, the program has demonstrated an exceptional ability to influence participation in postrelease continuing care. From September 2007 to February 2008, the Our House TC treatment program reported mean postrelease aftercare rates of 81 percent, a rate far higher than all other prison-based TC programs in the state (G Freitas, personal communication, July 28, 2008). If this trend continues, it may demonstrate an effective model for increased post-release aftercare, a variable shown time and again to be instrumental for reduced recidivism.

REFERENCES

Aos, S., Miller, M., & Drake, E. (2006). *Evidence-based adult corrections programs: What works and what does not*. Olympia, WA: Washington State Institute for Public Policy.

Cate, M. L. (2007). Office of the Inspector General. Special review into in-prison substance abuse programs managed by the California Department of Corrections and Rehabilitation. Retrieved April 16, 2007, from http://www.oig.ca.gov/reports/pdf/SubstanceAbusePrograms.pdf.

De Leon, G. (2000). *The therapeutic community: Theory, model, and method.* New York: Springer Publishing.

Farabee, D., Prendergast, M., Cartier, J., Knight, K., Wexler, H., & Anglin, M. (1999). Barriers to the implementation of prison-based substance abuse treatment programs. *Prison Journal, 79*(2), 150–162.

Friedmann, P. D., Taxman, F. S., & Henderson, C. E. (2007). Evidence-based treatment practices for drug-involved adults in the criminal justice system. *Journal of Substance Abuse Treatment, 32,* 267–277.

Inciardi, J. A., Martin, S. S., & Butzin, C. A. (2004). Five-year outcomes of therapeutic community treatment of drug involved offenders after release from prison. *Crime and Delinquency, 50,* 88–107.

Knight, K., Simpson, D. D., & Hiller, M. L. (1999). Three-year reincarceration outcomes for in-prison therapeutic community treatment in Texas. *The Prison Journal, 79,* 337–351.

Lipton, D. S., Pearson, F. S., Cleland, C. M., & Yee, D. (2002). The effects of therapeutic communities and milieu therapy on recidivism. In J. McGuire (Ed.), *Offender rehabilitation and treatment: Effective programmes and policies to reduce re-offending* (pp. 39–78). Etobicoke, Canada: John Wiley.

Martin, S. S., Butzin, C. A., Saum, C. A., & Inciardi, J. A. (1999). Three year outcomes of therapeutic community treatment for drug-involved offenders in Delaware: From prison to work release and aftercare. *The Prison Journal, 79,* 294–317.

Messina, N., Burdon, W., Hagopian, G., & Prendergast, M. (2004). One year return to custody rates among co-disordered offenders. *Behavioral Sciences and the Law, 22,* 503–518.

Pearson, F. S., & Lipton, D. S. (1999). A meta-analytic review of the effectiveness of corrections-based treatment for drug abuse. *The Prison Journal, 79,* 384–410.

Prendergast, M. L. (2002). *Using evaluation to improve program implementation.* Presented as testimony before the Little Hoover Commission. Retrieved October 10, 2007, from http://www.lhc.ca.gov/lhcdir/drug/PrendergastMay23.pdf.

Prendergast, M. L., Hall, E. A., Wexler, H. K., Melnick, G., & Cao, Y. (2004). Amity prison-based therapeutic community: 5-year outcomes. *The Prison Journal, 84*(36), 36–60.

Saum, C. A., O'Connell, D. J., Hiller, M. L., Bacon, G. A., Simpson, D. D. (2007). Tempest in a TC. Changing treatment providers for in-prison therapeutic communities. *Criminal Justice and Behavior, 34*(9), 1168–1178.

Welsh, N. W. (2007) A multisite evaluation of prison-based therapeutic community drug treatment. *Criminal Justice and Behavior, 34,* 1481–1498.

Wexler, H. K., Melnick, G., Lowe, L., & Peters, J. (1999). Three-year reincarceration outcomes for Amity in-prison therapeutic community and aftercare in California. *The Prison Journal, 79,* 321–336.

Wormith, J. S., Althouse, R., Simpson, M., Reitzel, L. R., Fagan, T. J., & Morgan, R. D. (2007). The rehabilitation and reintegration of offenders. The current landscape and some future directions for correctional psychology. *Criminal Justice and Behavior, 34*(7), 879–892.

Chapter **19**

Advances in Corrections-Based Treatment: Building the Addiction Treatment Workforce

Igor Koutsenok, MD, MS, and Scott E. McClure, PhD

BIASES AND PRECONCEPTIONS AS BARRIERS TO EFFECTIVE TREATMENT IN CORRECTIONAL SETTINGS

The relationship between substance use and criminal incarceration is an undeniable factor in the nation's prison crisis. The 2003 Arrestee Drug Abuse Monitoring Program (ADAM) report identified that nearly 74 percent of all male and female offenders tested positive for either drugs or alcohol. Because of the high prevalence of substance use disorders among criminal offenders, rigorous efforts to develop effective treatment interventions have occurred. Over the past 20 years there has been substantial growth in addiction treatment research for criminal offenders (Friedmann, Taxman, & Henderson, 2007). In the past decade efforts to implement research into practical application has made tremendous progress and has identified the utilization of evidence-based practices as an essential factor for the improved quality of treatment in correctional settings (Friedmann et al., 2007).

Advances in neuroscience, brain imaging, molecular neurobiology, and the behavioral sciences have significantly enhanced our understanding of addictive behaviors and their treatment (Koutsenok, Deitch, Marsolais, & Franc, 2004). Regardless of these advances, there remains an unfortunate gap between the scientific research and its implementation in correctional treatment settings. One barrier to the implementation of evidence-based principles in correctional treatment programs is that many addiction treatment counselors trained to

work in correctional environments come with preconceived biases of what works in treatment. These biases are most often based on personal experience, previous training experiences, varied expectations, professional orientation, personal beliefs about addiction, and an overall lack of knowledge about addiction and treatment, especially for offender populations. In addition, many addiction treatment counselors are in recovery themselves and have been heavily influenced by the treatment models that made their recovery successful. Unfortunately, these biases are rarely evidence-based or scientifically driven, often resulting in a resistance to utilize scientifically proven treatment strategies under the premise that their way is the only way (Leshner, 1997). This attitude can have a detrimental influence over treatment delivery, especially if it remains with individuals as they advance into leadership positions and permeate their views throughout treatment staff.

In addition to treatment biases shared by addiction treatment professionals, there are also many common counterproductive beliefs held by custody staff, such as: "nobody gets better," "they don't deserve treatment anyway," and that treatment professionals are an additional threat to prison safety as they may participate in illegal activities with inmates or are obstacles in the way of correctional staff maintaining order. These biases can serve as major obstacles for treatment as they increase tensions between corrections and treatment staff, and they are an unfortunate side effect of the misperceptions of job duties and functions of both correctional and treatment staff.

Biased attitudes in addiction treatment can arrest the development of effective treatment implementation. This is especially problematic in correctional treatment programs where offender treatment needs are escalated because of stressors unique to the prison environment. These stressors include so-called prison yard politics, lockdowns, prison guard interactions, lack of social and familial support, and often increased incidents of violence compared to civilian environments. The consequences for not implementing treatment principles proven to work in correctional settings include programmatic failure, increased recidivism rates, and stressful work environments for staff who are trained in evidence-based principles during professional trainings but are not supported by leadership when they make attempts to implement these principles. As a result, many treatment professionals adopt a business as usual attitude, which is unacceptable in our current system, where recidivism is the norm. By far, the largest consequence is a failure to effectively change the addictive behaviors of offenders. This failure contributes to the revolving door of offender recidivism, leaving society to pay the consequence for those who resist the implementation of scientifically proven addiction treatment principles.

DEVELOPING THE MODERN DAY ADDICTION TREATMENT COUNSELOR

The need for well-trained addiction treatment counselors was recognized as early as the 1960s when most physicians, psychologists, psychiatrists, and social workers did not want to be involved with substance use treatment (Mustaine, West, & Wyrick, 2003). In the early 1970s the Comprehensive Alcohol Abuse and Alcohol Prevention, Treatment, and Rehabilitation Act was passed by Congress, which resulted in large sums of money to be utilized for the treatment of alcohol use disordered patients as well as for the training of treatment staff to implement proper care for this population (Banken & McGovern, 1992). Congress additionally established the National Institute of Drug Abuse (NIDA) and the National Institute for Alcohol Abuse and Alcoholism (NIAAA), which marked the beginning of specialized trainings for the counseling skills needed to treat individuals with substance use disorders (Banken & McGovern, 1992).

The fast-growing and immediate need for substance use treatment counselors and the lack of counselors available limited the field's ability to require formal academic graduate level training of substance use counselors. As a result, the addiction treatment field has relied more heavily on counselors' education and experience, which is primarily gained through workshops, training seminars, or college courses as well as two to three years of experience working in the field under the guidance of experienced counselors (Mustaine et al., 2003). In an attempt to develop a minimum standard for the training and education for addiction treatment counselors, addiction treatment certifications were developed. The immediate need for addiction treatment counselors and lack of participation in addiction treatment practices from other professional groups has lead to certification training requirements that remain controversial in regard to the minimal training standards deemed acceptable for certification when compared to the higher academic qualifications needed for other counseling professions (Mustaine et al., 2003).

Regardless of the growing body of evidence that suggests a significant increase in quality of treatment outcomes with increased training and education, the educational requirements for addiction counselor certifying bodies are significantly less than those required by other counseling professions (Mustaine et al., 2003). Though there are some college- and graduate-level addiction treatment specialists in the workforce, the majority of the workforce is composed of adults from various cultural and vocational backgrounds, without college education, and often with personal histories of substance use rehabilitation.

Kerwin, Walker-Smith, and Kirby (2006) performed a comparative analysis of state requirements for the training of substance abuse and mental health counselors and identified that 45 percent of states do not require a college degree to qualify as a substance abuse counselor, whereas 98 percent of states require at least a master's degree to qualify as a mental health counselor. Only one-half of the states require a credential to be qualified as substance abuse counselors compared to 86 percent of states that require a credential to be qualified as a mental health counselor.

The current obstacle of balancing the need for treatment versus the lack of qualified applicants, due to low salaries and often undesirable work locations, hinders the substance use treatment field's ability to hire individuals with the required background and training for optimal treatment outcomes. This is especially true in correctional treatment programs, often located in undesirable areas to live or in areas where the cost of living far exceeds the pay of treatment staff. Unfortunately, these barriers remain despite the growing demands faced by modern-day addiction treatment counselors, who are expected to understand the co-occurring complexities of mental health issues, family dynamics, as well as the gender and cultural differences of the individuals they treat.

Ideally, all addiction treatment professionals working in correctional environments would meet the education and training standards utilized by medical and other professional treatment models. In reality, the need for qualified treatment counselors in correctional settings far exceeds the availability of graduate-trained professionals. This has resulted in the addiction treatment field making its best efforts to develop qualified individuals at the pace of placement need.

Today's addiction treatment counselors are required to understand and utilize the latest evidence-based treatment procedures (motivational interviewing, therapeutic community, cognitive-behavioral therapy, and risk-needs assessment), case management skills, as well as the ability to recognize mental and physical health issues and learning disabilities. Most of these requirements were traditionally performed by master's-and doctorate-level clinicians with much more education and training. These increased expectations and demands have led the addiction treatment field to look to the scientific communities of university's and other professionals to aid in the dissemination of the latest evidenced-based practices and theories needed for the treatment of addiction disorders in correctional settings. The immediate and vast need to disseminate evidence-based addiction treatment for offenders has opened the door to the new and evolving field of professional training for addiction treatment professionals.

TECHNOLOGY TRANSFER AND BARRIERS TO SUCCESSFUL KNOWLEDGE DISSEMINATION

The promotion and adoption of evidence-based practices and state-of-the-art addictions research is essential for the improvement of frontline addiction treatment (Addis, 2002). Current approaches to disseminate advances in scientific research, such as conference presentations and journal articles, are geared mostly toward academia and not frontline addiction treatment staff. Even though evidence-based principles are often translated into manuals or practice guidelines, this process does not guarantee the actual implementation or behavioral change by providers in correctional settings. Likewise, addiction treatment agency leaders may be quick to adopt new practices without taking into account the fact that frontline addiction counselors may lack the education and training needed to successfully implement these practices. In addition, because many corrections-based addiction treatment counselors have not been educated in traditional graduate degree programs, their attitudes about research are often skeptical, leading to a lack of research-practice partnerships and little awareness of the benefits of evidence-based approaches.

The successful dissemination of evidenced-based knowledge into correctional addiction treatment programs depends largely on the knowledge, skills, and attitudes of the organizations and individuals being trained. Though training is an essential tool for technology transfer, it is only one aspect of the overall knowledge dissemination process. Technology transfer involves creating a mechanism by which change is accepted and reinforced at all organizational levels (Addiction Technology Transfer Center Network [ATTC], 2004). In essence, successful technology transfer in corrections-based addiction treatment needs to be designed to develop a sustainable model and mechanism for workforce development specific to the needs of this unique environment. For clients in treatment, change may be slow, difficult, occur in stages, and produce natural resistance. In a similar way, any attempt to change practices among practitioners and treatment programs may be met with discomfort, doubt, and hesitancy.

Successful knowledge dissemination, whether through professional training or other means, is best performed when the disseminating body takes into consideration an organization's stages of change (Prochaska & DiClemente, 1982) and addresses each of them with appropriate strategies. The use of incremental and gradual interventions along with contingency planning and ongoing monitoring of new treatment principles and implementation are essential for effective knowledge dissemination. Effective knowledge dissemination will address the following goals: (1) Establish systems to measure performance and

ensure *accountability;* (2) build, enhance, and maintain newly acquired treatment infrastructure and *capacity;* and (3) enable all trainees and providers to deliver *effective* treatment services.

The common barriers for successful training and knowledge dissemination often overlap. It is essential that trainers are prepared to manage an expected level of resistance to change with trainees just as knowledge dissemination experts are prepared for organizational resistance to change. Several barriers for successful training that often overlap with barriers for technology transfer have been identified (Addiction Technology Transfer Center Network [ATTC], 2004; Bartholomew et al., 2007; Farabee et al., 1999; Koutsenok et al., 2004):

- Staff and funding limitations that dictate the type and frequency of training treatment professionals can attend.
- Lack of time for trainings including client overload, too much paperwork, and other duties to meet organizational demands.
- New material conflicts with agency philosophy.
- Maintenance of preconceived beliefs about substance use treatment regardless of scientific evidence.
- Leadership is not supportive of changes.
- Access to training and education is limited for providers in rural settings.
- High turnover rates for substance use disorder treatment staff.
- Conflicting goals between corrections and treatment.
- Correctional officers are frequently transferred to different yards, limiting the ability for corrections and treatment staff to establish relationships.

Barriers to change, whether on the individual trainee or organizational level, are to be expected, and strategies to address these barriers will expedite the change process. To minimize resistance, trainers should skillfully and directly address it. This can be achieved through discussion of the pros and cons of change, listening to the fears and concerns of individuals, actively listening to resistors, providing incentives and rewards, and celebrating small victories (ATTC, 2004). In addition, it is important to involve as many people as possible in the knowledge transfer process, including frontline providers, supervisors, directors, correctional staff, and, when appropriate, offender clients who are leaders in their communities. This will ensure that everyone affected by the change process is involved and will provide a safe platform for cross-agency discussions about the implementation of change that is mediated by knowledge transfer experts. Though many barriers exist between science and the implementation of evidence-based practices in correctional settings, there is no barrier too great to account for the insufficient training of scientifically validated treatment strategies (Koutsenok et al., 2004).

TRAINING ADULT LEARNERS

Adults are a unique group of learners who have often acquired a significant amount of life experience, knowledge, and skills that can both help and hinder their ability to learn. In order to maximize training effectiveness for adult learners, trainers must use educational concepts that are personally relevant to them and their work environment (Koutsenok et al., 2004). Adult learners perceive themselves as responsible, self-directing, and independent persons who demand that the trainer accept them as such. Resistance to learning often solidifies under conditions that do not maintain the concept of self-direction and instances when training content is not relevant to practice. The best approach to training adults is to view training as a planned and sequential process designed to provide meaning to the learner. This process is best when it activates previous knowledge, elicits self-awareness, and allows adult learners to utilize the skills, knowledge, and attitudes needed to perform particular tasks relevant to the learner (Koutsenok et al., 2004.; Stuart, Tondora, & Hoge, 2004).

In order to foster the most effective training environment for adult learners, trainers must maintain a balance between making directive decisions for trainees while, whenever possible, encouraging trainees to be responsible for suggestions regarding training content and design. This increases feelings of ownership and fosters personal engagement with the training. In addition, adult learners have been found to identify believability, acceptability, and usefulness of information as requirements for successful training implementation (Stuart et al., 2004). The most effective trainings for adults are designed with the following points in mind:

- A climate of mutual respect must exist between trainer and trainees.
- An open, friendly, and casual atmosphere that facilitates the exchange of differing viewpoints and ideas should be fostered.
- Trainees should be aided in identifying their own skill and growth needs, as well as in analyzing the specific training elements needed by their agency.
- Adults learn best from that which is relevant and useful to them. "Here and now" challenges should be the focus of training. Whenever possible, training ideas should be applied to current work situations. The emphasis should always be on illustrating new concepts as they relate to the life and work experiences drawn from the trainees (Kolb, 1984).

EFFECTIVE TRAINING TECHNIQUES FOR ADDICTION COUNSELORS IN CORRECTIONAL SETTINGS

Professional trainers for corrections-based addiction treatment are most effective if open-minded, culturally sensitive, and well-educated in the specific

needs of the individuals who work in correctional settings. The typical training participants from corrections-based addiction treatment programs include correctional professionals, addiction treatment counselors, and medical and educational staff. Individuals who provide frontline addiction treatment in correctional settings are faced with many dynamics and demands unique to the prison environment. Treatment participants are usually coerced into treatment, frequently do not believe they need treatment, are forced to deal with inmate politics, have to cope with inmate racial tensions, and often share both an addiction to substance use and criminal behavior. In addition to unique inmate dynamics, corrections-based addiction treatment counselors come from several cultures and backgrounds, may or may not be in recovery, may have served time in jail or prison, or may have no history of legal involvement or substance use. Corrections-based treatment programs are made up of a diverse mixture of treatment staff and inmates who often have different worldviews, philosophies, and life experiences. Thus, it is essential that professional trainers are aware and sensitive to the unique needs of the individuals with whom they train.

Without a doubt, the implementation of efficient and effective training is needed for addiction treatment counselors working in correctional environments. The 2007 SAMHSA action plan for behavioral health administration reported significant concern by stakeholders regarding the nature of training and education currently offered to the workforce. Currently, there is a need for more evidence-based training research in the addiction treatment field (Bartholomew, Joe, Rowan-Szal, & Simpson, 2007). Because there is a lack of research on evidence-based training practices for addiction treatment counselors, we must look to the scientific literature regarding effective teaching and skill development strategies utilized by the medical and behavioral health fields.

First and possibly most important—the solitary use of didactic lecture, the method of instruction by which an individual delivers knowledge through lecture to individuals who do not yet possess this knowledge (Stuart et al., 2004)—has been shown to increase knowledge but yields no scientific evidence of building skills among trainees (Korte, 2006; Stuart et al., 2004). Thus, the traditional teaching technique that involves an active teacher and passive participants is not an effective method of training for adult learners. Typical errors trainers make in addition to the sole use of didactic lecture include:

- Not thoroughly understanding the material they train.
- Not staying with the training objective.
- Not relating the subject content to the audience.
- Being underprepared.
- Poor time management.
- Not involving the audience.

- Underestimating the audience.
- Providing false information.

SKILLS FOR EFFECTIVE TRAINING DELIVERY

Effective trainers have not only successfully learned to avoid the typical training errors but have also developed enhanced skills in communication and public speaking. Effective trainers are adept at creating audience participation. They use several means to encourage audience discussion, including: the strategic use of disagreement to stimulate discussion, pulling multiple audience-delivered ideas together, positive affirmations and compliments to validate participation, as well as interactive role plays.

Prior to training delivery, it is essential that trainers are well-prepared. When possible, it is best for trainers to have direct communication with the organization in need of their services. This allows the trainer to identify and research the specific needs of the organization in order to thoroughly prepare for the training session. It is recommended that trainers develop an abstract of the training needs, take time to study relevant research or other relevant materials, anticipate and prepare for problems that may occur during training, and take time to practice the material to be delivered.

In addition to being well-prepared, trainers must remain open-minded and fluid in their approach. The specific needs of the training may shift or be changed spontaneously before or during the training session. Trainers who are well-educated and experienced in the training topic and have solid technique will make this transition feel natural to the audience without interrupting the flow of the training.

Prior to training delivery, it is important to arrive 30–45 minutes early in order to properly set up the room, organize training materials, and resolve any problems associated with the facility or training equipment. When trainees begin to arrive, trainers are to maintain a positive attitude, greet the audience, pass out outlines and materials, and then highlight three to five major points that will be covered during the training. This ensures the audience knows who the trainer is, why he is there, and what they can expect to learn from the training.

While presenting the training material, trainers should be sensitive to the attention span of the trainees, taking frequent breaks if needed. A good rule of thumb for adult attention span is to add 15 minutes to the age of the adult. For example, a 40-year-old adult is likely to have an attention span of 55 minutes. Though attention spans will vary, it is advised to take short breaks every 1.5 to 2 hours, encourage participant involvement, and have energizing activities throughout the training if presenting in a didactic mode.

Slide presentations are to be aimed at providing both direction and structure for the information presented. They are best if the information is accompanied by illustrations, pictures, short videos, and humor when appropriate. A good slide presentation will both provide needed information and stimulate the senses of the trainee. Slide presentations should be concise, orderly, have visual stimulus, and use the least amount of words possible to deliver the point. They should avoid language above the 10th-grade reading level, and if new vocabulary or jargon is used, these terms should be explained by the trainer upon first use.

During the presentation delivery, trainers are most effective if they make good eye contact, involve the audience, monitor their delivery time, continuously connect and summarize the delivered information, and end with major points of discussion. It is very important for trainers to maintain control of the group discussion and not allow for participants to highjack the training with comments or discussions that move away from the goals of the training. The successful use of humor and positive reinforcement for appropriate audience participation can ease this process. Most importantly, the trainers and the trainees should have fun while training. Enthusiastic and compassionate trainers will absorb the audience in the material and allow the training to be a fun learning experience.

EFFECTIVE TRAINING MODALITIES IDENTIFIED THROUGH RESEARCH

Training success is measured by its impact on organizational change and the utilization of new skills by individuals within the organization. Even if trainings are delivered by the most charismatic and interesting trainers in the world, if no behavioral change occurs after the training, it was ineffective. In addition to effective training delivery, it is important for trainers to utilize the modalities and techniques of training as identified through scientific research. This will increase the likelihood that the training will have a lasting impact on individual and organization change. Training modalities that have been identified as effective include: interactive training sessions; sequenced, longitudinal learning; outreach; auditing of practice with feedback; reminders; and the utilization of opinion leaders (Bartholomew et al., 2007; Davis et al., 1999; Korte, 2006; Stuart et al., 2004).

Interactive training is a form of active learning that identifies learners at the center of the educational process, involves problem-solving and solutions related to the learner, is self-directed and facilitator-guided, and utilizes a two-way cooperative engagement between the facilitator and learner (Stuart et al.,

2004). Interactive learning is often achieved through the use of small task-oriented groups, large problem-solving groups, role-play activities, and interactive exchanges between facilitators and trainees. Bartholomew et al. (2007) found that engagement in training materials and comfort with the applicability of the material while training was predictive of later use of training materials in practice.

Sequenced, longitudinal learning (academic detailing) is marked by face-to-face interactions with the intent of changing a trainee's behavior and his clinical practices. Academic detailing techniques include: conducting individual interviews to investigate baseline knowledge and motivations for current practice behavior patterns; defining clear educational and behavioral objectives; using concise, graphic educational materials; highlighting and repeating essential messages; referencing unbiased scientific literature while presenting both sides of controversial issues; engaging active trainee involvement in the academic detailing process; and providing positive reinforcement of improved practices in follow-up visits.

Outreach visits are performed by specialized learning facilitators who meet with trainees at their place of occupation to examine trainees while working and provide information for enhanced performance of treatment techniques.

Auditing of practice with feedback is any summary of counselor performance over a given period of time (Mazmanian & Davis, 2002). It may or may not include recommendations for improvements and differs from outreach visits in that audits may be achieved through examination of inmate treatment records, databases, inmate interviews, or direct observation.

Reminders are any interventions that prompt trainees to utilize the skills acquired from trainings (Stuart et al., 2004). Reminders can include stickers, posters, follow-up reports of techniques used, visits from trainers, and any other mechanism that prompts the use of newly acquired skills.

The training of *opinion leaders* involves focused training on organizationally identified influential peers that will in turn have influence on training other staff through formal or informal education (Stuart et al., 2004). Often, organizations need influential staff members to enhance readiness for change when new treatment modalities are identified. Accurate identification and training of opinion leaders can speed the process of knowledge dissemination and reduce resistance to change from coworkers.

To date, the aforementioned training techniques have been identified to be effective through scientific investigation and should serve as the golden standard for effective knowledge dissemination. Other training techniques such as didactic lecture, single-session presentations and workshops, as well as providing handouts, audiovisual materials (e.g., videos), or electronic publications

have been identified as having limited impact on changing trainee behavior (Stuart et al., 2004) but are still helpful if used in combination with the previously listed evidence-based approaches and should not be discarded entirely.

Though many techniques have been identified as effective when used alone, combining multiple techniques has been shown to significantly increase training effectiveness. The positive effects of combining training interventions was demonstrated by Davis, Thomson, Oxman, and Haynes (1995) when they found that including two or more interventions resulted in a 64 percent increase in positive provider behavior change and that adding an additional intervention resulted in a 79 percent positive behavior change. Thus, training has the most impact on behavioral change when mixtures of several techniques are combined. Combining multiple techniques allows the learner to take on new information through multiple channels, helps decrease boredom, and increases the odds of affecting individuals with different learning styles (Koutsenok et al., 2004; Stuart et al., 2004).

CROSS-TRAINING IN CORRECTIONS-BASED ADDICTION TREATMENT

Cross-training is a unique and essential training for addiction treatment staff who work in correctional settings. It unites addiction treatment staff and corrections staff for several days to provide a safe, mediated environment for both staffs to work together to develop an increased understanding of their job roles in the correctional environment. Unfortunately, both treatment staff and corrections staff alike share many misconceptions about each other and their job duties. For example, treatment staff often jokingly refers to corrections staff as "knuckle draggers," while corrections staff refer to treatment staff as "hug a thugs." Though seemingly humorous, such names paint a picture of the misconceptions of the job duties of both staffs.

It is important to have cross-trainings early in a new treatment program's development and as frequently as possible to help train new staff. Cross-training serves a vital role in improving relationships between treatment and custody staff. This relationship is essential for effective treatment delivery as both treatment and corrections staff have constant direct contact with offenders in treatment and share the overarching goal of reducing offender recidivism. During a cross-training, both staffs participate in educational and team-building activities. These activities provide factual information about job roles, safety awareness, and allow individuals to interact in a less restrictive manner, which helps them understand each other's similarities and differences beyond their job descriptions.

While training, it is not unusual to be met with marked resistance from both corrections and treatment staffs. Comments commonly made by one or both staffs include, "You don't understand our jobs," "They aren't children, kids, or clients; we call them inmates," or "Treatment or rehabilitation means going soft." These beliefs are usually superficially based and often resolved before the end of training. Effective trainers will allow for open discussion, even if heated, and will monitor the mood and appropriateness of the discussants. In doing this, trainers will provide factual evidence to clarify myth and opinion, validate individual emotions and experiences, help both staffs better understand each other's job roles, and work to help both corrections and treatment staff understand their shared goal of reduced recidivism.

MULTIPLE DOMAINS OF LEARNING

The successful implementation of several learning techniques can be demonstrated through the utilization of multiple domains of learning while providing professional training. Learning theory has identified three independent domains of learning that can be experienced simultaneously: cognitive, affective, and psychomotor. The *cognitive* domain is the mind-based domain, which is focused on acquisition of information, intellectual ability, and the thinking process. Training strategies to stimulate this domain often include didactic lecture, manuals and handouts, and one-on-one interactions. The *affective* domain is the feeling domain and is focused on the internalization and increased connection to emotions, attitudes, values, beliefs, and personalized interests. Training strategies that stimulate the affective domain include Socratic questioning, case vignettes, role-playing, gaming, and group discussions. The *psychomotor* domain is a skill-based domain that focuses on motor activities. Training strategies to stimulate the motor domain are focused on imitation, practice, and habit development. Techniques may include demonstration and practice of new techniques while training.

Effective trainings will include activities to elicit each of the learning domains, and many activities may elicit several domains simultaneously. For example, imagine a role-play activity that addresses conflict resolution through communication and body language. Participants are asked to pair up and role play an offender client and counselor involved in conflict with one another. Each trainee must first understand the didactic lecture and handouts provided prior to the activity, which reflects cognitive learning. Each participant will also utilize her own personal emotional experiences, which will likely change throughout the role-play activity during conflict escalation and resolution. In addition, each trainee will be asked to provide the role-play demonstration to a larger group

in order to have his skills evaluated and critiqued. This single training activity elicits the cognitive, emotional, and psychomotor learning domains, is interactive, evaluative, and a good method of multidimensional learning acquisition.

MAINTENANCE OF SKILLS LEARNED POSTTRAINING: IMPLEMENTING CLINICAL SUPERVISION

The success of knowledge dissemination is dependent on the implementation and maintenance of the newly trained skills in the workplace. Though several training techniques, as discussed earlier, have been shown to increase skill comprehension and utilization in the field, more efforts are needed to ensure quality implementation and longevity of change with frontline treatment providers working in correctional settings. As mentioned in the technology transfer section of this chapter, organizational acceptance of evidenced-based practices is instrumental for long-term program change and growth. If acceptance is achieved, the implementation of a clinical supervisor well-versed in evidence-based practices for addiction treatment in correctional settings will substantially increase the speed of change as well as the quality of services delivered.

Addiction treatment professionals who receive quality clinical supervision have been shown to demonstrate increased job performance, cope better with stress, and are less prone to work burnout (Roche, Todd, & O'Conner, 2007). Because of this, clinical supervision may be an essential component for improved treatment outcomes in correctional settings. Clinical supervision can protect against treatment staff burnout, which often leads to increased staff turnover. Staff turnover often places undue demands on the remaining staff as it temporarily increases caseloads and responsibilities, which often include training new treatment staff. Adding licensed clinical supervisors, such as psychologists, social workers, or other clinically trained professionals can provide a source of support and guidance needed for treatment staff to help cope with work-related issues, protect against burnout, and enhance clinical skills. In addition, like inmates, corrections-based addictions counselors have to cope with unique stressors such as highly resistant clients, violence, and philosophical differences between corrections staff and treatment staff. The presence of a clinical supervisor who is well-versed in addiction treatment, corrections policy, and addiction counselor needs may serve as the necessary support needed to reduce staff stress and improve treatment outcome.

In order to accurately implement clinical supervision, it is essential to make the distinction between line-management supervision and clinical supervision (Roche et al., 2007). Although line-management supervision does share some

overlap with clinical supervision, such as professional development delivery and professional practice appraisal, it is primarily focused on organizational quality control and performance appraisals that are utilized for promotions, dismissal, and career development. Thus, because of its hierarchical nature, line-management can be a barrier for some individuals to obtain the objective support they need for skill enhancement and other job-related issues. In contrast, the effective implementation of clinical supervision minimizes power differential issues by not exercising authority over supervisees but providing a safe environment with minimal disclosure over issues discussed in supervision, with exception to mandated topics dictated by law or organizational policy.

An additional method to enhance the maintenance of skills learned in trainings is to increase the number of hours required for workforce development. Currently, the education and experiential requirements needed to be an addiction treatment counselor remain controversial, as most all clinical and health-related fields have substantially higher standards for professional certification (Mustaine et al., 2003). Though the exceptional demand for addiction treatment professionals renders it unreasonable to expect all treatment staff to be licensed clinicians with extensive training in substance use disorders, increasing the amount and quality of trainings along with the implementation of clinical supervision is a reasonable solution to enhance the current workforce. An increase in overall training hours is necessary to enhance the skills learned in trainings. Increased training hours would allow for follow-up visits by trainers to assess for new skill implementation, improvement needs, and organizational barriers to change. In addition, increased training-based education incentives, such as higher pay and promotions for treatment staff who choose to increase their formal education and treatment experience, can help develop new leaders for the future of addiction treatment with an enhanced understanding and acceptance of evidence-based practices and principles.

The implementation of clinical supervisors, increased training hours aimed at skill development and program skill utilization assessment, as well as increased incentives for individuals who make efforts to increase their formal education and training experiences in the addiction treatment field can assure that knowledge dissemination of evidence-based practices is successful.

CONCLUSION

Over the past 20 years, substantial change and growth has occurred in corrections-based addiction treatment. In the past decade significant advances in evidence-based treatment for offenders have been made. These advances have given scientific evidence to the most effective treatment modalities to be used

with offender populations and will likely become the norm for offender addiction treatment as government funding bodies increase the standards of program effectiveness based on outcome measurement.

The addiction treatment field originated as a community-driven model and did not adhere to the path of development of the medical model. The medical model is based on a top-down hierarchy in which individuals are required to obtain the appropriate training, education, and experience prior to providing treatment services. The overall lack of participation by psychology, psychiatry, and other behavioral health professionals in the treatment of individuals with addiction along with the vast need and rapid growth of the addiction treatment field has led it to be developed from the bottom up.

With time, formalized trainings emerged as the specific needs for addiction counselors were identified, eventually leading to the presently controversial certification process. Though controversial because of minimal education and training standards, certification is a necessary step to ensure the quality of training that addiction treatment counselors receive. Ideally all addiction treatment counselors would receive formal education and training, similar to that of other medical and behavioral health professionals, but at this time it is unrealistic if we are to meet the needs and demands of the addiction workforce. Thus, as in other professional models, the implementation of effective training aimed precisely at the unique demands and requirements of addiction treatment counselors is a viable option for the development of the field.

When utilizing a professional training and certification model for workforce development, it is essential that the quality of training and trainers is at its highest capacity. Though there is a lack of scientific research specifically aimed at training in addiction treatment, there is literature about the training modalities found to be most effective with adult learners. Training modalities that have been identified as effective include: interactive training sessions; sequenced, longitudinal learning; outreach; auditing of practice with feedback; reminders; and the utilization of opinion leaders (Bartholomew et al., 2007; Davis et al., 1999; Korte, 2006; Stuart et al., 2004). In addition to effective training modalities, trainer style is also a key component for successful knowledge dissemination. Trainers who are open-minded, authentic, honest, and allow adult learners to actively participate in the training process are most effective.

Cross-trainings are unique to correctional addictions treatment environments and have emerged to help foster productive relationships between treatment and correctional staff. Cross-trainings aim to help both staffs develop an enhanced understanding of their role in offender treatment. Trainers facilitate the cross-training process, allowing individuals to discuss their perceptions and misperceptions of job responsibilities. Trainers serve as mediators to ensure a safe learning environment. They present fact-based information, challenge

biased opinions and myths, and facilitate group-oriented activities that influence interaction and team building between corrections and treatment staff. Cross-trainings are an essential component in corrections-based addiction treatment in that they help develop the camaraderie and understanding needed by both staffs to cooperate toward the common goal of reducing offender recidivism.

Fortunately, with specialized trainings aimed at disseminating the latest evidence-based research for addictions treatment, the addiction treatment field can grow to meet the capacity of needs in the field. With this being said, the success of knowledge dissemination is dependent on its utilization in the field after individuals are trained. For this to occur, trainings need to follow scientifically driven principles, be facilitated by well-trained and educated professional training staff, and be reinforced through clinical supervision, organizational acceptance, follow-up trainings, and implementation analysis. The ability to successfully improve the addiction treatment workforce is highly dependent on these factors. Hence, the future success of corrections-based addiction treatment is highly dependent on organizational, leadership, and treatment counselor attitudes, as well as the economic availability to maintain qualified treatment staff, implement clinical supervision, develop well-rounded leadership, and increase the amount of trainings and evaluations necessary to bring science to frontline practice.

REFERENCES

Addis, M. E. (2002). Methods for disseminating research products and increasing evidence-based practice: Promises, obstacles, and future directions. *Clinical Psychology, 9*(4), 367–378.

Addiction Technology Transfer Center Network. (2004). *The change book: A blueprint for technology transfer* (2nd ed.). Kansas City, MO: CSAT. Retrieved April 10, 2007, fromhttp://www.nattc.org/pdf/The_Change_Book_2nd_Edition.pdf

Banken, J. A., & McGovern, T. F. (1992). Alcoholism and drug abuse counseling: State of the art consideration. *Alcoholism Treatment Quarterly, 9*(2), 29–51.

Bartholomew, N. G., Joe, G. W., Rowan-Szal, G. A., & Simpson, D. D. (2007). Counselor assessments of training and adoption barriers. *Journal of Substance Abuse Treatment, 33,* 193–199.

Davis, D., O'Brien, M.A.T., Freeman, N. Wolf, F. M., Mazmanian, P. , Taylor-Vaiset, A. (1999). Impact of formal continuing medical education: Do conferences, workshops, rounds, and other traditional continuing education activities change physician behavior or health care outcomes? *Journal of the American Medical Association, 282,* 867–874.

Davis, D. A., Thompson, M. A., Oxman, A. D., & Haynes, B. R. (1995). Changing physician performance: A systematic review of continuing medical education strategies. *Journal of the American Medical Association, 274,* 700–705.

Farabee, D., Prendergast, M., Cartier, J., Wexler, H., Knight, K., & Anglin, M. D. (1999). Barriers to implementing effective correctional drug treatment programs. *The Prison Journal, 79,* 150–162.

Friedmann, P. D., Taxman, F. S., & Henderson, C. E. (2007). Evidence-based treatment practices for drug-involved adults in the criminal justice system. *Journal of Substance Abuse Treatment, 32,* 267–277.

Kerwin, M. E., Walker-Smith, K., & Kirby, K. C. (2006). Comparative analysis of state requirements for the training of substance abuse and mental health counselors. *Journal of Substance Abuse Treatment, 30,* 173–181.

Kolb, D. A. (1984). Experiential learning: experience as the source of learning and development. Englewood Cliffs, NJ: Prentice-Hall.

Korte, R. F. (2006). Training implementation: Variations affecting delivery. *Advances in Developing Human Resources, 8,* 514–527.

Koutsenok, I, Deitch, D. A., Marsolais, K., & Franc, M. (2004). Enhancing substance abuse treatment in correctional settings. In K. Knight & D. Farabee (Eds.), *Treating addicted offenders: A continuum of effective practices* (pp. 38-1–38-8). Kingston, NJ: Civic Research Institute.

Leshner, A. (1997). Addiction is a brain disease, and it matters. *Science, 278,* 45–47.

Mazmanian, P. E. & Davis, D. A., (2002). Continuing medical education and the physician as a learner. *Journal of the American Medical Association, 288,* 1057–1060.

Mustaine, B. L., West, P. L., & Wyrick, B. K., (2003). Substance abuse counseling certification requirements: Is it time for a change? *Journal of Addictions and Offender Counseling, 23,* 99–107.

Prochaska, J. O., & DiClemente, C. C. (1982). Transtheoretical therapy: Toward a more integrative model of change. *Psychotherapy, Theory, Research and Practice, 19,* 4–6.

Roche, A. M., Todd, C. L., & O'Conner, J. (2007). Clinical supervision in alcohol and other drugs field: An imperative or an option? *Drug and Alcohol Review, 26,* 241–249.

Stuart, G. W., Tondora, J., & Hoge, M. A. (2004). Evidence-based teaching practice: Implications for behavioral health. *Administration and Policy in Mental Health, 32,* 107–130.

U.S. Department of Health and Human Services. (2007). *An action plan for behavioral health workforce development.* Cincinnati, OH: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, The Annapolis Coalition on the Behavioral Health Workforce .

Chapter **20**

Sacred Medicines for Harm Reduction and Substance Abuse Rehabilitation

Michael Winkelman, MPH, PhD

The concept of sacred medicines refers to substances also known as psychedelics, hallucinogens, entheogens, and psychointegrators (see Rätsch, 2005; Schultes & Hofmann, 1992; also see Winkelman 1996, 2007a, for overview).[1] Humans have used plants as sacred medicines for thousands of years for their vision-inducing properties, their ability to shift awareness and consciousness to a spiritual domain, and their ability to induce healing. While these substances have been largely excluded from human research and treatment since their broad prohibition in the 1970s, there has emerged significant evidence that these substances have important applications in the treatment of a variety of conditions, including addictions (see Winkelman & Roberts, 2007a for assessment and 2007b, vol. 2, for review articles).

It might seem unconscionable to treat addicts with drugs that are generally classified by the federal government as Schedule I substances, which is to say that they are considered to have a high abuse potential and no established therapeutic effectiveness. Nonetheless, many forms of evidence suggest that this classification is political rather than scientific (Feeney, 2007), and that these substances constitute both safe and effective treatments for many. First, the psychedelics have a much greater safety profile than the major addictive drugs, legal and illegal (see Frecska, 2007). Second, they produce little if any physical dependence (McKim, 1991). Third, they directly interact with the serotonin neurotransmitter system, with afterglow effects that appear to reflect their enhancement of neurotransmitter systems imbalanced by the use of addictive drugs. Finally, there is a variety of forms of evidence for the efficacy of the

sacred medicines such as peyote, ibogaine, and ayahuasca, as well as similar chemical substances such as LSD, in the treatment of substance abuse.

A review of the use of these sacred substances as harm-reduction approaches and drug abuse and treatment strategies, combined with general considerations of their mechanisms of efficacy, illustrate why there is a moral imperative to apply them in substance abuse rehabilitation. Halpern (2007) points out that given the limited efficacy of current treatments for drug addiction, the use of psychedelic substances for treatment of drug dependence is an ethical responsibility of the medical field. These treatment approaches are more ethical than maintenance/treatment programs that employ drugs of high abuse or substitutes (e.g., methadone treatment, or supervised application of heroin)[2] because of their abuse and overdose potential.

SACRED MEDICINES AS PSYCHOINTEGRATORS

Since the 1960s, the principal effects of the major sacred plants (as well as similar synthetic substances such as LSD) have been recognized as being derived from their effects on the serotonergic neurotransmitter system (Aghajanian, 1994; Vollenweider, 1998).[3] While activation of other neurotransmitter systems is also established, it is the effect on serotonin neurotransmission that underlie the well-known phenomenological, physical, emotional and cognitive dynamics of these substances (see Winkelman, 1996, 2001, 2007b, for general overviews). While specific knowledge of these mechanisms is primarily based on studies of LSD, generalization to the broader group of substances is substantiated by the cross-tolerance among most of these substances. While there are additional mechanisms of action, and specific differences in effects among these plant substances, they share similar effects in their neurochemical mechanisms involving serotonin. The common effects on serotonergic neurotransmission and the roles of serotonin in the brain have guided my reconceptualization of these substances as *psychointegrators*.

The Concept of the Psychointegrator

In a series of publications (Winkelman, 1996, 2001, 2007b), I have provided a synthesis of laboratory, clinical, and ethnographic findings on the sacred medicines that illustrates why the term *psychointegrator* is a more appropriate characterization of the similar effects of these diverse substances. The concept of psychointegrators replace less accurate terms (such as *hallucinogens* and *psychomimetics*) with a referent that reflects their effects on the brain and experience. The root "psyche"—referring to the soul—also reflects the concep-

tualization of the basic effects of these sacred medicines as seen from the perspective of the many cultures that have employed these substances in religious activities.

This concept of psychointegration reflects similar effects revealed by neurophysiologic, clinical, and cross-cultural studies, reflecting their action within the serotonin system, which has multiple roles reflected in its characterization as a neuromodulator that regulates the balance among many neurotransmitter systems. Psychointegrative effects are epitomized by the functions of serotonin in modulating the activities of dozens of bodily and brain processes and neurotransmitter systems and by psychointegrators' systemic effects on the serotonergic neurotransmitter system that enhances the integration of information across levels of the brain. This enhanced integrative function is epitomized by the systemic effects at the level of the raphe and reticular formation, where there is an enhancement of excitatory effects, and at the level of the limbic brain, where emotions and memories are formed.

The effects of psychointegrators are reflected in synchronized alpha and theta brain wave hyperactivity across the neuraxis, the nerve bundle linking the structural levels of the brain from the brain stem to the frontal cortex. In the process of inducing synchronous discharges across this nerve bundle, psychointegrators elicit processes central to awareness and fundamental aspects of self, emotions, and attachments. These processes of psychointegration are manifested physiologically in the typical coherent theta wave discharges that produce a synchronization of brain waves across the neuraxis and lobes of the brain. Psychointegration is also manifested in psychological experiences, particularly those related to emotional healing and the sense of interconnectedness (e.g., cosmic consciousness and other transpersonal experiences). Psychointegrators' effects on neural, sensory, emotional, and cognitive processes enhance consciousness through integrating normally unconscious emotional and self information into the frontal cortex and consciousness. This integration of the unconscious into consciousness underlies the general potentials of psychointegrators as a harm-reduction approach.

SACRED MEDICINES USED IN THE TREATMENT OF ADDICTIONS

One of the areas in which there is a substantial and growing body of literature regarding the efficacy of the psychointegrators in the treatment of illness is in the field of addictions. While we still lack the highly esteemed but sporadically applied double-blind clinical trials, there is evidence regarding the effectiveness of psychointegrators in the treatment of additions. As Alper

and Lotsof (2007) point out, we find validation of the effectiveness of these substances in the treatment of addictions from triangulation and synthesis of many forms of evidence—animal studies, laboratory data, medical case studies, personal case accounts, and case series. Treatment practices involving ritual and clinical use of the sacred plants peyote, ibogaine, and ayahuasca, as well as synthetic analogues such as LSD, are reviewed to illustrate their potential as part of harm-reduction and substance abuse rehabilitation strategies.

The Native American Church: Peyote as Addiction Treatment

One of the widely used psychointegrators in the treatment of addictions is peyote (*Lophophora williamsii*), employed as a sacrament within the Native American Church (NAC, the Peyote Religion or Peyote Way). NAC members consume peyote in an all-night meeting during which all the community may participate in the singing, prayers, chanting, and drumming. The amount of peyote ingested may be quite small, often less than the minimal dose required for notable visual psychoactive effects. The pharmacological effects are not the only mechanism of treatment. The effectiveness of the NAC as a health care delivery system is also dependent on a variety of supportive psychotherapeutic modalities: a master guide, marathon group sessions, ego reduction techniques, social networks, self-actualization, and psychotropic drugs (Calabrese, 1997, 2007; Wiedman, 1990). Many addictions professionals consider the NAC the only effective treatment of alcoholism among Native Americans, and the U.S. Indian Health Service provides reimbursement for the treatment of Native American alcoholics by so-called road men, the peyote ritual leaders (Calabrese, 2007). The Peyote Religion rejects the use of alcohol, making it a natural support group for recovering alcoholics.

Peyote contains mescaline, with stimulant properties similar to ephedrine and amphetamines; while different from indole alkaloids typical of the psychointegrators, its transformation in the body produces similar visionary effects. It is also accepted that part of the effects of mescaline are mediated through the serotonergic system (McKim, 1991). Alternate models of the physiological effects of peyote in healing addictions are characterized by Jilek (1994) as involving altered states of consciousness (ASC) that he points out can be induced through nondrug means (e.g., drums, chanting, prolonged sleeplessness; also see Winkelman, 2000). The overnight nature of the activity also contributes to the typical visionary experiences predicting one's future ruin as an addict. This awareness contributes to a sense of hitting bottom and a heightened consciousness of the need to change one's lifestyle.

The physiological effects of peyote in addressing the dynamics of addiction should not, however, be completely discounted. In addition to the general ASC-inducing effects, peyote also produces an afterglow (e.g., see Halpern, 1996, for review) that has been noted as conducive to therapeutic interventions. This after-effect lasts up to six weeks and includes not only enhanced affect but an increased openness to communication regarding one's problems, making them more accessible to therapeutic intervention.

The NAC also addresses addiction through social psychological mechanisms, forming a sense of community that promotes a new identity and a social support group that does not use nor tolerate alcohol use. The NAC brings hope to Native American communities, instilling a moral code of devotion to family and obligation to the community and producing feelings of spirituality and unity (Aberle, 1966). Peyotism provides Native Americans with religious healing, transcendence, release from guilt, and guidance and a sense of purpose (Aberle, 1966). A significant aspect of the effects of peyote is inducing a sense of control in meeting challenges and initiating change. Heggenhougen (1997) suggests therapeutic effects involve managing cultural alienation experienced by young Native Americans, providing a context for a ritual death-and-rebirth, and constructing a positive identity with one's culture. Jilek (1994) conveys the peyotists' perspective that the peyote ritual combats alcoholism through reducing physical and mental stress and enhancing mental and physical strength through contact with the supernatural.

Calabrese (1997, 2007) describes the Peyote Way as a cultural psychiatry involving meaning-manipulative therapies that engage social and intrapsychic processes. Calabrese uses the term desematicizing agent to refer to how the effects of peyote facilitate a reinterpretation of self. De Rios, Grob, and Baker (2002) similarly characterize the effects of peyote as a nonspecific facilitator that contributes to the development of a new sense of identity through grounding and connection with a community that gives a sense of belonging. Calabrese (1997) assesses the effects in terms of a "social manipulation of consciousness states and symbols to support socially-valued patterns of ritual experience, self awareness and emotional control . . . [rendering] adults more suggestible and thus more open to education and mental health interventions" (pp. 238–239). In this sense, peyote rituals heal by shaping consciousness in ways that facilitate symbolic healing processes. Calabrese (2007) also notes that unlike many traditional etiologies that attribute personal misfortune to supernatural causes (e.g., witches, taboo violations), the Peyote Way instead places the causal factors in a lack of personal responsibility for one's own behavior.

The practices of the NAC have many effects on social, psychological, and emotional well-being. Specific pharmacological actions of peyote in the treat-

ment of addictions may be best characterized in terms of the general effects of inducing ASC and the associated transcendental spiritual experiences. Aberle (1966) points to the ability of peyote to induce a sense of connection with the spiritual world that was lost by many Native American individuals and groups in their forcible assimilation to American culture. The significance of spiritual experiences for rehabilitation of the addicted self has been noted in many therapeutic traditions and may be particularly relevant for Native American groups deprived of their own spiritual traditions.

While further research on specific physiological mechanisms of peyote and its diverse compounds is definitely needed, evidence exists regarding its relative safety and effectiveness. While there are limited toxicology studies that constitute Phase I of the Food and Drug Administrations's ideal evaluation process, there is no evidence of physical harm found in long-term peyote users (see Halpern, Sherwood, Hudson, Yurgelun-Todd, & Pope, 2005). The Phase II evaluations that are used to provide evidence of proof of concept—specific effectiveness of an agent for a health condition—can be derived from case studies of Peyote Church members (also see Halpern, 1996). Evidence of Phase III—approved therapeutic use—is found in the practice of the Indian Health Services, a branch of the U.S. federal government, which approves reimbursement for road men who use of peyote for treatment of alcoholism among Native Americans (see Calabrese, 2007).

Ibogaine Use in Withdrawal Cessation

Ibogaine is an indolealkylamine extract from an alkaloid found in the root of iboga (*Tabernanthe iboga*); it is characterized as both a stimulant and hallucinogen. Dosage and other factors (additives, diet, activity) determine whether iboga induces dreamy visions, and possibly stupor and unconsciousness, or acts as a powerful stimulant (De Rios, 1984). The traditional uses of iboga in west central Africa were primarily ritual, but it was also employed for relief of fatigue, to enhance hunting skills, and for divinatory purposes. Iboga was also imbibed in large quantities to produce stupor by the Bwiti secret societies of Gabon and Congo and in all-night village or intervillage initiation ceremonies (Fernandez, 1972). The Bwiti cult combined Christian and traditional symbols in a revitalization movement that addressed community dislocation caused by colonialism. Communicating with and affirming relationships with the ancestors reintegrated individuals into traditional kinship ties and systems of social control that helped reestablish social stability and well-being (De Rios, 1984). Iboga was interpreted as provoking experiences of contact with the ancestors and a sense of the relationship of the individual to group spirit.

Ibogaine also has a history of informal treatment of addiction in addict communities, functioning largely within the context of both addict and medical subcultures in the public sector rather than in conventional medical settings (but see Alper & Lotsof, 2007; Alper, Lotsof, Frenken, Luciano, & Bastiaans, 1999; Alper, Lotsof, & Kaplan, 2008; Mash et al., 1998; Mash et al., 2001). Iboga is used as treatment for narcotic addiction, particularly the amphetamines, opiates, and cocaine, with a reputation for terminating withdrawal. Informal research on iboga began within the United States in the 1960s and, while placed on Schedule I in 1967, has remained unscheduled in many other countries where various forms of research have continued (Alper & Lotsof, 2007). Ibogaine became an important element in heroin self-help groups in the Netherlands, where it became a central feature of the harm-reduction movement there. The U.S. National Institute of Drug Abuse (NIDA) funded Phase I studies of toxicity in the early 1990s (Mash et al., 1998) but in spite of promising results failed to continue funding the study for Phase II evaluations. It has nonetheless remained available in alternative nonmedical treatment settings around the world and through more conventional medical approaches in countries where permitted by law. This persistent use is based not only on the widespread success encountered among the addict and medical subcultures involved (see Alper, Lotsof, & Kaplan, 2008) but also because of the findings of a wide range of studies that illustrate that ibogaine ought to be considered as having passed the equivalent of Phase I and II evaluations.

Although the FDA withheld funding for the planned and approved Phase I and II protocols, the continuing use of ibogaine for addiction treatment in the United States and around the world constitutes a vast uncontrolled experiment. This has provided many forms of information—including case reports, preclinical toxicological evaluations, and initial Phase I trials of safety pharmacokinetics—which constitute a substantial body of evidence constituting preclinical proof of the concept and the equivalence of Phase I and II trials (see Alper, 2001; Alper & Lotsof, 2007; Alper et al., 1999; Mash, et. al., 1998).

The typical use of ibogaine in addict communities and medical subcultures is without the ritual and ceremonial aspects associated with the indigenous traditions, but there are also other models, including spiritual ones (see Lotsof & Wachtel, 2003). Ibogaine is typically used with the patient located in a dark room and with a bed for reclining during the majority of the session. Administration (see Alper & Lotsof, 2007, for details) is with large doses that provoke visions incorporating repressed memories. While often characterized as a hallucinogen, the effects may be better conceptualized as oneiric, referring to the dreamlike experiences evoked in the internal mental space experienced with eyes closed. The visual effects are characterized as high density images

generally of autobiographical nature and central to life narrative, although archetypal and cartoonlike imagery is also reported. Notable effects reported by addicts include the loss of craving and the lack of withdrawal symptoms and an aversion to the drug, generally eliminating the addict's desire to further use drugs. The patient's experience under ibogaine typically includes visions that provide psychological insight into his drug use, particularly understandings that help overcome psychological blocks (Alper & Lotsof, 2007). Iboga's ability to induce a cosmic consciousness experience may also underlie iboga's effectiveness in treatment of drug addiction (Sanchez-Ramos & Mash, 1996). Iboga's effectiveness in facilitating psychotherapy is attributed to its ability to evoke repressed memories, clarify thoughts, promote introspection, and facilitate manipulation and reenactment of images and scenes (Sanchez-Ramos & Mash, 1996).

Human and animal research (see Alper & Lotsof, 2007, for review) suggests that ibogaine has physiological efficacy in reducing withdrawal cravings and addictions. In reviewing nonmedical clinical studies in the United States and the Netherlands, Alper and Lotsof (2007) point to the "attenuation of opioid withdrawal symptoms within several hours of ingestion, and lasting resolution of the acute opioid withdrawal syndrome within 12 to 18 hours" (p. 49). These limited studies also suggest a short-term effect in reducing cocaine addictions that lasts several weeks to months. Physiological effects (rather than placebo effects) are illustrated in animal studies that show ibogaine may reduce the reinforcing effects of opiates and decrease the experience of withdrawal (see Alper & Lotsof, 2007, for review and original sources).

Alper and Lotsof (2007) review research indicating that the mechanisms of action of ibogaine are different from other substances used to address the neurobiology of addiction, being neither opiate agonists nor opiate antagonists, nor related to the agonism and antagonism of serotonin receptors. Alper and Lotsof (2007) suggest "that ibogaine treatment results in the 'resetting' or 'normalization' of neuroadaptations thought to underlie the development of dependence" (p. 44). They point to evidence indicating that ibogaine has a selective effect on the "learning encoding of drug salience" (p. 54), a selective interference with learning related to prior drug exposures. Other mechanisms of ibogaine action include effects on the inferior olive of the brain stem and the Purkinje cells, where it induces prolonged tremors induced by the release of glutamate. Studies in animals suggest that effects on these centers controlling behavioral routines could produce cessation of addiction by destroying or exhausting these cells, eliminating previous behavioral patterns. In humans, this brain stem excitation is hypothesized to ascend into the brain circuitry underlying REM sleep (dreams) and the thalamus and *locus coeruleus*. These

natural mechanisms involved in forgetting may produce similar destruction of previous behavior patterns reinforcing addiction.

Ayahuasca in Addictions Treatment

Ayahuasca typically refers to a combination of two plants, *Banisteriopsis caapi* and *Psychotria viridis*. The *Banisteriopsis* vine (also called ayahuasca) contains several monoamine oxidase (MAO) inhibitors that render the N,N-Dimethyltryptamine (DMT) of *Psychotria* orally active (see McKenna, 2007, for sources of primary research). Scientific characterizations of the active ingredient from ayahuasca brews implicate the DMT from the *Psychotria*, but the *Banisteriopsis* is generally considered to be the source of the more important psychoactive ingredients among many cultures that use it ritually.

In tribal contexts, ayahuasca was used for a wide range of purposes (e.g., see Schultes & Winkelman, 1996, for review). Primary contemporary uses include: contacting spirits of the dead; telepathy, clairvoyance, diagnosis, prophecy, and healing; and adult transition rites and collective ceremonies. Contemporary rural Amazonian basin groups take ayahuasca in collective adult rituals to strengthen group cohesion and identity and to facilitate psychosocial and conceptual adaptation to rapid cultural change (Andritzky, 1989). Ayahuasca traditions have also diffused to urban areas, where broad segments of the population employ them for healing psychosomatic and ethnomedical conditions, particularly those associated with anxiety and stress (De Rios, 1984). Among these urban traditions are the Brazilian ayahuasca churches (see Labate & Araujo 2002) that have incorporated ayahuasca as a sacrament in syncretistic Christian religions.

The Takiwasi Program

Mabit, Giove, and Vega (1996; also see Mabit, 2007) review the Takiwasi program that incorporates ayahuasca in ritual treatments for rehabilitation of cocaine addicts in the Peruvian Amazon. The program integrates traditional ayahuasca rituals and physical, psychological, and spiritual activities into treatments that address a range of factors contributory to addiction. The program claims widespread evidence of clinical success, although formal clinical studies are lacking. The success is attributed not to the ayahuasca alone, but to the ritual setting and interactions with therapists. The traditional medicines and rituals are combined within the Takiwasi program with modern transpersonal psychology and social techniques to guide the personal transformation of addicts, using the ayahuasca ritual to produce profound ASC that change their outlook on life and their spiritual strength and faith.

The Takiwasi program takes the ritual approach to treatment seriously, incorporating traditional medicine approaches at organic, psychological, and spiritual levels. These three levels of treatment are synchronized with the induction of ASC to enhance access to unconscious levels of the individual. This shamanic approach takes the addict's desire to engage in an alteration of consciousness and initiates him into a kind of vision quest to accesses deep levels of meaning and a sacred dimension of experience that produces personal reorganization.

The Takiwasi treatment program begins with a focus on the organic level of the body, providing a physical detoxification to eliminate toxins acquired through drug use. The consequences of withdrawal and dependency are also addressed. Treatments at the psychological level address the emotional dynamics that contributed to drug addiction. Ayahuasca induces ASC that bypass rational functions to provide access to deep levels of the personal unconsciousness, particularly repressed memories. The patient is empowered through the ayahuasca to become his own healer, discovering the cause of his problems in his visions and taking personal responsibility for the healing processes that he must engage in. The verbalization of these realizations with their therapists allows patients to reflect on their personal conditions and receive feedback regarding their self-perceptions, attitudes, and emotions. These ayahuasca-induced experiences also provide access to spiritual and transpersonal levels that take the patient beyond himself, instilling a value orientation that provides new meaning to life. This spiritual restructuring is an essential aspect of the healing process. It requires a deflation of the ego that produces reconciliation with self, others, nature, and the universe at large.

The first phase of the Takiwasi program begins with a 10-day isolation, followed with up to two months of detoxification to deal with the long-term consequences of cocaine paste addiction and withdrawal symptoms. This includes the use of purgatives, rituals, chants, massages, and tobacco smoke to address tension and anxiety and balance the patient's energy level. Meditation, music therapy, saunas, and relaxing teas are the focus after the isolation period. Following a two-month isolation from family, visits from family members are allowed. This third month also begins a period of retreats into the isolation of the jungle to take additional plants specific for the personality of the individual and his therapeutic needs. A folk healer administers the plants and a carefully controlled diet and observes the patient for energetic disturbances. This experience, similar to an initiation, helps reinforce the will to heal and overcome addiction. The treatment program continues up to seven or more months, using ayahuasca and other ASC-inducing techniques (e.g., meditation, holotropic breath work) to reveal unconscious material for therapeutic

work. Dream material is also the focus of therapeutic processes, integrating the unconscious dynamics into a fuller understanding of self. This enhanced self-knowledge contributes to an increasing sense of serenity and a focus on transcendent concerns that reflect a spiritual deepening in patients lives.

The efficacy of ayahuasca ritual treatments are considered the consequence of many factors—the physiological properties of the ayahuasca, their interaction with the patients' psychological conditions, the environment, and the social relations with therapists and other participants in the treatment center. The therapists consider a central aspect of the treatment effects to be produced through the tobacco smoke blown on the patient and the songs sung throughout the sessions. The physiological action of ayahuasca has been characterized as a disinhibitor that promotes the manifestation of the most basic perceptual capacities of the organism. This is manifested in the visions and other experiences loosely characterized as hallucinations but reflecting symbolically important information for the person. The patient's psychological condition, particularly attitudes of openness, trust, and surrender accompanied by commitment to the process, evoke the healing power of ayahuasca. Inappropriate diet, drug use, and sex before or after sessions are considered to undermine the therapeutic process or even cause deleterious effects.

Effects of Uniao do Vegetal on Drug Use

Studies (e.g., Callaway, Airaksinen, McKenna, Brito, & Grob, 1994; Callaway, McKenna, et al., 1999; McKenna, Callaway, & Grob, 1998) of the adherents to the ayahuasca church Uniao do Vegetal (UDV) revealed that people with a history of alcoholism underwent profound life changes leading to sobriety shortly after joining the church. The effects were seen as having derived from the changes in their worldview and having developed from a generous attitude towards others. There was also an effect on social relations, restoring stability to personal and familial relationships. Case study approaches (e.g., Guimaraes dos Santos, Carvalho de Moraes, & Holland, 2006) have also implicated ayahuasca in recovery mechanisms. Case-control studies showed that long-term regular religious ayahuasca users were healthier than ayahuasca nonusers in terms of drug abuse and psychiatric symptomatology (Da Silveira et al., 2005; Doering-Silveira, Grob, et al., 2005; Grob et al., 1996) and were similar on neuropsychological assessments, suggesting no negative effects from ayahuasca use. There is evidence that participation in the UDV ayahuasca churches reduces the risk of adolescent alcohol use, as well as anxiety, depression, and psychiatric symptomatology (Doering-Silveira, Grob, et al., 2005; Doering-Silveira, Lopez, et al., 2005).

Although ayahuasca treatment programs for drug addicts have shown considerable promise (see Guimaraes dos Santos et al., 2006; Mabit et al., 1996; Mabit, 2007), formal assessments are still lacking. There is however, justification for further applications. The equivalence of basic Phase I studies have been carried out (for reviews see Grob et al., 1996; McKenna, 2007), and there is a limited amount of Phase II evidence (see Doering-Silveira, Grob, et al., 2005; Doering-Silveira, Lopez, et al., 2005; Mabit, 2007; McKenna, 2007).

Lysergic Acid Diethylamide (LSD) in the Treatment of Alcoholism

While LSD is considered to be a synthetic chemical, it has its natural analogues in the ergot alkaloids found in a number of plants (Rätsch, 2005). The use of LSD in the treatment of alcoholism is the most widely investigated clinical application of the psychointegrators to addictions treatment (see Abuzzahab & Anderson, 1971; Halpern, 1996, 2007; Mangini, 1998, for reviews). The early (e.g., 1950s and 1960s) applications of LSD to the treatment of alcoholism suggested the potential to curtail use and reduce recidivism (Halpern, 1996). These applications began with the hypothesis that the delirium tremors that often led alcoholics to change their behavior might be induced by LSD. This hypothesis was rejected but lead to other insights about LSD's potential efficacy as a treatment for addictions. LSD sessions could produce a vivid awareness of one's personal problems, presenting graphic images of the immediate and long-term deleterious effects of the alcohol. The recovering alcoholics often credited these realizations as providing the motivation to change their behavior. Furthermore, some studies indicated that those who received the greatest effects in achieving sobriety were those who experienced a profound spiritual or transcendent experience from LSD. Such spiritual motivation for their recovery was often attested to by the former alcoholics, echoing the main theme of Alcoholics Anonymous: alcoholism is a disease characterized by a deep spiritual craving that can be overcome only by a spiritual awakening.

The limited methodological designs (i.e., lacking double-blinds and placebo controls) and limited follow-up, however, left the findings in a poorly defensible position. This has led many people to dismiss them. Halpern (1996, 2007), however, has shown that there are some valuable findings and applications to be culled from this early research.

Abuzzahab and Anderson (1971) performed a meta-analysis of 31 early (1953–1969) studies using LSD with alcoholics, illustrating the potential of LSD in addictions treatment. Single-dose studies showed an improvement of greater than 50 percent in treatment over control groups. However, these and

other reviews indicate that the improvement differences decline across time. Studies employing LSD with narcotics addicts are far fewer than those assessing effects with alcoholics; Halpern nonetheless finds that the studies provide encouraging results. These studies indicate that a more effective treatment of alcoholism derives from a psychedelic model (using large doses to produce spiritual experiences), rather than a psycholytic model (using small repeated doses to facilitate insight) (see Passie, 2007). Large psychedelic doses that produce life-transforming cosmic experiences appear to be a critical factor in changing one's self-concept and lifestyle in ways that contribute to a resolution of alcoholism.

The limited research done with LSD in the treatment of addictions prior to general prohibition of such studies lead Halpern to conclude that doses of LSD (as well as indolealkylamines such as DMT, ibogaine, and psilocybin, and phenethylamines such as mescaline and MDMA [3,4-methylenedioxymethamphetamine, commonly known as Ecstasy]) have finite effects in diminishing the use of addictive substances. There is the essence of Phase I assessment, however (see Farber, Hanslick, Kirby, McWilliams, & Olney, 1998) and limited Phase II treatment evidence (also see Grof, 1979, 1992; Halpern, 2007). Halpern's assessment of the literature suggests that the antiaddictive effects of a single dose may last only one to two months. They nonetheless appear to provide a time-limited afterglow where the addict experiences a reduction in craving for drugs and an increased openness to communication about personal problems that can contribute to their therapeutic resolution.

Yensen and Dryer (2007) place the therapeutic effects of LSD in the context of the relationship between addiction and peak experience and the reactions of hopelessness and despair to emotional (and physical) pain that leads to addiction as a substitute solution. Peak experiences provide a fundamental shift in consciousness that lead one from anger, despair, and false gratification to a shift in consciousness that provides a profound motivation for positive change. They attribute the therapeutic outcomes less to the drug effect alone than to its ability to contribute to the complex reliving of memories in a safe and supportive context in which a healing relationship facilitates psychotherapeutic transformation.

MECHANISMS OF EFFICACY AND HARM REDUCTION FROM PSYCHOINTEGRATORS

The effects of psychointegrators in reducing addictive behaviors and associated harm are best understood in terms of biopsychosocial interactions, where their physiological effects are molded by ritual, set, and setting. The various

psychointegrators nonetheless also have physiological effects that can directly reduce addictive tendencies, independent of ritual, set, and setting, as illustrated in animal studies (for example see Alper and Lotosf's [2007] review of ibogaine research). Nonetheless, prominent effects of the psychointegrators are derived from their ability to function as nonspecific facilitators of the dynamics derived from set and setting—the social factors, the ritual, social setting, and personal expectations regarding treatment.

Physiological Effects

The psychointegrators produce a wide range of physiological effects as a function of dosage and other factors (see Winkelman, 1996, 2001, 2007a, for reviews). Halpern (1996) suggests that the general physiological aspects of therapeutic efficacy may involve an afterglow effect involving serotonergic mechanisms. The noted reduction of craving provides the addict with additional resources for gaining a sense of self-control that contributes to the development of an abstinent lifestyle.

Serotonin Enhancement

A principal physiological aspect of the efficacy of psychointegrators in the treatment of addiction likely comes from their effects on the serotonergic neurotransmitter systems. The research on LSD provides ample evidence with regard to these effects on serotonin. Serotonin is depleted by long-term drug use, and low serotonin levels predispose depression, a contributory factor in drug use (Walton & Levitsky, 1994, p. 110). The effects of the LSD-like psychointegrators on serotonin are presumably responsible for the widely noted mood-enhancing effects, as well as the reported reduction of anxiety, depression, aggression, and compulsions. Serotonin's role as a neuromodulator, with direct effects on other neurotransmitter systems, enables serotonin-enhancement effects to cascade into other neurotransmitter systems affected by addictions. In the case of ibogaine, both case studies with humans and laboratory research with animals illustrate the reduction of craving that follows from such treatments, although these are not likely serotonergic mechanisms (see Alper & Lotsof, 2007, for review).

Psychointegration and Systemic Brain Function

Psychointegration (Winkelman, 1996, 2001) characterizes the physiological effects of these substances, reflected in highly synchronized and coherent brain wave patterns that propagate from the brain stem through the emotional

(limbic) brain and into the frontal cortex (Mandell, 1980). These brain wave patterns integrate information from physiological, behavioral, and emotional levels of the brain into the frontal cortex. These synchronized theta waves also produce a relaxation response and physiological integration of behavioral, emotional, and cognitive processes (see Winkelman, 2000, for review).

Altered States of Consciousness and Unity Experiences

A wide variety of means of inducing ASC, including drug and nondrug procedures, have basic similarities (Mandell, 1980; Winkelman, 1992, 2000). This high voltage slow wave electroencephalograph activity (alpha, theta, and delta, especially 3–6 cycles per second) provokes slow wave hypersynchronous discharges across the hippocampal-septal-reticular-raphe circuit, which spread to the frontal lobes, producing pleasurable sensations and feelings of well-being (Mandell, 1980). General physiological aspects of ASC—parasympathetic dominance, interhemispheric synchronization, and limbic-frontal integration—are collectively an inherent aspect of human consciousness, the integrative mode of consciousness (Winkelman, 2000). These manifestations of consciousness have inherent therapeutic effects related to addiction; these involve stress reduction and activation of aspects of the brain related to mental and emotional integration. They also produce spiritual experiences, particularly with large doses, that produce life-changes that help in ending addictive behaviors. This paradigm of LSD use known as the psychedelic paradigm suggests that the changes in worldview and perceptions produced by these dramatic experiences are key to the treatment effects. While psycholytic therapy with small doses may have a role in addictions treatment, the general consensus is that the best results come from the large psychedelic doses that produce profound effects (see Passie, 2007). These psychointegrator-induced changes in worldview include a sense of connection with nature and others, providing a sense of wholeness and connection often lacking in the addict.

Access to Repressed Memories

A basic technique of psychotherapy involves enhanced access to repressed memories. Increased access to memories and repressed trauma elicited by psychointegrators reflects their physiological actions on the limbic brain and associated structures that mediate memory processes and emotions. The well-recognized effects of psychointegrators in promoting the emergence of repressed material into consciousness contributes to therapeutic effects through enhancing awareness of the nature of the consequence of one's addictive behavior. Integration of these deeper levels of human identity, reflecting the

reptilian and paleomammalian brains' automated and emotional processes, is important for resolving addictions. Psychointegrators stimulate the brain stem and limbic structures of the brain and their behavioral, emotional, and social processes, producing ascending nerve impulses. These and other physiological effects enhance information availability, producing limbic-frontal integration and interhemispheric fusion, which contribute to the insight necessary to change behavior. Interhemispheric fusion is the enhanced communication between the two hemispheres of the frontal brain through the corpus collosum. Psychointegrators enhance the rate of transfer of information, increasing the integration of cognition from the nondominant, nonverbal hemisphere and the dominant, language-based information processes, producing insight and cognitive-emotional integration (Mandell, 1985).

Psychophysiological Effects

Effects of psychointegrators also reflect psychophysiological enhancement of ritual and contextual effects on the experiences. De Rios et al. (2002, p. 239) suggest that these substances are efficacious because the changes that they are capable of producing in overall psychosocial functioning enables them to exert effects as nonspecific facilitators of set and setting (also see Bravo & Grob, 1989).

Internal Focus of Attention

Large dosages of psychointegrators produce a withdrawal from the external world and a focus on the internal environment. Contextual factors may reinforce the internal focus of attention on memories, personal relationships, behavioral conflicts, traumas, and other subconscious and unconscious material. This introspection facilitates awareness of the personal psychodynamics underlying addiction.

Suggestibility

A recognized effect of psychointegrators is cathartic expression and increased suggestibility. Ritual healing processes capitalize on this suggestibility to facilitate the induction of cultural scripts or dramas for a resocialization and social incorporation of the addict. The extreme suggestibility helps the individual overcome his current reality, escaping his sense of a traumatized self, and instead experiencing a sense of emotional relief and connectedness, unity, and community with others.

Emotional Enhancement

A recognized effect of psychointegrators is the enhancement of emotional experiences, particularly manifestations of emotional conflicts and issues central to personal identity and relationships with others. Other effects include the enhanced sense of emotional harmony, joy, euphoria, and sense of connection and integration with others.

Behavioral Dishabituation

Psychointegrators increase arousal and information flow but interfere with habitual behavioral patterns. This often leads to reversal of habituation and typical response patterns. This reversal of habituation often leads to the development of new behavioral patterns.

Ego Suspension

Another psychophysiological effect involves the suspension of ego structures, which allows the addict to view his own behavior and criticisms and feedback in a less defensive way. This allows new perspectives on the psychodynamic material released by the psychointegrators. There is also a tendency to maintain an increased openness to others and a willingness to communicate with others.

Psychological Integration

The psychointegrators provide a physiologically based set of changes that enhance the ability of ritual to produce an integrated sense of self, personally, socially, and cosmologically (an enhanced sense of connection with one's worldview about the cosmos). A notable aspect of this psychological integration is the enhancement of the sense of one's soul and personal spiritual contact. Many consider these spiritual dynamics to be at the foundations of addiction rehabilitation.

Psychosocial Effects

The engagement with psychointegrators may be a very solitary process typical of the shamanic vision quest, but these substances also produce a variety of social effects that are notable in their ability to counteract some of the central dynamics of addictions.

Redemption as Social Reintegration

De Rios et al. (2002) characterize the effects as producing an alleviation of addiction by providing redemption. Redemption, whether secular or religious, involves restoring a person's relationship with god and community by freeing him from an undesirable state (e.g., sin, addiction). The community and spiritual connection provided by redemption are pillars of the major alcoholism treatment approaches, embodied in Alcoholics Anonymous.

Prosocial Effects

Some of the most important effects of the psychointegrators include the setting. Significant applications to the treatment of addictions may be achieved by enhancing the context effects, in particular the dynamics of intensive, small-group interactions. These settings generally develop high degrees of relaxed intimacy; the widely recognized prosocial effects of MDMA has led to the coining of the term "empathogen." Psychointegrators can be seen as having important applications in treatment of addiction in terms of reducing, preventing, and interpersonally managing aggressive behavior.

CONCLUSIONS: POLICY DIRECTIONS IN THERAPEUTIC DEVELOPMENT

Federal prohibitions on psychointegrators and similar medicines are considered by many to lack constitutional grounds, since there is no article in the constitution or amendments to the constitution that allows the federal government to regulate what we put into our bodies (Boire, 2007). Remember, it took the 18th Amendment to the U.S. Constitution to restrict the use of alcohol by the federal government. After Timothy Leary won his U.S. Supreme Court case regarding the illegality of federal laws concerning marijuana, the Nixon administration made an end-run on the constitution and regulated such substances through the channels of administrative law. This processes of placing them in Schedule I—substances considered to be without evidence of medical efficacy and with a high potential risk for abuse—has, however, been achieved through political actions rather than on the basis of scientific evaluations (Boire, 2007).

So how can we proceed? Winkelman and Roberts (2007a) have provided an overview of the multiple levels of society at which we need to act to change the current political climate that regulates these substances. Political pressure on federal regulatory agencies remains a central approach for opening up experimental use of these substances. This pressure involves many forms of

action, including general education, education of the media, activities in public health and policy organizations, private funding of research, and perhaps even corporate developments. What remains key is applying the cumulative scientific, clinical, ethnographic, and cross-cultural evidence regarding the immense potentials of these substances to public education to facilitate professional, media, and popular pressure to effect administrative changes in federal regulation.

With *Psychedelic Medicine* (Winkelman & Roberts, 2007b), we have laid a groundwork for this public health and harm-reduction endeavor. Education, public policy development, and collective political action, rather than just more science, is necessary for changing opportunities for the use of psychointegrators in treatment of some of the most ravaging social diseases of our times such as the addictions to alcohol, tobacco, methamphetamines, and opiates and their synthetic derivatives. The acknowledged success rates of the conventional addictions treatment industry are not much different from the spontaneous remission rate. In contrast, the case study and other evidence effectiveness of the psychointegrators—particularly peyote, ibogaine, ayahuasca, and LSD—is substantial for those who are willing to consider the evidence. In general, we must say that the Phase II evidence is limited, as it lacks double-blind controls; nonetheless, the principles of Phase II assessment are strongly suggested by other forms of validation such as animal research, medical case studies, personal accounts, and case series.

Physicians have a moral imperative to seek the applications of these more effective venues of treatment for these devastating psychosocial diseases. Because of the great cost of a Phase III studies, and the imitations on patenting natural substances, we won't see these developments without changes in the government funding or industry approaches to make these substantial investments. Even where there are approved drugs that also have a demonstrated effectiveness in addictions treatment, such as ketamine, the drug industry is disinterested in further clinical trials. Ketamine is already classified as a Schedule III drug, with approved uses. Phase I, Phase II, and Phase III clinical trials in the ketamine psychedelic psychotherapeutic treatment of alcoholism and opioid dependence have been carried out, and currently there is research that constitutes the beginning of Phase IV (see Krupitsky et al., 2002; Krupitsky & Kolp, 2007). But the drug company that owns ketamine is not interested in the further development of a treatment that requires only one or two lifetime doses, as such a treatment has negligible potential revenues.

Psychotherapists have a limited range of therapeutic options for the use of psychointegrators. Boire (2007) provides guidelines for adapting to the legal constraints on the psychotherapeutic use of these substance created by the

current administrative and professional regulations. Boire outlines a medical necessity defense, a justification for using a substance as an alternative when lawful medical treatments have been found to be ineffective. Individual users may also be able to make such claims directly. Boire contends that one can base a valid defense on the assertion that although the treatment is prohibited by Schedule I classification, its use reduces the patient's severe suffering without causing disproportionate harm to others (patient, other people, or to the state's interest). Such a valid defense surely could be mounted for cluster headaches (see Sewell & Halpern, 2007). The general failure of the addictions treatment industry might justify similar conclusions about the applications of psychointegrators to the treatment of addictions.

Fenney (2007) expands our understanding of the possible permissible uses of these substances. The constitutional and legislative provisions relating to protection of religious freedoms and the federal government's special protections of the religious rights of the Native American nations has resulted in a variety of legal precedents affecting the rights to use peyote and other psychoactive sacraments. The federal rulings regarding Native Americans' rights to use peyote have established rights for members of federally recognized tribes. Feeney contends that our legal system cannot logically—or perhaps even legally—deny the same rights to other ethnic groups in society. The inconsistencies in rulings lead Feeney to conclude that the use of peyote, or by extension any psychoactive sacrament, cannot be restricted to specific groups, Native American or otherwise, as long as they are sincere believers. In addition, other venues for increasing opportunities for the use of these sacred medicines may be found in the development of religious-based uses such as the Uniao do Vegetal and Santo Daime churches, which use ayahuasca as a sacrament (Groisman & De Rios, 2007; Labate & Araujo, 2002).

NOTES

1. This chapter includes sections of material based on information previously published in Winkelman (1996, 2001, 2007a&b) and Winkelman and Roberts (2007a).
2. For instance, regarding such risks of the standard treatments, see Lintzeris et al. (2006) regarding the practice of intrafemoral heroin administration.
3. Other principal sources consulted for characterization of serotonin and psychointegrator neurophysiology and mechanisms include Aghajanian (1994), Glennon (1990), Kruk and Pycock (1991), Mandell (1980, 1985), McKim (1991), Miller and Gold (1993), Ribeiro (1991), and Vollenweider (1998). It is generally accepted that the primary effects of the psychedelic medicines is through their action on the 5-HT1A and 5-HT2A serotonergic neurons; there is also binding with the

5-HT5 and 5-HT7 receptors (Vollenweider, 1998). Serotonin 5-HT1A receptors in the raphe system, which mediate responses of the serotonergic neurons with respect to their own transmitters, show a strong sensitivity to LSD-like substances (Aghajanian 1994, p. 140), inhibiting their firing in the raphe area and depressing neuronal firing in lower areas of the brain. LSD affinity for 5-HT2A and 5-HT2c receptors facilitates the functioning of the locus coeruleus, which receives numerous somatosensory and visceral inputs and projects diffusely to most of the brain (Miller & Gold, 1993). LSD effects the hippocampus by blocking or suppressing the typical depressant functions of serotonin, permitting the release of responses similar to dreaming and contributing to production of the typical visual experiences by disinhibiting postsynaptic neurons in the limbic and visual areas. Indoleamine and phenethylamines cause greater activation of 5-HT2 serotonin receptors relative to other serotonin receptors (Aghajanian, 1994). Primary effects of LSD-like psychointegrators involve their action on the 5-HT2 serotonergic neurons. Large concentrations of serotonin 5-HT2 receptors are in the limbic system in the hypothalamus and basal ganglia; these sensory processing functions are antagonized by LSD (Kruk & Pycock, 1991). LSD-like psychointegrators also affect the cerebral cortex and the locus coeruleus 5-HT2 receptors (Aghajanian, 1994).

REFERENCES

Aberle, D. (1966). *The peyote religion among the Navaho*. Chicago: Aldine Publications.

Abuzzahab, F., & Anderson, B. (1971). A review of LSD treatment in alcoholism. *International Pharmacopsychiatry 6*, 223–35.

Aghajanian, G. (1994). Serotonin and the action of LSD in the brain. *Psychiatric Annals, 2463*, 137–141.

Alper, K. R. (2001). Ibogaine: A review. The alkaloids. *Chemistry and Biology, 56*, 1–38.

Alper, K. R., & Lotsof, H. S. (2007). The use of Ibogaine in the treatment of addictions. In M. Winkelman & T. Robert (Eds.), *Psychedelic medicine* (vol. 2, p. 43–66). Westport, CT: Praeger Perspectives.

Alper, K. R., Lotsof, H. S., Frenken, G. M., Luciano, D. J., & Bastiaans, J. (1999). Treatment of acute opioid withdrawal with ibogaine. *American Journal of Addiction, 8*(3), 234–242.

Alper, K. R., Lotsof, H. S., & Kaplan, C. D. (2008). The ibogaine medical subculture. *Journal of Ethnopharmacology 115*, 9–24.

Andritzky, W. (1989). Sociopsychotherapeutic functions of ayahuasca healing in Amazonia. *Journal of Psychoactive Drugs, 21*(1), 77–89.

Boire, R. (2007). Psychedelic medicine and the law. In M. Winkelman & T. Robert (Eds.), *Psychedelic medicine* (vol. 1, pp. 217–232). Westport, CT: Praeger Perspectives.

Bravo, G., & Grob, C. (1989). Shamans, sacraments and psychiatrists. *Journal of Psychoactive Drugs, 21*(1), 123–128.

Calabrese, J. (1997). Spiritual healing and human development in the Native American Church: Toward a cultural psychiatry of peyote. *Psychoanalytic Review, 84*(2), 237–255.

Calabrese, J. (2007). The therapeutic use of peyote in the Native American Church. In M. Winkelman & T. Robert (Eds.), *Psychedelic medicine* (vol. 2, pp. 29–42). Westport, CT: Praeger Perspectives.

Callaway, J. C., McKenna, D. J., Grob, C. S., Brito, G. S., Raymon, L. P., Poland, R. E., et al. (1999). Pharmacokinetics of hoasca alkaloids in healthy humans. *Journal of Ethnopharmacology, 65,* 243–256.

Callaway, J. C., Airaksinen, M. M., McKenna, D. J., Brito, G. S., & Grob, C. S. (1994). Platelet serotonin uptake sites increased in drinkers of ayahuasca. *Psychopharmacology, 116,* 385–387.

Da Silveira, D. X., Grob, C. S., De Rios, M., Lopez, E., Alonso, L., & Talca, C. (2005). Ayahuasca in adolescence: A preliminary psychiatric assessment. *Journal of Psychoactive Drugs, 37*(2), 129–133.

De Rios, M. (1984). *Hallucinogens: Cross-cultural perspectives.* Albuquerque: University New Mexico Press.

De Rios, M., Grob, C., & Baker, J. (2002). Hallucinogens and redemption. *Journal of Psychoactive Drugs, 34*(3), 239–248.

Doering-Silveira, E., Grob, C., De Rios, M., Lopez, E., Alonso, L., Talca, C., et al. (2005). Report on psychoactive drug among adolescents using ayahuasca within a religious context. *Journal of Psychoactive Drugs, 37*(2), 141–144.

Doering-Silveira, E., Lopez, E., Grob, C., De Rios, M., Alonso, L., Talca, C., et al. (2005). Ayahuasca in adolescence: A neuropsychological assessment. *Journal of Psychoactive Drugs, 37*(2), 123–128.

Farber, N. B., Hanslick, J., Kirby, C., McWilliams, L., & Olney, J. W. (1998). Serotonergic agents that activate 5HT2A receptors prevent NMDA antagonist neurotoxicity. *Neuropsychopharmacology, 18,* 57–62.

Feeney, K. (2007). The legal bases for religious peyote use. In M. Winkelman & T. Robert (Eds.), *Psychedelic medicine* (vol. 1, pp. 233–250). Westport, CT: Praeger Perspectives.

Fernandez, J. W. (1972). *Bwiti: An ethnography of religious imagination in Africa.* Princeton, NJ: Princeton University Press.

Frecska, E. (2007). Therapeutic guidelines: Dangers and contra-indications in therapeutic applications of hallucinogens. In M. Winkelman & T. Robert (Eds.), *Psychedelic medicine* (vol. 1, pp. 69–95). Westport, CT: Praeger Perspectives.

Glennon, R. (1990). Serotonin receptors: Clinical implications. *Neuroscience and Biobehavioral Reviews, 14,* 35–47.

Grob, C. S., McKenna, D. J., Callaway, J. C., Brito, G. S., Neves, E. S., Oberlaender, G., et al. (1996). Human psychopharmacology of hoasca, a plant hallucinogen used in ritual context in Brazil. *The Journal of Nervous and Mental Disease, 184,* 86–94.

Grof, S. (1979). *LSD psychotherapy.* Pomona, CA: Hunter House.

Grof, S. (1992). *The holotropic mind*. San Francisco, CA: Harper Collins.

Groisman, A., & De Rios, M. (2007). Ayahuasca, the U.S. Supreme Court, and the UDV-U.S. government case. In M. Winkelman & T. Robert (Eds.), *Psychedelic medicine* (vol. 1, pp. 271–298). Westport, CT: Praeger Perspectives.

Guimaraes dos Santos, R., Carvalho de Moraes, C., & Holland, A. (2006). Ayahuasca e reducao do uso abusivo de psicoactivos: Eficacia terapeutica [Ayahuasca and reduction of abusive use of psychoactives: Therapeutic efficacy]. *Psicologia: Teoria e Pesquisa, 22*(3), 363–370.

Halpern, J. (1996). The use of hallucinogens in the treatment of addiction. *Addiction Research, 4*(2), 177–189.

Halpern, J. (2007). Hallucinogens in the treatment of alcoholism and other addictions. In M. Winkelman & T. Robert (Eds.), *Psychedelic medicines* (vol. 2, pp. 1–14). Westport, CT: Praeger Perspectives.

Halpern, J. H., Sherwood, A. R., Hudson, J. I., Yurgelun-Todd, D., & Pope, H. G., Jr. (2005). Psychological and cognitive effects of long-term peyote use among Native Americans. *Biological Psychiatry, 58*(8), 624–631.

Heggenhougen, C. (1997). Reaching new highs: Alternative therapies for drug addicts. Northvale, NJ: Jason Aronson.

Jilek, W. G. (1994). Traditional healing in the prevention and treatment of alcohol and drug abuse. *Transcultural Psychiatric Research Review, 31*, 219–258.

Kruk, Z., & Pycock, C. (1991). *Neurotransmitters and drugs*. London: Chapman and Hall.

Krupitsky, E., Burakov, A., Romanova, T., Dunaevsky, I., Strassman, R., & Grinenko, A. (2002). Ketamine psychotherapy for heroin addiction: immediate effects and two-year follow-up. *Journal of Substance Abuse Treatment, 23*(4), 273–283.

Krupitsky, E., & Kolp, E. (2007). Ketamine psychedelic psychotherapy. In M. Winkelman & T. Robert (Eds.), *Psychedelic medicines* (vol. 2, pp. 67–86). Westport, CT: Praeger Perspectives.

Labate, B., & Araujo, W. (Eds.). (2002). *O uso ritual do ayahuasca [The ritual use of ayahuasca]*. Campinas, Brazil: Mecado de Letras Edicoes e Liveria.

Lotsof, H., & Wachtel, B. (2003). Manual for ibogaine therapy screening, safety, monitoring and aftercare (2nd ed.). Retrieved May 31, 2006, from http://www.ibogaine.org/Ibogaine.pdf.

Mabit, J. (2007). Ayahuasca in the treatment of addictions. In M. Winkelman & T. Roberts (Eds.), *Psychedelic medicine* (vol. 2, pp. 87–105). Westport, CT: Praeger Perspectives.

Mabit, J., Giove, R., & Vega, J. (1996). Takiwasi: The use of Amazonia shamanism to rehabilitate drug addicts. In M. Winkelman & W. Andritzky (Eds.), *Sacred plants, consciousness and healing: Cross-cultural and interdisciplinary perspectives. Yearbook of cross-cultural medicine and psychotherapy* (pp. 257–286). Berlin: Verlag und Vertrieb.

Mandell, A. (1980). Toward a psychobiology of transcendence: God in the brain. In D. Davidson & R. Davidson (Eds.), *The psychobiology of consciousness* (pp. 379–464). New York: Plenum.

Mandell, A. (1985). Interhemispheric fusion. *Journal of Psychoactive Drugs, 17*(4), 257–266.

Mangini, M. (1998). Treatment of alcoholism using psychedelic drugs: A review of the program of research. *Journal of Psychoactive Drugs, 30*(4), 381–418.

Mash, D. C., Kovera, C. A., Buch, B. E., Norenberg, M. D., Shapshak, P. , Hearn, W. L., et al. (1998). Medication development of ibogaine as a pharmacotherapy for drug dependence. *Annals of the New York Academy of Sciences, 844*, 274–292.

Mash, D. C., Kovera, C. A., Pablo, J., Tyndale, R., Ervin, F. R., & Kamlet, J. D. (2001). Ibogaine in the treatment of heroin withdrawal. *Alkaloids Chemical Biology, 56*, 155–171.

McKenna, D. J. (2007). The healing vine: Ayahuasca as medicine in the 21st century. In M. Winkelman & T. Roberts (Eds.), *Psychedelic medicine* (vol. 1, pp. 21–44). Westport, CT: Praeger Perspectives.

McKenna, D. J., Callaway, J. C., & Grob, C. S. (1998). The scientific investigation of ayahuasca: A review of past and current research. *The Heffter Review of Psychedelic Research, 1*, 65–76.

McKim, W. (1991). Drugs and behavior: An introduction to behavioral pharmacology. Englewood Cliffs, NJ: Prentice-Hall.

Miller, N., & Gold, M. (1993). LSD and ecstasy: Pharmacology, phenomenology and treatment. *Psychiatric Annals, 24*(3), 131–134.

Passie, T. (2007). Contemporary psychedelic therapy: An overview. In M. Winkelman & T. Roberts (Eds.), *Psychedelic medicine* (vol. 1, pp. 45–68). Westport, CT: Praeger Perspectives.

Rätsch, C. (2005). The encyclopedia of psychoactive plants: Ethnopharmacology and its applications. (J. Baker, Trans.). Rochester, VT: Park Street Press.

Ribeiro, C. 1991. Pharmacology of serotonin neuronal systems. *Human Psychopharmacology*, 6, 37–51.

Sanchez-Ramos, J., & Mash, D. (1996). Takiwasi: The use of Amazonian shamanism to rehabilitate drug addicts. In M. Winkelman & W. Andritzky (Eds.), *Sacred plants, consciousness and healing: Cross-cultural and interdisciplinary perspectives. Yearbook of cross-cultural medicine and psychotherapy* (pp. 353–68). Berlin: Verlag und Vertrieb.

Schultes, R., & Hofmann, A. (1992). *Plants of the gods*. Rochester, VT: Healing Arts Press.

Schultes, R., & Winkelman, M. (1996). The principal American hallucinogenic plants and their bioactive and therapeutic properties. In M. Winkelman & W. Andritzky, (Eds.), *Sacred plants, consciousness and healing: Cross-cultural and interdisciplinary perspectives. Yearbook of cross-cultural medicine and psychotherapy* (pp. 205–240). Berlin: Verlag und Vertrieb.

Sewell, A., & Halpern, J. H. (2007). Response of cluster headaches to psilocybin and LSD. In M. Winkelman & T. Roberts (Eds.), *Psychedelic medicine* (vol. 1, pp. 97–124). Westport, CT: Praeger Perspectives.

Vollenweider, F. (1998). Recent advances and concepts in the search for biological correlates of hallucinogen-induced altered states of consciousness. *The Heffter Review of Psychedelic Research, 1,* 21–32.

Walton, K., & Levitsky, D. (1994). A neuroendocrine mechanism for the reduction of drug use and addictions by transcendental meditation. In D. O'Connell & C. Alexander (Eds.), *Self-recovery: Treating addictions using transcendental meditation and Maharishi Ayur-Veda,* (pp. 13–88). New York: Hayworth Press.

Wiedman, D. (1990). Big and little moon peyotism as health care delivery systems. *Medical Anthropology, 12,* 371–87.

Winkelman, M. (1992). Shamans, priests and witches. A cross-cultural study of magico-religious practitioners. Anthropological Research Papers 44. Tempe, AZ: Arizona State University.

Winkelman, M. (1996). Psychointegrator plants: Their roles in human culture and health. In M. Winkelman & W. Andritzky (Eds.), Sacred plants, consciousness and healing: Cross-cultural and interdisciplinary perspectives. Yearbook of cross-cultural medicine and psychotherapy (pp. 9–53). Berlin: Verlag und Vertrieb.

Winkelman, M. (2000). Shamanism: The neural ecology of consciousness and healing. Westport, CT: Bergin and Garvey.

Winkelman, M. (2001). Psychointegrators: Multidisciplinary perspectives on the therapeutic effects of hallucinogens. *Complementary Health Practice Review, 6*(3), 219–237.

Winkelman, M. (2007a). Therapeutic bases of psychedelic medicines: Psychointegrative effects. In M. Winkelman & T. Roberts (Eds.), *Psychedelic medicine* (vol. 1, pp. 1–19). Westport, CT: Praeger Perspectives.

Winkelman, M. (2007b). Shamanic guidelines for psychedelic medicines. In M. Winkelman and T. Roberts (Eds.), *Psychedelic medicine* (vol. 2, pp. 143–168). Westport, CT: Praeger Perspectives.

Winkelman, M., & Roberts, T. (2007a). Conclusions: Guidelines for implementing the use of psychedelic medicines. In M. Winkelman & T. Roberts (Eds.), *Psychedelic medicine* (vol. 1, pp. 271–298). Westport, CT: Praeger Perspectives.

Winkelman, M., & Roberts, T. (Eds.). (2007b). *Psychedelic medicine: New evidence for hallucinogenic substances as treatments* (vols. 1 and 2). Westport, CT: Praeger Perspectives.

Yensen, R., & Dryer, D. (2007). Addiction, despair and the soul: Psychedelic psychotherapy. In M. Winkelman & T. Roberts (Eds.), *Psychedelic medicine* (vol. 2, pp. 15–28). Westport, CT: Praeger Perspectives.

This page intentionally left blank

Chapter **21**

Entheogen-Enhanced Transpersonal Psychotherapy of Addictions: Focus on Clinical Applications of Ketamine for Treating Alcoholism[1]

Eli Kolp, MD, Evgeny Krupitsky, MD, PhD, DMedSci, Harris Friedman, PhD, and M. Scott Young, PhD

Undeniably, alcoholism is an extremely destructive problem that resists most conventional treatment approaches (Miller et al., 1995; Nathan, 1986). In the United States, alcohol is the most abused drug, with an estimated 17.6 million adults suffering alcohol-related problems, while 115 million use alcohol regularly (National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2004). The use of alcohol is related to numerous maladies, including accidents, cancer, cardiovascular disease, fetal alcohol syndrome, liver disease, and suicide. NIAAA estimated that approximately 200,000 deaths per year in the United States are attributed to alcohol-related problems, incurring an estimated annual economic cost of approximately $185 billion. Similarly, the cost of other addictions, both economically and to human lives, is steep.

Ketamine hydrochloride (ketamine) is a prescription medication widely used for general anesthesia. It was first synthesized in 1962 and later patented by Parke-Davis in 1966 as a human anesthetic. During the Vietnam War, ketamine was the most widely used battlefield anesthetic. The U.S. Food and Drug Administration (FDA) approved ketamine anesthesia for use with all ages (children, adults, and the elderly) in 1970. Since then, ketamine has been adopted by many hospitals and medical offices because of its rapid onset, proven safety, and short duration of action. There are more than 7,000 publications that describe its high effectiveness and safety (e.g., Bauman, Kish, Baumann, & Politis, 1999; Dachs & Innes, 1997; Ersek, 2004; Reich & Silvay, 1989; Ross & Fochtman, 1995; Shapiro, Wyte, & Harris, 1972). It is also noteworthy that numerous clinical studies have detected no long-term psychological impairment following

ketamine use (Siegal, 1978). Furthermore, according to several studies, ketamine even prevents brain damage in some high-risk circumstances, such as from low blood sugar, low oxygen levels, epileptic seizure, head trauma, heart attack, and stroke (Hirota & Lambert, 1996; Rothman, Thurston, Hauhart, Clark, & Solomon, 1987; Shapira, Lam, Eng, Laohaprasit, & Michel, 1994; Weiss, Goldberg, & Choi, 1986), suggesting that it is more than merely safe but actually may be protective in many adverse circumstances.

But ketamine also has some unusual properties. As a rapid-acting, nonbarbiturate and nonnarcotic agent, ketamine has been called a dissociative anesthetic since it appears to create a sense of disconnection between so-called mind and body. This is quite unlike the properties of conventional anesthetics, which basically extinguish consciousness. The dissociative property of ketamine creates what can be described as an emergence phenomena, which is a nonordinary consciousness state that can be viewed as psychedelic or transpersonal. In this state, inner awareness and perception of the outer world are dramatically altered, which is characteristic of psychedelic substances. Also, ketamine induces experiences that can be called transpersonal, such as the following: identity loss or ego dissolution; mythological, archetypal, or mystical interconnectedness/unity; out-of-body or nonphysical being; rebirth, dying, or even reliving past incarnations (Khorramzadeh & Lofty, 1976; Krupitsky & Grinenko, 1997; Krystal et al., 1994; Lilly, 1988; Moore & Alltounian, 1978; Weil & Rosen, 1983). Substances that produce such transpersonal states are often called entheogens for the frequent religious and spiritual significance of these experiences.

Although ketamine can be classified with other psychopharmacological compounds called psychedelics because of its mind-altering effects, it is the only psychedelic that is legal for physicians to prescribe for therapeutic purposes in the United States, because it has been so extensively researched for safety as an anesthetic. Ketamine is not banned under any of the provisions of the U.S. Controlled Substances Act, so it is readily available for use as an anesthetic—and it can also be legally administered off-label in the United States by licensed physicians for treating various medical concerns. Some of its possible off-label uses have been studied, such as for treating severe intractable pain, such as ischemic limb and neuropathic pain disorder, as well as refractory cancer pain (Carr et al., 2004), and for treating mood disorder (Zarate et al., 2006). In this context, it should be noted that there has been a recent renewal of interest in psychedelic therapy for treating a wide variety of psychological and psychiatric problems, including alcoholism and various forms of substance abuse (Friedman, 2006). Many have argued that transpersonal or spiritual experience is a crucial factor in recovery from addiction to various substances

(e.g., Amodia, Cano, & Eliason, 2005; Corrington, 1989; Grof, 1980; Halpern, 1996; Krupitsky & Grinenko, 1997; Kurland, Savage, Pahnke, Grof, & Olson, 1971; Robinson, Brower, & Kurtz, 2003; Whitfield, 1984). This is also the core teaching of many self-help movements, such as Alcoholics Anonymous (AA). AA's cofounder, Bill Wilson, recovered from alcoholism after a spontaneous spiritual experience in 1934; after that, he never again doubted God's existence, nor took another drink (Alcoholics Anonymous World Services, 1984, pp. 120–121). It is well-known that, subsequently, AA and various Twelve-Step programs adopted a spiritual orientation (Corrington, 1989; Whitfield, 1984). It is lesser known, however, that Wilson in 1956 took a psychedelic drug (LSD) under supervision by a psychiatrist at the Los Angeles Veterans Administration Hospital. He had a profound experience, similar to his previous spiritual experience, and felt that this second spiritual experience eliminated "many barriers erected by the self, or ego" and thought he "might have found something that could make a big difference to the lives of many who still suffered" from alcoholism (Alcoholics Anonymous World Services, 1984, pp. 368, 371). A therapy enhancing the likelihood of spiritual experience, therefore, could have great utility for treating alcoholism and other substance use disorders. Consequently, this opens an avenue to how using psychedelics such as ketamine may be therapeutic for treating such concerns.

MECHANISMS FOR UNDERSTANDING POSSIBLE THERAPEUTIC APPLICATIONS OF KETAMINE

Most contemporary U.S. researchers exploring the therapeutic application of ketamine seem to assume it operates through a biochemical mechanism, similar to the way actions of other medications are generally construed. Even other psychedelics are primarily seen as a psychotomimetic agent affecting neurotransmitters, most often the serotonin system. Interestingly, ketamine is not primarily based on serotonergic actions; rather, its biochemical mechanism appears primarily based on glutamatergic N-methyl-D-aspartate (NMDA) antagonism. In this way, ketamine is often seen as a more appropriate model for psychosis than are serotonergic psychedelics, such as lysergic acid diethylamide (LSD), N,N-Dimethyltryptamine (DMT), and psilocybin (Krystal et al., 1994).

In addition to biochemical perspectives, psychedelics are sometimes seen as operating through neurobiological, rather than neurochemical, mechanisms. For example, visual effects of ayahuasca could be related to alterations in cortical oscillations in visual pathways, a top-down, as opposed to bottom-up, mechanism (Frecska, White, & Luna, 2003, 2004). In this regard, ketamine

seems to affect different cortical regions, based on positron emission tomography data, than do other psychedelic drugs like psilocybin (Vollenweider, 1992–1993). The neurobiological mechanisms involving ketamine and most other psychedelics are complex and not yet fully understood, but they provide a plausible alternative to neurochemical explanations that seem to prevail (Friedman, 2006).

In addition to these possible neurochemical and neurobiological mechanisms, effects of ketamine may be explained psychologically. Ketamine often induces psychodynamic experiences, such as a life-review similar to what is sometimes described in near-death experiences (NDEs) when one's past history is vividly seen unfolding (Kolp et al., in-press). These experiences may reveal patterns that are sometimes portrayed in richly symbolic forms of visionary experiences. Such revelations can bring into awareness previously unconscious maladaptive themes that, when made conscious, allow possibilities for life decisions to change these patterns, including changing ingrained destructive addictions such as alcoholism and other forms of substance use disorders.

Ketamine may also produce other effects that are extraordinary by conventional standards, namely transpersonal phenomena (e.g., experiences of death/rebirth, ego dissolution, and unity with humanity, nature, and the supernatural). These transpersonal emergence phenomena have been well-documented in multiple studies and appear in general not to result in ensuing psychological problems (White, Way, & Trevor, 1982), though they can be disturbing for some. Many have speculated that psychedelic drugs, including ketamine, might be useful primarily because of transpersonal, rather than neurochemical or neurobiological, effects (Friedman, 2006). Previous psychedelic psychotherapy studies (e.g., Grinspoon & Bakalar, 1979; Grof, 1980; Jansen, 1997, 2001; Khorramzadeh & Lofty, 1973, 1976; Krupitsky et al., 2002; Krupitsky et al., 1997; Krupitsky et al., 1992; Kurland et al., 1971; Leary, Metzner, & Alpert, 1964; Roquet, 1974; Strassman, 1995) suggested that transpersonal experiences may be beneficial through contributing to catharsis, stabilizing positive changes, enhancing growth and self-awareness, catalyzing insights, increasing creativity, broadening spirituality, and harmonizing relationships with people and the world.

Grof (1980) developed the most comprehensive transpersonal theory of psychedelic psychotherapy, presenting not only theory but also supporting data that psychedelics can facilitate experiences of symbolic death/rebirth, which allows clients to work through unconscious trauma. Grof applied psychedelic psychotherapy successfully with over 750 clients by discouraging analyzing psychological problems and, instead, encouraging transcending them through emphasizing their growth potential.

One specific transpersonal mechanism that may inure uniquely to ketamine, as compared to other psychedelic substances (in addition to ketamine being available legally by off-label prescription), is that it reliably replicates NDEs (Grinspoon & Bakalar, 1979; Lilly, 1988). Ketamine-induced NDEs produce all of the common features associated with natural NDEs, including a sense of being truly dead, of consciousness being separated from the body or out-of-body, that what is being experienced is absolutely real and that it cannot be adequately described in usual language, and of transcendence of space/time (Jansen, 1997, 2001). It is noteworthy that ketamine-induced psychedelic experience differs significantly from that caused by serotonergic hallucinogens. Most (approximately 70%) NDEs result in feelings of peacefulness, while some (approximately 30%) result in fear. Memories are often reorganized in a life-review, and transcendent states with visions, such as of important figures in one's life like parents and teachers (who may be dead or alive at the time), as well as of archetypal images including angels and gods/goddesses, may be experienced. Frequently, the experience of a sense of God is reported as luminescent white light (Jansen, 1997, 2001). Similar to natural NDEs, these ketamine experiences are often transformative, changing worldviews and deepening spiritual understandings (Ring, 1984). Thus, NDEs provide a plausible transpersonal mechanism to understand the possible efficacy of clinical applications of ketamine. Specifically, we speculate that ketamine-induced NDEs provide opportunity for intense life-review that can facilitate powerful experiential shifts in attitudes toward alcohol and substance use, motivating clients to change therapeutically.

HISTORY OF KETAMINE USE IN PSYCHOTHERAPY

Lilly (1988) first began to explore the psychotherapeutic use of ketamine during the 1960s. He administered ketamine, often using sensory isolation tanks to enhance its effects. Lilly documented relationships between dosage levels and the qualities of ketamine experiences. He noted clear dose relations such that intramuscular injections of ketamine at 25 mg did not cause visual images, whereas at above 30 mg it produced such images when the subject's eyes were closed. At above 75 mg, visual images increased and feelings of detachment of the mind from the body began. At above 150 mg, feelings of complete dissociation of the mind from the body were common, as were intense visual experiences, whereas doses above 300 mg produced unconsciousness.

Khorramzadeh and Lofty (1973) were the first to study ketamine in the practice of psychiatry by administering ketamine to 100 clients with psychiatric and/or psychosomatic diagnoses, including anxiety, conversion reaction,

depression, hypochondriasis, hysteria, neurosis, obsessive-compulsive phobias, tension headaches, and ulcerative colitis. They reported that 91 of these patients did well after six months, while 88 did well after a year. Complications from ketamine were minimal, leading them to conclude that the mind-expanding potential of ketamine related to abreactive (cathartic) healing. Later, Khorramzadeh and Lofty (1976) studied the relationship of client personality to types of emergence phenomena induced by ketamine. Using a well-known personality inventory (Eysenck Personality Inventory) with 606 clients who took ketamine as anesthesia during surgery, Khorramzadeh and Lofty reported a variety of meaningful patterns.

Fontana (1974) employed ketamine adjunctive to psychotherapy for depression to facilitate "a deep regression to prenatal level, experienced as disintegration and death, followed by a progression experienced as a rebirth" and observed that ketamine "allows the therapist not only to witness the dream but also to introduce himself into it and to correct, in situ, the primitive experience through the bi-personal relationship" (p. 39). He emphasized the advantages of ketamine as making it possible "to reach such levels of regressions as had never been observed before" (p. 39).

Roquet (1974) used ketamine in group psychotherapy, combining psychoanalytic techniques with indigenous Mexican healing ceremonies, and created an approach to psychotherapy he called psychosynthesis (not the psychosynthesis developed by Robertos Assagioli, 1965). Roquet primarily treated neurotic clients, although he also treated personality disorders and some psychotic clients. His therapeutic regimen also incorporated additional psychedelic substances, such as LSD, peyote, psilocybin, and *Datura ceratocaulum*.

Grof (1980), who developed a comprehensive psychedelic psychotherapy, primarily employed LSD, but he also acknowledged ketamine as holding great potential because of its "affinity for positive dynamic systems" and reported that the effects of ketamine were so powerful that "it catapults the patient beyond the point of impasse from the previous LSD session, and can make it possible for him or her to reach the better level of integration" (Grof, 1980, p. 214).

As mentioned previously, most contemporary ketamine research in the United States stems from a biochemical approach in which the ketamine psychedelic state is often seen as a model for psychosis. Many of these recent studies have been conducted by John Krystal and have concentrated on the perceptual and cognitive effects of ketamine (Krystal et al., 1994). However, his research team has also completed one clinical study of ketamine, showing it to be very effective for treatment-resistant major depression (Berman et al., 2000). In addition, other recent work suggests ketamine may be highly effective for treatment-resistant major depression (Zarate et al., 2006).

KRUPITSKY'S CLINICAL RESEARCH WITH KETAMINE

One of us, Krupitsky (who formerly worked with Krystal), has conducted the most extensive and rigorous clinical research on ketamine to date. He began administering ketamine in 1985 as part of psychotherapy and has treated without complications more than 1,000 clients. His early exploration used ketamine as part of an aversive conditioning treatment of alcohol dependence, but his later work gradually shifted to an existential and transpersonal model focused on ketamine treatment of alcohol and, to a lesser extent, heroin dependence.

Among his noteworthy findings, Krupitsky has shown that ketamine-enhanced psychotherapy is dose-related, being more effective with a relatively high dose (2.0 mg/kg to 2.5 mg/kg), which is seen as psychedelic, than with a relatively low (or subpsychedelic) dose (0.2 mg/kg) (Krupitsky et al., 2002). Quantitative assessment of the ketamine-induced effects within the high-dose group using the Hallucinogen Rating Scale (HRS; Strassman, Qualls, Uhlenhuth, & Kellner, 1994) were comparable to those using a tryptamine hallucinogen (DMT) described as fully psychedelic by experienced users of psychedelic (Strassman, 1996). In that regard, Krupitsky argued that the salutary effects of ketamine are likely related to its psychedelic properties. It should be noted, however, that subpsychedelic doses do induce some psychedelic effects but not a fully psychedelic experience. High-dose subjects in this study had psychedelic peak experiences, while low-dose subjects experienced something less potent, sometimes called ketamine-facilitated guided imagery (Leuner, 1977). Consequently, Krupitsky argued that the salutary effects of ketamine are likely psychedelic in nature.

Other studies by Krupitsky (e.g., Krupitsky et al., 2002; Krupitsky & Grinenko, 1997; Krupitsky et al., 1992) have shown that clients treated with ketamine reported better ability to attribute meaning and purpose in their lives, as well as changes in values that reflected an increased understanding of positive alternatives to alcoholism and substance abuse. This is congruent with Frankl's (1978) existential approach, which considers alcohol and substance use disorders to be related to loss of meaning in life—and these findings suggest that ketamine psychotherapy might address existential voids rooted in human existence. These same studies by Krupitsky also indicated that ketamine treatment affected Minnesota Multiphasic Personality Inventory personality profiles in beneficial ways, showing that clients became more confident, emotionally open, and optimistic about their futures and, conversely, less anxious, depressed, and neurotic. Other psychological measures used in these studies demonstrated positive transformations of nonverbal, unconscious aspects of self-concept and attitudes toward other people and increases in levels of spiritual development.

All of these psychological changes were judged to favor sober, nonabusing lifestyles.

Krupitsky has also used electroencephalography data in studying ketamine (Krupitsky & Grinenko, 1997), demonstrating that ketamine increases delta and theta activity in the cortex, evidencing limbic system activation as well as limbic-cortex interaction. This can be considered indirect evidence of strengthening of interactions between conscious and subconscious levels of mind. Krupitsky also provided rich data from biochemical studies showing that pharmacological action of ketamine affects opioidergic and monoaminergic neurotransmitter metabolism, neurochemical systems involved in pathogenesis of alcoholism (Krupitsky et al., 1990). So, though Krupitsky favors a transpersonal understanding of the clinical effectiveness of ketamine, he is also open to concomitant biochemical processes as well.

Krupitsky's studies (Krupitsky & Grinenko, 1997; Krupitsky et al., 1992) demonstrated that ketamine psychotherapy increases abstinence rates in clients with alcoholism. Of many subjects who received ketamine, 69.8 percent were judged abstinent for one year, while only 24 percent in a control group remained abstinent during a one-year follow-up. Krupitsky et al.'s (2002) research with heroin addicts similarly showed the one-year abstinence rate in a group receiving ketamine was five times higher (25%) than of a control group, while corresponding relapse rates were also lower. This study also showed that ketamine reduced cravings for heroin and produced no significant negative reactions. This study, a double-blind clinical trial, clearly supported the efficacy of ketamine in psychotherapy for addictions due to its scientific rigor. Last, it should be noted that many of Krupitsky's clients in these studies became more spiritual through transpersonal experiences obtained with ketamine.

THERAPEUTIC APPLICATIONS OF KETAMINE

The present body of research clearly documents that ketamine can be effective for the treatment of addiction. Although most studies to date have concentrated on alcoholism and, to some degree, opioid dependency, research results may be extrapolated to treatment of other addictions and depression, as well as possibly in the treatment of neurotic, psychosomatic, and personality disorders.

The techniques of ketamine psychedelic psychotherapy have now evolved as a procedure with the specific goal of inducing ego-dissolving transpersonal experiences. This method generally includes one to three sessions using ketamine with a conducive set, or expectations, and setting, or clinical environment. We consider it important to emphasize that ketamine alone is not a

healing agent, as in a biochemical model of medicine, but rather is adjunctive to a psychotherapeutic process involved in treating underlying biopsychosociospiritual problems.

Ketamine psychotherapy as used by Krupitsky and, to a lesser degree, by another one of us (Kolp) in his practice, involves three stages: preparing, administering, and integrating. In the preparation, preliminary psychotherapy is conducted with clients, who are informed that psychedelic sessions may allow important insights concerning personal problems, systems of values, notions of self and world, and life-meaning to become conscious. Patients are encouraged to view all of these possible insights as leading to potentially positive changes important for healing problems and embracing sobriety. At least 5 and up to 10 hours of psychotherapy may be provided before the initial ketamine session to establish psychospiritual goals for a possible transpersonal experience. This information is not simply explained to the client; rather, it is discussed at length in an individualized fashion. The therapist closely attends to issues such as personal motives for seeking treatment, goals for sobriety, and thoughts concerning causes and consequences of the problem being treated. The client and therapist form an individualized psychotherapeutic myth, which creates a set favoring confidence and mutual trust. In our view, this is the most important psychotherapeutic factor responsible for success during the second stage.

The second stage involves the induction of a transpersonal experience through administration of ketamine. Breakfast is omitted prior to a morning ketamine administration, and clients refrain from food and drink for eight hours prior to administration. Clients are instructed that they will have some unusual experiences and are encouraged to surrender to them.

Clients are asked to assume a comfortable supine position using eyeshades to reduce external visual stimulation. Ketamine is then injected intramuscularly in psychedelic doses of 2.0 mg/kg to 2.5 mg/kg. The intramuscular route provides a more gradual onset and psychedelic experiences last longer. With intravenous psychedelic doses (i.e., from 0.7 mg/kg to 1.0 mg/kg), effects last only 15 to 20 minutes, while with intramuscular injection from 45 minutes to an hour is typical.

Administration of ketamine is accompanied by specially chosen music, generally classical or New Age, and clients typically have ego-dissolving transpersonal experiences. Afterwards, there is a recovery period, which usually takes one to two hours, and clients begin to feel the return of ordinary reality. At this point, clients usually describe their experiences, and discussion begins with the psychotherapist. After the session, there is a rest period in which clients are asked to write detailed self-reports of the transpersonal aspects of the ketamine experience.

The third stage involves integration of transpersonal experiences. It is generally conducted in group psychotherapy because we (Kolp and Krupitsky) have clinically judged group therapy to be more powerful therapeutically than individual sessions for this treatment. Typically three to five hours of group psychotherapy are provided to facilitate integration of the experiences into clients' everyday lives. Clients discuss and interpret personal significances of the symbolic content in their ketamine-induced experiences. This discussion is aimed toward helping clients forge a relation between their psychedelic experiences and their problems, both intra- and interpersonal. The therapist assists clients with integrating the spiritual transformations that can result from direct transpersonal experience, often by helping clients generate fresh insights and new, often unexpected, attitudes, meanings, and values about self and the world.

Two additional points need to be addressed. First, it is noted that some clients do not fully benefit from just one ketamine session. Such so-called treatment-resistant clients may require one or two additional ketamine sessions. In addition, data from Krupitsky et al. (2007) most recent study provide evidence that three ketamine sessions may work better than one in providing a higher abstinence rate in heroin addicts, suggesting that increasing sessions may increase treatment efficacy. Second, despite the power of this type of psychotherapy, it may not have long-lasting effects without appropriate support structures, which is why we stated earlier that ketamine alone is not adequate as a treatment—it must be provided in a therapeutic context. Many clients may experience what has been called psychedelic afterglow after receiving psychedelic psychotherapy (Grinspoon & Bakalar, 1979). Such afterglow includes positive changes induced by the psychedelic experience that usually last only up to a few weeks, providing only a transient psychotherapeutic benefit that, though often pronounced for several weeks after treatment, may start declining rapidly (Halpern, 1996).

POTENTIAL FOR KETAMINE MISUSE

Ketamine is part of the group of chemicals including phencyclidine (i.e., Sernyl, PCP). These chemicals are called arylcyclohexylamines and are classified as hallucinogens or psychedelics. Ketamine, although legal for medical administration, is a controlled substance requiring prescription and is listed on Schedule III (Controlled Substances Act of 1970). Medical evidence regarding possibilities of it being abused or leading to dependence suggests that its abuse potential is similar to that of phencyclidine and other hallucinogens (Siegal, 1978). Although phencyclidine and the other hallucinogens do not meet criteria for causing drug dependence, since they do not lead to tolerance or with-

drawal symptoms, individuals with a history of heavy psychedelic drug use have reported cravings. In addition, ketamine may affect cognition (thinking), mood (feelings), and perception (imagery) in ways that may motivate some people to repeatedly use. Therefore, ketamine should only be used under direct supervision by a licensed physician.

FUTURE DIRECTIONS FOR TREATMENT AND RESEARCH

Research supports that ketamine used as part of psychotherapy may be highly effective in the treatment of both alcoholism and opioid dependency (Krupitsky et al., 2002; Krupitsky & Grinenko, 1997; Krupitsky et al., 1992). It is also possible that ketamine psychotherapy may be equally effective for treating similar dependencies, such as to depressants including benzodiazepines and barbiturates. In addition, preliminary results (Krupitsky & Grinenko, 1997) have suggested that ketamine psychotherapy may be effective for treating dependency on ephedrine, a stimulant, as well as other psychostimulants, such as amphetamines, caffeine, cocaine, and nicotine. More broadly, ketamine may find use in many areas of psychiatry, in line with studies demonstrating significant improvements with various psychiatric disorders and psychosomatic illnesses (Khorramzadeh & Lofty, 1973; Zarate et al., 2006).

This opens the possibilities for many productive research efforts. More rigorous research could explore the effectiveness and efficacy of ketamine for treating a variety of psychological problems in which preliminary data appear promising. More basic research of both a biological and psychological nature could also occur, such as to further explore the connection between clients' personality profiles and types of ketamine transpersonal experiences, as well as relationships between both of these phenomena and clients' subsequent response to ketamine treatment. Another potential research area would be to explore similarities between ketamine-induced transpersonal experiences and NDEs, following up on Jansen's (1997, 2001) work in this area. Last, the very materialistic assumptions engrained in modern science are strained by some of the powerful phenomenological reports generated by ketamine experiences; in this sense, ketamine may offer a particularly valuable tool for exploration of fundamental epistemic and ontic mysteries (Friedman, 2006).

CONCLUSION

Ketamine is an anesthetic that can legally be administered in the United States off-label by a licensed physician for psychiatric treatment as an adjunct

to psychotherapy. In this way, it differs from other psychedelics that are illegal to use in the United States, except in closely controlled research contexts. In subanesthetic doses, ketamine is a psychedelic that reliably induces transpersonal experiences. In addition, ketamine has advantages over other psychedelics due to its rapid onset, safety, and short duration of action.

A body of careful scientific research documents that ketamine psychotherapy is effective in the treatment of alcoholism and opioid dependency, as well as to some extent on abused stimulants (e.g., ephedrine). Additional data show that ketamine may be successfully used for treating various psychiatric illnesses (e.g., depression, obsessive-compulsive disorder, posttraumatic stress disorder, and phobias), some personality disorders (e.g., avoidant and histrionic personality disorder), and also some psychosomatic illnesses (e.g., tension headaches and ulcerative colitis). These studies demonstrate impressive benefits for many clients who showed improvements after receiving ketamine, including becoming more responsible, emotionally mature and open, self-confident and self-sufficient, and having increased ego strength and less anxiety and depression. In addition to these many benefits, clients often experienced both existential and spiritual growth. These transformations may reinforce positive attitudes toward sobriety and healthy lifestyles that supported stable recovery from alcohol and substance use disorders. Continued scientific research on ketamine in well-controlled studies is indicated now because of its demonstrated therapeutic effectiveness, biological safety, and pragmatic availability as one of the few legally available psychedelics and the only one that has shown clinical value.

NOTE

1. This chapter is based on earlier works by the first two authors (Kolp & Krupitsky, 2007) and all of the authors (Kolp, Friedman, Young, & Krupitsky, 2006).

REFERENCES

Alcoholics Anonymous World Services. (1984). The story of Bill Wilson and how the A.A. message reached the world. New York: Author.

Amodia, D., Cano, C., & Eliason, M. (2005). An integral approach to substance abuse. *Journal of Psychoactive Drugs, 37*(4), 363–371.

Assagioli, R. (1965). Psychosynthesis: A manual of principles and techniques. New York: Hobbs Dorman.

Bauman, L., Kish, I., Baumann, R., & Politis, D. (1999). Pediatric sedation with analgesia. *American Journal of Emergency Medicine, 17*, 1–3.

Berman, R., Cappiello, A., Anand, A., Oren, D., Heninger, G., Charney, D., et al. (2000). Antidepressant effects of ketamine in depressed patients. *Biological Psychiatry, 47*(4), 351–354.

Carr, D., Goudas, L., Denman, W., Brookoff, D., Staats, P. , Brennen, L., et al. (2004). Safety and efficacy of intranasal ketamine for the treatment of breakthrough pain in patients with chronic pain: A randomized, double-blind, placebo-controlled, crossover study. *Pain, 108,* 17–27.

Controlled Substances Act of 1970. Pub. L. No. 91-513, 84 Stat. 1236 (October 27, 1970), codified at 21 U.S.C. § 801 et. seq.

Corrington, J. (1989). Spirituality and recovery: Relationship between levels of spirituality, contentment and stress during recovery from alcoholism in AA. *Alcoholism Treatment Quarterly, 6,* 151–165.

Dachs, J., & Innes, M. (1997). Intravenous ketamine sedation of pediatric patients in the emergency department. *Annals of Emergency Medicine, 29,* 146–150.

Ersek, A. (2004). Dissociative anesthesia for safety's sake: Ketamine and diazepam—a 35-year personal experience. *Plastic and Reconstructive Surgery, 113*(7), 1955–1959.

Fontana, A. (1974). Antidepressant Therapy with Ketamine. *Psychiatric and Psychological Act of Latin America, 20,* 32.

Frankl, V. (1978). *The unheard cry for meaning.* New York: Simon and Schuster.

Frecska, E., White, K. D., & Luna, L. E. (2003). Effects of the Amazonian psychoactive beverage ayahuasca on binocular rivalry: Interhemispheric switching or interhemispheric fusion? *Journal of Psychoactive Drugs, 35*(3), 367–374.

Frecska, E., White, K. D., and Luna, L. E. (2004). Effects of ayahuasca on binocular rivalry with dichoptic stimulus alternation. *Psychopharmacology, 173,* 79–87.

Friedman, H. (2006). The renewal of psychedelic research: Implications for humanistic and transpersonal psychology. *The Humanistic Psychologist, 34*(1), 39–58.

Grinspoon, L., & Bakalar, J. (1979). Psychedelic drugs reconsidered. New York: Basic Books.

Grof, S. (1980). *LSD psychotherapy.* Alameda, CA: Hunter House Publishers.

Halpern, J. (1996). The use of hallucinogens in the treatment of addiction. *Addiction Research, 4,* 177–189.

Hirota, K., & Lambert, D. (1996). Ketamine: Its mechanism of action and unusual clinical use. *British Journal of Anaesthesia, 77*(4), 441–444.

Jansen, K. (1997). The ketamine model of the near-death experience: A central role for the NMDA receptor. *Journal of Near-Death Studies, 16,* 5–27.

Jansen, K. (2001). *Ketamine: Dreams and realities.* Sarasota, FL: MAPS.

Khorramzadeh, E., & Lofty, A. (1973). The use of ketamine in psychiatry. *Psychosomatic Journal, 14,* 344–346.

Khorramzadeh, E., & Lofty, A. (1976). Personality predisposition and emergence phenomena with ketamine. *Psychosomatic Journal, 17,* 94–95.

Kolp, E., Friedman, H., Young, M. S., & Krupitsky, E. (2006). Ketamine enhanced psychotherapy: Preliminary clinical observations on its effectiveness in treating alcoholism. *The Humanistic Psychologist, 34*(4), 399–422.

Kolp, E., & Krupitsky, E. (2007). Ketamine psychedelic psychotherapy. In M. Winkelman & T. Roberts (Eds.), *Psychedelic medicine: Addictions medicine and transpersonal healing* (vol. 2, pp. 67–85). Portsmouth, NH: Praeger.

Kolp, E., Young, M. S., Friedman, H., Krupitsky, E., Jansen, K., & O'Connor, L. (in-press). Ketamine enhanced psychotherapy: Preliminary clinical observations on its effectiveness in treating death anxiety. *International Journal of Transpersonal Studies*.

Krupitsky, E., Burakov, A., Dunaevsky, I., Romanova, T., Slavina, T., & Grinenko, A. (2007) Single versus repeated sessions of ketamine-assisted psychotherapy in people with heroin dependence. *Journal of Psychoactive Drugs, 39*(1), 13–19.

Krupitsky, E., Burakov, A., Romanova, T., Dunaevsky, I., Strassman, R., & Grinenko, A. (2002). Ketamine psychotherapy for heroin addiction: Immediate effects and two-year follow-up. *Journal of Substance Abuse Treatment, 23*, 273–283.

Krupitsky, E., & Grinenko, A. (1997). Ketamine psychedelic therapy (KPT): A review of the results of ten years of research. *Journal of Psychoactive Drugs, 29*(2), 165–183.

Krupitsky, E., Grinenko, A., Berkaliev, T., Paley, A., Petrov, V., Mashkov, K., et al. (1992). The combination of psychedelic and aversive approaches in alcoholism treatment: The affective contra-attribution method. *Alcoholism Treatment Quarterly, 9*(1), 99–105.

Krupitsky, E., Grinenko, A., Karandashova, G., Berkaliev, T., Moshkov, K., & Borodkin, Y. (1990). Metabolism of biogenic amines induced by alcoholism narcopsychotherapy with ketamine administration. *Biogenic Amines, 7*(6), 577–582.

Kurland, A., Savage, E., Pahnke, N., Grof, S., & Olson, E. (1971). LSD in the treatment of alcoholics. *Pharmacopsychiatrie Neuropsychopharmakolgie, 4*, 83–94.

Krystal, J., Karper, L., Seibyl, J., Freeman, G., Delaney, R., Bremner, J., et al. (1994). Subanesthetic effects of the noncompetitive NMDA antagonist, ketamine, in humans. Psychotomimetic, perceptual, cognitive, and neuroendocrine responses. *Archives of General Psychiatry, 51*, 199–214.

Leary, T., Metzner, R., & Alpert, R. (1964). The psychedelic experience: A manual based on the Tibetan Book of the Dead. New York: University Books.

Leuner, H. (1977). Guided affective imagery: An account of its developmental history. *Journal of Mental Imagery, 1*, 73– 91.

Lilly, J. (1988). *The scientist: A metaphysical autobiography*. New York: Bantam / Lippincott.

Miller, W., Brown, J., Simpson, T., Handmaker, N., Bien, T., Luckie, L., et al. (1995). What works?: A methodological analysis of the alcohol treatment outcome literature. In R. Hester & W. Miller (Eds.), *Handbook of alcoholism treatment approaches* (2nd ed., pp. 12–44). Boston: Allyn and Bacon.

Moore, M., & Alltounian, H. (1978). *Journeys into the bright world*. Gloucester, MA: Para Research.

Nathan, p. N. (1986). Outcomes of treatment for alcoholism: Current data. *Annals of Behavioral Medicine, 8*, 40–46.

National Institute on Alcohol Abuse and Alcoholism. (2004). *Alcoholism: Getting the facts* (NIH publication number 96-4153). Washington, DC: U.S. Department of Health and Human Services, National Institutes of Health, National Institute on Alcohol Abuse and Alcoholism.

Reich, L., & Silvay, G. (1989). Ketamine: An update on the first twenty-five years of clinical experience. *Canadian Journal of Anesthesiology, 36,* 186–197.

Ring, K. (1984). *Heading toward omega.* New York: William Morrow.

Robinson, E., Brower, K., & Kurtz, E. (2003). Life-changing experiences spirituality and religiousness of persons entering treatment for alcohol problems. *Alcoholism Treatment Quarterly, 21*(4), 3–16.

Roquet, S. (1974). Operacion Mazateca: Estudio de hongos y otras plantas allucinoganas—Mexicanastratamiento psicotherapeutico depsicosintesis [The Mazateca Operation: A study of hallucinogenic plants and mushrooms—Mexican psychotherapeutic psychosynthesis]. Mexico City: Asociacion Albert Schweitzer.

Ross J., & Fochtman, D. (1995). Conscious sedation: A quality management project. *Journal of Pediatric Oncology Nursing, 12,* 115–121.

Rothman, S., Thurston, J., Hauhart, R., Clark, G., & Solomon, J. (1987). Ketamine protects hippocampal neurons from anoxia in vitro. *Neuroscience, 21,* 673–683.

Shapira, Y., Lam, A., Eng, C., Laohaprasit, V., & Michel, M. (1994). Therapeutic time window and dose response of the beneficial effects of ketamine in experimental head injury. *Stroke, 25,* 1637–1643.

Shapiro, M., Wyte, R., & Harris, B. (1972). Ketamine anesthesia in patients with intracranial pathology. *British Journal of Anesthesiology, 44,* 1200–1204.

Siegal, R. (1978). Phencyclidine and ketamine intoxification: A study of four populations of recreational users. *National Institute of Drug Abuse Research Monograph, 21,* 119–147.

Strassman, R. (1995). Hallucinogenic drugs in psychiatric research and treatment. Perspectives and prospects. *Journal of Nervous and Mental Disease, 183,* 127–138.

Strassman, R. (1996) Human psychopharmacology of N,N-dimethyltryptamine. *Behavior and Brain Research, 73,* 121–124.

Strassman, R., Qualls, R., Uhlenhuth, H., & Kellner, R. (1994). Dose-response study of N,N-dimethyltryptamine in humans. II. Subjective effects and preliminary results of a new rating scale. *Archives of General Psychiatry, 51,* 98–108.

Vollenweider, F. X. (1992–1993). New avenues in the search for biological correlates of altered states of consciousness—From model to practice. Retrieved May 6, 2006, from http://www.maps.org/news-letters/v03n4/03418pet.html.

Weil, A., & Rosen, W. (1983). *Chocolate to morphine: Understanding mind-active drugs.* Boston: Houghton Mifflin.

Weiss, J., Goldberg, M. P., & Choi, D. W. (1986). Ketamine protects cultured neocortical neurons from hypoxic injury. *Brain Research, 380,* 186–190.

White, p. , Way, W., & Trevor, A. (1982). Ketamine: Its pharmacology and therapeutic uses. *Anesthesiology, 56,* 119–136.

Whitfield, C. (1984). Stress management and spirituality during recovery: A transpersonal approach. Part 1: Becoming. *Alcoholism Treatment Quarterly, 1,* 3–54.

Zarate, C., Singh, J., Carlson, p. , Brutsche, N., Ameli, R., Luckenbaugh, D., et al. (2006). A randomized trial of an N-methyl-D-aspartate antagonist in treatment-resistant major depression. *Archives of General psychiatry, 63,* 856–864.

This page intentionally left blank

Index

About the Editor and Contributors

EDITOR

Angela Browne-Miller, PhD, DSW, MPH, is the Set Editor of the *Praeger International Collection on Addictions* (2009), Director of Metaxis Institute for Personal, Social, and Systems Change, based in northern California, United States; Director of Browne and Associates Violence, Substance Abuse, and Trauma Treatment and Prevention Program, also in northern California; has been a keynote speaker at conferences around the world on addiction, violence, and behavior change; and is the author of numerous books, including *To Have and to Hurt: Seeing, Changing or Escaping Patterns of Abuse in Relationships* (2007) and *Rewiring Your Brain to Change Your Behavior* (2009). Dr. Browne-Miller earned two doctorates and two master's degrees at the University of California, Berkeley, where she lectured in three departments for 14 years, and has served as a National Institute of Mental Health Postdoctoral Fellow, a U.S. Department of Public Health Fellow, the public relations director for Californians for Drug Free Youth, the Research Education and Treatment Director for the Cokenders Alcohol and Drug Program, a member of the board of directors of the Employee Assistance Society of North America, an advisor to addiction treatment programs in the United States and several other countries, and project director on three California Department of Health violence prevention projects.

CONTRIBUTORS

Lyndsay Ammon, MPH, is a doctoral candidate in epidemiology at University of California Berkeley's (UCB) School of Public Health in Berkeley, California, United States. Ms. Ammon received an MPH from Boston University's School of Public Health. Currently, she is a research associate at the Alcohol Research Group (ARG) in Emeryville, California and a predoctoral research fellow in ARG's and UCB's joint National Institute of Alcohol Abuse and Alcoholism's Training Program. She has conducted research on the relationship between formal and informal drug and alcohol services and alcohol-related outcomes. In addition, she has published in the area of substance use in relation to sexual risk behavior. In her dissertation, Ms. Ammon is evaluating whether prenatal multivitamin use modifies the relationship between prenatal exposure to alcohol and perinatal and reproductive outcomes.

Kelly Anders, MAL, is a research associate working on national and international research projects with Maria Dinis, PhD, MSW, associate professor at the Division of Social Work, California State University Sacramento in Sacramento, California, United States. Ms. Anders does research in the area of leadership studies. Her interests are organizational sociology, leadership, international studies, and educational policy. She received her master's of arts in leadership from Augsburg College in Minneapolis, Minnesota.

Thomas Beers, PhD, graduated from the University of Cincinnati in Cincinnati, Ohio, United States, with an MA and PhD in clinical psychology. Following graduation he worked as a clinical psychologist at the Alcohol Counseling Program at a CMHC in Pensacola, Florida, United States. Dr. Beers was director of the Chemical Dependency Recovery Program with Kaiser Permanente, San Diego, California, United States, from 1981 to 2003. Since 2003, Dr. Beers has served as clinical director of the California Screening, Brief Intervention, Referral and Treatment program (CASBIRT). He has also supervised US Navy addictions counselors for the last 25 years. Dr. Beers is a licensed psychologist in California and holds APA Certification for Proficiency in Treatment of Substance Disorders.

Robert K. Brooner, PhD, is a professor of medical psychology and psychiatry in the Department of Psychiatry and Behavioral Sciences at the Johns Hopkins University School of Medicine in Baltimore, Maryland, United States. He received a BA at the University of California–Santa Cruz and a PhD at the California School of Professional Psychology, both in California, United States. He completed an internship in medical psychology at The Johns Hopkins University School of Medicine in Baltimore Maryland, United States, and one

year of additional postdoctoral training. He is a licensed clinical psychologist and director of Addiction Treatment Services at the Johns Hopkins Bayview Medical Center. Current research interests include: assessment of substance use disorder with and without other psychiatric comorbidity; testing interventions for substance dependent patients with specific types of psychiatric comorbidity; testing of combination treatments that incorporate behavioral reinforcement to motivate adherence; and the development and testing of adaptive treatment models to improve treatment-seeking behaviors and maximize therapeutic response in substance-dependent patients with and without other psychiatric and medical comorbidities.

James Burns, PhD, is a senior staff psychologist and senior research fellow at the Danielsen Institute of Boston University in Boston, Massachusetts, United States. He also holds appointments at the University of St. Thomas Graduate School of Professional Psychology in Twin Cities, Minnesota, United States, and as an instructor at Harvard Medical School in Boston, Massachusetts, United States. He currently coordinates consultation services for clergy, their families, and congregations. He was ordained as a priest in 1993 and holds a double diploma in behavioral medicine and counseling psychotherapy. He is a licensed psychologist and has received various awards for clinical expertise having completed clinical education at Yale School of Medicine, McLean Hospital/Harvard Medical School, the Center for the Study of Religion and Psychology, and the Boston Psychoanalytic Society. He holds a PhD in counseling psychology from Northeastern University. Dr. Burns's research to date has examined the relationship between levels of anger and aggression in adolescent males and their levels of spirituality, as well as general mental health issues and risk factors in adolescence and the relationship of these issues to religion and spirituality. Additional research studies currently underway are examining the way the sexual abuse crisis in the Catholic Church and church closings have affected morale of priests, pastoral leadership, and community dynamics. He has been involved in studying the impact that Catholic schools in inner cities have on the lives of youth and their communities. His international studies engage questions of peace and conflict particularly as these impact globalization and mental health resources.

Giuseppe Carrà, MD, MSc, PhD, consultant psychiatrist and research fellow at University College London, (UCL), England, studied Medicine at Milan University, and did his psychiatric training at Pavia University in Pavia, Lombardy, Italy. He has a PhD in public mental health from the University of Pavia. Dr. Carrà did an MSc in psychiatric research at UCL. Since 1995, he has been a consultant psychiatrist at S. Paolo University Hospital Trust,

Milan, Italy. Since 2004 Dr. Carrà has been a research fellow at UCL. He is a board member of the International Society of Addiction. His governmental and institutional appointments include: member of Guideline Development Group for Schizophrenia at the Italian National Institute of Health; member of Addiction Panel at Italian Department of Health; member of the Panel of Independent Experts assisting the European Commission at Directorate General of Justice, Freedom and Security in the priority field "Prevent Drug Consumption and Inform the Public," July 2007–June 2010. His research interests include dual diagnosis of psychosis and substance misuse; psychoeducational interventions in schizophrenia; and adaptation and implementation of local guidelines.

Massimo Clerici, MD, PhD, studied medicine and did his psychiatric training at Milan University, Milan, Italy. He has a PhD in psychiatry and relational sciences from the University of Milano in Italy. Since 1991, he has held the position of assistant professor (university researcher) at the University Hospital of S. Paolo in Milan, Italy, and of associate professor at the University of Milano Bicocca, also in Italy, (second University of Milan) from the academic year of 2007–2008. He is now the president of the Italian Society of Addiction Psychiatry (SIP. Dip, special section of the Italian Society of Psychiatry) since 2002, vice president of the Association of Research on Schizophrenia (ARS, member Society of WPA) from 2007 and board member at several professional and scientific societies. He is also the editor of the Addiction's Book Collection of one of the most important Italian publishers. Dr. Clerici also serves as a member of the National Department for Politics against Drugs, National Scientific Committee against Addictions of the Italian Government; and the Italian Panel of Scientific and Professional Societies against Addictions.

Jana Cook, MD, PhD, joined the Center for Criminality and Addiction Research, Training and Application (CCARTA) in San Diego, California, United States, in 2006 as assistant project scientist. She trains the workforce of substance abuse counselors and criminal justice professionals on addiction treatment issues in custody and community-based treatment programs in the state of California and serves as a member of the Executive Committee of the Forensic Addictions Corrections Treatment (FACT) Board of Directors. Dr. Cook graduated from the Medical School at the Comenius University, Bratislava, Slovakia. She is trained in general psychiatry and specialized in substance abuse treatment and forensic psychiatry and is certified by the Ministry of Justice of the Slovak Republic as an expert witness in psychiatry. Jana worked as assistant professor of psychiatry at the Comenius University, Bratislava. She is a member of the World Council of Young Psychiatrists and one of the found-

ers of the World Association of Young Psychiatrists and Trainees, and she has worked with the Council of Europe as consultant.

Ann N. Dapice, PhD, is director, Education and Research, of T. K. Wolf, Inc., a nonprofit American Indian organization in Tulsa, Oklahoma, United States. With a PhD in psychology, sociology, and philosophy from the University of Pennsylvania, United States, she has served as professor and administrator at a number of universities in the United States, including the University of Pennsylvania, Widener University, Penn State University, and Goddard College, teaching social sciences and Native American studies. Her cross-cultural research has been reported in professional journals, books, and academic presentations regionally, nationally, and internationally. Her speaking engagements include topics related to addiction, stalking, and American Indian issues. She has served on the Elders Council for the American Indian Chamber of Commerce, is a member of the Board of Directors of Mental Health Association in Tulsa, and the Tulsa Metropolitan Ministry/Domestic Violence Intervention Services Committee. She consults with the University of Pennsylvania on Native American issues, where she is founder/chair of the Association of Native Alumni.

Maria Dinis, PhD, MSW, is an associate professor for the Division of Social Work at California State University Sacramento (CSUS), in Sacramento, California, United States. Dr. Dinis is the admissions director for the Master's of Social Work Program at CSUS. Dr. Dinis is the chair of the CSUS Committee for the Protection of Human Subjects (CPHS) during every spring term and is a member of the California Health and Human Services Agency, a CPHS committee. She has published in the areas of alcohol/drug treatment, policy, and prevention. Her interests are in multicultural issues in regards to alcohol/drugs, women, and global social work. Dr. Dinis received her PhD from the School of Social Welfare at the University of California, Berkeley, in Berkeley, California, United States.

Nady el-Guebaly, MD, is professor and head, Division of Addiction, Department of Psychiatry at the University of Calgary in Calgary, Alberta, Canada, and past chair of the department. He is the founding medical director of the Calgary Health Region's Addiction Centre and Program in Canada. Currently, he is also board chair of the Alberta Gaming Research Institute, also in Canada; third term chair, Addiction Psychiatry Section of the World Psychiatric Association; and executive medical director and past founding president of the International Society of Addiction Medicine. He holds recognition awards from the American Society of Addiction Medicine, the Mexican Psychiatric Association, the Italian Society of Addiction Psychiatry, the University of Calgary's Guenther Distinguished Achievement Award in International Health, a Queen Elizabeth II Golden Jubilee medal and an Alberta

Centennial medal. Major research interests have resulted in 200 peer-reviewed papers and chapters, 450 abstracts, and 60 past and current research grants.

Ann Fabean, PhD, of Greenburg, Pennsylvania, United States, holds her PhD in family studies, an MA in counseling and BS in psychology. A licensed marriage and family therapist, she is now retired from practice. Her extensive work in the drug and alcohol field has been with addicts and their families in inpatient and outpatient settings, and in advocacy, prevention, and on various mental health boards.

Harris Friedman, PhD, is research professor in the Counseling Psychology program at the University of Florida in Gainesville, Florida, United States. He is also professor emeritus at Saybrook Graduate School and holds a number of other academic appointments. He received his PhD from Georgia State University in Atlanta, Georgia, United States, and Diplomate from the American Board of Professional Psychology, both in clinical psychology. He has taught at a number of colleges and universities as well as chaired a doctoral clinical psychology program and served as a graduate school academic dean. He is also retired from private practice as a clinical and consulting psychologist, where he specialized in humanistic-transpersonal psychotherapy and community-organizational change psychology. He serves as associate editor of *The Humanistic Psychologist* and editor of the *International Journal of Transpersonal Studies*, and has published over 100 professional papers and books.

Hamid Ghodse, MD, PhD, DSc, is professor of psychiatry and international drug policy at the University of London, England, the director of the International Centre for Drug Policy at St. George's University of also in London, England, medical director of the National Advisory Committee on Clinical Excellence Awards (ACCEA) for England and Wales, UK, nonexecutive director of the UK National Patients Safety Agency (NPSA), and the chairman of the RCPsych Honours Committee in the UK. He is a distinguished leader in the fields of psychiatry and addictive behavior from the perspective of research and policy development and implementation. He was awarded the highest honor of the Royal College of Psychiatrists in the UK, by being elected an honorary fellow in 2006. He has published over 300 papers and chapters and a number of books, of which those on legal and clinical aspects have become standard works, both in their 3rd edition. His new book, *Addiction at Work*, is another groundbreaking work. He has been an advisor on analgesic and psychotropic medication to the British National Formulary in the UK since 1982. His research paper won the SIGP 2004 Prize for the best psychopharmacological paper in the *British Journal of Psychiatry*. He was awarded CBE in 1999.

Sean R. Hogan, PhD, MSW, is assistant professor in the Department of Social Work at California State University Fullerton, California, United States. In 2005, he received his doctorate from the School of Social Welfare at the University of California, Berkeley, Berkeley, United States. Dr. Hogan's teaching interests are in the areas of substance abuse and social welfare policy. He also has an interest in the effectiveness of service learning as a pedagogical method for teaching substance abuse education to social welfare students. Dr. Hogan's research focuses on the effects of welfare reform legislation on marginalized populations, with a specific interest in the effects of eliminating welfare benefits to low-income substance abusers. He also has an interest in the history and construction of substance abuse policy in the United States and its relationship to social inequality.

Melinda Hohman, PhD, is a professor at the School of Social Work, San Diego State University, San Diego, California, United States, where she has worked since 1995. Dr. Hohman teaches courses in substance abuse treatment, research, and social work practice. Her research interests include substance abuse assessment and treatment services and the overlap of substance abuse treatment and child welfare services. She has been a trainer in motivational interviewing (MI) since 1999, training community social workers, child welfare workers, probation officers, and addiction counselors across southern California. Dr. Hohman is the Co-PI/evaluator on two substance abuse and mental health services expansion grants for women receiving drug treatment in a residential setting. She serves as a consultant on the California Screening Brief Intervention, Referral and Treatment Services (CASBIRT) project.

Rev. Robert Davis Hughes, III, PhD, is Norma and Olan Mills Professor of Divinity and Professor of Systematic Theology at the School of Theology, University of the South (Sewanee), Sewanee, Tennessee, United States, where he has taught since 1977. His degrees are from Yale, Episcopal Theological School, and St. Michael's College, University of Toronto. During this time he was a National Merit Scholar and a Kent Fellow of the Danforth Foundation. He is a graduate fellow of the Episcopal Church Foundation and a member of the Fellows' Forum planning group. He also serves on the General Board of Examining Chaplains and publishes frequently in *Sewanee Theological Review*, *Anglican Theological Review*, and elsewhere. He has served parishes in the Dioceses of Southern Ohio, Ohio, United States, and Toronto, Canada. He is past president and chair of the Missions Committee for Global Outreach for Addiction Leadership (GOAL) Project, www.goalproject.org, an ecumenical mission society that takes Twelve-Step recovery where it is not and supports it where it is new. Bob's wife, Barbara, is a noted sculptor, completing the new per-

manent crèche for the National Cathedral. They have two sons and two grandchildren.

Michael Kidorf, PhD, is an associate professor of psychiatry and behavioral sciences at Johns Hopkins University School of Medicine in Baltimore, Maryland, United States. He received a BA at Emory University and a PhD in clinical psychology at Florida State University. He also completed an internship at Brown University and postdoctoral training at the Behavioral Pharmacology Research Unit of the Johns Hopkins University. He is a licensed psychologist and associate director of Addiction Treatment Services at Johns Hopkins Bayview Medical Center. His current research interests include the study of stepped care approaches to improve the treatment response of drug users and the study of motivational interventions to encourage out-of-treatment drug abusers to enroll in substance abuse treatment.

Van L. King, MD, is an associate professor of psychiatry and behavioral sciences at the Johns Hopkins University School of Medicine in Baltimore, Maryland, United States. He received a BS at the University of Wisconsin–Madison and an MD at the University of Rochester School of Medicine. He completed postdoctoral psychiatry residency training at the Massachusetts General Hospital and is board certified by the American Board of Psychiatry and Neurology. He has been the medical director of Addiction Treatment Services at Johns Hopkins Bayview Medical Center since 1993. His research interests have focused on co-occurring psychiatric disorders in patients with substance use disorders and integration of co-occurring psychiatric disorder treatment with substance use disorder treatment. More recently, he has developed adaptive treatment strategies to extend the continuum of care for patients requiring long-term substance use treatment to minimize the intrusiveness of treatment while ensuring adequate monitoring and treatment response.

Eli Kolp, MD, received the MD degree from Moscow Medical Stomatological Institute in Moscow, Russia, and received residency training in both surgery and psychiatry in Russia, as well as additional residency training in psychiatry at the University of Texas Medical School in Texas, United States. In 1994, he began researching ketamine psychedelic psychotherapy while on staff at the James A. Haley Veterans Hospital in Tampa, Florida, United States, where he continued his work through 1999. He also was a fellow in geriatric psychiatry at University of South Florida College of Medicine. He is board certified in psychiatry and presently maintains a private practice in the Tampa Bay area of Florida, United States, specializing exclusively in ketamine psychedelic psychotherapy.

Igor Koutsenok, MD, MS, is assistant professor of psychiatry at the University of California San Diego (UCSD) School of Medicine in San Diego, California, United States, and director of the Center for Criminality and Addiction Research, Training and Application (CCARTA) also in San Diego. He has an MD from the University in Kiev (1982) in Kiev, Russia. In 1990 he received a degree as psychiatrist from the Medical University in Sofia in Bulgaria. In 1994–1996 he worked at the University of London, UK, Department of Addictive Behavior and Psychological Medicine, where he received a master's degree in addictive behavior. During the last 20 years, he served as an expert for many international organizations such as the Council of Europe, European Union, and the United Nations. Dr. Koutsenok joined UCSD in 1997. He is the Forensic Addiction Treatment Certification Board of Education director and director of the Offender Substance Abuse Treatment Institute. He has authored and coauthored over 30 scientific publications and book chapters, such as *Substance Abuse: A Comprehensive Textbook* (4th edition, 2004), and *Treatment of Drug Offenders* (2007).

Evgeny Krupitsky, MD, PhD, DMedSci, received an MD from Leningrad Institute of Hygiene and Sanitation, in Leningrad, Russia, a PhD from Leningrad Institute of Experimental Medicine in Psychopharmacology, also in Leningrad, and a DMedSci in psychiatry from St. Petersburg Bekhterev Research Psychoneurological Institute in St. Petersburg, Russia. He is the chief of the Laboratory of Clinical Pharmacology of Addictions at St. Petersburg State Pavlov Medical University and the chief of the Department of Addictions at the Bekhterev Research Psychoneurological Institute, as well as the main specialist in Addiction Medicine of the Health Care Committee of the Government of the Leningrad Region in Russia. In 1996–1997, he was visiting scientist in the Department of Psychiatry, Yale University School of Medicine in New Haven, Connecticut, United States. He has more than 150 publications dealing with the treatment of various forms of addictions and alcoholism.

Scot Lord, CATC, FACT, is a senior learning skills counselor at the University of California San Diego (UCSD) Center for Criminality and Addiction Research, Training and Application (CCARTA), in San Diego, California, United States. He is a certified addiction treatment specialist with over three years of frontline experience working with substance-addicted individuals. He has provided treatment services for the Mental Health Systems East County Drug Court program and was the lead counselor at a San Diego County Proposition 36 diversionary treatment program. Scot has extensive background and knowledge with evidence-based addiction treatment approaches, criminality, and addictive behaviors. Most recently, Scot was the lead trainer in

the CMC peer mentor training program in which he taught long-term prison inmates the therapeutic community treatment model, fundamentals of addiction science, and cross-trained correctional officers with prison inmates in order to develop an entirely inmate-driven therapeutic community.

Julian G. Martins, MA, MB, BS, qualified in medicine from the University of Cambridge, Cambridge, England, (BA [Hons], MA pharmacology) with first-class honors and from the Royal London Hospital (MBBS) in London, England, with honors in surgery and further prizes in medicine, surgery, and therapeutics. He obtained both the MRCP and MRCPsych and worked for many years as a university lecturer and consultant psychiatrist. He is currently the director of an independent academic research company, the Dr. Julian G. Martins Academy of Nutritional Medicine, Cambridge, England.

Scott E. McClure, PhD, is currently employed as a senior learning skills counselor with the University of California San Diego (UCSD), Department of Psychiatry, Center for Criminality and Addiction Research, Training, and Application (CCARTA) also in San Diego. At CCARTA, he provides technology transfer of the most current evidence-based treatment procedures for substance use disorders to substance use treatment staff throughout the state of California. Scott has over seven years of experience working with individuals who have co-occurring mental health and substance use issues across a broad scope of populations ranging from the severally mentally ill to university students. He has facilitated groups on posttraumatic stress disorder (Seeking Safety), adult attention deficit hyperactivity disorder, dialectical behavior therapy, as well as implemented motivational interviewing and life skills groups while employed at the UCSD Co-occurring Disorder Outpatient Clinic. Scott's clinical and research interests include criminality and addiction, trauma, ADHD, and multicultural issues.

Patrick McNamara, PhD, is an associate professor of neurology and psychiatry at Boston University School of Medicine in Boston, Massachusetts, United States. He is based at the Department of Neurology at the Veterans Administration New England Healthcare System. He has received U.S. Veterans Administration (VA) Merit Review Awards (2000–2003) there as well as two U.S. National Institute of Health (NIH) awards (current) to fund his research in neurology and evolutionary psychology. He is the cofounder (with Wesley Wildman) of the nonprofit research Institute for the Biocultural Study of Religion (www.ibcsr.org). The institute's most recent venture is the launching of a new scientific journal called *Religion, Brain and Behavior,* which will be devoted to publishing new studies in the field. He has authored several publications on religion and brain and related topics including addictions. In 2005–2006 he edited a three-volume series of books on evolutionary, cognitive, and neurological associations of religiousness (Praeger Press). Prior

to joining the VA's Neurology Department he taught experimental design, research methods, and neuropsychology at SUNY College Buffalo and Keene State College. He graduated with honors in psychology at Boston University in Massachusetts, United States, and received a PhD in behavioral neuroscience from Boston University in 1991.

Joseph R. Merighi, PhD, MSW, is an associate professor of human behavior at Boston University School of Social Work in Boston, Massachusetts, United States. Dr. Merighi is the Co-editor-in-Chief of the *Journal of Nephrology Social Work* and he serves as a consulting editor for *Social Work* and the *Journal of Social Work Education*. His research focuses on social work practice in health care settings, with an emphasis on renal social work, and cross-national comparisons of job-related well-being of social work practitioners. In addition to his research and teaching, Dr. Merighi has served as a consultant for organizations such as the National Kidney Foundation's Council of Nephrology Social Workers and United Cerebral Palsy. Dr. Merighi received his MSW and PhD from the School of Social Welfare at the University of California, Berkeley in Berkeley, California, United States.

Lorraine T. Midanik, PhD, is dean and professor in the School of Social Welfare at the University of California at Berkeley in Berkeley, California, United States. She received her PhD from the School of Public Health at the Johns Hopkins University in Baltimore, Maryland, United States. Following graduation, she was a postdoctoral fellow at the School of Public Health at U.C. Berkeley and a health evaluation analyst at Kaiser Permanente. Dr. Midanik has an extensive career in research and publication in the alcohol field beginning as a clinician, then as a researcher and teacher. Her work has focused primarily on the epidemiology of alcohol use and alcohol-related problems and on methodological issues—primarily in the area of self-report validity. In 2004, she received a Fulbright Fellowship to examine alcohol research policy at Stockholm University in Sweden. In 2006, she wrote *Biomedicalization of Alcohol Studies: Ideological Shifts and Institutional Challenges*, published by Aldine Transaction.

Karin J. Neufeld, MD, MPH, is assistant professor of psychiatry and behavioral sciences at Johns Hopkins University School of Medicine in Baltimore, Maryland, United States. She received her BS and MD from the University of Manitoba, Canada, completed postdoctoral psychiatry residency training at the Johns Hopkins University, School of Medicine, and an MPH at the Johns Hopkins School of Public Health. She is board certified by the American Board of Psychiatry and Neurology and a fellow of the Royal College of Physicians and Surgeons of Canada. Since 2002, she has been an associate medical director

of Addiction Treatment Services at Johns Hopkins Bayview Medical Center. Her research interests have focused on co-occurring psychiatric and medical disorders in patients with substance use and the ways in which appropriate care can be integrated. Currently she is developing innovative strategies for patients with severe and persistent drug use problems who are not responding to routine treatment.

Jessica Peirce, PhD, is assistant professor of psychiatry at Johns Hopkins University School of Medicine in Baltimore, Maryland, United States. She received a BA from University of Chicago in Chicago, Illinois, and a PhD in biological psychology from University of Oklahoma Health Science Center, Oaklahoma, United States, respecialized in clinical psychology at Oklahoma State University, and completed internship and postdoctoral training at the Boston VA Medical Center. She is a licensed psychologist and associate director of the Addiction Treatment Services program at Johns Hopkins Bayview Medical Center. Dr. Peirce's research and clinical interests are in gender differences and comorbidity of substance use with other psychiatric problems, particularly trauma and posttraumatic stress disorder.

Flavio Poldrugo, MD, PhD, received his MD at Trieste, Italy (1975), the specialization in psychiatry at Zagreb, Croatia, 1979, and the PhD in pharmacology in Birmingham, Alabama, United States, 1985. Dr. Polgrugo has held numerous academic positions, including: assistant professor of psychiatry (Trieste, 1975), visiting professor of neuroscience (Birmingham, 1980), associate professor of psychiatry (Trieste, 1985) and visiting professor of social psychiatry (Zagreb, 1992). His research interests span from preclinical research on alcoholism to prevention of alcohol problems, pharmacological and nonpharmacological treatment of alcoholism, and utilization and cost-effectiveness of alcohol treatment. Dr. Poldrugo has been very active in international relationships: he was a founding member of the Association of the Alcoholics under Treatment (1979), the Association for the Study and Treatment of Alcoholism in Europe (1991), the International Society of Addiction Medicine (1999), chairman of Section of the International Council on Alcoholism and Addictions (1995), board member of the International Society of Addiction Medicine Journals (2000), past president of the Addiction Section of the European Psychiatric Association (2006) and secretary of the same section in the World Psychiatric Association (2005). Dr. Poldrugo is deputy editor of the journal *Alcoholism: Journal on Alcoholism and Related Addictions* (1992) and serves in the scientific board of seven other international journals. He is author of more than 200 articles in international journals and 15 chapters of books.

Basant K. Puri, MA, PhD, MB, BChir, MRCPsych, MMath, of the Medical Research Council (MRC) Clinical Sciences Centre (CSC), Hammersmith Hospital and Imperial College London, England, received his primary and postgraduate degrees in medicine from the University of Cambridge, England, and carried out postdoctoral work in molecular genetics at the MRC and University of Cambridge, and in imaging at the Royal Postgraduate Medical School at Hammersmith Hospital, London. He also has a first-class honors degree in mathematics and postgraduate degrees in mathematics and is a member of the Royal College of Psychiatrists in the United Kingdom.

Kathleen Sciacca, MA, of Sciacca Comprehensive Service Development for Mental Illness, Drug Addiction and Alcoholism (MIDAA) in New York City, New York, United States and on the Web at http://users.erols.com/ksciacca/, has been a forerunner in the development of treatment methods and programs for mental illness, drug addiction, and alcoholism (MIDAA) since 1984, when there were no program models or attention given to this population. Her programs have been replicated across New York state, Alaska, Delaware, Georgia, Tennessee, North Carolina, Louisiana, Connecticut, Michigan, Minnesota, Washington, DC, Texas, Virginia, and other states, cities, counties, and agencies in the United States and internationally. She has lectured in colleges and universities; participated in national reports; authored the MIDAA service manual; produced video invited into the Library of Congress; and has been reviewed by *Time* magazine. She is an international consultant, lecturer, and trainer. She is former director of the MICAA Training Site for Program and Staff Development, statewide for the New York State Office of Mental Health (1984–1992). Ms. Sciacca is a trainer and consultant on motivational interviewing.

Ramadugu Shashikumar, MD, is a consultant psychiatrist based in Jallandhar, Punjab, India. He graduated from one of the most prestigious colleges of India, the Armed Forces Medical College Pune, in December 1991. After having done service obligations, he received an MD in psychiatry in 1999. He has been working on treatment and prevention of alcoholism at a community level since 1999. Apart from this he has worked in child psychiatry. He has been awarded the best paper award for the paper titled "Best Paper Presentation on Psychosocial Co-relates of Service Personnel's Children with Recurrent Abdominal Pain." He is a keen wildlife enthusiast. He has been instrumental in creating information booklets for use by general public and patients on subjects like alcoholism, substance abuse, and child psychology in multiple languages. He has authored a chapter on psychological aspects of a cold environment in a textbook on environmental emergencies published by the Department of Medicine of Armed Forces Medical College.

Daniela Sisneros, MSW, received her master's degree in social work, with an emphasis in administration, from San Diego State University, San Diego, United States. Ms. Sisneros has worked in nonprofit and academic settings for six years and is currently the center coordinator at the Center for Alcohol and Drug Studies and Services. The programs she has served on have specialized in: alcohol and drug prevention; child welfare–related research and program evaluation; consensus organizing; screening; brief intervention and referral to treatment services; and short-term psychiatric rehabilitation treatment. Ms. Sisneros has made presentations at scientific meetings and currently educates on substance abuse and cultural competency issues.

Katharine Slack, MSW, received a BS in psychology from Guilford College in Greensboro, North Carolina, United States, and an MSW from San Diego State University in San Diego, California, United States. Katie Slack currently works as a brief treatment counselor for the California Screening Brief Intervention, Referral and Treatment Services (CASBIRT) Project. Her work history includes nearly 10 years of experience in the social services field, including several years of case management and program management for an independent living skills program working with teenagers and young adults in foster care. She also worked for nearly three years doing community education and outreach on domestic violence and sexual assault. Additionally, she held a one-year internship placement offering individual therapy for persistently mentally ill adults, as well as an additional one-year internship placement conducting individual, family, and child therapy with survivors of domestic violence and sexual assault.

Ray B. Smith, PhD, received his doctorate at the University of Texas in Austin, Texas, United States, in physiological psychology in 1976. Since then he has taught at the State University of New York at Cortland, the University of Virginia Graduate School, the Washington Theological Coalition, and has served as adjunct advisor for doctoral dissertations at several universities, including the University of Michigan. Since 1972 he has researched CES in major addiction treatment programs in Washington, DC, Dallas/Ft. Worth, Santa Barbara, Los Angeles, Seattle, and St. Louis. After serving as research consultant on CES at the veterans hospital in Houston, Texas, he presently serves as a consultant based in Forestville, Maryland, United States, as research advisor to a CES research project in a field army hospital in Iraq, and as a member of a scientific board of a major pharmaceutical company. He has presented papers at national and international conferences on CES, and in 2007 published a research monograph on CES. He presently serves as a private research consultant for research projects around the nation.

Kenneth B. Stoller, MD, is an assistant professor of psychiatry and behavioral sciences at the Johns Hopkins University School of Medicine in Baltimore, Maryland, United States. He received his medical degree from Johns Hopkins in 1992. He completed his residency in psychiatry at the Johns Hopkins Hospital, followed by a two-year postdoctoral fellowship in addiction psychiatry at the Behavioral Pharmacology Research Unit at Johns Hopkins. He then joined the faculty group at Addiction Treatment Services at Hopkins Bayview, where he is an ABPN-certified psychiatrist and associate medical director. Dr. Stoller's research interests center on cost issues as they relate to drug abuse and treatment, methods of enhancing treatment adherence, managed care organization approaches to members with drug abuse, and co-occurring psychiatric, medical, and substance use disorders.

Mary Theresa Webb, PhD, is the founder and past Director of the Global Outreach for Addiction Leadership and Learning (GOAL) Project based in Aliquippa, Pennsylvania. She was one of the founders of the International Substance Abuse and Addiction Coalition (ISAAC), based in the UK, and the OPORA Training Center in Moscow, Russia. (*Opora* is the Russian word for *support*. Moscow-based nongovernmental OPORA, which works with the Russian government to fight addiction, has trained more than 3,000 persons in addiction and 12 Step programs, and implemented 60 recovery groups in 31 cities in Russia.) As a global educator, she has personally and with others facilitated addiction trainings in the United States, Russia, Romania, Tajikistan, Bulgaria, Kenya, Tanzania, Honduras, and Egypt. Currently, Dr. Webb is the Project Director of the Kenya-based, USAIDS-funded Substance Abuse Rehabilitation and HIV/AIDS (SARAH) Network in Kenya. Dr. Webb holds a master's in Education from the University of Pittsburgh and a Doctor of Philosophy Degree in Family Systems/Addiction Intervention from the University of America. She is the author of *Tree of Renewed Life, Church Training Manual*, and coauthor of *Codependence in Family Systems*.

Michael Winkelman, MPH, PhD (University of California-Irvine), MPH (University of Arizona), is an associate professor at Arizona State University in Tempe, Arizona, United States. He served as president of the Anthropology of Consciousness Section of the American Anthropological Association and was the founding president of its Anthropology of Religion Section. Winkelman has engaged in cross-cultural and interdisciplinary research on shamanism, focusing principally on the cross-cultural patterns of shamanism and identifying the associated biological bases of shamanic universals and altered states of consciousness. His principal publications on shamanism include *Shamans, Priests and Witches* (1992) and *Shamanism: The Neural Ecology of Consciousness*

and Healing (2000). He has also addressed the role of psychedelic medicines in shamanism, developing neurophenomenological perspectives that link the experiences to the underlying physiological processes (*Sacred Plants, Consciousness and Healing* [1996] and *Psychedelic Medicine* [2008]). He has also explored the applications of shamanism to contemporary health problems of addiction.

Susan I. Woodruff, PhD, is a professor at San Diego State University School of Social Work in San Diego, California, United States. She is an epidemiologist with over 20 years of experience in the area of health research, much of it with underserved, high need, and understudied populations. She has a master's degree in experimental psychology and a doctorate in epidemiology from the University of California San Diego/San Diego State University both in San Diego, California. Her areas of expertise include substance use epidemiology and intervention, adolescent health, program evaluation (i.e., pre-post change analysis), Latino health promotion, measurement/methodological issues, research design, and univariate/multivariate statistical data analysis. Dr. Woodruff has served as investigator, statistician, and data analyst on several large epidemiological and intervention studies, and has authored and coauthored over 150 articles in scientific journals. Currently she is the evaluator on the California Screening Brief Intervention, Referral and Treatment Services (CASBIRT) project.

M. Scott Young, PhD, received an MS in management information systems, an MA in clinical psychology, and a PhD in experimental psychopathology from University of South Florida in Florida, United States. He works as coordinator of statistical research in the Department of Mental Health Law and Policy at the Louis de la Parte Florida Mental Health Institute, University of South Florida in Tampa, Florida, United States. His research currently focuses on substance abuse treatment program evaluation, alternatives to incarceration for nonviolent drug offenders, and coerced substance abuse treatment. He has published a number of articles and book chapters on substance abuse and mental health program evaluation, and he has received grant and contract funding to analyze mental health and substance abuse treatment provider administrative data sets.

The Praeger International Collection on Addictions

Recent Titles in
Abnormal Psychology

Mental Disorders of the New Millennium, Volumes 1–3
Thomas G. Plante, editor

Bleeding to Ease the Pain: Cutting, Self-Injury, and the Adolescent
Lori G. Plante

Understanding and Treating Depression: Ways to Find Hope and Help
Rudy Nydegger

THE PRAEGER INTERNATIONAL COLLECTION ON ADDICTIONS

Volume 4
Behavioral Addictions from Concept to Compulsion

Edited by Angela Browne-Miller

Praeger Perspectives

Abnormal Psychology

Thomas G. Plante, Series Editor

PRAEGER

Westport, Connecticut
London

Library of Congress Cataloging-in-Publication Data

The Praeger international collection on addictions / edited by Angela Browne-Miller.
v. cm. — (Abnormal psychology, ISSN 1554–2238)
Includes index.
Contents: v. 1. Faces of addiction, then and now—v. 2. Psychobiological profiles—v. 3. Characteristics and treatment perspectives—v. 4. Behavioral addictions from concept to compulsion.
ISBN 978–0–275–99605–5 (set : alk. paper)—ISBN 978–0–275–99607–9 (v.1 : alk. paper)—ISBN 978–0–275–99609–3 (v.2 : alk. paper)—ISBN 978–0–275–99611–6 (v.3 : alk. paper)—ISBN 978–0–275–99613–0 (v.4 : alk. paper) 1. Substance abuse. 2. Alcoholism. 3. Compulsive behavior. 4. Addicts. I. Browne Miller, Angela, 1952–
HV4998.P72 2009
616.86—dc22 2008051468

British Library Cataloguing in Publication Data is available.

Library of Congress Catalog Card Number: 2008051468
ISBN: 978–0–275–99605–5 (set)
978–0–275–99607–9 (Vol. 1)
978–0–275–99609–3 (Vol. 2)
978–0–275–99611–6 (Vol. 3)
978–0–275–99613–0 (Vol. 4)
ISSN: 1554–2238

First published in 2009

Praeger Publishers, 88 Post Road West, Westport, CT 06881
An imprint of Greenwood Publishing Group, Inc.
www.praeger.com

Printed in the United States of America

The paper used in this book complies with the Permanent Paper Standard issued by the National Information Standards Organization (Z39.48–1984).

10 9 8 7 6 5 4 3 2 1

Contents

This page intentionally left blank

Series Foreword

Tragically, most people across the globe have either struggled with a health- and relationship-damaging addiction or know someone who has. Addictions, broadly defined, have touched the lives of the majority of people in multiple cultures and locations. For centuries, numerous people have suffered with their addictions to alcohol and drugs as well as with other addictions, with often devastating outcomes. Sadly, important relationships, jobs and careers, and many lives have been lost due to the destructive power of addiction. These tragedies not only occur for those who suffer from addiction, but for their loved ones, coworkers, and community members, and for innocent victims who are perhaps in the wrong place at the wrong time when an addiction-related accident, crime, or violence occurs. The enormous cost of addiction in health care, traffic accidents, crime, violence, loss of workplace productivity, and broken families is too large to quantify. The global spread and success of organizations such as Alcoholics Anonymous (as well as related organizations such as Narcotics Anonymous, Sexoholics Anonymous, and Overeaters Anonymous) is a testament to the numerous people trying to recover from their addictions. Sadly, for every person seeking help for his addiction problem, there are likely to be many more people who never do. Clearly we need help to better understand, evaluate, treat, and cope with those who suffer from addictions.

In this remarkable four-volume set, *The Praeger International Collection on Addictions,* Angela Browne-Miller, PhD, DSW, MPH, has assembled an all-star and diverse team of leading experts from across the globe to provide a state-of-the-art understanding of the various facets of addiction. Each chapter

is written in a manner that is suitable for professionals working in the field as well as educated lay readers and those who either struggle with addictions or live or work with someone who does. What is especially remarkable about the four-volume set is its emphasis on addiction from around the globe, examining multicultural and international issues in addiction, as well as its coverage of so many multifaceted aspects of diverse addictions. For example, it certainly makes sense to cover fully addiction topics such as alcohol abuse and illegal drug use of, say, cocaine and heroin, yet chapters are also offered that examine addictions to caffeine, Internet pornography, work, television, intimate relationship abuse, and shopping. The chapters highlight biological, psychological, social, spiritual, and public health perspectives, with chapter authors who are psychologists, psychiatrists, other physicians, nurses, social workers, counselors, clergy, and other professionals. Dr. Browne-Miller is uniquely qualified to assemble this project as she is someone who has worked in the field of addiction for many years and has training in a unique blend of both the policy and the clinical sides of psychology, social work, education and public health.

The set is complete, state of the art, and highly informative and engaging. There is something for everyone interested in the field of addiction for professional or personal reasons. It is hoped that professionals and lay readers will greatly benefit from this important work and, in doing so, will find a way to improve the lives of those touched by addiction. It is my hope that both research and practice in the field of addiction will be greatly improved thanks to this set. The lives of those who either struggle with addiction or live with those who do will ultimately be improved thanks in part to this critical series. I am grateful to Dr. Browne-Miller and her assembled contributors for providing us all with such important and high-quality volumes that are now available to the public and professional communities. If only one life is saved or improved thanks to this set, it will be a great success in my view; yet I expect that many lives will ultimately be saved or greatly improved thanks to *The Praeger International Collection on Addictions*.

Thomas G. Plante, PhD, ABPP
Santa Clara University and Stanford University School of Medicine,
Praeger Series Editor, Abnormal Psychology

Preface

Angela Browne-Miller, PhD, DSW, MPH

Welcome to *The Praeger International Collection on Addictions,* addressing the insidious, pervasive, worldwide problem of human addiction. Addiction is clearly a global issue, touching every population, every nation, and every age group, people from all walks of life everywhere, directly or indirectly. Indeed, we are talking about an affliction of epic and epidemic proportions. We cannot look away. This is the health of the human species we are talking about.

The World Health Organization (WHO, 2008, p. 1) reports that "psychoactive substance use poses a significant threat to the health, social and economic fabric of families, communities and nations. The extent of worldwide psychoactive substance use is estimated at 2 billion alcohol users, 1.3 billion smokers and 185 million drug users" (p. 1). The WHO has estimated there to be at least 76.3 million persons with alcohol use disorders worldwide, and at least 15.3 million persons with drug use disorders worldwide. Alcohol use and abuse as well as the use and abuse of other psychoactive substances contributes to substantial individual and public health costs. Alcohol is but one substance playing a major role in this global addiction epidemic, but clearly there are many others, despite efforts to prevent new addictions and addicts, and to contain world drug markets (UN, 2008, p. 7).

For example, cocaine shares the stage with other abused drugs. Its prevalence is estimated to be up to 3 percent of the population in developing countries, with severe medical, psychological, social, and economic consequences including, but not limited to, the spread of infectious diseases (e.g., AIDS, hepatitis, and tuberculosis), plus crime, violence, and neonatal drug exposure.

Amphetamine-type stimulant (ATP) abuse is more widespread than cocaine abuse in at least 20 countries. Methamphetamine is presumed to lead in ATP addiction rates, with massive meth epidemics affecting several whole countries and entire regions of others. Social and public health costs of methamphetamine production and use via smoking, sniffing, inhaling, and injecting are staggering and growing in many regions. Additionally, there has been a global increase in the production, transportation, and use of opioids, especially heroin, with worldwide heroin production doubling or even tripling since the mid-1980s. Global estimates are that 13.5 million persons consume opioids, with 9.5 million of these being heroin users who face health risks including hepatitis, HIV, and death. And cocaine, meth, and heroin are just one piece of the picture.

The hotly debated drug cannabis—or the *Cannabis* family of drugs with the euphoric tetrahydrocannabinols, or THCs, including marijuana and hashish preparations—is said to be the most widely abused drug. Research is now suggesting the risk for acute health effects of long-term, chronic cannabis use, including potential impairment of cognitive development, learning, memory, recall, attention, and coordination. (Certainly the presence and extent of long-term effects of casual, of regular and of chronic use are as yet not entirely ascertained.) Both casual use of marijuana and medical use of forms of what is termed medical marijuana (e.g., dronabinol sold as Marinol, the cannabidiols, or CBDs) are subsets of all forms of cannabis use. There are legitimate therapeutic uses of this substance, and these uses make it all the more difficult to regulate marijuana drugs fairly and effectively.

We have here, and in the use of any psychoactive medication for therapeutic purposes, a gray area in which illicit and licit use overlap and can confuse many adult and youth consumers, researchers, and policy makers, among others. In the emergence (or reemergence in history, some will argue) of cannabis as medicine, we have a model for asking which, if any, abused substances may, and perhaps should, be repurposed for medicinal or treatment purposes, and how this is best done against the backdrop of the global addiction epidemic.

Regarding marijuana, we are confronted with the age-at-first-use issue, which suggests that early onset of regular cannabis use may affect not only the academic and social performance of children and teens, but also their future susceptibility to addictions. It was in the 1960s that the hotly debated label "gateway drug" was applied to marijuana, perhaps to scare off its use, and only in the decades since have we understood better what this might actually mean to us. It may not be that marijuana provides the training wheels for drug addiction, but rather that it may serve as an indicator of future use of the same or other drugs. Of course, today, with so many young people having access, and taking advantage of their access, to the whole range of psychoactive substances,

the question of which drug might be a gateway to which other drug dissolves into the fury of the countless addiction conundrums of our constantly changing times.

There is always a new, or rediscovery of an old, addiction on the horizon. There is also always a new (or rediscovered) psychoactive substance for exploratory, research, and perhaps even treatment purposes emerging (or re-emerging). Labeling all of these substances as addictive right out the gate may or may not serve science or even humanity itself. How can we be certain the approach we take will be a constructive one? With new legal (where licensed for development and experimentation) and illegal (where not being utilized under protection of law) so-called designer drugs emerging at a staggering rate, we must admit that we cannot know what is coming, nor whether the new compound will be addictive, or popular, or of medicinal value, or even accessible. We can only imagine what the brave new world of chemistry will continue to bring and whether any benefits can be made available without accompanying risks and detriments.

Moreover, the desire to explore and achieve various altered states of consciousness in religious, spiritual, ritual, and perhaps even treatment settings, is unfolding into debates about rights (Browne-Miller, 1989, pp. 258–260). When there is no demonstrated risk to self or others, we have to ask ourselves whether this right should be protected, especially in circumstances of traditional uses for religious purposes. Again, this dilemma arises against the backdrop of the global and runaway epidemic of substance addiction. How do we balance pressures from opposite directions (freedom protecting the right to use versus control to stop injury and costs of using), when these pressures are not balancing themselves?

Also against the backdrop of global addiction levels, is the massive level of addiction to legal drugs, many of which are heavily marketed to consumers. The legal drug tobacco is said to be the substance causing the most damage globally, with at least one-third of the global population smoking. While smoking rates may be dropping in some countries, the reverse is true globally. As just one of its effects, smoking accounts for some 90 percent of all lung cancer in men and 70 percent of all lung cancer in women. And yet tobacco use is overwhelmingly viewed as being "the single most avoidable cause of disease, disability and death" in the United States (CDC, 2008, p. 2).

And perhaps nothing here has touched so many lives as the regularly consumed, legal drug caffeine, perhaps because coffee drinking is considered so very normal and acceptable, even necessary, in everyday life. However, we must ask whether there is a level of caffeine use that is abuse—or perhaps self-abuse. Surely we do not want to throw caffeine use onto this list of substance abuses

and addictions. Still, a collection on addiction would not be complete without at least touching on this matter, and therefore we do address caffeine herein.

And then there are also the addictions to prescription drugs (such as Vicodin, Percocet, OxyContin, and Darvon), which we find increasing rapidly and already a worldwide phenomenon, with the most commonly abused prescription drugs being opiates. The U.S. National Institute of Mental Health characterizes prescription drug addiction as the second most common illegal use of drugs in the United States, second only to marijuana.

We must also note that unusual, virtually invisible psychoactive substances are working their way into our everyday lives. Household and workplace products contain many volatile substances, exposure to which can be not only damaging, but also intoxicating, and perhaps addicting. Although this domain of substance use and abuse is not specifically addressed herein, we must acknowledge the severe and perhaps largely unmeasured effects of this domain of even routine, legal substance use as well as unintentional and intentional abuse.

So as not to exclude nonsubstance addictions in this overview of addiction today, the fourth volume in this collection on addiction reminds us that work, television, shopping, food (with its particularly difficult-to-call-addiction nature), intimate partner relationship, gambling, Internet, and even pornography addictions make their marks in our lives, either indirectly or directly. These behavioral, nondrug addictions, which occur alone and co-occur with each other, also do co-occur with substance uses, abuses, and addictions. Every human being is in some way affected by the prevalence of behavioral addictions, either directly or indirectly. The study of behavioral addictions teaches us a great deal about addiction itself.

All this suggests the picture of an addiction-prone and largely chemically dependent human species. And this is just the tip of the iceberg. With this truly incomplete laundry list of human fallibilities—or better stated, perhaps, human *vulnerabilities*—this four-volume collection on addiction is truly that: a collection of perspectives, approaches, and findings. Each chapter is a snapshot of the work and thinking taking place in many fields of addiction. Contributors to this collection work with addiction on the various social, philosophical, psychological, spiritual, policy, political, economic, biological, and even cellular levels, all places where this thing we call "addiction" lives. Certainly this collection would have to comprise hundreds of volumes, rather than the four that it does, to address addiction in all its iterations.

Here we give voice to a diverse cross section of perspectives on addiction. This is in no way an exhaustive cross section (of either perspectives or addictions); rather, this collection suggests the diversity of perspectives, theories, practices, and types of addiction in the field—or better stated, *fields*—of addiction. The four volumes of this collection represent the voices of those who have

graciously and even bravely stepped forward from their numerous countries and arenas of work to contribute their ideas, research, and experiences. Certainly there are many others out there, many other aspects of addiction, and many other drugs and objects of addiction not addressed in these volumes.

This work is divided into four volumes, with the first three addressing addictions to substances and the fourth addressing behaviors that show characteristics of addiction. Volume 1, *Faces of Addiction, Then and Now,* offers a sampling of the depth and breadth of addiction today and in the past; volume 2, *Psychobiological Profiles,* surveys some of the interlinked psychological and biological aspects of addiction; volume 3, *Characteristics and Treatment Perspectives,* samples the range of addiction treatment perspectives and approaches; and volume 4, *Behavioral Addictions from Concept to Compulsion,* gives the reader a glimpse of behavioral addictions other than substance addictions.

Readers will observe that the content of these volumes is indeed diverse and in no way represents any one view or theory of addiction. There are many other voices out there who must also be heard, and only in the interest of time and space are we stopping here, at these volumes. The content of these volumes in no way expresses the opinion of this editor, nor of this publisher, regarding what is right, best proven, or even most en vogue in the addiction world; rather, this *International Collection on Addictions* seeks a display of, a sampling of, the diversity of effort to quell the detrimental effects of addiction on individuals, families, communities, societies, economies, and international relations; on ecologies; and in fact, on the human population of planet Earth.

REFERENCES

Browne-Miller, Angela. (2008, reprint of 1989). Synaptic rights. In *Working dazed: Why drugs pervade the workplace and what can be done about it* (pp. 258–260). Houston, Texas: Questia Media. Also available at http://www.questia.com/library/book/working-dazed-why-drugs-pervade-the-workplace-and-what-can-be-done-about-it-by-angela-browne-miller.jsp

Centers for Disease Control (CDC). (2008). *Targeting tobacco use: The nation's leading cause of preventable death* (March 2008 revision). Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.

United Nations (UN). (2008). 2008 world drug report. UNODC (United Nations Office on Drug Control), Vienna, Austria: Vienna International Centre, Full report in English available at http://www.unodc.org/documents/wdr/WDR_2008/WDR_2008_eng_web.pdf

World Health Organization, Management of Substance Abuse, Facts and Figures. (2008). "The Global Burden," 2008, (Accessed 9-01-08), p. 1 http://www.who.int/substance_abuse/facts/global_burden/en/index.html. **See Also:** http://www.who.int/substance_abuse/facts/en/index.html

This page intentionally left blank

Introduction to Volume 4: Behavioral Addictions from Concept to Compulsion

Angela Browne-Miller, PhD, DSW, MPH

This is volume 4, *Behavioral Addictions from Concept to Compulsion*, of *The Praeger International Collection on Additions*, where we see among the many faces of addiction the nonsubstance addictions. Here we move into the tangled territory of evolving and expanding definition. We can ask whether nondrug addictions are actually addictions. If so, why? If not, why not? We can argue all sides of this matter. Yet, where there is continued engagement, excessive engagement, in the face of harm to self or others, in an activity generating temporary and too often lasting shifts, changes, adaptations, even damages—on social, psychological, biological, even biochemical levels—we encounter *addiction-like* behavior that, for all intents and purposes, is indeed addiction. Certainly we may label these as *non*substance addictions, yet we are in so many ways talking about drug-like effects where there is *excessive engagement in* working, television viewing, shopping, intimate partner abusing, food consuming, as well as in pornography, Internet, and gaming and gambling activities. As our contributors will show, engagement in these activities to excess shows symptoms of addiction such as trigger-response, urge-craving, reinforcement, tolerance, and withdrawal.

Part I, "Compulsions," opens with a penetrating look at something to which we may all be vulnerable, that is, work addiction. Ronald J. Burke, PhD, of the Schulich School of Business, York University, in Toronto, Ontario, Canada, contributes chapter 1, "Work Addiction: Causes, Consequences, and Choices." Working hard and working long hours are highly respected activities signifying (at least to onlookers) dedication and commitment. Yet, working hard—

excessive working—may be an addiction to working or workaholism. While employers may even reward workaholism, workaholics may or may not be productive. Symptoms of workaholism have been seen as parallel to those of other addictions, with workaholics often being unhappy, obsessed, exhibiting a reluctance to disengage from work (e.g., a tendency to work, to think about work, and to talk about work far more than others do), with willingness to work anytime and anywhere, to choose work over leisure, to lose sight of boundaries between work and the rest of life. Burke reviews definitions of workaholism, the term itself first being used in 1971, when a workaholic was defined as "a person whose need for work has become so excessive that it creates noticeable disturbance or interference with his bodily health, personal happiness, and interpersonal relationships, and with his smooth social functioning." The workaholic gradually becomes "emotionally crippled and addicted" as this "progressive, potentially fatal disorder" consumes him or her. Burke encourages employers to assist in the development of workplace values and to promote healthy lifestyles among employees, which will support the "workaholism types" in addressing their addiction. However, intervention in a workaholic pattern may be quite challenging because "work addicts almost always are in denial." We might add that their employers may be as well.

Chapter 2, "Addiction to Television: With Commentary on Dependence on Video Games and the Internet," by Robert W. Kubey, PhD, of the Center For Media Studies at Rutgers University in New Brunswick, New Jersey, United States, adds another face to behavioral addiction. Kubey notes that "The time people spend viewing is nearly astonishing. People throughout the industrialized world, from the United States and Latin America to Europe and parts of Asia, the Middle East, and Africa, typically devote about three hours a day to watching television. In many societies, this easily constitutes half of all a person's leisure time or, calculated another way, 9 full years of a 75-year lifespan." Kubey adds that "the three hour figure *is* an average. . . . 'addicted' or so-called 'heavy viewers' . . . might watch five or six hours a day . . . about 30% to perhaps 38% of their waking time watching TV, or as many as 20 years of the 50 years they will be awake if they live to 75." That television viewing can indeed be self-perpetuating, and can indeed produce a dependency, is of grave concern. Addiction to television can produce effects such as aggression, obesity, poorer academic performance, and impaired imagination. Kubey does not decry all television viewing, as there are significant cultural, educational, and entertaining programs. "Television also surely provides much needed distraction and escape. Still, "viewing begets more viewing" and "one must generally keep watching in order to keep feeling relaxed." Note that where television is used for relaxation,

the "quick onset of relaxation is particularly telling when compared to that produced by certain drugs that are known to be habit forming or 'addictive.'"

Chapter 3, "Excessive Buying as a Genuine Addictive Behavior" by Paul Rose, PhD, and Dan J. Segrist, PhD, of Southern Illinois University, Edwardsville, Illinois, United States, turns to "hyperconsumption." Here the case for "classifying excessive buying as a genuine behavioral addiction" is presented. The benefit of such a classification is a better understanding of, and therefore treatment of, this condition as an addiction. Rose and Segrist define excessive buying through a review of the literatures on compulsive, impulsive, and nonfrugal purchasing. Only in the past few decades has research begun to establish a demographic of this worldwide problem—shopping addiction. Excessive buyers are more likely to experience feelings of depression after shopping, rather than pleasure after a new purchase. The emotional pathway is mapped something like this: the individual may be driven to shop for any of a variety of reasons, then shop excessively, and then feel out of control, then afterward experience guilt and shame, negative self-views, and depression. Shopping exposes the excessive buyer to numerous mental health problems. The sensation of "diminished impulse control" is seen as parallel to that found in other addictions. Preceding excessive buying is any number of states of mind including low self-esteem and narcissism—likely serving as classical triggers.

Offering another look at compulsive buying, we shift in chapter 4 to perspectives from another part of the globe, exemplifying the universality of the conditions we read about in this collection on addictions. "Compulsive Buying Disorder" is contributed by Mamta Sood, MD, and Meera Vaswani, PhD, both of the All India Institute of Medical Sciences in New Delhi, Delhi, India. Sood and Vaswani accept that buying is a "universal, everyday human experience and has been practiced since ancient times." As we have progressed through history, our awareness of, and labeling of, a buying disorder—the *compulsive* buying disorder—have emerged. As long ago as the early twentieth century, terms such as *oniomania* (for sale—mania) have focused on impulsiveness in shopping. Compulsive buying has been conceptualized as various disorders including impulse, obsessive-compulsive, mood, and substance abuse disorders. Sood and Vaswani describe the "increasing sense of arousal or tension prior to the act and an experience of pleasure, gratification, or release of tension at the time of committing the act." Sood and Vaswani also note that, while they find no formal guidelines being emphasized, there are psychotherapeutic, pharmacological, and other treatments available. Long-term outcome studies and the establishment of the reliability and validity of diagnostic criteria are called for.

At this point, we pause to consider which of the compulsive and addictive behaviors are indeed criminal and which of these are mistaken for criminal behaviors. To this end, chapter 5, "Association with Criminality of Habit and Impulse-Control Disorders," is contributed by Ian H. Treasaden, MB, LRCP, MRCS, FRCPsych, LLM, of the Three Bridges Medium Secure Unit, West London Mental Health NHS Trust, Middlesex, England, and Basant K. Puri, MA, PhD, MB, MRCPsych, MMath, of the Medical Research Council (MRC) Clinical Sciences Centre, Hammersmith Hospital and Imperial College, London, England. Treasaden and Puri emphasize that "Impulse-control disorders are a disparate group of conditions with different characteristics and epidemiologies." The authors establish the parameters of the debate, or better stated, perhaps, of the confusion, surrounding impulse control disorders: "whether the urges and impulses and resulting criminality are irresistible is open to question. Perhaps no impulse is irresistible, if an individual is motivated to try hard enough to resist." If an impulse is generally controlled and then not controlled in an instance of "momentary excitement," or heightened stimulation (triggering), the question may be as follows: is this a "disordered function" or merely an "irresistible impulse?" Treasaden and Puri note that the psychopathologies of the many conditions included in habit and impulse-control disorders are not identical. "Pathological gambling is a more complex condition, requiring attention to the whole person, than an impulse-control disorder such as tricotillomania. A pathological gambler shows features akin to substance addiction, with characteristic histories of escalation from use, abuse, and then addiction with tolerance and withdrawal symptoms, with gambling becoming the center of a sufferer's life, unlike . . . pyromania or tricotillomania."

Surely at this point numerous other detrimental habitual behaviors come to mind. The next chapter, chapter 6, "Troubled IPR Addiction: Habitual Attraction, Abuse, and Violence in Intimate Partner Relationships," which I, Angela Browne-Miller, PhD, DSW, MPH, contribute here, addresses an addiction-like behavior that may be seen for political, philosophical, and perhaps even emotional reasons as being in the "gray zone" of unproven and debated addictions. The existence of some degree of intimate partner relationship (what I call IPR) addiction is virtually a given, in that even emotional and sexual attractions can, and do, in their extremes bear habitual, even compulsive and disturbingly addictive, components. After all, if excessive working, television viewing, and shopping can be described as addictions or addiction-like behaviors, we must look at other excesses in which large portions of our species might engage. These might be emotional, sexual, or otherwise violent, cruel, or excessive extremes. Most troubling perhaps is the reality that interpersonal violence, when between two intimate partners, may exhibit addiction-like

characteristics. As I note in chapter 6, "In fact, the intimate partner relationship (IPR) itself—or its dynamics—can become habitual, can under certain circumstances turn not only sour but detrimental to one or both partners, the family around them including child witnesses, the surrounding community including the workplace, and beyond to the economy, in the form of not only lost productivity and lost work days but also ongoing . . . mental and physical health care costs. And, at its extreme, one possible outcome of a troubled IPR is that of intimate partner violence (IPV), which can be quite dangerous, even lethal." That the various emotional and physical abuses and violences found under the umbrella of intimate partner violence can be addictive is perhaps obvious and at the same time controversial. I emphasize that this is in no way what is called a "blame the victim" approach to intimate partner violence. Still, we are best able to assist persons (persons being abused and persons doing the abusing) at risk for being caught in, or stuck in, or returning to, detrimental and even dangerous patterns when we can tell it like it is: these are patterns that one might be at risk of "getting hooked on." Add in any form of co-occurring addiction (such as substance abuse), and both IPR and IPV addiction can be magnified exponentially.

Part II, "Eating as a Special Issue," is set apart from the other chapters in this volume, as eating "disorders," "compulsions," and "habits" are so variable in etiology and in interpretation when it comes to "addiction" itself. For what is and is not a medical and or psychological condition, while often the central question in the fields of addictions, is key to deciphering eating extremes, whatever these may be. Thus, we have chapter 7, "Craving Pizza? This Is Your Brain on Drugs: Eating Disorders as Addiction," written by Amanda Ruiz, MD, of the University of San Francisco, San Francisco, California, United States; Hugo Barrera, MD, and Norman Jackson, MS, both of the Center for Criminality and Addiction Research, Training and Application (CCARTA) in San Diego, California, United States. Ruiz, Barrera, and Jackson look at the areas of the brain that are "involved in this addictive process," to better establish eating disorders as addictions. Opening with a review of the various types of eating disorders, the authors demonstrate that "Multiple similarities exist between the neurotransmitters that regulate eating disorders and addiction." Eating disorders are bio-psycho-social conditions. Where the genetic explanation with regard to the human obesity gene appears to account for some 20 percent of variation in the basal metabolic index (BMI) in young women (for example), this may leave 80 percent not explained by this gene.

We then turn to what Cynthia Kalodner, PhD, at Towson University in Baltimore, Maryland, United States, has to say on this matter of eating disorders and addictions in chapter 8, "Eating Disorders and Disturbances: The

Continuum of Eating Disturbances." Kalodner discusses eating "disturbances" as a *continuum of eating behavior*, with normal eating at one end, eating disturbances in the middle, and eating disorders at the far end. Dieting, binge eating, and forms of purging can occur in the middle of this continuum and of course at the disorder end. Certainly, not every behavior taking place along this continuum is habitual, compulsive, or addictive; and Kalodner does not make the case that any or all of these are. Yet, within the context of the chapters in this and the other volumes of this collection on addiction, we cannot help but see that there are parallel patterns.

From here, we move into Part III of this volume, "Pornography, Internet, Gaming, and Gambling," which opens with chapter 9, "Addiction to Pornography and Its Psychological and Behavioral Implications," contributed by Robert Kubey, PhD, who, as noted earlier, is at the Center for Media Studies, Rutgers University, New Brunswick, New Jersey, United States. Pornography is, Kubey tells us, a phenomenon often said to be addictive, both in its print media forms and in its video and newer "interactive erotic" forms. Pornography is virtually all around us and is worldwide. Adults and children everywhere are exposed to pornography either directly or indirectly. Certainly, not all uses of pornography are excessive. However, sexual media can be addicting; they can be used excessively and in the face of harm to self or others. Yet, the "harm" quotient is perhaps most hotly debated. Note that when pornography addiction co-occurs with Internet addiction, the harm can be magnified exponentially.

Chapter 10, "Assessment and Treatment of Internet Addiction," is contributed by Kimberly S. Young, PhD, of St. Bonaventure University's Center for Internet Addiction Recovery, in St. Bonaventure, New York, United States. Young delineates the troubled category of Internet addiction: "In contrast to chemical dependency, the Internet offers several direct benefits as a technological advancement in our society . . ." The Internet differs from chemical dependence as "chemical dependence is not an integral part of our professional lives, nor does it offer any direct benefit." The tug of war over the true meaning of addiction surfaces again here: "many researchers argue that the term *addiction* should be applied only to cases involving the ingestion of a drug." However, "pathological Internet use" is continued use in the face of harm. It can cause addicts to hurt or lose significant real-life relationships, to feel worthless and unlovable; plus it can occur with depression, obsessive-compulsion, and likely even substance abuse. Cyberpsychology has its work cut out for it.

Next we move to chapter 11, "Gambling Addictions," by Mark Griffiths, PhD, of the International Gaming Research Unit, Nottingham Trent University, Nottingham, England. Griffiths tells us that "the majority of people have gambled at some time in their life" and that "The introduction of national lotteries

and new casinos, the proliferation of electronic gaming machines, and the introduction of remote gambling (e.g., Internet gambling, mobile phone gambling, interactive television gambling), has greatly increased the accessibility and popularity of gambling all over the world." And certainly impulses to gamble are up as a result of growing opportunities to gamble. Gambling behavior and "maladaptive gambling" are complex, "a multifaceted rather than a unitary phenomenon." Excessive gambling is harmful to self or others. These symptoms manifest (in some mix) as follows: being chronically and progressively unable to resist impulses (to gamble); compromising and disrupting one's life and the lives of those around one to support gambling; being arrested for any number of crimes including forgery and fraud to obtain money for gambling; disrupting family and significant other relationships due to gambling; borrowing money from illegal sources; and more.

Adding to this investigation of gambling and Internet addictions, is chapter 12, "Youth Gambling Problems: An International Perspective," by Isabelle D. Lussier, MA, Jeffrey L. Derevensky, PhD, and Rina Gupta, PhD, all at the International Centre for Youth Gambling Problems and High-Risk Behaviors, McGill University, Montreal, Quebec, Canada. "Recent reviews suggest that upwards of two-thirds of underage North American youth have gambled in regulated and licensed gambling venues . . . with adolescents having been reported to have pathological gambling prevalence rates two to four times those of adults." The problem of gambling too often begins in the latency age. Moreover, "excessive gambling among adolescents has been shown to be positively correlated with participation in increased delinquency and criminal behaviors, substance use, and antisocial behaviors. The serious nature of gambling problems is especially disconcerting considering that gambling is perceived to be a highly socially acceptable activity among adults and adolescents, with little recognition of the inherent risks."

The four volumes of this *Praeger International Collection on Addictions* has looked at the many faces of addiction. The authors have considered addiction as a moral problem, a social problem, a medical problem—akin to an allergy, a brain disease, and more. We have also discussed the marketing of alcohol and other drugs as an example of great business interest in addicting consumers. Here, in this part of volume 4, we are looking at addiction not only as a business but as a huge *industry*—the gambling industry. Gambling is so internationally pervasive that we have dedicated a significant portion of this volume to it. The above chapter is therefore followed by three chapters, providing examples of the global gambling invasion, written by Richard A. McGowan, MA, MDiv, ThM, DBA, associate professor at the Carroll School of Management at Boston College and Research Associate at Harvard Medical, Division on Addictions in

Boston, Massachusetts, United States. These chapters, as discussed below, are chapter 13, "Macau: China's Entry into the World Of Gambling;" chapter 14, "Native American Gambling: Economic Development or Dependence?" and chapter 15, "The Current Climate of Gambling in the United States."

In chapter 13, McGowan reports on what we will call the "addiction industry" in Macau. Gambling is not a new phenomenon, as it can be traced back at least hundreds of years, if not as far back as recorded history. Virtually no nation remains untouched. "Recently, Macau has been dubbed the 'Monte Carlo of the Orient.' This nickname is becoming well deserved." With the marked growth in the Macau gambling industry, the economy affected by it is booming. "This increase in casinos and hotels has resulted in a similarly strong increase in visitors to the island. In 2005, Macau welcomed 10.5 million visitors from China, 2.5 times as many as it had seen in 2002. Chinese visitors accounted for 56 percent of all visitors in 2005, up from 37 percent in the same time frame. . . . [At the same time], local authorities have been citing a sharp increase in casino-related crimes, such as money laundering. . . . Many companies are vying for a strong foothold in Macau, and some have a set strategy already."

In chapter 14, McGowan then turns to what we are calling the "addiction industry" in "Native America": "Tribal gaming is a $19.6 billion per year industry, and it is getting larger. . . . The number of tribes with gaming facilities grew about 3 percent in 2004 (from 221 to 228), and the number of Indian gaming facilities saw growth at about 5 percent in 2004 (from 385 to 405). Although they are highly correlated with the development of new gaming facilities, the numbers of gaming tables and gaming machines have seen tremendous growth as well. . . . Therefore, in coming years, we can only hope that the size and power of the Indian gaming industry influences public and private parties to engage in further studies of the effects that Indian gaming has had on the Native American population as well as the American population at large."

And third, in chapter 15, McGowan discusses this "addiction industry" in the United States: "A look at how gambling revenues stack up against revenues from other recreational/leisure time activity sectors reveals not only that the gambling revenues outweigh both music sales and movies combined, but also that the gambling industry is the only one of the industries in this study to have shown consistent growth in each of the last three years." Moreover, "The rise of Internet gambling has been the primary driver of the intensifying levels of competition within the industry. No longer is consumers' ability to place wagers limited by their geographic proximity to gambling operators. . . . Internet gambling operators can serve a truly global customer base, and with the financial barriers to entry relatively low in comparison with the huge profits

being reaped through Internet gambling, new entrants can enter at will and attempt to compete with the market leaders."

From McGowan's overview of this addiction-based industry, we move to chapter 16, contributed by Kimberly S. Young, PhD, as noted earlier of the Center for Internet Addiction Recovery, St. Bonaventure University in St. Bonaventure, New York, United States. Young discusses "Online Gaming Addiction: Symptoms, Risk Factors, and Treatment." Note the ongoing use of the broader term, "gaming," which can include gambling but is not limited to gambling. Young presents her definition for purposes of her chapter: "Online gaming addiction is an addiction to online video games, role-playing games, or any interactive gaming environment available through the Internet. Online games such EverQuest, the Dark Age of Camelot, or Diablo II—dubbed 'hero-inware' by some players—can pose much more complex problems. Extensive chat features give such games a social aspect missing from offline activities, and the collaborative/competitive nature of working with or against other players can make these games a hard habit to break." Interactive gaming addiction, like so many of the other global addictions, is pervasive. Its consequences are increasingly apparent. Interactive gaming can lead to divorce, job loss, and health problems. Not yet as prevalent as addictions to cyberporn and online chatting, interactive online gaming is already reaching millions of users. "Globally, recent reports have indicated that interactive online gaming has reached addictive proportions in China, Korea, and Taiwan. About 10 percent of China's more than 30 million Internet gamers are said to be addicted." The global picture is bleak; however, efforts are being made to stem the tide. "To battle what has been called an epidemic in some reports, Chinese authorities regularly shut down Internet cafes, many illegally operated, in crackdowns that also include huge fines for their operators. The Chinese Government . . . in 2005 opened the first treatment center for Internet addiction in Beijing. Online gaming addiction continues to raise such serious concerns that the first detox center for video game addiction has opened in Amsterdam, and . . . the American Medical Association, at its annual policy meeting, has considered calling video game overuse an addiction."

We complete this volume with chapter 17, "Youth Gambling Prevention and Resilience Education: A Harm Reduction Approach," by Isabelle D. Lussier, MA, Jeffrey L. Derevensky, PhD, and Rina Gupta, PhD, all of the International Centre for Youth Gambling Problems and High-Risk Behaviors, McGill University, Montreal, Quebec, Canada. Lussier, Derevensky, and Gupta remind us that "Gambling, in its many forms, has permeated every society and culture all the way back to ancient times. While gambling has been a source of entertainment for countless people, a minority of people exhibit compulsive

gambling behaviors that lead to personal harm and suffering." Young people are reported to be at greatest risk from gambling behavior and addiction. Programs and initiatives seeking to quell this mounting predicament recognize the "significant overlap in risk and protective factors for youth with respect to problem gambling and other problem behaviors." This "has led to the creation of prevention initiatives that target multiple risk behaviors." The authors call for "resilience research regarding youth gambling behaviors," as "resiliency skills have long been incorporated into prevention programs for a wide variety of risky behaviors." However, the authors note, "resilience researchers have become increasingly cautious in using the term resilience, opting instead for more specific terms such as *educational resilience*, *emotional resilience*, and *behavioral resilience*."

We hear the hope in the work of these authors and the other contributors to the four volumes in this *International Collection on Addictions*. It is in their work, their solutions, and the work and solutions of their colleagues around the world, that we can envision an ever more, rather than an ever less, resilient human species, one that faces, understands, and overcomes detrimental addictions of all forms everywhere. In the face of daunting addictions data from around the globe, this may seem an impossible dream. However, with the talent and vision of the cadre of persons fighting this war on addictions internationally, the paths to healing and cures for people everywhere can become realities.

Part I

COMPULSIONS

This page intentionally left blank

Chapter 1

Work Addiction: Causes, Consequences, and Choices

Ronald J. Burke, PhD

Workaholism and long working hours have positive connotations such as dedication, commitment, and organizational citizenship behavior as well as negative connotations such as ill health and damaged family relationships (Killinger, 1991). The number of hours worked per week, while obviously an element of workaholism, does not capture one's degree of work involvement, psychological state, or attitude. Hours worked per week, however, are a behavioral manifestation of workaholism (Ng, Sorensen, & Feldman, 2007). Although the popular press has paid considerable attention to workaholism, very little research has been undertaken to further our understanding of it (McMillan, O'Driscoll, & Burke, 2003). It should come as no surprise, then, that opinions, observations, and conclusions about workaholism are both varied and conflicting (McMillan, O'Driscoll, Marsh, & Brady 2001). Some writers view workaholism positively from an organizational perspective. Machlowitz (1980) conducted a qualitative interview study of 100 workaholics and found them to be very satisfied and productive. Others view workaholism negatively (Fassel, 1990; Killinger, 1991; Oates, 1971). These writers equate workaholism with other addictions and depict workaholics as unhappy, obsessive, tragic figures who are not performing their jobs well and are creating difficulties for their co-workers (Porter, 1996). The former would advocate the encouragement of workaholism; the latter would discourage it.

McMillan and O'Driscoll (2006) see workaholism as a value system regarding the importance of working and achieving that typically does not meet the scientific criteria for addiction. They propose an integrated model of workaholism that includes antecedents, behaviors, and consequences. Antecedents

include a reluctance to disengage from work, an obsessive style, and a strong enjoyment of work, driven by internal positive reasons. Behaviors include working more than others, thinking about work more than others, talking about work more than others, and stability in these areas over time. Consequences include working anytime and anywhere, choosing work/chores over leisure, and unclear work/relationship boundaries. Workaholism, to McMillan and O'Driscoll may in fact represent an approach to work (with intensity) rather than a frequency captured only by hours.

DEFINITIONS OF WORKAHOLISM

Oates (1971), generally acknowledged as the first person to use the word *workaholic*, defined a workaholic as "a person whose need for work has become so excessive that it creates noticeable disturbance or interference with his bodily health, personal happiness, and interpersonal relationships, and with his smooth social functioning" (Oates, 1971, p. 4). Killinger (1991, p. 61) defines a workaholic as "a person who gradually becomes emotionally crippled and addicted to control and power in a compulsive drive to gain approval and success." Robinson (1998, p. 81) defines workaholism "as a progressive, potentially fatal disorder, characterized by self imposed demands, compulsive overworking, [and an] inability to regulate work to the exclusion of most other life activities." Porter (1996, p. 70) defines workaholism as "an excessive involvement with work evidenced by neglect in other areas of life and based on internal motives of behavior maintenance rather than requirements of the job or organization." Most writers use the terms *excessive work*, *workaholism*, and *work addiction* interchangeably.

Spence and Robbins (1992, p. 162) define the workaholic as a person who "is highly work involved, feels compelled or driven to work because of inner pressures, and is low in enjoyment at work." Most writers view workaholism as a stable individual characteristic (Scott, Moore, & Miceli, 1997; Spence & Robbins, 1992). Most definitions of workaholism portray it in negative terms.

TYPES OF WORKAHOLICS

Some researchers have proposed the existence of different types of workaholic behavior patterns, each having potentially different antecedents and associations with job performance, work outcomes, and life outcomes (Burke, 1999b; Naughton 1987; Scott et al., 1997; Spence & Robbins, 1992). The existence of different types of workaholics might reconcile conflicting views as to whether workaholics are productive and satisfied or tragic and unfulfilled.

Scott et al. (1997) suggest three types of workaholic behavior patterns: compulsive-dependent, perfectionist, and achievement oriented. They hypothesize that compulsive-dependent workaholism will be positively related to job performance and job and life satisfaction. Perfectionist workaholism will be positively related to levels of stress, physical and psychological problems, hostile interpersonal relationships, low job satisfaction and performance, and voluntary turnover and absenteeism. Finally, achievement-oriented workaholism will be positively related to physical and psychological health, job and life satisfaction, job performance, low voluntary turnover, and pro-social behaviors.

Spence and Robbins (1992) propose three workaholic patterns based on their workaholic triad notion. The workaholic triad consists of three concepts: work involvement, feeling driven to work because of inner pressures, and work enjoyment. Data were collected in Spence and Robbins's study from 368 social workers holding academic appointments. Profile analysis resulted in the same six profiles for women and men—three workaholic types and three nonworkaholic types. These profiles were as follows. Work addicts (WAs) score high on work involvement, high on feeling driven to work, and low on work enjoyment. Work enthusiasts (WEs) score high on work involvement, low on feeling driven to work, and high on work enjoyment. Enthusiastic addicts (EAs) score high on all three workaholism components. Unengaged workers (UWs) score low on all three workaholism components. Relaxed workers (RWs) score low on feeling driven to work and work involvement and high on work enjoyment. Disenchanted workers (DWs) score high on feeling driven to work and low on work involvement and work enjoyment.

RESEARCH FINDINGS

The following sections of this chapter will review research findings that compare the personal demographics, job behaviors, work outcomes, extrawork outcomes, and psychological health of the three types of workaholics proposed by Spence and Robbins (1992).

Personal Demographic and Work Situational Characteristics

A critical question involves potential differences between the three workaholism types on both personal demographic and work situation characteristics including hours worked per week. If the workaholism types were found to differ on these (e.g., organizational level, marital status, hours worked per week), these differences would account for any differences found in work and health outcomes.

A number of studies (Bonebright, Clay, & Ankenmann, 2000; Burke, 1999d; Burke, Burgess, & Oberklaid, 2002; Spence & Robbins, 1992) have reported essentially no differences between the three workaholism types on a variety of personal and work situation characteristics. The workaholism types work the same number of hours and extra hours per week; the workaholism types work significantly more hours per week and more extra hours per week than the nonworkaholism types.

Job Behaviors

There has been considerable speculation regarding the work behaviors likely to be exhibited by workaholics (see Mudrack, 2007). This list includes hours worked per week, extra hours worked per week, job involvement, job stress, nondelegation of job responsibilities to others, high (or low) levels of job performance, and high levels of interpersonal conflict and lack of trust. There is empirical research that examines some of these hypothesized relationships.

Burke (1999d) considered these relationships in a large sample of Canadian MBA graduates. Comparisons of the three workaholism types on a number of behavioral manifestations provided considerable support for the hypothesized relationships. First, there were no differences between WAs, EAs, and WEs on hours worked per week or extra hours worked per week; workaholism types worked significantly more hours and extra hours per week than did the three nonworkaholism types. Second, EAs devoted more time to their jobs in a psychological sense than did both WEs and WAs. Third, WAs reported greater job stress than did EAs, both reporting greater job stress than did WEs. Fourth, both EAs and WEs reported greater job involvement than did WAs. Fifth, WAs exhibited greater inability and unwillingness to delegate than both WEs and EAs. Sixth, EAs were more perfectionistic than were WEs.

Spence and Robbins (1992) found that WAs reported higher levels of job stress, perfectionism, and unwillingness to delegate job duties to others than did WEs. Kanai, Wakabayashi, and Fling (1996), using the Spence and Robbins measures, reported that WAs and EAs scored higher than WEs on measures of job stress, perfectionism, nondelegation, and time committed to job.

In summary, WAs reported higher levels of work stress, more perfectionism, and greater unwillingness or difficulty in delegating than one or both of the other workaholism types.

ANTECEDENTS OF WORKAHOLISM

Four potential antecedents of workaholism have received some conceptual and research attention. Three of these, family of origin, Type A behavior, and

personal beliefs and fears, are the result of socialization practices within families and society at large. The fourth, organizational support for work–personal life imbalance, represents organizational values and priorities.

Family of origin. Robinson (1998) has written about work addiction as a symptom of a diseased family system. Work addiction, like other addictive behaviors, is intergenerational and passed on to future generations through family processes and dynamics. In this view, work addiction is seen as a learned addictive response to a dysfunctional family of origin system.

Personal beliefs and fears. Burke (1999g) examined the relationship between personal beliefs and fears and workaholism. Beliefs and fears are a reflection of values, thoughts, and interpersonal styles. Three measures of beliefs and fears developed by Lee, Jamieson, and Early (1996) were used: "Striving against others," "No moral principles," and "Prove yourself." Burke compared the three workaholism types on these measures of beliefs and fears. WAs scored significantly higher than WEs and EAs on the measures of striving against others and no moral principles, as well as on the composite measure. In addition, WAs scored higher on the need to prove self than did WEs. Workaholism thus emerges as work behaviors in response to feelings of insecurity and low self-worth. This is best reflected in managers' feelings of being driven to work. Paradoxically, these beliefs and fears were also found to be associated with lower levels of work enjoyment.

Kaiser and Kaplan (2006) offer some observations on the wellsprings of "overdoing it" at work, based on their coaching and consulting work with executives. They emphasize intrapersonal issues, describing how psychological wounds sensitize managers to be anxious about being hurt again. When managers feel threatened, their behavior frequently goes to the extreme, either overdoing or underdoing. Kaiser and Kaplan propose the following sequence: first one's sensitivity becomes activated; this influences (or distorts) one's perceptions of resources and demands in the environment, precipitating feelings of threat that in turn promote compulsion—overdoing, leading to working extreme hours, striving to prove oneself, impatience with the performance of others, overcontrolling, nondelegating and micromanaging. This list reads like a template for the work addict.

Type A Behavior

Zhdanova, Allison, Pui, and Clark (2006), using meta-analysis, provided support for Type A behavior as an antecedent of workaholism. Type A behavior has been shown to be associated with levels of job stress, psychological distress, and coronary heart disease. Pred, Helmreich. and Spence (1987) factor analyzed the Jenkins Activity Survey, a self-report measure of Type A behav-

ior, producing two independent factors: Achievement Striving (AS), which they found to be predictive of positive work attitudes and performances, and Impatience-Irritation (II), found to be predictive of psychological distress.

Burke, Richardsen, and Martinussen (2004), in a study of 171 Norwegian owners and senior managers of construction companies, found that WAs scored higher than WEs on Impatience-Irritation; EAs scored higher than WEs on Achievement Striving, both being dimensions of Type A behavior. Impatience-Irritation has been shown to be predictive of psychological distress.

Organizational values. Burke (1999f) compared perceptions of organization culture values supporting work–personal life imbalance across the three workaholism types. Organizational values encouraging work-family imbalance were measured by scales developed by Kofodimos (1993). Organizational values encouraging balance were measured by nine items (e.g., "Setting limits on hours spent at work"). Organizational values supporting imbalance were measured by eight items (e.g., "Traveling to and from work destinations on weekends"). A total imbalance score was obtained by combining both scales, reversing the balance scores. WAs reported higher imbalance values than both WEs and EAs. Thus, WAs see their workplaces as less supportive of work–personal life balance than do the two other workaholism types.

Johnstone and Johnston (2005), using two of the three Spence and Robbins workaholism components (work enjoyment, feeling driven to work because of inner pressures), examined the relationship of these to four aspects of organizational climate: work pressures, involvement, supervisor support, and co-worker cohesion. Data were collected in two occupation groups: business services (law firms, management consulting, accounting firms) and social services (schools, social workers in government agencies, workers in a hospice). Involvement, supervisor support, and co-worker cohesion were positively related to work enjoyment while work pressures were negatively related to work enjoyment. Only work pressures were significantly related (positively) to feeling driven.

Regression analyses including age, occupation type, and hours worked along with the four organizational climate measures showed that only co-worker cohesion and supervisor support predicted work enjoyment. Age, work pressures, and occupational type were significant predictors of feeling driven. Those in business services had higher levels of feeling driven and lower levels of work enjoyment.

Work Outcomes

The relationship between workaholism and indicators of job and career satisfaction and success is difficult to specify. It is likely that different types

of workaholics will report varying work and career satisfactions (Scott et al., 1997).

Burke (1999a) compared levels of work and career satisfaction and success among the workaholism profiles observed by Spence and Robbins (1992). Four work outcomes, all significantly intercorrelated, were used. Intent to quit was measured by two items (e.g., "Are you currently looking for a different job in a different organization?"). Work satisfaction was measured by a seven-item scale developed by Kofodimos (1993). One item was "I feel challenged by my work." Career satisfaction was measured by a five-item scale developed by Greenhaus, Parasuraman, and Wormley (1990). One item was "I am satisfied with the success I have achieved in my career." Future career prospects were measured by a three-item scale developed by Greenhaus et al. (1990). One item was "I expect to advance in my career to senior levels of management."

WAs scored lower than WEs and EAs on job satisfaction, career satisfaction, and future career prospects, and higher than WEs on intent to quit. It should be noted that all three workaholic profiles (WAs, EWs, WEs) worked the same number of hours per week and had the same job and organizational tenure.

WORKAHOLISM TYPES AND FLOW AT WORK

Csikszentmihalyi (1990) uses the term *optimal experience* to refer to times when individuals feel in control of their actions and masters of their own destinies. Optimal experiences commonly result from hard work and meeting challenges head on. Would the workaholism types differ in the experience of flow? In a study of 211 Norwegian journalists, Burke and Matthiesen (2004) found that journalists scoring higher on work enjoyment and lower on feeling driven to work because of internal needs indicated higher levels of flow or optimal experience at work. In this same study, Burke and Matthiesen found that WEs and EAs indicated higher levels of flow than did WAs.

PSYCHOLOGICAL WELL-BEING

There is considerable consensus in the workaholism literature on the association between workaholism and poorer psychological and physical well-being. In fact, some definitions of workaholism incorporate aspects of diminished health as central elements. It is not surprising that this relationship has received research attention.

Burke (1999e) compared the three workaholism types identified by Spence and Robbins (1992) on three indicators of psychological and physical well-being

in a sample of 530 employed women and men MBA graduates. Psychosomatic symptoms were measured by 19 items developed by Quinn and Shepard (1974). Respondents indicated how often they experienced each physical condition (e.g., "headaches") in the past year. Lifestyle behaviors were measured by five items developed by Kofodimos (1993). One item was "I participate in a regular exercise program." Emotional well-being was measured by six items developed by Kofodimos (1993). One item was "I actively seek to understand and improve my emotional well-being."

Once again, the comparisons of the workaholism types on the three measures of psychological and physical well-being provided considerable support for the hypothesized relationships. WAs had more psychosomatic symptoms than both WEs and EAs and poorer physical and emotional well-being than WEs.

In a study of 171 Norwegian construction company owners and senior managers, Burke et al. (2004) found that WAs reported higher levels of emotional exhaustion than both WEs and EAs; the three workaholism types were similar on levels of cynicism and personal efficacy.

EXTRA-WORK SATISFACTIONS AND FAMILY FUNCTIONING

A number of writers have hypothesized that workaholism is likely to impact negatively on family functioning (Killinger, 1991, Porter 1996; Robinson, 1998). Burke (1999c) considered the relationship of the three workaholism types identified by Spence and Robbins (1992) with extrawork satisfactions. Three aspects of life or extrawork satisfaction were included, using measures developed by Kofodimos (1993). These aspects were family satisfaction, relationship satisfaction, and relationship satisfaction and community satisfaction. The comparisons of the workaholism types on the three measures of life or extrawork satisfactions provided moderate support for the hypothesized relationships. WAs reported less satisfaction on all three extrawork satisfaction measures than did WEs and less satisfaction on one (family) than did EAs.

"GOOD" AND "BAD" WORKAHOLICS

Schaufeli, Taris, and Bakker (2007) make a distinction between "bad" workaholics, exhibiting a negative psychological state, and "good" workaholics, characterized by high levels of work engagement. They differentiate between work addiction and work engagement. Engaged workers work hard because it is fun; work addicts work hard because they are compulsively driven. They conducted a study in the Netherlands, involving 2164 employees in a wide variety

of jobs who participated in an Internet survey They developed a measure of workaholism having two components: working excessively, and working compulsively (also see Taris, Schaufeli & Verhoeven, 2005). Scores on these two components were related to measures of overwork, employee well-being, and job performance. Both measures of workaholism were significantly and positively correlated with indicators of overwork, Working excessively correlated with these at a slightly higher level. Both workaholism components were also significantly and negatively correlated with psychological health and happiness. Work engagement was positively correlated with indicators of overwork and psychological health and happiness, and negatively correlated with absenteeism. And somewhat surprisingly, both work addiction components and work engagement were positively related to indicators of job performance.

Schaufeli, Taris, and Bakker (2008), again using their two-component measure of work addiction (working excessively and working compulsively), report the results of two further studies. In the first study, involving 7594 Dutch workers, hours worked were correlated with both work addiction scales, and different occupational groups differed on the two work addiction scales. Managers and entrepreneurs scored higher on both, as did men when compared to women.

In the second study, data were collected from 2115 medical residents. Although there were a few differences in the predictors of working excessively and working compulsively, the majority of relationships between these two components and other variables (e.g., job demands, job resources, burnout recovery, life satisfaction) were statistically significant. Schaufeli and his colleagues conclude that both working excessively and working compulsively formed a syndrome —two characteristics that occur together and are associated with potential predictors in the same way.

They also divided their sample into four groups based on either high or low scores on the two workaholism scales. These groups were labeled workaholics, hard working, compulsively working, and relaxed. These four groups were then compared on 22 variables with significant group differences found on all but one. Workaholics (those residents scoring high on both components) had the poorest scores on 16 of the 21 variables on which significant group differences were found.

The work of Schaufeli and his colleagues has highlighted the distinction between work engagement and work addiction, between good and bad workaholics, and put the spotlight on different motivational systems. Workaholics are motivated by performance goals; engaged workers are motivated by mastery goals. The former are external; the latter internal. Workaholics work hard to prevent themselves from feeling bad—an avoidance motivation; engaged workers work hard for learning and development —an approach motivation.

PASSION VERSUS ADDICTION

Why do people work hard, and does their motivation for working long hours matter in terms of their satisfaction and well-being? Several streams of research bear on these questions. First, a growing body of research on workaholism has shown that different types of workaholics exist (Scott et al., 1997; Spence & Robbins, 1992) and that some types seem to be work satisfied and psychologically healthy while other types are dissatisfied with their jobs and careers, are dissatisfied with their family relationships, and are in psychological distress (Buelens & Poelmans, 2004; Burke, 2007; Kanai et al., 1996; Machlowitz, 1980; Robinson, 1998; Spence & Robbins, 1992).

Second, extensive research on sources of motivation (e.g., the effects of intrinsic versus extrinsic goals) and different processes or motivations for realizing these goals (e.g., internal versus external motivations)—the "what" and "why" of goal pursuits—has shown that individuals motivated by extrinsic goals and external sources of motivation report lower levels of satisfaction and psychological health (Deci, Koestner, & Ryan, 1999; Deci & Ryan, 1985, 2000; Ryan & Deci, 2000; Srivastava, Locke, & Bartol, 2001). Burke (2006) has shown that different types of workaholics are motivated by different beliefs and fears about people and their larger social and work environment.

Third, although working long hours has generally been associated with more negative work and health outcomes, dramatic exceptions to this trend have also been observed. Hewlett and Luce (2006) reported some work and family experiences of men and women working in "extreme jobs," jobs in which they worked 70 or more hours per week and under high work intensity (e.g., an unpredictable flow of work, responsibility for clients 24/7, and a fast-paced flow of work). Respondents were senior level managers and executives in large U.S. and international based corporations earning huge salaries and working in prestigious jobs having lots of perks. Their respondents indicated great work satisfaction resulting in part from the challenge, meaning, and rewards from their jobs. They were passionate about their work and their jobs. Respondents did indicate, however, that they hoped to work a few fewer hours in the future, and some were concerned about potential negative effects of their work hours on personal and family lives. Brett and Stroh (2003), in a sample of alumni of a prestigious U.S. business school, also reported positive reasons among both men and women for working over 61 hours per week.

Fourth, it is only recently that passion in the workplace has begun to be explored. Vallerand and his colleagues (Vallerand et al., 2003; Vallerand et al., 2007) have proposed a dualistic approach to passion. Passion is defined as a strong inclination toward an activity (e.g., work in our case) that is important,

liked, and involves investing considerable time in its pursuit. They distinguish between a harmonious passion (HP) that is well integrated into one's identity and undertaken freely and willingly and an obsessive passion or addiction (OP) that is not well integrated into one's identity and is the result of internal pressure (e.g., to increase one's self-esteem in the eyes of others). The activity controls the person under OP; the person controls the activity under HP. Vallerand and colleagues hypothesized and found that HP leads to more positive affect, less negative affect, and higher levels of flow, while OP produces the opposite effects. Because the activity is freely chosen under HP, the individual is engaged in the activity more fully and flexibly, leading to greater concentration, absorption, flow, and positive affect. Vallerand and colleagues developed measures of both types of passion and found they were significantly and positively correlated with each other, and similarly and positively correlated with evaluations of and liking for a self-chosen activity. They also found that levels of HP were higher than levels of OP for the chosen activity. They further suggest that HP would likely be correlated with psychological health and OP with psychological distress. Thus, passion can create motivation, increase well-being, and provide meaning in one's life, but it can also lead to negative emotions, rigid persistence, and an unbalanced life.

Fifth, two of the three workaholism components in the most widely used measure of workaholism developed by Spence and Robbins (1992), feeling driven to work because of inner pressure(D) and work enjoyment (WE), have been found to relate in different directions and to different outcomes (see Burke, 2006, for a review). WE, not surprisingly, was shown to be positively related to various work outcomes whereas D was shown to be negative related to many of these work outcomes. On the other hand, D was found to be negatively related to measures of psychological health, while WE was unrelated to these health indicators. In addition, WE and D related differently to potential antecedents of workaholism, such as perceptions of organizational climate supporting work–personal life balance and perceptions of people and their motives and how to succeed in the world. Other researchers have also reported different relations between WE and D and a number of different work and well-being outcomes (e.g., Graves, Ruderman, & Ohlott, 2006; Johnstone & Johnston, 2005; Schaufeli et al., 2007; Virick & Baruch, 2007).

These studies indicate different patterns of correlations, both antecedents and consequences, of WE and D. WE and D represent different underlying motivations or orientations to work and therefore have different effects in terms of both work and well-being outcomes. D is likely to hamper performance. WE is likely to facilitate performance (Vallerand et. al., 2003, 2007). D is likely to be associated with persistence, rigidity, perfectionism, and heightened levels

of job stress. D is likely associated with working harder, not smarter. D may also be associated with the setting of unrealistic performance expectations and deadlines. The positive emotions of WE are likely to spur higher levels of performance through increasing social resources and creativity, building trust with colleagues, and reducing levels of debilitating stress.

Vallerand and his colleagues (Vallerand et al., 2003; Vallerand & Houlfort, 2003; Vallerand et al., 2007) suggest four hypotheses relating to their two types of motivation, passion and addiction. First, passion and addiction are likely to be positively correlated. Second, respondents will generally score higher on passion than on addiction. Third, passion is likely to be related to positive work outcomes and psychological health. And fourth, addiction is likely to be associated with negative work outcomes and psychological distress.

Burke and his colleagues conducted three exploratory studies involving different occupational groups and conducted in different countries that examine the four hypotheses suggested by Vallerand and his colleagues (Vallerand et al., 2003, 2007)

Study 1—Managers and Professionals in Canada

Mail questionnaires were sent to about 1,000 male and 1,000 female MBA graduates of a single university in Canada. Responses were received from 591 individuals, a response rate of about 35 percent, with elimination of questionnaires returned because the person had moved. The sample decreased to 530 when individuals who indicated they were no longer working full-time were excluded. A fairly wide range of response was present on most personal demographic items.

Measures included single-item measures of personal and work situation characteristics; the measures of passion and addiction (Spence & Robbins, 1992); potential antecedents of passion and addiction including beliefs and fears (Lee et al., 1996) and organizational values supporting work–personal life imbalance (Kofodimos, 1993); four indicators of work investment including hours worked; extra hours worked; job involvement (Spence & Robbins, 1992), and one's psychological sense of *time committed to the job* (Spence & Robbins, 1992), two job behaviors—perfectionism and nondelegation—both measured by scales developed by Spence and Robbins (1992), four work outcomes including measures of job satisfaction (Kofodimos, 1993); career satisfaction (Greenhaus et al., 1990); future career prospects (Greenhaus et al., 1990); and job stress (Spence & Robbins, 1992), three extra work satisfactions—family, friends, and community—using measures developed by Kofodimos (1993); and three indicators of psychological well-being including

emotional and physical health (Kofodimos, 1993) and psychosomatic symptoms (Quinn & Shepard, 1974).

The following results were obtained. First, passion and addiction were significantly and positively correlated ($r = .25$, $p<.001$, $N = 524$). Second, respondents indicated similar levels of passion and addiction, the mean scale values being 2.5 and 2.7, respectively, contrary to predictions .Third, the correlations between the measures of both passion and addiction with four indicators of investment were positive and significantly different from zero ($p<.001$). As hypothesized, managers and professionals scoring higher on passion, and on addiction, were more involved with their jobs and work (e.g., worked more hours, more extra hours, more job involved). Fourth, both passion and addiction were significantly correlated with the two potential antecedents. Managers scoring higher on passion scored lower on the measure of beliefs and fears and lower on the measure of organizational support for work–personal life imbalance; managers scoring higher on addiction also scored higher on the measure of beliefs and fears and higher on the measure of organizational support for work–personal life imbalance. As hypothesized, the pattern of correlations was the direct opposite. Fifth, let us now consider the correlations between passion and addiction and the two job behaviors. All correlations were significantly different from zero ($p<.001$). Managers scoring higher on passion and managers scoring higher on addiction also scored higher on perfectionism; however, managers scoring higher on passion scored lower on nondelegation, whereas managers scoring higher on addiction scored higher on nondelegation. These findings provide partial support for our hypotheses. Sixth, let us turn to the correlations between passion and addiction and the four work outcomes. Most correlations were significantly different from zero. Managers scoring higher on passion also indicated more favorable work outcomes across the board (more satisfaction, lower levels of stress); managers scoring higher on addiction also indicated less job and career satisfaction and higher levels of stress. Scores on addiction were not correlated with perceptions of future career prospects. Once again, as hypothesized, the pattern of correlations was in the opposite direction. Seventh, let us consider the correlations of scores on passion and addiction with three indicators of extrawork satisfaction. All correlations were significantly different from zero ($p<.05$). Managers scoring higher on passion were also more satisfied in all three extrawork areas; managers scoring higher on addiction were less satisfied in all three areas of extrawork satisfaction. As hypothesized, the pattern of correlations was in the opposite direction, but weak. Finally, let us consider the relationship of passion and addiction with three indicators of psychological health. All correlations between passion and addiction were significantly different from zero ($p<.001$). Managers

scoring higher on passion reported higher levels of psychological health, whereas managers scoring higher on addiction reported lower levels of psychological health.

In summary, the pattern of findings shown provides strong support for the proposed hypotheses. In almost all instances, passion and addiction showed opposite relationships with the antecedent and outcome variables under investigation.

Study 2—Female Psychologists in Australia

Mail questionnaires were sent to 35,61 members of the Australian Psychological Society in the state of Victoria. A total of 658 completed surveys were returned, a 19 percent response rate. Respondents were similar to the total membership of the Australian Psychological society on some dimensions (age, sex) for which data were available. Only female respondents were chosen for this analysis. A wide range of response was again present on most demographic items.

The measures used were identical to those employed in the study of Canadian managers and professionals discussed above. Let us now consider the results. First, passion and addiction were significantly and positively correlated (r = .25, p<.001). Second, levels of passion and addiction were similar, mean values being 2.7 and 2.5, respectively. Third, scores on both passion and addiction were significantly and positively correlated with each of the four measures of work investment (e.g., work hours, job involvement). Fourth, scores on passion were significantly negatively correlated with beliefs and fears and organizational values supporting work–personal life imbalance, whereas scores on addiction were significantly positively correlated with scores on these two measures. Fifth, correlations of passion and addiction with the two job behaviors (perfectionism, nondelegation) were significant but in opposite directions, passion being negative and addiction being positive. Sixth, almost all the correlations of passion and addiction with the four work outcomes were significantly different from zero. Female psychologists scoring higher on passion also indicated more favorable work outcomes across the board (more satisfaction, lower levels of stress); female psychologists scoring higher on addiction indicated less job satisfaction and higher levels of stress. However, scores on both passion and addiction were positively correlated with perceptions of future career prospects. Once again, as hypothesized, the pattern of correlations was generally in opposite directions. Seventh, most of the correlations of passion and addiction with the three measures of extrawork satisfaction were significantly different from zero (p<.05). Female psychologists scoring higher on passion were also

more satisfied in two extrawork areas; female psychologists scoring higher on addiction were less satisfied in all three areas of extrawork satisfaction. Finally, almost all the correlations of passion and addiction and the three indicators of psychological health reached statistical significance. Female psychologists scoring higher on passion reported higher levels of psychological health, whereas female psychologists scoring higher on addiction reported lower levels of psychological health.

In summary, these findings were generally consistent with our hypotheses and almost identical to those obtained in a large Canadian sample of female and male managers and professionals.

Study 3—Journalists in Norway

Data were collected from 211 journalists working in the city of Bergen, Norway, using anonymously completed questionnaires, representing a response rate of 43 percent. Five hundred questionnaires were sent out by the journalists' union, and completed questionnaires were returned to a university address. Measures originally appearing in English were translated into Norwegian by members of the research team using the back-translation method; other measures (e.g., the Maslach Burnout Inventory) had already been translated into Norwegian from English and used by others in their research projects.

Measures included a number of personal and work situation characteristics. The same measures of passion and addiction used in the two previous studies, two measures of work investment (hours worked, work-family conflict; Lindstrom et al., 1997), four work outcomes including intrinsic motivation (Lindstrom et. al., 1997), intrinsic motivation (Lindstrom et al., 1997); organizational commitment (Lindstrom, et al., 1997) and flow (Jackson & Marsh, 1996), and psychological health including three scales from the Maslach Burnout Inventory (Schaufeli, Maslach, Leiter, & Jackson, 1996), and measures of positive and negative affect—the PANAS scales (Watson, Clark, & Tellegen, 1988).

Let us now consider the findings. First, passion and addiction were significantly and positively correlated ($r = .29$, $p<.001$). Second, levels of both passion and addiction were generally similar, mean values being 2.8 and 2.6, respectively. Third, passion tended to be positively correlated with hours worked ($p<.10$) and with work-conflict; addiction was significantly correlated with both hours worked and levels of work-family conflict. Thus, as predicted, both passion and addiction were significantly related to level of work investment. Fourth, six of the eight resulting correlations between passion and addiction and the four work outcomes were significantly different from zero ($p<.05$), and the

direction of these correlations was opposite in the two work motivation sources. Journalists scoring higher on passion also scored higher on flow, organizational commitment, and intrinsic motivation; passion was uncorrelated with levels of extrinsic motivation (but negative in sign). Addiction was negatively correlated with flow and organizational commitment and positively correlated with level of intrinsic motivation, and tended to be positively correlated with level of extrinsic motivation. Thus, passion and addiction were differentially correlated with flow, organizational commitment, and extrinsic motivation (but in the latter case, only one correlation approached statistical significance) and similarly correlated with levels of intrinsic motivation. Finally, 7 of the 10 correlations between passion and addiction and the five indicators of psychological health reached statistical significance ($p<.01$). Journalists scoring higher on passion also scored lower on exhaustion, cynicism, and negative affect and scored higher on positive affect. Journalists scoring higher on addiction also scored higher on exhaustion and cynicism and lower on positive affect. Neither passion nor addiction was correlated with levels of efficacy; addiction was also uncorrelated with levels of self-reported negative affect.

In summary, the pattern of findings reported here were both consistent with our hypotheses and very similar to those obtained in two other studies employing different samples obtained in different countries. In almost all instances, passion and addiction showed opposite relationships with the outcome variables under investigation. These findings show rather convincingly that different sources of work motivation—passion and addiction—had dramatically different relationships with a range of job satisfactions, work outcomes, and indicators of psychological health. Though themselves moderately and positively correlated, passion and addiction had opposite relationships with these outcomes.

As predicted, both passion and addiction were significantly correlated with job and work investment (e.g., hours worked, job involvement).

There were also widespread differences in the direction of correlation of passion and addiction with antecedents, job behaviors, work and extrawork satisfactions, and indicators of psychological well-being. Passion was always correlated with favorable work, extrawork, and psychological well-being outcomes and less obsessive job behaviors. These findings were consistent with an emerging view that positive emotions are likely to be associated with favorable outcomes (Fredrickson, 1998, 2001; Lyubormirsky, King, & Diener, 2005; Pressman & Cohen, 2005) Addiction was almost always correlated with less favorable work, extrawork, and psychological well-being indicators, and with less constructive job behaviors (e.g., more difficulty delegating). In summary, our results suggest significant differences in the effects of a healthy commit-

ment to one's work versus a harmful, psychologically and physically damaging compulsion to work.

ADDRESSING WORKAHOLISM

There is a large speculative literature suggesting ways to reduce levels of workaholism. One part of this work focuses on individual and family therapy (Killinger, 1991, Robinson, 1998); a second part emphasizes organizational and managerial interventions.

Individual counseling. Workaholics Anonymous chapters have sprung up in some North American cities. These groups, patterned after Alcoholics Anonymous self-help groups, endorse the 12-step approach common to the treatment of a variety of addictions. Killinger (1991) and Robinson (1998) include chapters outlining actions an individual might pursue to reduce levels of workaholism; Seybold and Salomone (1994) offer suggestions on counseling approaches. Chen (2006) shows how the use of rational emotive behavior therapy (REBT) can be effective in lessening work-life balance concerns and ameliorating the effects of workaholism.

Chen (2006) applied rational emotive behavior therapy (REBT) to the treatment of work addiction. Several factors likely cause work addiction. However a negative self-concept and related self-perceptions (e.g., lack of self-confidence, low self-worth) are the essential sources. An external environment that supports and reinforces work addiction interacts with these intrapersonal factors to produce the addiction to work (see Killinger, 1991). REBT addresses this distorted sense of self, irrational beliefs and emotions concerning self-worth and self-image; REBT seeks to build a confident and positive inner self. Work addicts are informed of the hazards of work addiction and ways of coping with it. Cognitive intervention lies at the heart of REBT: irrational beliefs are examined—the musts and shoulds that spur and sustain work addiction; individuals are also taught to use different language—there are other ways to think and behave. Individuals explore the emotional imagery of being less work addicted (i.e., working fewer hours); role playing is used to further examine feelings and thoughts.

Behavioral strategies are also part of REBT applied to work addiction. Individuals learn how to relax, to slowly desensitize the exhibition of particular behaviors (engaging in more or less), to improve self-management, to reorganize their workloads, to set clear boundaries, and to spend time in valued non-work activities.

Burwell and Chen (2008), in more recent writing, position their approach to treating work addiction within the broader field of positive psychotherapy

(PPT). Then they discuss therapeutic approaches stemming from quality of life therapy (QOLT).

Work addiction has its roots in the individual, the family, organizations, and the broader society. Work addicts are almost always in denial. The typical interventions for addressing work addiction are individual treatment (Fassel, 1990; Killinger, 1991; Robinson, 1998) and workplace changes (Fassel, 1990; Munck, 2001).

PPT encourages work addicts to increase positive emotions, engagement, and meaning in their lives. QOLT encourages individuals to identify, pursue, and fulfill their most valued needs and aspirations in important areas of their lives. Relationships and play are typically addressed as goals for change in QOLT.

Family therapy. Robinson and his colleagues, consistent with their clinical and consulting perspective, focus on treatment, both individual and family. This is not surprising given the central role they give to both family of origin and current family functioning in the development, maintenance, and intergenerational transmission of workaholism. The treatment recommendations Robinson offers (1998) are similar to those offered to alcoholic families.

Thus, denial is common among workaholics and their family members. Family members are reluctant to complain. Workaholics define their behavior and symptoms in a favorable light (Killinger, 1991; Porter, 1996). Parental expectations of children, often unrealistic, must be addressed. Family structures need to be identified. How do family members collude with the workaholic parent? Family members need help in expressing their negative feelings to the workaholic. Families need to learn to set boundaries around the amount of time they spend working together and talking about work. Family members can set goals to improve family dynamics (e.g., communication, roles, expression of feelings).

Workplace interventions. How can employers help workaholics and workaholics help themselves? Schaef and Fassel (1988) offer the following ideas. Employers should pay attention to the performance and work habits of employees and be alert to warning signs of workaholism. They should ensure that employees take vacation time away from work. Finally, job insecurity, work overload, limited career opportunities, and lack of control can make employees feel compelled to work longer. If these factors exist, employers should try to minimize their impact on the atmosphere within the organization.

Haas (1991) also highlights the role that managers can play in assisting their workaholic employees to change. Workaholic employees should be referred to an employee assistance program or a recovery program to start treatment processes. Managers should help prioritize projects for employees as long-term and

short-term assignments (Cartwright, 2000). Workaholics must be encouraged and helped to delegate their work. At the end of each day, the manager should meet with the employee to discuss what has been accomplished during that day and to plan (down to short intervals) for the following day. The employee should be given specific times to take breaks and to leave work. It may also be possible to reduce the negative effects of workaholism, particularly the well-being and health consequences, through stress-management training.

The development of workplace values that promote new, more balanced priorities and healthier lifestyles will support those workaholism types that want to change their behaviors (Austin Knight, 1995; Messenger, 2006; Munck, 2001). More people today want a life beyond work. Employees can work more effectively if they can integrate their work, families, and personal lives in more satisfying ways—they want "just enough success" (Nash & Stevenson, 2004a, 2004b). This becomes a win-win situation for all involved (Friedman, Christensen, & DeGroot, 1998).

ACKNOWLEDGMENTS

Preparation of this chapter was supported in part by York University, Toronto, Ontario, Canada. Several colleagues participated in the design and conduct of the research program: Lisa Fiksenbaum, Astrid Richardsen, Stig Mattheisen, Graeme McDermid, Mustafa Koyuncu, Zena Burgess, and Fay Oberklaid.

REFERENCES

Austin Knight. (1995). *Long hours culture.* London: Austin Knight.

Bonebright, C. A., Clay, D. L., & Ankenmann, R .D. (2000). The relationship of workaholism with work-life conflict, life satisfaction, and purpose in life. *Journal of Counseling Psychology, 47,* 469–477.

Brett, J. M., & Stroh, L. K. (2003). Working 61 plus hours a week: Why do managers do it? *Journal of Applied Psychology, 88,* 67–78.

Buelens, M., & Poelmans, S.A.Y. (2004). Enriching the Spence and Robbins typology of workaholism: Demographics, motivational and organizational correlates. *Journal of Organizational Change Management, 17,* 446–458.

Burke, R. J. (1999a). Are workaholics job satisfied and successful in their careers? *Career Development International, 26,* 149–158.

Burke, R. J. (1999b). It's not how hard you work but how you work hard: evaluating workaholism components. *International Journal of Stress Management, 6,* 225–239.

Burke, R. J. (1999c). Workaholism and extra-work satisfactions. *International Journal of Organizational Analysis, 7,* 352–364.

Burke, R. J. (1999d). Workaholism in organizations: Measurement validation and replication. *International Journal of Stress Management, 6*, 45–55.

Burke, R. J. (1999e). Workaholism in organizations: Psychological and physical well-being consequences. *Stress Medicine, 16*, 11–16.

Burke, R. J. (1999f). Workaholism in organizations: The role of organizational values. *Personnel Review, 30*, 637–645.

Burke, R. J. (1999g). Workaholism in organizations: The role of personal beliefs and fears, *Anxiety, Stress and Coping, 14*, 1–12.

Burke, R. J. (2006). *Research companion to working hours and work addiction*. Chichester, England: Edward Elgar.

Burke, R. J., Burgess, Z., & Oberklaid, F. (2002). Workaholism, job and career satisfaction among Australian psychologists. *International Journal of Management Literature, 2*, 93–103.

Burke, R. J., & Matthiesen, S. (2004). Workaholism among Norwegian journalists: Antecedents and consequences. *Stress and Health, 20*, 301–308.

Burke, R. J., Richardsen, A. M., & Martinussen, M. (2004). Workaholism among Norwegian senior managers: New research directions. *International Journal of Management, 21*, 415–426.

Burwell, R., & Chen, C. P. (2008). Positive psychology for work-life balance: A new approach in treating workaholism. In. R. J. Burke & C. L. Cooper (Eds.), *The long work hours culture: Causes, consequences and choices*. Bingley: Emerald Publishing..

Cartwright, S. (2000). Taking the pulse of executive health in the UK. *Academy of Management Executive, 14*, 16–23.

Chen, C. (2006). Improving work-life balance: REBT for workaholic treatment. In R. J. Burke (Ed.), *Research companion to working time and work addiction* (pp. 310–329). Cheltenham, England: Edward Elgar.

Csikszentmihalyi, M. (1990). *Flow: The psychology of optimal experience*. New York: Harper Collins.

Deci, E. L., Koestner, R., Ryan, R. M. (1999). A meta-analysis review of experiments examining the effects of extrinsic rewards on intrinsic motivation. *Psychological Bulletin, 125*, 627–668.

Deci, E. L., & Ryan, R. M. (1985). *Intrinsic motivation and self-determination in human behavior*. New York: Plenum.

Deci, E. L., & Ryan, R. M. (2000). The "what" and "why" of goal pursuits: Human needs and the self determination of behavior. *Psychological Inquiry, 11*, 227–268.

Fassel, D. (1990). *Working ourselves to death: The high costs of workaholism, the rewards of recovery*. San Francisco: HarperCollins.

Fredrickson, B. L. (1998). What good are positive emotions. *Review of General Psychology, 2*, 300–319.

Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist, 56*, 218–226.

Friedman, S. D., Christensen, P., & DeGroot, J. (1998). Work and life: The end of the zero-sum game. *Harvard Business Review, 76,* 119–129.

Graves, L. M., Ruderman, M., & Ohlott, P. J. (2006, August). E*ffect of workaholism on managerial performance: Help or hindrance?* Paper presented at the Academy of Management Annual Meeting, Atlanta.

Greenhaus, J. H., Parasuraman, S., & Wormley, W. (1990). Organizational experiences and career success of black and white managers. *Academy of Management Journal, 33,* 64–86.

Haas, R. (1991). Strategies to cope with a cultural phenomenon—workaholism. *Business and Health, 36,* 4.

Hewlett, S. A., & Luce, C. B. (2006, December). Extreme jobs: The dangerous allure of the 70-hour work week. *Harvard Business Review,* 49–59.

Jackson, S. A., & Marsh, H. W. (1996). Development and validation of a scale to measure optimal experience: The Flow State Scale. J*ournal of Sport and Exercise Psychology, 18,* 17–35.

Johnstone, A., & Johnston, L. (2005). The relationship between organizational climate, occupational type and workaholism. *New Zealand Journal of Psychology, 34,* 181–188.

Kaiser, R. B., & Kaplan, R. B. (2006). The deeper world of executive development: Outgrowing sensitivities. *Learning and Education, 5,* 463–483.

Kanai, A., Wakabayashi, M., & Fling, S. (1996). Workaholism among employees in Japanese corporations: An examination based on the Japanese version of the workaholism scales. *Japanese Psychological Research, 38,* 192–203.

Killinger, B. (1991). *Workaholics: The respectable addicts.* New York: Simon & Schuster.

Kofodimos, J. (1993). *Balancing Act.* San Francisco: Jossey Bass.

Lee, C., Jamieson, L. F., & Earley, P. C. (1996). Beliefs and fears and Type A behavior: Implications for academic performance and psychiatric health disorder symptoms. *Journal of Organizational Behavior, 17,* 151–157.

Lindstrom, K., Dallner, M., Elo, A. L., Gamberale, F., Knardahl, S., Skogstad, A., & Orhade, E. (1997). *Review of psychological and social factors at work and suggestions for the General Nordic Questionnaire (QPS).* Copenhagen: Nordic Council of Ministers.

Lyubormirsky, S. L., King, L., & Diener, E. (2005). The benefits of frequent positive affect: Does happiness lead to success? *Psychological Bulletin, 131,* 803–855.

Machlowitz, M. (1980). *Workaholics: Living with them, working with them.* Reading, MA: Addison-Wesley.

McMillan, L.H.W., & O'Driscoll, M. P. (2006). Exploring new frontiers to generate an integrated definition of workaholism. In R. J. Burke (Ed.), *Research companion to working hours and work addiction* (pp. 89–107). Cheltenham, England: Edward Elgar.

McMillan, L.H.W., O'Driscoll, M. P., & Burke, R .J. (2003). Workaholism in organizations: A review of theory, research and future directions. In C. L. Cooper & I. T.

Robertson (Eds.), *International Review of Industrial and Organizational Psychology* (pp. 167–190). New York: John Wiley.

McMillan, L.H.W., O'Driscoll, M. P., Marsh, N. V., & Brady, E. C. (2001). Understanding workaholism: Data synthesis, theoretical critique, and future design strategies. *International Journal of Stress Management, 8,* 69–92.

Messenger, J. C. (2006). Decent working time: Balancing the needs of workers and employers. In R. J. Burke (Ed.) *Research companion to working time and work addiction* (pp. 221–241). Cheltenham, England: Edward Elgar.

Mudrack, P. (2006). Understanding workaholism: The case for behavioral tendencies. In R. J. Burke (Ed.), *Research companion to working time and work addiction* (pp. 108–128). Cheltenham, England: Edward Elgar.

Munck, B. (2001, November). Changing a culture of face time. *Harvard Business Review,* 3–8.

Nash, L., & Stevenson, H. (2004a). *Just enough: Tools for creating success in your work and life.* New York: John Wiley.

Nash, L., & Stevenson, H. (2004b, February). Success that lasts. *Harvard Business Review,* 102–109.

Naughton, T. J. (1987). A conceptual view of workaholism and implications for career counseling and research. *Career Development Quarterly, 14,* 180–187.

Ng, T.W.H., Sorensen, K .L., & Feldman, D. C. (2007). Dimensions, antecedents, and consequences of workaholism: A conceptual integration and extension. *Journal of Organizational Behavior, 28,* 111–136.

Oates, W. (1971). *Confessions of a workaholic: The facts about work addiction.* New York: World of Work, Environment and Health.

Porter, G. (1996). Organizational impact of workaholism: Suggestions for researching the negative outcomes of excessive work. *Journal of Occupational Health Psychology, 1,* 70–84.

Pred, R. S., Helmreich, R. L., & Spence, J. T. (1987). The development of new scales for the Jenkins Activity Survey measure of the TABP construct. *Social and Behavioral Science Documents, 16,* 51–52.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131,* 925–971.

Quinn, R. P., & Shepard, L. J. (1974). *The 1972–73 Quality of Employment Survey,* Ann Arbor, MI: Institute of Social Research, University of Michigan.

Robinson, B. E. (1998). *Chained to the desk: A guidebook for workaholics, their partners and children and the clinicians who treat them.* New York: New York University Press.

Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist, 55,* 68–78.

Schaef, A. W., & Fassel, D. (1988). *The addictive organization.* San Francisco: Harper and Row.

Schaufeli, W. B., Maslach, C., Leiter, M. P., & Jackson, S. E. (1996). *Maslach Burnout Inventory—General Survey.* Palo Alto, CA: Consulting Psychologists Press.

Schaufeli, W., B., Taris, T. W., & Bakker, A. B. (2006). Dr. Jekyll or Mr. Hyde: On the difference between work engagement and workaholism. In R. J. Burke (Ed.), *Research companion to working time and work addiction*. Cheltenham, England: Edward Elgar.

Schaufeli, W. B., Taris, T. W., & Bakker, A. B. (2008). It takes two to tango: Workaholism is working excessively and working compulsively. In R. J. Burke & C. L. Cooper (Eds.), *The long workhours culture: Causes, consequences and choices*. Bingley: Emerald Publishing..

Scott, K. S., Moore, K. S., & Miceli, M. P. (1997). An exploration of the meaning and consequences of workaholism. *Human Relations, 50*, 287–314.

Seybold, K. C., & Salomone, P. R. (1994). Understanding workaholism: A view of causes and counseling approaches, *Journal of Counseling and Development, 73*, 4–9.

Spence, J. T. & Robbins, A. S. (1992). Workaholism: Definition, measurement, and preliminary results. *Journal of Personality Assessment, 58*, 160–178.

Srivastava, A., Locke, E. A., & Bartol, K. M. (2001). Money and subjective well-being: It's not the money, it's the motives. *Journal of Personality and Social Psychology, 80*, 959–971.

Taris, T. W., Schaufeli, W,. B., & Verhoeven, L. C. (2005). Internal and external validation of the Dutch Work Addiction Risk Test: Implications for jobs and non-work conflict. *Applied Psychology: An International Review, 54*, 37–60.

Vallerand, R. J., Blanchard, C. M., Mageau, G. A., Koestner, R., Ratelle, C., Leonard, M., & Gagne, M. (2003). Les passions de l'ame: On obsessive and harmonious passion. *Journal of Personality and Social Psychology, 85*, 756–767.

Vallerand, R. J., & Houlfort, N. (2003). Passion at work: Toward a new conceptualization. In D. Skarlicki, S. Gilland, & D. Steiner (Eds.), *Social issues in management*, Vol. 3 (pp. 175–204). Greenwich, CT: Information Age Publishing.

Vallerand, R. J., Salvy, S. J., Mageau, G. A. Elliot, A. J., Denis, P. L., Grouzet, F.M.E., & Bouchard, C. (2007). On the role of passion in performance. *Journal of Personality, 75*, 505–533.

Virick, M., & Baruch, Y. (2007, August). *Factors determining workaholism, its positive and negative consequences*. Paper presented at the Academy of Management Annual Meeting, Philadelphia.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070.

Zhdanova, L., Allison, L. K., Pui, S. Y. & Clark, M. A. (2006, May). A meta-analysis of workaholism antecedents and outcomes. Paper presented at the Society for Industrial and Organizational Psychology Conference. Dallas, TX.

This page intentionally left blank

Chapter **2**

Addiction to Television: With Commentary on Dependence on Video Games and the Internet

Robert W. Kubey, PhD

When a person becomes chronically dependent on a stimulus that is enjoyable but that in the long run is harmful, people often speak of addiction. This is not a precise scientific term, because what we think about certain activities is relative and depends on value judgments.

For this and other reasons, the official manual used by psychotherapists throughout North America, the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders,* fourth edition (1994; *DSM–IV*) and fourth edition text revision (2000; *DSM–IV–TR*),[1] no longer uses the term *addiction*. Instead, the committees that wrote the *DSM* use the term *substance dependence*. This does not resolve the problem, because of course we are all dependent on substances like oxygen, water, and dietary protein without being considered addicts.

For years, the addiction term has been extended to a whole host of behaviors from gambling and sexuality to video gaming and Internet use. My aim here is to bring some clarity to the question of how people develop the television viewing habits that now absorb so many hours of daily life in nearly all developed societies.

The time people spend viewing is nearly astonishing. People throughout the industrialized world, from the United States and Latin America to Europe and parts of Asia, the Middle East, and Africa, typically devote about three hours a day to watching television. In many societies, this easily constitutes half of all a person's leisure time or, calculated another way, 9 full years of a 75-year life span. Unless one does something else during commercials, two of those nine

years will be devoted to viewing advertisements; this viewing constitutes about 20 percent of all commercial television viewing time.

And of course, the three-hour figure *is* an average after all. For the "addicted" or so-called heavy viewers who may watch five or six hours a day, we may well be talking about 30 percent to perhaps 38 percent of their waking time watching TV, or as many as 20 years of the 50 years they will be awake if they live to 75.

If television viewing is indeed self-perpetuating, as will be suggested in the pages ahead, and if it indeed *can* produce a dependency, this is a matter of considerable significance, because this effect may be seen as perhaps the most powerful link in a chain of other effects. Indeed, many effects of television—from aggression and obesity to poorer academic performance and the impairment of imagination—are presumed to be effects of heavy or prolonged viewing.

Most people believe television viewing can be addictive. In North American surveys, roughly 10 percent of adults believed that *they* were addicted, but 65 to 70 percent reported believing that *others* were addicted. And many millions experience misgivings about how much they view. In a 1990 Gallup Poll (Gallup & Newport, 1990), 42 percent (up from 31 percent in the late 1970s) of adult Americans reported believing that they spent too much time watching television.

It needs to be stated at the outset that television viewing needn't be seen as purely problematic, or necessarily problematic at all. There are fine entertainment and information programs on television. One can learn and derive enjoyment from television. The author personally watches some escapist programs, and a fair amount of news, historical and scientific documentaries, HBO dramas, and bio-dramas that he thinks quite worthy.

The new television environment of 300 channels and DVD and downloadable films and other programs has made for a viewing world that is extraordinarily rich for adults and children alike and quite different from the one in which some of the data were collected, data that will be cited in this chapter and that date back to the television world of 1975, before most homes even had a VCR and when most homes only had the usual 8–15 channels. Readers should keep in mind that in citations to some of my work, or my work with Csikszentmihalyi, the research being drawn on was collected in that media environment, though not all of the research was conducted in the United States, as will be seen.

Furthermore, my book, *Creating Television* (Kubey, 2004), examines the personal creativity of many television artists involved in the creation of many programs that I consider very good or great and classic programs over the first 50 years of the medium's history. Moreover, I am something of a film buff. I regularly expose my sons to everything from early Buster Keaton and *One Flew*

Over the Cuckoo's Nest to Hitchcock and foreign works such as *Blow-Up* and Ingmar Bergman. Not to go on too long about this—and these are *films* after all—but they are viewed on a television monitor, and though I watch them commercial free, many people watch them over cable or broadcast television complete with commercial interruptions. These offerings, too, can be seen as television and some of them were available when my earliest data were collected in 1974–75.

Television also surely provides much-needed distraction and escape. Still, there is little doubt that a great many millions of people around the world have developed very significant viewing habits that might be deemed problematic and even a risk to mental and physical health.

So, first, what accounts for such a devotion to television, a devotion greater than to any other activity, save sleep and work?

HOW THE VIEWING HABIT IS FORMED

One of the first things to know is that television viewing typically involves less concentration and alertness—and is experienced more passively—than most other daily activities. This is known from my own studies and others using the same methodology with children, adolescents, adults, and the aged in the United States (see Kubey, 1984; Kubey & Csikszentmihalyi, 1990). Other researchers have confirmed these findings using the same method in other countries.

The findings are based on people's responses to a random signaling method, the Experience Sampling Method (ESM), that permits researchers to study human behavior and emotions as they occur naturally in the normal course of everyday life

The ESM involves having research subjects report what they are doing, and how they are feeling, each time they are signaled with a radio controlled, or preprogrammed, signaling device, or beeper. In most studies, each respondent is signaled six to eight times each day, from morning till night, for a week. The timing of the signals is predetermined by the research team to occur at random intervals, and participants do not know when to expect a signal.

The method has proved very useful in studying the contours of everyday life in a wide variety of populations around the world. Studies have focused on everything from worker satisfaction and the ways in which one spouse's mood affects the other spouse to student performance in school, eating disorders, and drug and alcohol abuse.

Along with the beepers, participants also carry a small booklet of self-report forms. After each signal, the individual stops to fill out a short report form

telling researchers how she felt on a number of standard psychological measures of mood and mental activity.

When people report how they are feeling and thinking when watching television, they report feeling relaxed and, as noted, exerting little mental effort or concentration. It is these related aspects of the experience, while perhaps obvious, that are key among the reasons why so many people watch for so many hours a day, why their habits are so well established, and why it is generally hard for people to reduce their viewing.

The ease with which viewing is accomplished is especially pronounced when compared with reading. And while it is not as dramatic as popular press accounts would have us believe, others' studies with the EEG show that reading *does* produce more beta activity ("fast waves" associated with increased mental activity and attention) than does television viewing, which causes somewhat more alpha activity ("slow waves" associated with relaxation and less mental arousal). Unlike reading, with its constant provision of moving images and sound, television can be thought of as doing the work of attention for us. Research also indicates that lower cortical arousal coincides with prolonged viewing.

Part of what holds our attention to television is the *orienting response*. First described by Pavlov in 1927, the orienting response is our instinctive, visual (or auditory) reaction to any sudden or novel stimulus in the environment. A well-functioning orienting response is necessary to the survival of almost every species.

Once stimulated, organisms orient their sensory receptors toward the stimulus that caused the response. The typical response set includes vasodilation of the blood vessels to the brain, decrease (or blocking) of the EEG alpha frequency, slowing of the heart, and vasoconstriction of blood vessels to major muscle groups. In other words, the attentional capacities of the organism are put on high alert, with stimulus intake rapidly becoming the first priority while the rest of the body quiets.

Byron Reeves of Stanford University and Esther Thorson of the University of Missouri and their colleagues (1986) first used the EEG in 1985 to test whether the simple *formal features* of television (cuts, edits, zooms, pans, sudden noises, and so on) might activate the orienting response, thereby causing attention to be drawn to the screen. While an increase in alpha frequency coincides generally with viewing, *alpha blocking* is associated with orienting responses. Reeves and Thorson and their team concluded that the formal features of cuts, edits, and movement did indeed command involuntary responses that may well "derive their attentional value through the evolutionary significance of detecting movement." "It is the form, not the content, of television that is unique."

Since 1985, various teams of researchers have delved deeper into how and when television's formal features affect encoding, storage, and message retrieval. The work has been extended to look at lighting levels, slow motion, animation, graphics, music, and narrative structure, and there is a growing literature on the formal features of Web sites as well. Researchers don't use the EEG as much now, preferring observation of the drop in heart rate that comes immediately after the orienting-eliciting stimulus and that continues for 4–6 seconds. Annie Lang's research teams at Indiana University have been among the most active (see Lang, 2000). Lang and her colleagues repeatedly show heart rate decelerations in response to cuts, edits, and video graphics. Recently the list has expanded to voice changes and special effects in radio messages.

The use of formal features to sustain attention isn't all bad. Many involved in the production of educational television for children have done research on the formal features of the medium so they know how edits, movement, slow versus fast zooms, or changes in volume can help hold attention on the screen for the purpose of learning. (Edits and slow zooms, for example, help.) The problem lies in how often such techniques are used (too often, in the opinion of some, in a program such as Sesame Street) to merely hold viewers so they don't channel surf or miss the next ad.

Lang shows improved recognition memory with increasing edits (changes from one camera to another in the same visual scene, i.e., giving different views of the same phenomenon). Increases in cuts (changes from one scene to a new visual scene) also improve recognition, but only to a point. A sharp drop-off in recognition occurs if the number of cuts exceeds 10 in 2 minutes. Lang concludes that the increase in resources allocated to encoding new information can't keep up with the increase in processing load, and, as a result, memory decreases.

Music videos and other forms of advertising that frequently use rapid intercutting of generally unrelated scenes are thus particularly well designed to hold attention, but they also overload the system, resulting in a decrease in overall memory for specific elements, despite the fact that more attentional resources have been activated by the scores of unrelated scene changes and jump cuts. Still, the name of the product may be well remembered, but the detail of the ad or video itself may be less well remembered.

The cost appears to be an overworked orienting response, one that continues to work, with the result that we still attend to the screen, but there is also an accompanying tired and worn-out feeling with little attendant psychological reward. Yale psychologist Jerome Singer expresses his concern about the exploitation of the orienting response in this way: "The TV set, and particularly commercial television with its clever use of constantly changing short sequences,

holds our attention by a constant sensory bombardment that maximizes orienting responses. . . . We are constantly drawn back to the set and to processing each new sequence of information as it is presented. . . . The set trains us to watch it" (1980, pp, 50–51).

It is the orienting response that may best explain typical viewer reports such as the following: "If a television is on, I just can't keep my eyes off it"; "I don't want to watch as much as I do but I can't help it. It makes me watch it"; and "I feel hypnotized when I watch television."

No one should think this is only a problem for those who are not well educated. Research psychologists such as Milton Rosenberg (1978) of the University of Chicago and Percy Tannenbaum (1980) of the University of California at Berkeley have reported on the strong attraction and hold of the small screen. Says Rosenberg, "When I've got television on in my home and I have to get up for one of the conventional reasons . . . I feel temporarily unfulfilled. Some part of the total sensory experience has suddenly been subtracted and I'm left in some slight state of tension until I can turn my gaze back to the screen" (1978).

Tannenbaum writes: "Among life's most embarrassing moments have been countless occasions when I am engaged in conversation in a room while a TV set is on, and I cannot for the life of me stop from periodically glancing over to the screen. This occurs not only during dull conversations but during reasonably interesting ones just as well. Judging from the behavior of the people with whom I was talking at the time and from reports of friends and colleagues, I am far from alone in this behavior and its accompanying chagrin" (1980, p. 112).

The embarrassment and chagrin, however, *may* be something of a class phenomenon. Indeed, research going back nearly four decades in the United States, England, and Japan has shown that TV viewing passivity often is associated with mild feelings of guilt and self-contempt (Bower, 1973; Furu, 1971; Himmelweit & Swift, 1976; Steiner, 1963)—especially among more affluent and educated viewers. The feelings of guilt in each study were associated with self-recrimination about the passivity of viewing and appear to be part of a sense among middle class individuals that they ought to use their time more productively.

Along with the overworked orienting response, I have also found evidence that the longer people view in a given period, the less satisfaction they report deriving from television (concentration also becomes more difficult) (Kubey, 1984). Perhaps like drug users who don't quite get the same response from the same dose as time goes on, in my studies, heavier viewers also report enjoying viewing less, on average, than do light viewers. And if viewers are middle class, a slight twinge of unease or guilt may also accompany prolonged viewing.

The natural attraction to television's sound and bright, colorful, and changing images starts very early. Israeli researcher Dafna Lemish (1987) documents neonates at six to eight weeks of age attending to television. I've observed slightly older infants, when lying on their backs on the floor, turning their necks around nearly 180 degrees to catch the light from a TV screen.

Of course, the ability of the television medium to attract and hold our attention is by no means purely a function of biology. Television provides ready opportunities to vicariously visit exotic places and experience high drama and suspense, view the most beautiful people in the world, and enjoy myriad forms of entertainment. Just as importantly, television producers are masters at finding clever ways to get people to view longer than they had originally intended. Cliffhangers are as common as ever, and some one-hour and most two-hour television dramas are written in seven acts with a rise in the action before each of the commercial breaks to hold viewers for advertising.

The set of programs that will follow those at present being viewed are also routinely "teased" with titillating suggestions that spike viewer interest and increase the possibility that we will view beyond the single program that we may have planned to watch. While the blame cannot all be laid at the feet of television producers, viewers do frequently report that they will sit down to watch one program, say, from 9 to 10 p.m., but then find themselves still watching beyond midnight.

Furthermore, there is a whole host of psychosocial variables to consider. Those who live alone and who feel lonely are especially vulnerable to developing dependence on the quasi-social experience the medium affords. Heavy viewers report having more time on their hands generally, and they also spend more time alone than do light viewers. Not surprisingly, among the demographic groups with higher proportions of heavy viewers in their ranks are the old, the unemployed, and those with more free time

One of the primary positive experiences people report while viewing is "relaxation," and associated with relaxation are consistent reports of passive bodily and mental states. But the benefit of relaxation appears to occur only while viewing, not afterward, whereas the feelings of passivity and lowered alertness associated with viewing seem to continue, spilling over into how people report feeling after they stop viewing—what I have called the "passive spillover effect" (Kubey, 1984). In this same research, I also found that people reporting more difficulty concentrating after viewing. In contrast, they appear to concentrate easily after reading.

These findings raise concerns about whether the quality of thinking after viewing is affected and whether viewing reduces the likelihood of engagement in more active cognitive and behavioral activities after viewing. Indeed, it is not

uncommon for people to report that television somehow absorbs or sucks out their energy. Author Marie Winn (1977) quotes this recollection from a college English teacher: "I find television almost irresistible. When the set is on, I cannot ignore it. I can't turn it off. I feel sapped, will-less, enervated. . . . So I sit there for hours and hours. . . . I remember that feeling of tiredness and anxiety that always followed those orgies, a sense of time terribly wasted. It was like eating cotton candy; television promised so much richness, I couldn't wait for it, and then it just evaporated into air. I remember feeling terribly drained after watching for a long time" (pp. 21–22).

People regularly use television to escape and distract themselves from negative and unpleasant moods, normal stress, and mild tension. In one study, the distraction function of TV was found to effectively reduce patients' reports of pain during dental procedures.

Dependence on the medium appears to develop for many as a result of a need to escape negative feelings or to help fill an emotional vacuum. In repeated studies by Robert McIlwraith (1991, 1998) of the Department of Psychiatry at the University of Manitoba, the 10 percent of university students and adults who call themselves "TV addicts" on surveys are shown to be significantly more likely than the 90 percent of self-reported "nonaddicted" viewers to report using television to cope with negative moods such as loneliness, sadness, anxiety, and anger. McIlwraith also reports finding self-proclaimed addicted viewers to be significantly more neurotic and introverted. On a measure called the Short Imaginal Processes Inventory (SIPI), McIlwraith finds the TV addicts to be more easily bored and distractible, and to have poorer attentional control than the nonaddicted. The addicted also often report using TV to distract themselves from unpleasant thoughts and to fill time. Interestingly, self-proclaimed addicts are significantly more likely to eat junk food but are significantly less likely than nonaddicts to drink alcohol (McIlwraith, Jacobvitz, Kubey, & Alexander, 1991).

My research using the ESM finds much the same thing. By comparison with light viewers in my research who watch less than two hours a day, heavy viewers (more than four hours) reported feeling significantly worse when they are alone, in unstructured situations such as waiting in line, or between activities. Heavier viewers appear to be particularly prone to fill up unstructured time or escape loneliness with a flick of the switch.

Using the American Psychiatric Association's (APA's) *Diagnostic and Statistical Manual* (*DSM–IV–TR;* 2000, http://psych.org/MainMenu/Research/DSMIV.aspx), I have suggested that were television a substance (and it may be—light is both particular and wavelike and *something* is taken into the body when people view), many people could be given a diagnosis of

dependence. Indeed, Allen J. Frances (as quoted in Goleman, 1990, p. C8), who oversaw the most recent revision of the manual, concluded that "Under the broader definition, many kinds of compulsive behavior could be considered addictive, including obsessive sex or compulsive television viewing."

APPLYING *DSM–IV* AND *DSM–IV–TR* SUBSTANCE DEPENDENCE CRITERIA

Though it is tempting to use the term addiction when referring to individuals who report upwards of 60 hours of viewing each week, the term connotes different things to different people. It seems likely that less confusion will result if we are more careful in the words we choose.

Indeed, the prime diagnostic manuals used by psychotherapists throughout North America, the American Psychiatric Association's *DSM–IV* and *DSM–IV–TR*, do not use the term addiction, nor did the previous edition published in 1987. Instead, the committees that wrote the *DSM* prefer the term substance dependence to conceptualize what others might call addiction. Still, there remain researchers and clinicians who use the term addiction, especially with regard to pornography. As a result, in some of these pages I have used the addiction term from time to time.

Using the *DSM* as a guide for making a diagnosis of television dependence is instructive. It is noteworthy that in the most recently revised edition of 2000, the word *drug* is explicitly used in the preface, perhaps to discourage those who have applied the diagnostic criteria to think about or conceptualize dependence on things that are not drugs, as have I since 1990.

DSM–IV lists seven possible criteria for making a diagnosis of substance dependence. Three of the seven must apply in order to make a diagnosis of dependence. Diagnosis also involves a time dimension wherein three (or more) criteria occur at any time in the same 12-month period.

In considering these criteria and the relevant literature on television viewing, it seems to me that perhaps all seven diagnostic criteria could be seen as applicable to television viewing habits in some people, along with their concomitant behaviors and effects. Each criterion is followed by observations regarding the way in which known television behaviors are related to it.

Criterion #1 in the DSM.: "Tolerance is defined by either of the following: (a) a need for markedly increased amounts of the substance to achieve intoxication or desired effect" or "(b) markedly diminished effect with continued use of the same amount of the substance" (p. 181).

Notably, viewers appear to obtain the benefit of relaxation only when they are viewing. It is for this reason, among others, that viewing often continues for

as long as it does. Heavier viewers also *enjoy* their viewing less on average than do light viewers (Kubey, 1984).

Criterion #2: Withdrawal is "manifested by either of the following: (a) the characteristic withdrawal syndrome for the substance" or "(b) the same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms." Withdrawal includes "a maladaptive behavioral change" and it is noted that "withdrawal symptoms vary greatly" (p. 178).

This criterion is a bit more difficult to apply to television viewing behaviors because we are largely limited to anecdotal reports and a small number of social science studies of withdrawal-like symptoms. Still, such reports are not hard to find.

Reports of withdrawal. Steiner (1963), for example, presented individuals' reports of a variety of behaviors of psychological interest that occurred following the loss of a television set. He was quite clever in his research approach, going to television repair shops in the early 1960s when many households still had only one TV set. Then he would interview those families who were without TV for a week or so while their TV set was in the shop.

Here are three examples: "The family walked around like a chicken without a head"; "It was terrible. We did nothing—my husband and I talked"; "Screamed constantly. Children bothered me and my nerves were on edge. Tried to interest them in games, but impossible. TV is part of them" (p. 99).

In her informal interviews, Winn has presented many similar anecdotes (1977, pp. 21–22). Today, such reports are less frequent, in part because most homes have more than one set. To be completely without a television set today is unusual, which is perhaps still another sign of how entrenched television viewing has become. So difficult is this sort of research to conduct that in the early 1970s, Tannis MacBeth Williams (1986) went to great lengths to study a small town in Canada that had been blocked by the Rocky Mountains before it was wired for cable. She was able to study the town before, during, and after the introduction of television in what I consider the most ambitious and convincing research on television's effects ever conducted—and all in a natural environment. Furthermore, Williams compared the small town, "No-Tel," to two other demographically matched towns of similar size that had television. Her book-length treatment is a classic in the field.

Like Steiner, Winick (1988) also offers a valuable review of studies of families whose television sets were being repaired:

> The first 3 or 4 days for most persons were the worst, even in many homes where viewing was minimal and where there were other ongoing activities. In over half of all the households, during these first few days of loss, the regular routines were disrupted, family members had difficulties in dealing with the newly avail-

> able time, anxiety and aggressions were expressed, and established expectations for the behavior of other household members were not met. People living alone tended to be bored and irritated. Over four-fifths of the respondents reported moderate to severe dislocations during this period. . . . The fifth to eighth day represented, in many cases, some form of readjustment to the new situation. By the second week, a move toward adaptation to the situation was common. (pp. 221–222)

Daley (1978) offers a similar account of his family's difficulties when they stopped viewing, also showing how easily the habit reformed itself after six months of abstinence. A number of newspapers, in the United States and elsewhere, have offered money as an incentive to get individuals or families to stop viewing television for some limited period of time, often a week or a month (reviewed in Condry, 1989; Kubey, 1984; Winick, 1988). Increased tension among family members has been described, and many families could not complete the period of abstinence agreed upon (Ryan, 1974). In a German study, it was reported that there was increased verbal and physical fighting after viewing stopped.

If a family has been spending the lion's share of its free time together over a period of years watching television—as is the case for many families today—it may take some days or weeks, or longer, for the family to reconfigure itself around a new set of activities. Particularly because watching television is so easy to do, family members may have become less imaginative about other ways to spend their time together.

Anecdotal reports from families that have tried the annual TV-turnoff week in the United States tell much the same story, with the early days being most difficult and some accommodation reached after a week, accompanied by a sense that it was a good experience for the family to have lived without TV if only for a week and that they had learned from the experience. Some families change their habits for good ,but most don't, typically returning to their former levels of viewing very quickly. The habit of viewing is readily formed, and socially sanctioned and supported within the context of the home and family, and thus the habit can be very difficult to break (see tips for reducing viewing, below).

A Television reduction program that worked. Perhaps the most successful group attempt to help get a number of children to successfully reduce their viewing was recently developed by Barbara Brock and described in her book, *Living outside the Box: TV-Free Families Share Their Secrets* (2007)

Bock, a teacher in eastern Washington State, worked with 130 fourth-, fifth- and sixth- graders from six different classrooms in 2003 and 2005. The experiment in giving up television for a full month worked better than Brock

or anyone, in advance, would have dreamed possible. There was a tremendously high compliance rate—keep in mind how hard, historically, it has been to get people, adults or children, even to try to give up the box for a week.

Hardly any of the children quit after a full month, and some continued on afterward and reported great benefits, all recounted in Brock's book. And it is clear to me, at least, that the group camaraderie and the knowledge of being part of the project are what made the difference. Had just one or two children tried to do it, they might not have succeeded and it wouldn't have been so easy to accomplish as it turned out to be.

Barbara Brock is to be given great credit for conducting this experiment and taking measurements before, during, and after the TV-free month. The most important finding, however, is that it was the group morale that made her experiment work. That hadn't really been a big part of her expectations, but now that the results are clear and to my knowledge unprecedented, in my opinion, the message can go forth that there *is* a way for children and families, since siblings and parents also participated to greater or lesser extents, to learn what it is like to go without television for an extended period of time.

I'm not a Luddite and I've never advocated that people stop viewing; after all, I love many television programs (see, e.g., Kubey, 2004). I just believe that we need to be more discriminating in how we use television and all other media. But families and children can rediscover strengths, activities, and hobbies that they didn't know they had or had forgotten about when they try a TV-turnoff week, or even a few days, and see how life changes in comparison with life when viewing four to five hours a day every day.

In sum, although there is not a great deal of hard empirical evidence, it does seem likely that some individuals—and perhaps entire families—go through something akin to withdrawal if television suddenly disappears. Furthermore, in congruence with section "b" of this criterion, other enjoyable leisure and media activities are typically used to replace TV viewing for those trying to give it up. It is also interesting to note that television is sometimes used by individuals seeking to withdraw from drugs such as heroin, cocaine, and alcohol as a less harmful means of escape and distraction (personal communication from Dyznskyi, October 20, 1994; Kubey & Csikszentmihalyi, 1990, pp. 184–185).

Criterion #3: "The substance is often taken in larger amounts or over a longer period than was intended" (p. 181).

It is common for viewers of all ages to report sitting down to watch just one program but ending up watching much more than was planned. Thus, this diagnostic criterion may fit many viewers.

In a Gallup Poll, 42 percent of the 1,241 U.S. adults who were surveyed reported that they spent too much time watching television (Gallup & Newport, 1990). Mander (1978) reported that some of the typical viewers he interviewed said things such as, "If a television is on, I just can't keep my eyes off it" and "I don't want to watch as much as I do but I can't help it. It makes me watch it" (p. 158).

Indeed, the viewing habit is so entrenched in many people that the choice to view is made almost automatically (Kubey, 1990a). Once dinner is done—or the dishes washed—many individuals sit down to watch television regardless of what programs are on.

Criterion #4: "There is a persistent desire or unsuccessful efforts to cut down or control substance use" (p. 181).

As noted above, it is common for people to report that they believe they spend too much time viewing. This belief itself appears to be on the rise. The percentage of adults in the United States who felt that they watched too much television in the late 1970s was 31 percent, 11 points lower than the 1990 figure of 42 percent (Gallup & Newport, 1990).

It is also relatively common for people to report that they feel powerless to stop viewing on their own, without abandoning the set altogether or interfering with it electronically (Daley, 1977). Some people have told me that they have given up their cable subscriptions precisely so that they have less choice and will thereby watch less. And as stated in *DSM–IV*, *technically* one need only have a "persistent desire . . . to cut down or control substance use" for the criterion to apply. Presumably, some of the Gallup Poll respondents would qualify.

Criterion #5: "A great deal of time is spent in activities necessary to obtain the substance . . . [or] . . . use the substance (e.g., chain smoking), or recover from its effects" (p. 181).

Clearly, with the vast majority of Americans spending two to four hours daily with television, or over half of all their leisure time, a great deal of time is spent using television.

Criterion #6: "Important social, occupational, or recreational activities are given up or reduced because of substance use." "The individual may withdraw from family activities and hobbies in order to use the substance in private" (p. 178).

There is a good deal of research showing that television can bring family members together, but also that it can reduce familial contact (Bronfenbrenner, 1973; Kubey, 1990b, 1990c; Maccoby, 1951; National Institute of Mental Health [NIMH], 1982). Not a few adults (e.g., so-called football widows) feel neglected by their partners who use television heavily. People have reported to

me that they feel that they must regularly compete with television personalities for the attention of family members (Kubey, 1994).

Many people also use television (not to mention other media) purposely to avoid contact with their family. Particularly disturbing is the suggestion that some children may be emotionally, and perhaps even physically, neglected because their caregivers are too engaged in television programs to attend to their needs (Desmond, Singer, & Singer, 1989; Shanahan & Morgan, 1989).

With regard to recreation, some viewers will necessarily engage less in other activities if they are spending three, four, or more hours each day watching television. For example, Williams (1986) found that adolescents and adults participated much less in community activities and sports when TV was available than when it was not.

As for occupational activities, there are undoubtedly people who bring work home from the office but do not do as much as they might (or perhaps do a lower-quality job) because of a television habit that is not under control.

Criterion #7: "The substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance" (p. 181).

As will be noted below, there may be a small percentage of people to whom this criterion could be applied, but the reference in the criterion to "knowledge" demands *awareness,* and awareness of having a significant physical or psychological problem due to TV use is probably rare. Still, it is almost certainly the case that *some* individuals recognize that their television viewing habit interferes with their social relations, level of physical exercise, or work habits. In these instances, television could be seen as exacerbating physical or psychological problems.

There *is* evidence that children and adolescents who view a great deal of television tend to be more obese than those who view less (Dietz & Gortmaker, 1985; Taras, Sallis, Patterson, Nader, & Nelson, 1989). And there is growing evidence now that a child's metabolism slows down when watching television. Furthermore, consumption of junk food by adult self-labeled TV addicts is higher than for nonaddicts (McIlwraith, 1991). Some people also report feeling more passive after viewing than before, and this passivity may decrease the likelihood that viewers will become involved in more active and potentially rewarding activities (Kubey, 1984; Kubey, 1990a: Kubey & Csikszentmihalyi, 1990).

There is also very strong evidence that children and adolescents who view a great deal of television tend to be more obese than those who view less. A study by researchers at Harvard, Tufts, and the New England Medical Center shows perhaps the strongest statistical relationships known among the thousands of studies on television viewing (Dietz & Gortmaker, 1985). This research dem-

onstrates a strong dose-response relationship between television viewing and weight. Even when controlling for a myriad of critical demographic variables, children ages 10–15 who watch over five hours each day were shown to be over five times more likely to be overweight than those who watch less than two hours. Put another way, 60 percent of this population's excess weight is attributed to viewing. Reduced exercise and more consumption of junk food (often advertised on TV) are implicated, as is the finding that a child's metabolism slows down when watching television.

SUMMING UP

As can be seen, when *DSM-IV* or the identical *DSM–IV–TR*'s diagnostic criteria are applied to television viewing habits, a diagnosis of substance dependence can be made for many people. The key missing feature, it would seem, is that we are not accustomed to thinking of television as a substance: it is neither a liquid (alcohol) nor a solid (a pill). Not to go too far, but technically, the viewing of television does, in some way, involve taking *something* into the body, even if that something is only light and sound, and even if no residue of the substance can later be found in the body. Though TV may not be a substance, millions of people nonetheless believe that they, or people they know, need to gain better control of their use of the medium. It is to that end that we next turn our attention. And it is the case that physicists believe that light is both wave-like and *particular*, that is, that it has particles.

One important caveat. Robert McIlwraith (1998) an experienced clinician, is dubious about actually including television dependence in the *DSM*, pointing out that there has yet to be a demonstration of a significant clinical impairment due directly to so-called television dependence. For McIlwraith, while a person not engaging in certain other activities due to their television habit may be social significance, it may well fall short of needing diagnosis. McIlwraith also points out that the establishment of a new diagnosis requires that we rule out more parsimonious explanations for the observed phenomenon in terms of already existing diagnoses such as depression, social phobia, agoraphobia, or avoidant personality disorder.

But the evidence that television is itself causative with regard to some behavioral or psychological phenomena cannot be denied. The comprehensive inventory of three Canadian communities in the early 1970s, two that had television and one that was just beginning to receive it, led Tannis MacBeth Williams and her colleagues to conclude that the use of television "seems unlikely to encourage the ability to tolerate aloneness with one's thoughts and ideas" (1986, p. 125). Williams's team also found that adolescents and adults participated

much less in community activities and sports when TV was available than when it was not. Again there is the indication of viewing leading to isolation and less activity and of viewing perpetuating itself.

In short, television viewing habits are often self-perpetuating. Viewing may lead to more viewing and may also elicit what has been described as *attentional inertia*, that is, the longer people look at television, the greater is the probability that they will continue to look.

Viewing begets more viewing, because one must generally keep watching in order to keep feeling relaxed (Kubey, 1984; Kubey & Csikszentmihalyi, 1990). A kind of psychological and physical inertia may develop. Although paying the bills might not have seemed difficult immediately after dinner, after two or three hours spent with TV, viewers become accustomed to having their experience effortlessly and passively structured. Getting up and taking on a more demanding task may begin to seem more formidable.

Within moments of sitting or lying down and pushing a TV set's power button, many viewers will report feeling more relaxed than they did before. And because relaxation occurs quickly, people readily learn to associate viewing with relaxation. The association is positively reinforced through simple operant conditioning because viewers remain relaxed throughout viewing, and it is negatively reinforced via the stress and dysphoric rumination that occurs during idle time or once the set is turned off.

The quick onset of relaxation is particularly telling when compared to that produced by certain drugs that are known to be habit forming or addictive. As Alvin Swonger and Larry Constantine (1976) have written: "The attribute of a drug that most contributes to its abuse liability is not its ability to produce tolerance or physical dependence but rather its ability to reinforce the drug-taking behaviors" (p. 235). This is why both the speed of a drug's effect and the speed with which it leaves the body are often critical factors in whether or not dependence develops. And, of course, reinforcement needn't be consciously experienced at all for it to occur or to be a powerful motivator of behavior.

Some tranquilizers, for example, whose half-lives are very short—half the drug leaves the body more rapidly compared to other drugs—are much more likely to cause dependence precisely because the user is more prone to become aware that the drug's effects are wearing off. In decades past, some attentive physicians were inclined to prescribe a less well-known tranquilizer, a benzodiazepine called Tranxene (clorazepate) because it was so slow-acting and much less likely than faster-acting benzodiazepines like Valium (diazepam), and especially Ativan (lorazepam), to be habit forming.

When a person starts to feel anxious more quickly while a tranquilizer with a short half-life's effect is on the wane, the tendency to turn to the drug again for relief will be that much greater than if its effects wear off more slowly.

Returning to the use of television, the change in mood that one experiences from the time of viewing to the time when one suddenly stops viewing may be key. The vague experiential knowledge on the part of viewers that one will feel less relaxed if one stops viewing may be a significant factor in not turning the set off.

And relative to other means available to bring about distraction and relaxation, television is perhaps the quickest, most readily available, and least expensive ever invented. Unlike conversation or games, one does not need anyone else to watch TV. And viewing provides more immediate and cheaper relaxation than that involving drugs or alcohol.

As with any activity, too much of the same thing may not always be best, especially for children. If other activities and experiences are not occurring with the frequency that a caregiver or parent deems to be appropriate because of a television, video game, or Internet habit, then, in my opinion, it is surely the right and responsibility of the caregiver to limit such activities. We must also encourage children to develop their own internal self-monitoring abilities so that they can increasingly make these determinations for themselves.

And we should encourage media education, which has been required in schools nationwide across Canada and Australia for two decades now. We need to encourage children's ability to critically analyze the media and to make them more mindful the use of all media (Kubey, 1991; Macedo & Steinberg, 2007).

Is viewing addictive? If we only mean by the term that one can easily develop dependence on the activity, the answer would be "yes." But to be more properly classified as a true dependence or addiction, an activity must also be harmful, interfering with the quality of the rest of one's life. And so, on this score, the answer is "it depends." Most people can benefit from viewing without the activity interfering with the rest of their lives. In its easy provision of relaxation and escape, in small doses it can be beneficial. For lonely people without other resources, it may be wonderful; I have heard this from many older individuals or people stricken with an illness that makes it hard or impossible for them to read.

But when the viewing habit interferes with the ability to grow, to learn new things, to lead an active life, then viewing indeed becomes an obstacle in life.

VIDEO GAMES, COMPUTER GAMES, AND THE INTERNET

Parents are often concerned about their children's heavy use of computers, video games, and the Internet. We can readily apply many of the same explanations offered earlier, as to how television dependence develops, to help explain the attraction of these games, and in some instances the attraction of the Internet. As with television, they offer escape and distraction, and as with

television, players quickly learn that they feel better when playing, hence a kind of reinforcement loop develops.

But computer and video games also pose challenges and the opportunity to overcome them—something largely or entirely missing from much television viewing—and most importantly, many games are designed to minutely increase in difficulty along with the increasing ability of the player.

In being programmed to constantly challenge a player's current ability, video and computer games offer a nearly perfect level of difficulty for the player who enjoys such challenges. And psychic pleasure—what Csikszentmihalyi (1990) has called flow—does accompany the improvement of one's skills and increased mastery of almost any human endeavor (the application of flow to video game play was first discussed by Kubey & Csikszentmihalyi (1990, pp. 143–144).

Before the introduction of video and computer games, one could search for months to find another tennis or chess player of comparable ability, but many programmed games immediately provide a near perfect matching of the challenges with one's skills. Thus, computerized games make extended play extraordinarily common because one is feeling neither bored by too easy an opponent nor too frustrated by an inability to match the level of competition. Plus, players are highly motivated to keep playing to achieve a new personal best score and are being psychologically rewarded throughout the game as they see themselves succeeding.

In computer play, as with sports, musical performance, and other activities that induce flow, the feedback is quick and clear, and insofar as it often occurs at the height of one's own personal level of performance, it is little wonder that the games are extremely engaging and, hence, for some they feel addictive.

The latest media addiction to emerge is so-called Internet addiction As with video and computer games, the Internet is also interactive *and,* importantly, it can be readily used to sustain or form social relationships.

Being connected with others when one is alone, any time of day, and so inexpensively is very attractive, and there can be little doubt that some individuals have grown dependent on the Internet for social contact. A small minority of new college students—perhaps 5 percent—are using the Internet so much that it appears to interfere with their social adaptation to college life, and for some, with their academic performance (Kubey, Lavin, & Barrows, 2001). New students can readily use the Internet to stay in touch with their old high school friends, and in maintaining these safe and familiar social links, or engaging in other parasocial activities on the Internet, a handful of students may be avoiding making new friends on campus.

Indeed, for growing numbers of us, the life we lead online may seem more important, more immediate, and more intense than the life we lead face-to-face.

We're not prepared to draw the conventional conclusion that being with people in real time is absolutely better than being with people on the Internet, but as with other activities, Internet activity can begin to eclipse the rest of life for some, and wisdom suggests that we at least raise the possibility that some of us have developed a habit of some significance that might be interfering with the quality of our relationships with those immediately around us.

CONTROLLING VIEWING AND OTHER MEDIA HABITS

There are ways in which individuals and families can achieve better control of their viewing habits, if that is their goal.

Raising awareness. As with other habits and dependencies, an early critical step is to become aware of how entrenched the viewing habit has become, how much time it absorbs, and the limited rewards of viewing. One way to do this is to keep a diary for a few days of all programs viewed. Some people may be assisted by also rating the quality of their experience with TV, noting how much they enjoyed or learned from various programs. Adding up the hours at the end of the week can be quite sobering. Multiplying by 365 can be even more so.

Promoting alternative activities. All too many individuals and families view automatically: that is, as soon as dinner is done, they watch television. To help break the repetitive, habitual, and self-perpetuating nature of the habit, people need to replace television viewing with other activities. Generating a list of enjoyable and/or constructive activities that can be done in or around the home may prove particularly helpful. The list might be posted on the refrigerator. (Not on the television if using a magnet, lest you harm the cathode ray tube.) Using such a list of enjoyable leisure activities has proved effective for patients suffering from mild depressive episodes. Instead of reflexively going to the television as soon as dinner is done, those interested in reducing their viewing can go to the list. (See the section above on a viewing reduction program for children that worked.)

Exerting willpower. Viewers often know that a particular program or movie is not to their liking within the first few minutes, but instead of switching off the set, they view for the full two hours. It can be helpful to consider that just five or ten minutes after turning off an only somewhat interesting mystery story, most viewers will rarely care what was going to happen next.

Enforcing limits. In addition to setting overall limits on viewing for one's self or one's children, one tip that works particularly well with younger children's video game or computer play is to tell a child, and his or her friends, that they may only play for some specific period. This might be 20 to 40 minutes. It

can be very effective to use a kitchen timer to set the time deemed appropriate. When it rings, the kids know to stop. For some parents, this works much better than announcing the deadline themselves. The kids may actually take the bell more seriously than the parent's words. For an extended discussion of parental responsibility for children's viewing habits, please see the section below.

Blocking channels/v-chip. Viewers can block particular channels or content. Nowadays, television sets come equipped with microchips that can be used to program the set to avoid violence. There are also electronic devices that can be attached to a television set and can be set to count how many hours each family member has viewed and not to permit access beyond a particular level.

Viewing selectively. Using a television guide can be helpful in cutting down on TV viewing. One chooses specific programs to watch ahead of time and then watches only those programs that have been preselected.

Using the VCR or recording device. A VCR, DVD-R, or Tivo can be very effective in time shifting and reducing time spent viewing. Many viewers never return to much of the material they've recorded.

Making television less available or going cold turkey. Many families have succeeded in substantially reducing viewing by limiting the household to one TV set and placing it in a remote room, often one with less than ideal seating. Others keep a set in a closet and bring it out only a few times a year for a particular event. Others end their cable subscription or disconnect the set from an antenna, only using the set to watch videotapes. And thousands of people jettison the set altogether. A group called TV-Free America will send a kit of free information about how to encourage complete abstinence or organize one's school or community for a TV-turnoff week.

Parental Responsibility for Children, with Regard to Dependent and Heavy Viewing

In making decisions and judgments about what is in the best interests of a child, and especially about children and media, it is critical to remember that every child is simultaneously unique and changing—often rapidly. Furthermore, every medium is different, and TV shows, video games, movies, and books offer an enormous range of content, form, and style. Still, there are some similarities among most children at different ages and across media and programs, games, and stories. So while idiosyncratic judgments with regard to each child are critical, there are also some general observations that can be made.

Today, we probably have more children at risk for developing a dependency on television than ever before. I have observed, as have others (personal com-

munication from James Hutchinson, M.D., April 1994), that a substantial number of parents do not believe that they can, or should, control their children's viewing. Some believe that there is no potential harm in anything that a child might watch. They believe that children can negotiate the television text on their own.

Many parents have reported to me that it is beyond their ability to limit their children's viewing. None of us wants to be a dictator, but in my opinion parents should not back off making decisions with regard to what their children may view on television. If parents are not in reasonable control of their households, and their children and their activities, we might conclude that the socialization process is, at best, undergoing change. At worst, we might expect all manner of social problems to ensue (Kubey, 1994, 1996).

Parental monitoring of children's viewing is important because there are many programs and materials that are unsuitable for some children, young children in particular. I am especially concerned about reality programming and news programs that often engender unnecessary and substantial fear. Indeed, I am as concerned about the fear-induction effects of violence as I am about the potential modeling effects (Kubey, 1987).

Children sometimes need to be supervised in their use of television, video games, computers, and other media, just as they sometimes need supervision when carving a pumpkin, walking downstairs, or riding a bicycle on the street. The idea that a great many different children's activities need to be monitored from time to time but somehow only media activities can be completely unsupervised is thoroughly illogical to me, yet some hold to such a position.

What I have just written will strike most readers as obvious. I have made these points for two reasons. The first is that some media scholars and researchers, most often those allied with the cultural studies approach to media studies—an approach that has made many important contributions to our understanding of how audiences experience and understand the media—believe that parents ought never to censor a child's media experience or prohibit a child from partaking in any medium that she might wish to experience. Some believe it is a presumptuous and arrogant act for a parent to intrude on or censor the media experience of a child.

For theoretical, political, and pedagogical reasons, some theorists have concluded that the media are a different kind of stimulus or phenomenon from things such as fire, dangerous strangers, candy, and unguarded cliffs and stairs. Television shows, books, and video games are all cultural products and can be actively negotiated by audience members, it is argued. Notably, much of the research on such negotiations, or readings, of media texts has been done with adolescents and adults, not children.

Indeed, some developmental psychologists have been criticized by some cultural studies advocates for being too proscriptive in their views about appropriate media content for children. There are leading cultural studies scholars who seem to suggest that the only media effect with which we need to concern ourselves is what these theorists see as the negative effect of developmental psychologists and other authorities pontificating to the culture at large through the media and causing moral panic about the potential harmfulness of media (Buckingham & Sefton-Green, 2001).

My second reason for emphasizing the need for caregivers to supervise children's media use is that many parents report that they were not restricted in their own viewing when they were young and yet they often watched a lot of violence on television. Insofar as they believe *they* were not psychologically harmed in any way, they now believe that they can safely permit their children to watch whatever they like.

Regardless of this belief, one only has to go to a movie rated PG-13 or R to observe parents or other caregivers with three- and four-year-old children on their laps or in the seat beside them witnessing material that is by most measures incredibly violent, frightening, and horrific. There is material that I myself turn away from on a large-screen film and I am in my mid-50s. I am doubtlessly being judgmental here in the view of some readers, but it is my view that young children should not be exposed to horrific violence or graphic sex when they are highly impressionable.

With regard to the idiosyncrasies of different children and the differences among media, let me relate a final story. My older son, now 23, was six when he first watched *The Wizard of Oz* on TV, a favorite movie of mine that his mother and I concluded was now appropriate for his viewing, even though it contains frightening scenes of flying monkeys and melting witches. But with another child we might have been wrong. During one commercial break, at around 7:30 p.m. CBS promoted a story to be shown a few days hence on its popular program *60 Minutes*. The ad included both a voiceover and words on screen promoting a story entitled "Kids Killing Kids?" The promotion was dominated by news footage of a child being rushed into a hospital emergency room on a stretcher, wrapped in gauze and bleeding. My son was frightened by what he saw and heard, and no wonder. He wanted to know whether or not kids really killed kids. We talked about it for a while, though it meant missing Dorothy's initial meeting with the cowardly lion.

I am not recommending that children be constantly supervised, only that for many children, today's media offerings—from television to video games and the Internet—demand more vigilance on the part of parents than was the case 30, 40, or 50 years ago.

ACKNOWLEDGMENTS

I wish to thank Hartmut Mokros, PhD, and James Hutchinson, MD, for their helpful suggestions on the application of the *DSM* to this work in the first public presentation of these ideas at the 98th annual meeting of the American Psychological Association, Boston, August 1990.

NOTE

1. Page numbers cited here for the *DSM* refer to the 1994 edition but it is important to note that the wording has gone unchanged in the 2000 edition. It can be obtained online.

REFERENCES

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Bower, R. T. (1973). *Television and the public*. New York: Holt, Rinehart, & Winston.

Brock, B. (2007). *Living outside the box: TV-free families share their secrets*. Spokane and Cheney, WA: Eastern Washington University Press.

Bronfenbrenner, U. (1973). Television and the family. In A. Clayre (Ed.), *The impact of broadcasting,* (p. 20). London: Compton Russell.

Buckingham, D., & Sefton-Green, J. (2001). Multimedia education: Media literacy in the age of digital culture. In Kubey, R. (Ed.), *Media literacy in the information age: Current perspectives*. Information and Behavior, 6 (pp. 285–305). New Brunswick, NJ: Transaction Publishers.

Condry, J. (1989). *The psychology of television*. Hillsdale, NJ: Erlbaum.

Csikszentmihalyi, M. (1990). *Flow: The psychology of optimal experience*. New York: Harper & Row.

Daley, E. A. (1978). *Father feelings*. New York: William Morrow.

Desmond, R. J., Singer, J. C., & Singer, D. G. (1988). Family mediation and children's cognition, aggression, and comprehension of television: A longitudinal study. *Journal of Applied Developmental Psychology, 9*, 329–347.

Dietz, W., & Gortmaker, S. L. (1985). Do we fatten our children at the television set: Obesity and television viewing in children and adolescents. *Pediatrics, 75*, 807–812.

Furu, T. (1971). *The function of television for children and adolescents*. Tokyo: Sophia University Press.

Gallup, G., & Newport, F. (1990, October 10). Americans love—and hate—their TVs. *San Francisco Chronicle*, p. B3.

Goleman, D. (1990, October, 16). How viewers grow addicted to television. *New York Times*, p. C1.

Himmelweit, H., & Swift, B. (1976). Continuities and discontinuities in media usage and taste: A longitudinal study. *Journal of Social Issues, 32*, 133–156.

Kubey, R. W. (1984). *Leisure, television, and subjective experience*. Unpublished doctoral dissertation, University of Chicago.

Kubey, R. (1987). Testimony before the Subcommittee on Antitrust, Monopolies and Business Rights of the Committee on the Judiciary, United States Senate, on a television violence antitrust exemption, June 25 (Serial No. J-100027). Washington, DC: U.S. Government Printing Office.

Kubey, R. (1990a, August 5). A body at rest tends to stay on the couch. *New York Times*, Section 2, p. 27.

Kubey, R. (1990b). Television and family harmony among children, adolescents, and adults: Results from the experience sampling method. In J. Bryant (Ed.), *Television and the American family* (pp. 73–88). Hillsdale, NJ: Erlbaum.

Kubey, R. (1990c). Television and the quality of family life. *Communication Quarterly, 38*, 312–324.

Kubey, R. (1991, March 6). The case for media education. *Education Week, 10*, 27.

Kubey, R. (1994). Media implications for the quality of family life. In D. Zillmann, J. Bryant, & A. C. Huston, (Eds.), *Media, children, and family: Social scientific, psychodynamic, and clinical perspectives* (pp. 61–69). Hillsdale, NJ: Erlbaum.

Kubey, R. (1996). Television dependence, diagnosis, and prevention: With commentary on video games, pornography, and media education. In T. MacBeth (Ed.), *Tuning in to young viewers: Social science perspectives on television* (pp. 221–260). Newbury Park, CA: Sage.

Kubey, R. (2004). *Creating Television: Conversations with the people behind 50 years of American TV*. New York: Routledge.

Kubey, R. (2006). Television dependence. In *Encyclopedia of children, adolescents, and the media*. Newbury Park, CA: Sage.

Kubey, R., & Csikszentmihalyi, M. (1990). *Television and the quality of life: How viewing shapes everyday experience*. Mahwah, NJ: Erlbaum.

Kubey, R., & Csikszentmihalyi, M. (2002, February). Television addiction is no mere metaphor. *Scientific American, 286*, 74–80.

Kubey, R., Lavin, M., & Barrows, J. (2001). Internet use and collegiate academic performance decrements: First findings. *Journal of Communication, 51*, 366–382.

Lang, A. (2000). The Limited Capacity Model of Mediated Message Processing. *Journal of Communication, 50*, 46–70.

Lemish, D. (1987). Viewers in diapers: The early development of television viewing. In T. Lindlof (Ed.), *Natural audiences: Qualitative research of media uses and effects*. Norwood, NJ: Ablex Publishing..

Maccoby, E. (1951). Television: Its impact on school children. *Public Opinion Quarterly, 15*, 421–444.

Macedo, D., & Steinberg, S. R. (2007). *Media literacy: A reader.* New York: Peter Lang.

Mander, J. (1978). *Four arguments for the elimination of television.* New York: Morrow Quill.

McIlwraith, Robert (1998). "I'm addicted to television": The personality, imagination, and TV watching patterns of self-identified TV addicts. *Journal of Broadcasting and Electronic Media, 42,* 371–386.

McIlwraith, R., Jacobvitz, R. S., Kubey, R., & Alexander, A. (1991). Television addiction: Theories and data behind the ubiquitous metaphor. *American Behavioral Scientist, 35,* 104–121.

National Institute of Mental Health. (1982). *Television and behavior: Ten years of scientific progress and implications for the eighties* (Vol. 1). Rockville, MD: U.S. Department of Health and Human Services.

Reeves, B., & Thorson, E. (1986). Watching television: Experiments on the viewing process. *Communication Research, 13,* 343–361.

Rosenberg, M. (1978). *Television and its viewers.* Radio broadcast of conversations at University of Chicago.

Ryan, B. H. (1974, June 9). Would you free your children from the monster? *Denver Post.*

Shanahan, J., & Morgan, M. (1989). Television as a diagnostic indicator in child therapy: An exploratory study. *Child and Adolescent Social Work, 6,* 175–191.

Singer, J. (1980). The power and limitations of television: A cognitive-affective analysis. In P. Tannenbaum (Ed.), *The entertainment functions of television* (pp. 31–65). Hillsdale, NJ: Erlbaum.

Steiner, Gary. (1963). *The people look at television.* New York: Alfred A. Knopf.

Swonger, Alvin, K., and Constantine, Larry, L. (1976). *Drugs and therapy: A psychotherapist's handbook of psychotropic drugs.* Boston: Little, Brown & Co.

Tannenbaum, P. (1980). Entertainment as vicarious emotional experience. In P. Tannenbaum (Ed.), *The entertainment functions of television* (pp. 107–131). Hillsdale, NJ: Erlbaum.

Taras, H. L., Sallis, J. F., Patterson, T. L., Nader, P. R., & Nelson, J. A. (1989). Television's influence on children's diet and physical activity. *Journal of Developmental and Behavioral Pediatrics, 10,* 176–180.

Williams, T. M. (Ed.). (1986). *The impact of television: A natural experiment in three communities.* New York: Academic Press.

Winick, C. (1988). The functions of television: Life without the big box. In S. Oskamp (Ed.), *Television as a social issue,* (pp. 217–237). Newbury Park, CA: Sage.

Winn, M. (1977). *The plug-in drug.* New York: Viking.

This page intentionally left blank

Chapter **3**

Excessive Buying as a Genuine Addictive Behavior

Paul Rose, PhD, and Dan J. Segrist, PhD

Addiction scientists have not always agreed on what is and is not an addictive behavior. Although there has been some longstanding consensus that particular substances can be addictive (e.g., alcohol, cocaine, heroin), cultural and technological evolution has forced us to reconsider whether the concept of addiction can also apply to certain behaviors. Can work be addictive? What about viewing soap operas, gambling, or playing video games? Anecdotal evidence suggests that these behaviors might sometimes reach "out of control" levels that produce clinically significant dysfunction or distress, but if the list of addictive behaviors is not carefully limited, the most dangerous addictive behaviors risk being trivialized by their association with less serious self-control problems.

The primary purpose of this chapter is to present the case for classifying excessive buying as an addictive behavior. While presenting this case, we wish neither to understate nor to overstate the gravity of the problem. In fact, we recognize that there may be several addictive behaviors that have worse consequences than excessive buying. But the primary issue with which we are concerned is whether the existing literature warrants the grouping of excessive buying with other behaviors that are widely accepted as addictive. To present the case for classifying excessive buying in this way, we first describe the nature of excessive buying and the general characteristics of people who are prone to it. We then present well-accepted definitions of addiction and demonstrate "goodness of fit" between existing definitions of addiction and excessive buying as it is presently understood. Following this, we emphasize that classifying excessive buying as a genuine behavioral addiction may stimulate considerable progress

in our understanding by facilitating knowledge transfer between the study of other addictive behaviors and the study of excessive buying.

WHAT IS EXCESSIVE BUYING?

Excessive buying is an enduring purchasing pattern that has reached a level so high that it is deemed problematic by either the consumer or people close to the consumer (such as family and friends). There are many reasons why such behavior might be experienced as problematic. Excessive buying can undermine a consumer's financial security (which has the potential to erode well-being; Drentea, 2000; Drentea & Lavrakas, 2000; Hatcher, 1994) but may also have harmful effects on relationships (Andersen, 2005; see also Dean, Carroll, & Yang, 2007) and mental health (Christenson et al., 1994). This behavior also has obvious negative consequences for the earth's ecosystem and may have indirect effects on other, international-scale problems, but in this chapter our focus is on excessive buying as an individual and interpersonal problem. We use the term *excessive buying* because this term is relatively free of unnecessary assumptions about the essence of the behavior (cf. Dittmar, 2004a; Swan-Kremeier, Mitchell, & Faber, 2005).

In empirical studies, excessive buying is most often operationalized with measures of "compulsive buying" (e.g., Edwards, 1993; Faber & O'Guinn, 1992; Valence, d'Astous, & Fortier, 1988). However, questionnaires labeled as indices of "impulse buying" (Mick, 1996; Rook & Fisher, 1995), "addictive buying" (Scherhorn, Reisch, & Raab, 1990), and (low levels of) "frugality" (Lastovicka, Bettencourt, Hughner, & Kuntze, 1999) appear to capture the same construct. Indeed, although each of these measures has been labeled differently, all of them measure the propensity to buy at high levels in spite of negative consequences. For example, the Valence et al. compulsive buying measure contains the item "When I have money, I cannot help but spend part or the whole of it"; the Rook and Fisher (1995) impulsive buying measure contains the item "Sometimes I am a bit reckless about what I buy"; and the Lastovicka et al. (1999) frugality measure contains the item "I believe in being careful in how I spend my money." As this sample of items suggests, the differing names of these scales belie the possibility that the scales assess a common latent construct.

Although no formal assessment of whether these various measures are actually measuring different constructs has been conducted, the evidence of which we are aware is consistent with a common-construct view. In unpublished data (Rose & Segrist, 2008), we have observed (with data obtained from a sample of 183 undergraduates) that scores on measures of impulse buying (Mick, 1996), compulsive buying (Faber & O'Guinn, 1992), and frugality (Lastovicka et al.,

1999) substantially intercorrelate (all *r*'s > |.42|; see also Troisi, Christopher, & Marek, 2006). (Frugality correlated negatively with impulse and compulsive buying, which correlated positively with each other.) All three measures also produced correlations of magnitude (*r*'s ranging from |.37| to |.43|) similar to those of scores from Eysenck, Pearson, Easting, and Allsopp's (1985) Impulsiveness Questionnaire. At this point, therefore, it seems justified to assume that measures labeled as indices of compulsive buying, impulse buying, addictive buying, and frugality are at least approximately assessing the same construct.

Having stated this, an appreciation of the equifinality principle should compel us to recognize that there may be several very different routes by which consumers arrive at an excessive level of buying (cf. DeSarbo & Edwards, 1996). For instance, it has been repeatedly observed that compulsive buying is related to low self-esteem (e.g., d'Astous, 1990; Faber & O'Guinn, 1992; Scherhorn et al., 1990; cf. Valence et al., 1988), but it has also been observed that compulsive buying is related to narcissism (Rose, 2007). These findings may seem somewhat surprising in light of ample evidence that trait narcissism and trait self-esteem are positively related (Campbell, Rudich, & Sedikides, 2002; Rhodewalt & Morf, 1995; Rose, 2002). (Initial evidence, based on clinical speculations, that people with narcissistic personalities might dislike themselves "deep down" [i.e., beyond the reach of the questionnaires; Jordan, Spencer, Zanna, Hoshino-Browne, & Correll, 2003] has recently been contradicted by additional evidence [Campbell, Bosson, Goheen, Lakey, & Kernis, 2007].) These and other findings suggest that excessive buying, like many other addictive behaviors, may arise from a variety of different traits.

WHO BUYS EXCESSIVELY?

Until recently, little was known about the demographic and personality characteristics of excessive buyers. But primarily within the last two decades, numerous studies have provided a clearer picture of the background characteristics of these consumers.

Gender. In a recent review of compulsive buying research, Dittmar (2004b) noted that many studies suggest that compulsive buying is far more common among women than among men (e.g., d'Astous, 1990; Faber & O'Guinn, 1992; Scherhorn et al., 1990). However, she also acknowledged that the gender difference that has emerged in some studies may be exaggerated because of the sampling techniques that some researchers have used. Many researchers have recruited participants who self-identified as having buying problems, and because participants often volunteer for such studies with the hope of receiving

some help with their problem, women may have been especially likely to volunteer. After all, research has consistently demonstrated that women hold more favorable attitudes toward seeking help, including help for their psychological problems (see Mosher, 2002). For example, with regard to gambling problems, Ladd and Petry (2002) found that, among first-time treatment seekers, women sought treatment significantly sooner than men did. In addition, with regard to alcohol problems, Thom (1986) found that women were less likely than men to report that the belief that one should be able to resolve one's own problems was a barrier to seeking treatment (a finding that further attests to women's greater willingness to seek help for their problems).

Given that women generally seem more prone to seeking treatment than men do, and given that many research participants volunteer for studies with the hope of eventually getting help with their problems, it seems likely that earlier studies may have overestimated the degree to which excessive buying is more common among women than among men. Indeed, a recent phone survey conducted with a random sample of Americans indicated that 6.00 percent of women and 5.50 percent of men could be classified as compulsive buyers using Faber and O'Guinn's (1992) screening instrument (Koran, Faber, Aboujaoude, Large, & Serpe, 2006). Furthermore, in two nationally representative surveys conducted in Germany, women scored only moderately higher (i.e., .31 of a standard deviation higher) than men on Scherhorn et al.'s (1990) addictive buying scale (Neuner, Raab, & Reisch, 2005). The most reasonable conclusion at this point is that women are somewhat more prone to excessive buying, but the difference is not nearly as dramatic as some small-sample studies previously suggested. The magnitude of the gender difference may also vary across cultures.

Age. Existing data support the commonly held idea that young adults and teenagers experience more buying problems than older adults. In Neuner et al.'s (2005) surveys, Germans between 14 and 32 years of age scored higher in addictive buying than Germans between 33 and 49 years of age, and this middle-aged group scored higher than Germans older than 49. Using a sample of 190 Canadians (ranging in age from under 20 to over 74), d'Astous (1990) observed a negative association between age and compulsive buying. Similarly, in two surveys of British adults (with a few adolescents included in the first survey), Dittmar (2005) also found negative associations between age and compulsive buying. Negative correlations have also emerged in U.S. samples (Koran et al., 2006; Troisi et al., 2006). Dittmar (2005) uncovered evidence that materialistic values mediate the age-compulsive buying relationship, which suggests that teenagers and younger adults are the most susceptible to excessive buying because they are more materialistic than older adults.

Socioeconomic status (*SES*). In the United States, Canada, and Germany, it appears that excessive buying is negatively related to SES. In Koran et al.'s (2006) American survey, compulsive buyers were more likely to have incomes lower than $50,000 per year than to have incomes above that amount. In a study of 135 adult (nonstudent) Americans, Rindfleisch, Burroughs, and Denton (1997) observed a small negative relationship between socioeconomic status and compulsive buying. In d'Astous's (1990) Canadian survey, a negative relationship also emerged between socioeconomic status and compulsive buying, although the very lowest SES group scored no higher than the second-lowest group. In addition, Scherhorn et al. (1990) reported a negative correlation between addictive buying and SES in their sample of German participants (who were not preselected for having buying problems).

Values. Values guide behavior toward desired ends (Rokeach, 1973) and may play an important, and perhaps underappreciated, role in the development of addiction problems (see Rose, 2007). Materialistic values are the most obvious values connected to excessive buying, and research confirms that measures of compulsive buying (Dittmar, 2005; Yurchisin & Johnson, 2004), impulsive buying (Rose, 2008), and frugality (Lastovicka et al., 1999) are all predictably correlated with materialism. Further insight into the values of excessive buyers comes from research on the broader values of materialistic consumers. Materialism is negatively correlated with the valuation of family, community, religion, universalism (care for all living things), and benevolence (concern for the welfare of close others); it is also positively correlated with the valuation of power, pleasure, achievement, and work (Burroughs & Rindfleisch, 2002). In addition, fame and image values (i.e., the valuation of popularity and looking good) are also positively correlated with materialism (Grouzet et al., 2005). These associations have been observed with *materialism*, however, so additional research is needed to determine whether excessive buying itself yields a similar pattern of correlations.

Personality. Because excessive buying is relatively stable over time, researchers have devoted substantial attention toward identifying how it is related to enduring aspects of personality. Excessive buying may be related to all of the big five personality traits (the basic traits thought to underlie all major dimensions of personality; Wiggins & Trapnell, 1997) except openness to experience (Mowen & Spears, 1999; Troisi et al., 2006). Several studies indicate that neuroticism, the tendency to consistently experience a variety of negative emotions, is positively related to excessive buying (Mowen & Spears, 1999; Stone, 2002), although the relationship is sometimes weak (e.g., Verplanken & Herabadi, 2001). As previously mentioned, excessive buying is also negatively related to self-esteem (d'Astous, 1990; Faber & O'Guinn, 1992; Scherhorn et al., 1990;

cf. Valence et al., 1988), and self-esteem is strongly negatively related to neuroticism (Robins, Tracy, Trzesniewski, Potter, & Gosling, 2001). Impulsivity, which is considered a component of neuroticism (Jang, Livesly, & Vernon, 1996), may be the personality trait to which excessive buying is most strongly related (Rose, 2007; see also Eysenck et al., 1985). The association between neuroticism (and its components) and excessive buying is consistent with the hypothesis that, in the absence of strong inhibitions, buying is sometimes used as a means of escaping distress (cf. Kyrios, Frost, & Steketee, 2004; Valence et al., 1988;).

Evidence of a relationship between extraversion and excessive buying is mixed. Although some studies indicate a positive relationship (e.g., Stone, 2002; Verplanken & Herabadi, 2001), others suggest no relationship (Mowen & Spears, 1999). This mixed evidence may be reasonably attributed to researchers' use of different extraversion measures in different studies. Sensation seeking, which is a component of extraversion (Jang et al., 1996; Olino, Klein, Durbin, Hayden, & Buckley, 2005) but is not captured to the same degree by all extraversion measures, correlates positively with impulse buying (Troisi et al., 2006). In contrast, extraversion measures that primarily emphasize sociability tend to yield near-zero correlations with compulsive buying (Mowen & Spears, 1999). In short, whether there is a significant association between extraversion and excessive buying may depend on how extraversion is measured and defined.

Although the relationship is weak and does not emerge in all studies (cf. Verplanken & Herabadi, 2001), there appears to be a positive relationship between agreeableness (i.e., kindness) and excessive buying (Mowen & Spears, 1999; Stone, 2002). Strange as this association may initially seem, it is understandable in light of evidence that excessive buyers are highly susceptible to others' influence (d'Astous, 1990; see also Yurchisin & Johnson, 2004). The desire to gain others' approval is a characteristic of materialistic consumers in general (Rose & DeJesus, 2007), and an orientation toward pleasing others may make resisting marketing messages particularly challenging.

Of all of the big five personality traits, conscientiousness appears to be the only one that is negatively related to excessive buying (Mowen & Spears, 1999; Verplanken & Herabadi, 2001). Conscientious people probably tend to plan their purchases carefully, and considering established plans when confronted with a buying impulse may be an effective strategy for resisting the temptation to buy (cf. Dholakia, 2000). Consistent with this possibility, Bearden, Money, and Nevins (2006) have demonstrated that their measure of planning tendencies is positively related to frugality and negatively related to compulsive buying.

Beyond the big five, recent research has established that trait narcissism is positively related to compulsive buying (Rose, 2007), apparently because narcissists tend to be both materialistic and impulsive. Several motivational variables have also been linked to excessive buying. Mowen and Spears (1999) have established that the need for arousal (similar to sensation seeking) is positively related to compulsive buying, possibly because people with a strong desire to experience excitement tend to be more materialistic (Troisi et al., 2006). Furthermore, Verplanken and Herabadi (2001) have observed that the need for structure (i.e., the motive to simplify and reduce ambiguity) and the need to evaluate (i.e., the motive to assess things positively and/or negatively) are both inversely related to the cognitive aspects of impulsive buying, such as buying with little forethought. A cogent account of the reasons behind these associations will require additional research.

In summary, although people who buy excessively vary widely in their characteristics, it seems that women, adolescents and young adults, and people of lower socioeconomic status are somewhat more prone than others to excessive buying. However, personality traits and values may be more important risk factors than any of these demographic variables. All of the big five personality traits (except openness to experience) seem to be related to excessive buying, with neuroticism (and especially the more specific neuroticism facet, impulsivity) being the most consistent trait predictor that researchers have uncovered thus far. Among values, materialism clearly stands out as the value most closely related to excessive buying, and numerous research studies confirm that these two variables are positively associated.

WHAT IS AN ADDICTIVE BEHAVIOR?

To fairly assess whether excessive buying should be categorized as an addictive behavior, it is necessary to carefully consider how addiction should be defined. Shaffer (1996) noted that the addictions field is marked by "conceptual chaos" and that "clinicians, researchers, and policy makers are without a shared definition of addiction" (p. 463). Similarly, Doweiko (2002) observed that "there is no single definition of addiction and a universally accepted, comprehensive theory of addiction has yet to be developed" (p. 21, cited in Coombs & Howatt, 2005). Traditionally, however, addiction has been conceptualized as a phenomenon reserved only for psychoactive substances (Coombs, 2004; Holden, 2001; Orford, 2001; Shaffer & Freed, 2005), whereby an individual demonstrates tolerance and withdrawal as well as a variety of negative consequences subsequent to the increased use of a drug. Consequently, similarly problematic behaviors that are now known as behavioral or process addictions

(such as compulsive gambling) were excluded from the traditional framework of addictive behaviors. M. B. Walker (1989), for example, argued that gambling should not be considered an addictive behavior because, he contended, there is insufficient evidence of dependency or withdrawal, and dissimilar biochemical mechanisms are at work.

Peele (1995) pointed out that even the traditional conceptualization of addiction is a relatively recent phenomenon: "addiction was not applied especially to narcotics or alcohol until well into this century. Until the twentieth century, *addiction* simply meant liking to engage in a habit" (p. 22). Furthermore, Shaffer (1996) has argued that physical dependence is not a requisite indicator of addiction, and Smith and Seymour (2004) assert that physical dependence is "no longer our sole definition of addiction" (p. 26). In fact, while the current editions of the *International Classification for Diseases* (*ICD-10;* World Health Organization, 1990) and the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV–TR;* American Psychiatric Association, 2000) include tolerance and withdrawal as potential symptoms, neither is required for the diagnosis of substance dependence.

Unfortunately, the traditional view of addiction precludes many behaviors—behaviors that manifest dynamics quite similar to substance abuse and dependence—from fitting under the addiction umbrella. Orford (2001) asserts that because of the exclusive focus on drugs within the addictions field, "other forms of excessive appetite have been marginalized" (p. 2). Arguing against the narrow, traditional view of addiction, many authors have advocated that the concept of addiction can be generalized to other problem behaviors (e.g., Internet use; Young, 1996). For example, Peele (1995), a staunch critic of the disease model of addiction, suggests that in comparison with narcotics, activities such as gambling are more likely to be associated with a loss of control. Whelan, Steenbergh, and Meyers (2007) have also argued that gambling can be genuinely addictive. They noted that "Like other addictions, problem gambling includes loss of control, preoccupation, tolerance, withdrawal, escape, cravings, and other concomitant biopsychosocial problems" (p. 20).

Grant and Potenza (2005) point out that "mounting evidence supports phenomenological, clinical, epidemiological, and biological links between behavioral and drug addictions" (p. 303). Although M. B. Walker (1989) argued that the biochemistry underlying drug addiction is dissimilar to that of behavioral addictions such as gambling, the differences may be diminishing, as increasing evidence points to similarities between the neurological mechanisms implicated in substance-based addictions and behavioral addictions (Coombs & Howatt, 2005). For example, gambling has been shown to increase dopamine activity (Shizgal & Arvanitogiannis, 2003) and activate the brain's pleasure center (Coombs & Howatt, 2005). Within the area of eating disorders, Marrazzi and

Luby (1986) detail an "auto-addiction" model of anorexia implicating increases in endogenous opioids associated with starvation behaviors. They argue that this process contributes to the intractable nature of the disorder by creating an addictive cycle.

Other researchers have also pushed for a broader conceptualization of addictive behavior. Shaffer (1996) has asserted that "It is the relationship of the addicted person with the object of their excessive behavior that defines addiction" (p. 465), and Orford (2001) has argued that behaviors such as gambling, sex, and eating should be considered addictive behaviors because they "can get sufficiently out of hand" and "spoil the lives of many people" (p. 3). Coombs and Howatt (2005) have also conceptualized addictive behaviors broadly; they describe these behaviors as actions characterized by compulsive use, loss of control, and continued use in the face of harmful consequences. Consistent with these broader views, Grant and Potenza (2005) have outlined several similarities between drug and behavioral addictions: "(1) repetitive or compulsive engagement in the behavior despite adverse consequences; (2) diminished control over the problematic behavior; (3) an appetitive urge or craving state prior to engagement in the problematic behavior; and (4) a hedonic quality during the performance of the problematic behavior" (p. 304). With these perspectives in mind, we believe that one of the most reasonable definitions of addiction has been offered by Goodman (1990), who wrote that addiction is "a process whereby a behavior, that can function both to produce pleasure and to provide relief from internal discomfort, is employed in a pattern characterized by (1) recurrent failure to control the behavior (powerlessness) and (2) continuation of the behavior despite significant negative consequences (unmanageability)" (p. 1404).

A perusal of several major addiction journals attests to the fact that the zeitgeist within the addictions field is changing, leading to a more flexible characterization of what constitutes an addictive behavior. It is noteworthy that *Psychology of Addictive Behaviors*, a peer-reviewed journal of the American Psychological Association, reviews and publishes "articles on the full range of addictive behaviors, including alcohol use and addiction, drug use and misuse, eating disorders, tobacco and nicotine addiction, and *gambling and other excessive behavior patterns*" (emphasis added). Similarly, the leading journal *Addiction* "publishes peer-reviewed research on alcohol, illicit drugs, tobacco and *behavioural addictions*" (emphasis added).

DOES EXCESSIVE BUYING FIT WITHIN CURRENT CONCEPTUALIZATIONS OF ADDICTIVE BEHAVIOR?

Given the ways in which addictive behavior is now defined by many leading addiction scientists, we suggest that a behavioral pattern can be fairly classified

as addictive to the extent that the behavioral pattern (a) can produce pleasure (i.e., a "high") at least some of the time; (b) has reached an "out of control" level; and (c) persists in the face of harmful consequences (cf. Goodman, 1990). In this section, we demonstrate that excessive buying meets these criteria.

People who buy excessively experience buying as pleasurable. Many people seem to believe that buying can be a pleasurable experience, and both theory (Holbrook & Hirschman, 1982) and research (e.g., Cox, Cox, & Anderson, 2005) confirm that it can be a pleasurable experience in some circumstances. Some purchasing activities can even evoke excitement (Wakefield & Baker, 1998) and elation (Mano & Oliver, 1993). Indeed, the mere perception of a preferred product is associated with activation of the nucleus accumbens (Knutson, Rick, Wimmer, Prelec, & Loewenstein, 2007), a conglomeration of neurons in the forebrain that are involved in the experience of pleasure.

Consumers who buy excessively, however, seem especially prone to experiencing a rush of pleasure while purchasing. Faber and O'Guinn (1988) have demonstrated that compulsive buyers are much more likely than other consumers to report feeling high while shopping, and Lejoyeux, Mathieu, Embouazza, Huet, and Lequen (2007) have similarly shown that compulsive buyers find purchasing more gratifying than other consumers do. The strong high experienced by excessive buyers seems to stem from the purchasing act itself; both Valence et al. (1988) and Faber and O'Guinn (1988) have demonstrated that excessive buyers derive little satisfaction from owning the things they purchase.

People who buy excessively experience their buying as "out of control." A feeling of diminished volitional control is a cardinal feature of excessive buying. Qualitative studies have repeatedly documented that consumers who buy excessively feel as if their consumption is out of control (e.g., Dittmar, 2004b; Rook, 1987), and several measures of excessive buying include items that assess this experience. To provide a few examples (with italics added), Valence et al.'s (1988) compulsive buying measure includes the item "When I have money I *cannot help* but spend part or all of it"; Verplanken and Herabadi's (2001) impulsive buying measure contains the item "I sometimes *cannot suppress* the feeling of wanting to buy something"; and Lastovicka et al.'s (1999) frugality scale contains the item "There are things *I resist* buying today so I can save for tomorrow." Also, Faber and O'Guinn (1988) have demonstrated that, much more than other consumers, compulsive buyers report going on buying binges and feeling unable to stop.

The diminished sense of control experienced by excessive buyers seems very similar to that of people who experience other addictions. A substantial body of research suggests that diminished impulse control is associated with a wide variety of addictive behaviors (e.g., Acton, 2002; Miller & Brown,

1991), including excessive buying (Rose, 2007; Spinella, Yang, & Lester, 2007). Impulse control is partly heritable (Jang et al., 1996) and seems to be associated with prefrontal cortical functioning and the functioning of the orbitofrontal circuit in particular (Grace, Stout, & Malloy, 1999; Spinella, 2004). Indeed, O'Doherty, Kringelbach, Rolls, Hornak, and Andrews (2001) have demonstrated that experiencing hypothetical financial rewards and losses activates the orbitofrontal cortex. Given the existing research, therefore, we may hypothesize that suboptimal orbitofrontal functioning predisposes people to excessive buying and other impulse-control problems.

People who buy excessively persist in their purchasing in spite of adverse consequences. A third way in which excessive buying fits within current conceptualizations of addictive behavior is that it persists even when the consumer experiences harmful consequences from his or her buying. To provide evidence of this, in the section that follows, we document some of the negative consequences of excessive buying that research has uncovered thus far. Thereafter we demonstrate that excessive buyers persist in their extreme buying habits in spite of these negative consequences.

WHAT ARE THE ADVERSE CONSEQUENCES OF EXCESSIVE BUYING?

Financial stress and its sequelae. The most obvious negative consequence of excessive buying is that it undermines financial security (Faber, 2004). Financial problems may seem trivial in comparison to the negative effects of some other addictive behaviors, but in fact, such problems are likely antecedents of a wide range of health-, family-, and work-related problems. Financial strain is associated with general psychological distress (Whelan, 1992) and depressive symptoms in particular (Mills, Grasmick, Morgan, & Wenk, 1992). It has also been linked to smoking (Siahpush, Borland, & Scollo, 2003) and increases in alcohol consumption over time (when financial support from others is low; Peirce, Frone, Russell, & Cooper, 1996; see also Moos, Fenn, Billings, & Moos, 1989). Peirce et al.'s (1996) longitudinal study is an especially important contribution to our knowledge about the consequences of financial stress, because it rules out the possibility that financial problems are linked to alcohol use merely because purchasing large quantities of alcohol strains people's budgets. Peirce et al.'s (1996) study demonstrates increased drinking *following* financial strain (among people who have little financial support from others), suggesting that in some circumstances money problems may cause greater substance abuse.

Several studies also link financial difficulties with family problems. Financial strain among married people is associated with lower marital satisfaction,

both for the person reporting financial problems and for that person's spouse (Dean et al., 2007). Parents experiencing financial strain report higher levels of depression, as do their adolescent children (Clark-Lempers, Lempers, & Netusil, 1990). Moreover, children of parents with high levels of financial stress also experience more impulsive and antisocial behavioral problems (Takeuchi, Williams, & Adair, 1991).

Financial stress also seems to affect a variety of health problems. In O'Neill, Sorhaindo, Xiao, and Garman's (2005) survey of consumers participating in a debt management program, self-reported improvements in participants' financial situations were associated with reports of improved overall health. Although this finding (like all of the correlational findings in this section) raises questions about which way the causal arrow might point (because health problems might cause financial problems and vice versa), there are reasons to believe that financial stress might cause poorer health. For instance, Rosengren et al. (2004) found that compared to control patients, patients who had experienced a heart attack were more likely to report severe financial stress in the 12 months *prior* to their hospital admission.

Financial stress may also affect people's work lives, because employees experiencing greater financial stress exhibit lower job satisfaction (Bailey, Woodiel, Turner, & Young, 1998), lower organizational commitment (i.e., sense of attachment to the organization), and higher absenteeism (Kim & Garman, 2003). In short, financial stress seems to produce many of the same detrimental effects that other types of stress do, and these consequences are far from trivial. Because excessive buying has an obvious negative effect on many consumers' financial security, it may have an indirect effect on a wide variety of occupational, familial, and personal problems.

Affective distress. Excessive buyers seem to be highly prone to guilt and shame (Edwards, 1993; O'Guinn & Faber, 1989; Valence et al., 1988), emotions that are often classed with other "self-conscious" emotions (Tangney & Fischer, 1995). Studies suggest that guilt and shame are both related to hostility, anxiety, and depression as well as psychotic, paranoid, and obsessive-compulsive symptoms (Tangney, Wagner, & Gramzow, 1992). Shame in particular has been linked to posttraumatic stress symptoms and somatization symptoms, although guilt may also be indirectly related to these symptoms through shame (Pineles, Street, & Koenen, 2006). These findings are consistent with research on both substance-based addictive behaviors (see Marlatt & Witkiewitz, 2005) and other behavioral addictions such as gambling (see Shaffer & LaPlante, 2005), in which people with addiction problems have elevated rates of mood disorders. It is important to note that among people with substance use prob-

lems, negative emotional states are strongly linked to relapse (see Marlatt & Witkiewitz, 2005). In future research, it would be helpful to examine whether this link between negative states and relapse might also exist for people who are in treatment for excessive buying.

One reason why excessive buyers may be prone to aversive self-conscious emotions is that they have some awareness that their buying is a serious personal problem (cf. Scherhorn et al., 1990). Self-report measures of excessive buying, and the many studies that employ them, clearly suggest that many compulsive buyers recognize that their buying habits are problematic. For example, the Rook and Fisher (1995) impulsive buying scale includes the item "Sometimes I am a bit reckless about what I buy"; and the Valence et al. (1988) compulsive buying scale includes the item "There are some things I buy that I do not show to anybody for fear of being perceived as irrational in my buying behavior ('a foolish expense'). These and similar items require the respondent to admit that he or she may have a buying problem, and suggest that excessive buyers are prone to feeling guilty about their purchases and ashamed of themselves. As suggested above, these feelings may predispose people to a wide variety of mental health problems.

Consistent with evidence that excessive buying is linked to affective distress (see also Scherhorn et al., 1990), Faber and O'Guinn (1988) have demonstrated that excessive buyers are more likely than other consumers to feel depressed after shopping. (It may seem that these consumers should be especially happy with their new purchases, but as previously mentioned, several studies suggest that compulsive buyers gain little satisfaction from the objects they buy. It is the buying act itself that thrills them; Faber & O'Guinn, 1988; O'Guinn & Faber, 1989; Scherhorn et al., 1990.) Although no research has clearly delineated the reasons for a link between excessive buying and depression, existing studies lead us to tentatively propose a model of how excessive buying might contribute to such distressing feelings. First, when a consumer buys something he believes should not have been bought, he should experience guilt (as people often do when they act contrary to their personal standards). Once guilty feelings arise, the bad act (buying when one shouldn't have bought) may be interpreted as evidence of a bad self; thus, guilt may give rise to shame (cf. Tangney et al., 1992). When shame arises, depressive thoughts and feelings may naturally follow, because negative self-views are central to the experience of depression. Although we provide this as a possible (and as yet untested) explanation for how excessive buying might contribute to depression, we recognize that depression might also engender excessive buying (especially among consumers who believe that buying can make them feel better).

DO PEOPLE WHO BUY EXCESSIVELY PERSIST IN SPITE OF ADVERSE CONSEQUENCES?

By definition, excessive buying is an enduring pattern of buying, and as we have shown above, adverse consequences follow from this enduring pattern. The question posed in the heading of this section is thus easily answered by reflecting on the definition of excessive buying and why it is considered a problem. If excessive buyers persist in their extreme buying habits even after they experience harmful repercussions, it seems clear that excessive buying, like other addictive behaviors, is characterized by maladaptive persistence. Indeed, items commonly used to measure excessive buying suggest that excessive buyers frequently fail to factor adverse consequences into their buying choices. "How often have you bought things even though you knew you couldn't afford them?" (from Faber and O'Guinn's [1992] compulsive buying measure) captures a tendency to keep spending at high levels in spite of negative outcomes. Other items that suggest a disregard for adverse consequences include "I have often bought a product that I did not need, while knowing that I have very little money left" (from Valence et al.'s [1988] compulsive buying measure) and "When I see something that really interests me, I buy it without considering the consequences" (from Weun, Jones, and Beatty's [1997] impulsive buying measure). The failure to consider the deleterious consequences of purchasing decisions is clearly central to the experience of persistent buying in the face of harmful consequences.

Qualitative data also reveal a pattern of maladaptive persistence among people who buy excessively. Excessive buyers interviewed by Dittmar (2004b) mentioned an awareness of having a problem, but a perceived inability to stay below the limit on a credit card, to cut up a credit card, or to pass by a store without buying anything. Similarly, O'Guinn and Faber (1989) interviewed an excessive buyer who said she could not stop even though (she believed) her husband and children hated her for her problem. Given the desperate tone of some of these statements, it seems that many excessive buyers do have a goal of reducing their buying, but the temptation to buy overpowers this goal.

FUTURE DIRECTIONS IN THE STUDY OF EXCESSIVE BUYING

One of the substantial benefits of recognizing that excessive buying should be classed among other behavioral addictions is that it may facilitate knowledge transfer between our currently rudimentary understanding of excessive buying and our more mature understanding of some other addictive behaviors. As fur-

ther research identifies similarities between excessive buying and other addictive behaviors, clinicians should have better knowledge to draw upon as they seek to help people who experience excessive buying problems. Toward this end, we suggest that researchers investigating excessive buying would do well to model lines of research that have already produced valuable insights into substance abuse and dependence. In the paragraphs that follow, we highlight just a few constructs from substance use research—several of which draw from Marlatt and Gordon's relapse prevention model (see Larimer, Palmer, & Marlatt, 1999; Marlatt & Donovan, 2005; Marlatt & Gordon, 1985)—that might be profitably applied to research on excessive buying. We stress that the concepts that follow are only a limited sample of the ideas from substance abuse research that could be used to build a better understanding of excessive buying.

Outcome expectancies. Research on alcohol consumption has demonstrated that outcome expectancies—"anticipated effects that an individual expects will occur as a result of alcohol or drug consumption" (Marlatt & Witkiewitz, 2005, p. 10)—are predictive of use (e.g., Brown, 1985; Smith & Goldman, 1994), beverage selection (Corcoran & Segrist, 1993), and consequences related to use (e.g., Blume, Lostutter, Schmaling, & Marlatt, 2003). Goldman, Del Boca, and Darkes (1999) note that since the late 1980s there has been "an explosion of research applying the expectancy concept to alcohol use" (p. 205). An important aspect of expectancies, as pointed out by Walters and Baer (2006), is that "Whether accurate or not, these beliefs are related to drinking" (p. 38). For example, despite the fact that alcohol can impair sexual desire and performance (e.g., Miller & Gold, 1988), individuals may *believe* that alcohol can increase their sexual prowess and behave accordingly.

Measures of alcohol expectancies typically include anticipated physiological, psychological, and social outcomes that serve as potential predictors of use. Some of the alcohol expectancies that have been researched are positive (e.g., those assessed by the Alcohol Expectancy Questionnaire; Brown, Goldman, Inn, & Anderson, 1980), whereas others are negative (such as those assessed by the Negative Alcohol Expectancy Questionnaire; Jones & McMahon, 1994). Some studies, such as those employing the Comprehensive Effects of Alcohol Scale (CEOA; Fromme, Stroot, & Kaplan, 1993) have examined alcohol expectancies that are both positive (e.g., tension reduction: *I would feel calm*) and negative (e.g., self-perception: *I would feel guilty; my problems would seem worse*). The expectancy concept has been applied to other substance behaviors, including expectations and motivations for smoking (e.g., Copeland, Brandon, & Quinn, 1995; Gilbert, Sharpe, Ramanaiah, Detwiler, & Anderson, 2000), as well as expectations about marijuana (Gaher & Simmons, 2007) and cocaine use (Schafer & Brown, 1991).

Recently, the expectancy concept has been used to build knowledge of non-substance addictive behaviors such as eating disorders (see Collins, 2005; Collins & Ricciardelli, 2005) and gambling (e.g., Raylu & Oei, 2004). Nevertheless, to our knowledge, the study of outcome expectancies associated with buying has been relatively neglected. One relevant study (Babin, Darden, & Griffin, 1994) involved the development of a scale to assess what participants expected to gain from their shopping. Results indicated that the measure assessed two factors: hedonic value (e.g., "While shopping I was able to forget my problems.") and utilitarian value (e.g., "I accomplished just what I wanted on this shopping trip."). Although the hedonic factor correlated with compulsive buying, it is important to note that the scale developed in this study assessed participants' experience of a *specific* shopping occasion, not the experience of shopping in general. Furthermore, there are undoubtedly many other outcome expectancies that predict excessive buying. Given the contribution that the expectancies construct has made to the assessment and treatment of other addictive behaviors, further exploration of buying-related outcome expectancies would seem a particularly fruitful avenue for future research.

High-risk situations. In summarizing Marlatt and Gordon's (1985) relapse prevention model, Larimer, Palmer, and Marlatt (1999) describe situations that "pose a threat to the person's sense of control and, consequently, precipitate a relapse crisis" (p. 153). These high-risk situations are typically characterized by any or all of the following components: negative affect, interpersonal conflict, pressure from others to engage in the problem behavior, and positive mood states (Larimer et al., 1999). The identification of high-risk situations, as well as the development of strategies for navigating them, is an important focus of treatment within the relapse prevention model.

How might applying the concept of high-risk situations to excessive buying be useful? At least some (if not all) of the high-risk situations described by Larimer et al. (1999) may precipitate buying binges, but none of these situations (other than negative affect) have been investigated as antecedents of excessive buying. Knowledge about such high-risk situations would be extremely valuable, as clinicians might need to help clients recognize and respond appropriately to situations that are high risk. If a client with an excessive buying problem is to successfully overcome a buying addiction, it seems crucial that he develop a repertoire of strategies for handling such high-risk situations.

Seemingly unimportant decisions. An element of Marlatt and Gordon's (1985) relapse prevention model centers around the decisions an individual makes that place her in a high-risk situation. Some of these decisions are referred to as seemingly unimportant decisions (SUDs; e.g., Wheeler, George, & Stephens, 2005) or apparently irrelevant decisions (AIDs)—"a series of covert decisions or choices, each of them seemingly inconsequential, which in combination set

the person up for situations with overwhelmingly high risk" (Larimer et al., 1999, p. 154). For example, at first glance, Cindy's seemingly unimportant decision to take a different route from work to her home may appear unrelated to her ultimately buying $900 worth of new clothes. But Cindy's choice of a different route actually led Cindy to drive by the mall, the sight of which gave her an overwhelming urge to buy, which ultimately led her to make several unplanned and unneeded purchases at considerable expense.

Individuals with excessive buying problems face a multitude of opportunities for SUDs (e.g., deciding to go to a Wal-Mart supercenter, instead of a grocery store, for a gallon of milk). In future research, it may be insightful to focus on the role of such decisions in triggering or sustaining excessive buying, with the ultimate goal of helping clients identify and appropriately respond to the subtle but powerful precursors to high-risk situations that lead to buying binges. Such research may eventually enable clinicians to help clients prone to excessive buying reconceptualize "impulse purchases" as acts preceded by a series of decisions that only *seemed* unimportant.

Stages of change. Prochaska, DiClemente, and Norcross (1992) proposed that individuals pursuing treatment for addictive behaviors progress through a series of stages: precontemplation, contemplation, preparation, action, and maintenance (also see Prochaska & Norcross, 2007). At the precontemplation stage, an individual has no interest in changing her behavior or is not aware of the potential consequences of the behavior (Dimeff, Baer, Kivlahan, & Marlatt, 1999). The contemplation stage entails the recognition of a problem, but no consistent dedication to change. The preparation stage is characterized by an intention to make changes soon. During the action stage, a client makes changes in his behavior or environment. Finally, a client in the maintenance stage works to sustain positive changes he has previously made and prevent relapse.

The stages of change model has been applied to several addictive behaviors including gambling (Petry, 2005; Shaffer & LaPlante, 2005), marijuana use (Walker, Roffman, Stephens, Berghuis, & Kim, 2006), eating disorders (Collins, 2005), smoking (Schuman et al., 2005), and others (Prochaska et al., 1994). However, we are not aware of any research on stages of change for people pursuing treatment for buying problems. Such research might be extremely useful to therapists, because as Blume (2004) has noted, "by establishing at what state of change a person might be, therapists can determine what might be the best therapeutic strategies to use with the patient" (pp. 89–90). Global measures to assess stages of change have been developed (e.g., University of Rhode Island Change Assessment: Long Form; McConnaughy, Prochaska, & Velicer, 1983) and so have specific measures designed to assess stages of change across a variety of behaviors (see *Cancer Prevention Research Center Measures*), including those of alcohol use (Alcohol Stages of Change: Short Form; Laforge,

Maddock, & Rossi, 1998), smoking (Smoking: Stage of Change, Short Form; DiClemente et al., 1991; Velicer et al., 1995), and health activities (General Health Survey; Nigg et al., 1999). Because understanding a client's motivation level has implications for treatment, creating measures to assess and further our understanding of stages of change in the context of excessive buying makes sense from both a research and treatment standpoint.

Social norm perceptions. The social norms framework has become an important approach for understanding substance use, particularly alcohol use (see Perkins 2003). Substance use research has consistently demonstrated a tendency among adolescents and young adults to believe that others use psychoactive substances more frequently and in greater quantities than oneself (e.g., Perkins, Meilman, Leichliter, Cashin, & Presley, 1999; Segrist, Corcoran, Jordan-Fleming, & Rose, 2007). These perceived norms, even though they are often inaccurate, are predictive of greater consumption (Perkins, Haines, & Rice, 2005). Consequently, social norms interventions seek to correct misperceptions of peer drinking behaviors (Perkins, 2003). Given the extremely high level of exposure people usually have to marketing messages that promote buying, it would not be surprising if many consumers similarly hold an inflated perception of how much buying is actually occurring among their peers. Furthermore, previous research on the association between perceived norms and substance use suggests that perceptions of high peer consumption may lead to greater personal consumption. From a treatment standpoint, an understanding of the role of norm perceptions in sustaining excessive buying may be particularly valuable, because these beliefs may need to be addressed and corrected before a client is ready to make significant changes in her buying.

In summary, a considerable benefit of recognizing that excessive buying is a genuine addictive behavior is that hypotheses that have already proven useful in the study of other addictive behaviors might be fruitfully applied to increasing our understanding, recognition, and treatment of excessive buying. The brief list of potentially applicable concepts that we have reviewed above could provide much-needed direction in the excessive buying literature, as could many other concepts in the addictions literature that researchers of excessive buying have yet to exploit. Ultimately, an extensive analysis of the ways in which excessive buying is similar to and different from other addictive behaviors should be helpful as therapists seek to develop effective and specific treatments for excessive buying.

CONCLUSION

Our primary objective in this chapter has been to demonstrate that excessive buying should be classed with other addictive behaviors. Toward this end, we

have briefly reviewed some of the characteristics of people who buy excessively, considered how addictive behaviors should be defined, and established that excessive buying fits within contemporary definitions of addictive behavior. To illustrate one potential benefit of classifying excessive buying as an addictive behavior, we have emphasized that considerable progress in theory construction and treatment might be made by applying concepts developed in the study of other addictive behaviors to research on excessive buying.

REFERENCES

Acton, G. S. (2002). Measurement of impulsivity in a hierarchical model of personality traits: Implications for substance use. *Substance Use and Misuse, 38,* 67–83.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Andersen, J. D. (2005). Financial problems and divorce: Do demographic characteristics strengthen the relationship? *Journal of Divorce and Remarriage, 43,* 149–161.

Babin, B. J., Darden, W. R., & Griffin, M. (1994). Work and/or fun: Measuring hedonic and utilitarian shopping value. *Journal of Consumer Research, 20,* 644–656.

Bailey, W. C., Woodiel, D. K., Turner, M. J., & Young, J. (1998). The relationship of financial stress to overall stress and satisfaction. *Personal Finances and Worker Productivity, 2,* 198–206.

Bearden, W. O., Money, R. B., & Nevins, J. L. (2006). A measure of long-term orientation: Development and validation. *Journal of the Academy of Marketing Science, 34,* 456–467.

Blume, A. W. (2004). Understanding and diagnosing substance use disorders. In R. H. Coombs (Ed.), *Handbook of addictive disorders: A practical guide to diagnosis and treatment* (pp. 63–93). Hoboken, NJ: Wiley.

Blume, A. W., Lostutter, T. W., Schmaling, K. B., & Marlatt, G. A. (2003). Beliefs about drinking behavior predict drinking consequences. *Journal of Psychoactive Drugs, 35,* 395–399.

Brown, S. A. (1985). Expectancies versus background in the prediction of college drinking patterns. *Journal of Consulting and Clinical Psychology, 53,* 123–30.

Brown, S. A., Goldman, M. S., Inn, A., & Anderson, L. (1980). Expectations of reinforcement from alcohol: Their domain and relation to drinking pattern. *Journal of Consulting and Clinical Psychology, 48,* 419–426.

Burroughs, J., & Rindfleisch, A. (2002). Materialism and well-being: A conflicting values perspective. *Journal of Consumer Research, 29,* 348–370.

Campbell, W. K., Bosson, J. K., Goheen, T. W., Lakey, C. E., & Kernis, M. H. (2007). Do narcissists dislike themselves "deep down inside"? *Psychological Science, 18,* 227–229.

Campbell, W. K., Rudich, E., & Sedikides, C. (2002). Narcissism, self-esteem, and the positivity of self-views: Two portraits of self-love. *Personality and Social Psychology Bulletin, 28,* 358–368.

Cancer Prevention Research Center Measures. Retrieved April 18, 2008, from http://www.uri.edu/research/cprc/measures.htm.

Christenson, G. A., Faber, R. J., de Zwaan, M., Raymond, N. C., Specker, S. M., & Ekern, M. D. (1994). Compulsive buying: Descriptive characteristics and psychiatric comorbidity. *Journal of Clinical Psychiatry, 55,* 5–11.

Clark-Lempers, D. S., Lempers, J. D., & Netusil, A. J. (1990). Family financial stress, parental support, and young adolescents' academic achievement and depressive symptoms. *Journal of Early Adolescence, 10,* 21–36.

Collins, R. L. (2005). Relapse prevention for eating disorders and obesity. In G. A. Marlatt & D. M. Donovan (Eds.), *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors* (pp. 248–275). New York: Guilford Press.

Collins, R. L., & Ricciardelli, L. A. (2005). Assessment of eating disorders and obesity. In D. M. Donovan & G. A. Marlatt (Eds.), *Assessment of addictive behaviors* (pp. 305–333). New York: Guilford Press.

Coombs, R. H. (Ed.). (2004). Handbook of addictive disorders: A practical guide to diagnosis and treatment. Hoboken, NJ: Wiley.

Coombs, R. H., & Howatt, W. A. (2005). *The addiction counselor's desk reference.* Hoboken, NJ: Wiley.

Copeland, A. L., Brandon, T. H., & Quinn, E. P. (1995). The Smoking Consequences Questionnaire—Adult: Measurement of smoking outcome expectancies of experienced smokers. *Psychological Assessment, 7,* 484–494.

Corcoran, K. J., & Segrist, D. J. (1993). Personal expectancies and group influences affect alcoholic beverage selection: The interaction of personal and situational variables. *Addictive Behaviors, 18,* 577–582.

Cox, A. D., Cox, D., & Anderson, R. D. (2005). Reassessing the pleasures of store shopping. *Journal of Business Research, 58,* 250–259.

d'Astous, A. (1990). An inquiry into the compulsive side of normal consumers. *Journal of Consumer Policy, 13,* 15–31.

Dean, L. R., Carroll, J. S., & Yang, C. (2007). Materialism, perceived financial problems, and marital satisfaction. *Family and Consumer Sciences Research Journal, 35,* 260–281.

DeSarbo, W. S., & Edwards, E. A. (1996). Typologies of compulsive buying behavior: A constrained clusterwise regression approach. *Journal of Consumer Psychology, 5,* 231–262.

Dholakia, U. M. (2000). Temptation and resistance: An integrated model of consumption impulse formation and enactment. *Psychology and Marketing, 17,* 955–982.

Doweiko, H. (Ed.) (2002). *Concepts of chemical dependency* (5th Ed.). Pacific Grove, CA: BROOKS/COLE.

Dimeff, L. A., Baer, J. S., Kivlahan, D. R., & Marlatt, G. A. (1999). *Brief alcohol screening and intervention for college students: A harm reduction approach.* New York: Guilford Press.

Dittmar, H. (2004a). Are you what you have? *The Psychologist, 4,* 206–210.

Dittmar, H. (2004b). Understanding and diagnosing compulsive buying. In R. H. Coombs (Ed.), *Handbook of addictive disorders: A practical guide to diagnosis and treatment* (pp. 411–450). Hoboken, NJ: Wiley.

Dittmar, H. (2005). Compulsive buying—a growing concern? An examination of gender, age, and endorsement of materialistic values as predictors. *British Journal of Psychology, 96*, 467–491.

Drentea, P. (2000). Age, debt and anxiety. *Journal of Health and Social Behavior, 41*, 437–50.

Drentea, P., & Lavrakas, P. J. (2000). Over the limit: The association among health, race and debt. *Social Science and Medicine, 50*, 517–529.

Edwards, E. A. (1993). Development of a new scale for measuring compulsive buying behavior. *Financial Counseling and Planning, 4*, 67–85.

Eysenck, S. B., Pearson, P. R., Easting, G., & Allsopp, J. F. (1985). Age norms for impulsiveness, venturesomeness and empathy in adults. *Personality and Individual Differences, 6*, 613–619.

Faber, R. J. (2004). Self-control and compulsive buying. In T. Kasser & A. D. Kanner (Eds.), *Psychology and consumer culture: The struggle for a good life in a materialistic world* (pp. 169–188). Washington, DC: American Psychological Association.

Faber, R. J., & O'Guinn, T. C. (1988). Compulsive consumption and credit abuse. *Journal of Consumer Policy, 11*, 97–109.

Faber, R. J., & O'Guinn, T. C. (1992). A clinical screener for compulsive buying. *Journal of Consumer Research, 19*, 459–469.

Fromme, K., Stroot, E., & Kaplan, D. (1993). Comprehensive effects of alcohol: Development and psychometric assessment of a new expectancy questionnaire. *Psychological Assessment, 5*, 19–26.

Gaher, R. M., & Simmons, J. S. (2007). Evaluations and expectancies of alcohol and marijuana problems among college students. *Psychology of Addictive Behaviors, 21*, 545–554.

Gilbert, D. G., Sharpe, J. P., Ramanaiah, N. V., Detwiler, F.R.J., & Anderson, A. E. (2000). Development of a situation x trait adaptive response (STAR) model-based smoking motivation questionnaire. *Personality and Individual Differences, 29*, 65–84.

Goldman, M. S., Del Boca, F. K., & Darkes, J. (1999). Alcohol expectancy theory: The application of cognitive neuroscience. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (pp. 203–246). New York: Guilford Press.

Goodman, A. (1990). Addiction: Definition and implications. *British Journal of the Addictions, 85*, 1403–1408.

Grace, J., Stout, J. C., & Malloy, P. F. (1999). Assessing frontal behavior syndromes with the Frontal Lobe Personality Scale. *Assessment, 6*, 269–284.

Grant, J. E., & Potenza, M. N. (2005). Pathological gambling and other "behavioral" addictions. In R. J. Frances, S. I. Miller, & A. H. Mack (Eds.), *Clinical textbook of addictive disorders* (pp. 303–320). New York: Guilford Press.

Grouzet, F.M.E., Kasser, T., Ahuvia, A., Dols, J.M.F., Kim, Y., Lau, S., et al. (2005). The structure of goal contents across 15 cultures. *Journal of Personality and Social Psychology, 89,* 800–816.

Hatcher, S. (1994). Debt and deliberate self-poisoning. *British Journal of Psychiatry, 164,* 111–114.

Holbrook, M. B., & Hirschman, E. C. (1982). The experiential aspects of consumption: Consumer fantasies, feelings and fun. *Journal of Consumer Research, 9,* 132–140.

Holden, C. (2001). "Behavioral" addictions: Do they exist? *Science, 294,* 980–982.

Jacobs, D. F. (1986). A general theory of addictions: A new theoretical model. *Journal of Gambling Behavior, 2,* 15–31.

Jang, K. L., Livesly, W. J., & Vernon, P. A. (1996). Heritability of the big five personality dimensions and their facets: A twin study. *Journal of Personality, 64,* 577–591.

Jones, B. T., & McMahon, J. (1994). Negative and positive alcohol expectancies and predictors of abstinence after discharge from a residential treatment program. A one-month and three-month follow-up study in men. *Journal of Studies on Alcohol, 55,* 543–548.

Jordan, C. H., Spencer, S. J., Zanna, M. P., Hoshino-Browne, E., & Correll, J. (2003). Secure and defensive high self-esteem. *Journal of Personality and Social Psychology, 85,* 969–978.

Kim, J., & Garman, E. T. (2003). Financial stress and absenteeism: An empirically derived model. *Financial Counseling and Planning, 14,* 31–42.

Knutson, B., Rick, S., Wimmer, G. E., Prelec, D., & Loewenstein, G. (2007). Neural predictors of purchases. *Neuron, 53,* 147–156.

Koran, L. M., Faber, R. J., Aboujaoude, E., Large, M. D., & Serpe, R. T. (2006). Estimated prevalence of compulsive buying behavior in the United States. *American Journal of Psychiatry, 163,* 1806–1812.

Kyrios, M., Frost, R. O., & Steketee, G. (2004). Cognitions in compulsive buying and acquisition. *Cognitive Therapy and Research, 28,* 241–258.

Ladd, G, & Petry, N. M. (2002). Gender differences among pathological gamblers seeking treatment. *Experimental and Clinical Psychopharmacology, 10,* 302–309.

Laforge, R. G., Maddock, J. E., & Rossi, J. S. (1998). Comparison of five stage methods for alcohol abuse among college students [Abstract]. *Annals of Behavioral Medicine, 20,* 170.

Larimer, M. E., Palmer, R. S., & Marlatt, G. A. (1999). Relapse prevention: An overview of Marlatt's cognitive-behavioral model. *Alcohol Research and Health, 23,* 151–160.

Lastovicka, J. L., Bettencourt, L. A., Hughner, R. S., & Kuntze, R. J. (1999). Lifestyle of the tight and frugal: Theory and measurement. *Journal of Consumer Research, 26,* 85–98.

Lejoyeux, M., Mathieu, K., Embouazza, H., Huet, F., & Lequen, V. (2007). Prevalence of compulsive buying among customers of a Parisian general store. *Comprehensive Psychiatry, 48,* 42–46.

Mano, H., & Oliver, R. L. (1993). Assessing the dimensionality and structure of the consumption experience: Evaluation, feeling, and satisfaction. *Journal of Consumer Research, 20,* 451–466.

Marlatt, G. A., & Donovan, D. M. (Eds.). (2005). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors* (2nd ed.). New York: Guilford Press.

Marlatt, G. A., & Gordon, J. R. (Eds.) (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York: Guilford Press.

Marlatt, G. A., & Witkiewitz, K. (2005). Relapse prevention for alcohol and drug problems. In G. A. Marlatt & D. M. Donovan (Eds.), *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors* (pp. 1–44). New York: Guilford Press.

Marrazzi, M. A., & Luby, E. D. (1986). An auto-addiction opioid model of chronic anorexia nervosa. *International Journal of Eating Disorders, 5,* 191–208.

McConnaughy, E. N., Prochaska, J. O., & Velicer, W. F. (1983). Stages of change in psychotherapy: Measurement and sample profiles. *Psychotherapy: Theory, Research, and Practice, 20,* 368–375.

Mick, D. G. (1996). Are studies of dark side variables confounded by socially desirable responding? The case of materialism. *Journal of Consumer Research, 23,* 106–119.

Miller, N. S., & Gold, M. S. (1988). The human sexual response and alcohol and drugs. *Journal of Substance Abuse Treatment, 5,* 171–177.

Miller, W. R., & Brown, J. M. (1991). Self-regulation as a conceptual basis for the prevention of addictive behaviours. In N. Heather, W. R. Miller, & J. Greeley (Eds.), *Self-control and the addictive behaviours* (pp. 3–79). Sydney, Australia: Maxwell Macmillan.

Mills, R. J., Grasmick, H. G., Morgan, C. S. & Wenk, D. (1992). The effects of gender, family satisfaction, and economic strain on psychological well-being. *Family Relations, 41,* 440–445.

Moos, R. H., Fenn, C. B., Billings, A. G., & Moos, B. S. (1989). Assessing life stressors and social resources: Applications for alcoholic patients. *Journal of Substance Abuse, 1,* 135–152.

Mosher, C. E. (2002). Impact of gender and problem severity upon intervention selection. *Sex Roles: A Journal of Research, 46,* 3–4, 113–119.

Mowen, J. C., & Spears, N. (1999). Understanding compulsive buying among college students: A hierarchical approach. *Journal of Consumer Psychology, 8,* 407–430.

Neuner, M., Raab, G., & Reisch, L. A. (2005). Compulsive buying in maturing consumer societies: An empirical re-inquiry. *Journal of Economic Psychology, 26,* 509–522.

Nigg, C. R., Burbank, P., Padula, C., Dufresne, R., Rossi, J. S., Velicer, W. F., Laforge, R. G., & Prochaska, J. O. (1999). Stages of change across ten health risk behaviors for older adults. *The Gerontologist, 39,* 473–482.

O'Doherty, J., Kringelbach, M. L., Rolls, E. T., Hornak, J., & Andrews, C. (2001). Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience, 4,* 95–102.

O'Guinn, T. C., & Faber, R. J. (1989). Compulsive buying: A phenomenological exploration. *Journal of Consumer Research, 16,* 147–157.

Olino, T. M., Klein, D. N., Durbin, C. E., Hayden, E. P., & Buckley, M. E. (2005). The structure of extraversion in preschool aged children. *Personality and Individual Differences, 39,* 481–492.

O'Neill, B., Sorhaindo, B., Xiao, J. J., & Garman, E. T. (2005). Financially distressed consumers: Their financial practices, financial well-being, and health. *Financial Counseling and Planning, 16,* 73–87.

Orford, J. (2001). *Excessive appetites: A psychological view of addictions.* Chichester, United Kingdom: Wiley.

Peele, S. (1995). *Diseasing of America: How we allowed recovery zealots and the treatment industry to convince us we are out of control.* New York: Jossey-Bass.

Peirce, R. S., Frone, M. R., Russell, M., & Cooper, M. L. (1996). Financial stress, social support, and alcohol involvement: A longitudinal test of the buffering hypothesis in a general population survey. *Health Psychology, 15,* 38–47.

Perkins, H. W. (2003). The emergence and evolution of the social norms approach to substance abuse prevention. In H. W. Perkins (Ed.), *The social norms approach to preventing school and college age substance abuse: A handbook for educators, counselors, and clinicians* (pp. 3–17). San Francisco: Wiley.

Perkins, H. W., Haines, M. P., & Rice, R. (2005). Misperceiving the college drinking norm and related problems: A nationwide study of exposure to prevention information, perceived norms, and student alcohol misuse. *Journal of Studies on Alcohol, 66,* 470–478.

Perkins, H. W., Meilman, P. W., Leichliter, J. S., Cashin, J. R., & Presley, C. A. (1999). Misperceptions of the norms for frequency of alcohol and other drug use on college campuses. *Journal of American College Health, 47,* 253–258.

Petry, N. M. (2005). Stages of change in treatment-seeking pathological gamblers. *Journal of Consulting and Clinical Psychology, 73,* 312–322.

Pineles, S. L., Street, A. E., & Koenen, K. C. (2006). The differential relationships of shame-proneness and guilt-proneness to psychological and somatization symptoms. *Journal of Social and Clinical Psychology, 25,* 688–704.

Prochaska, J. O., DiClemente, C. C., & Norcross, J. C. (1992). In search of how people change: Applications to addictive behaviors. *American Psychologist, 47,* 1102–1114.

Prochaska, J. O., & Norcross, J. C. (2007). *Systems of psychotherapy: A transtheoretical analysis.* Belmont, CA: Thomson Brooks/Cole.

Prochaska, J. O., Velicer, W. F., Rossi, J. S., Goldstein, M. G., Marcus, B. H., Rakowski, W., et al. (1994). Stages of change and decisional balance for 12 problem behaviors. *Health Psychology, 13,* 39–46.

Raylu, N. & Oei, T.P.S. (2004).The Gambling Related Cognitions Scale (GRCS): Development, confirmatory factor validation and psychometric properties. *Addiction, 99,* 757–769.

Rhodewalt, F., & Morf, C. C. (1995). Self and interpersonal correlates of the narcissistic personality inventory. *Journal of Research in Personality, 29,* 1–23.

Rindfleisch, A., Burroughs, J. E., & Denton, F. (1997). Family structure, materialism, and compulsive consumption. *Journal of Consumer Research, 23,* 312–325.

Robins, R. W., Tracy, J. L., Trzesniewski, K., Potter, J., & Gosling, S. D. (2001). Personality correlates of self-esteem. *Journal of Research in Personality, 35,* 463–482.

Roffman, R. A., & Stephens, R. S. (2005). Relapse prevention for cannabis abuse and dependence. In G. A. Marlatt & D. M. Donovan (Eds.), *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors* (pp. 179–207). New York: Guilford Press.

Rokeach, M. (1973). *The nature of human values.* New York: Free Press.

Rook, D. W. (1987). The buying impulse. *Journal of Consumer Research, 14,* 189–199.

Rook, D. W., & Fisher, R. J. (1995). Normative influences on impulsive buying behavior. *Journal of Consumer Research, 22,* 305–313.

Rose, P. (2002). The happy and unhappy faces of narcissism. *Personality and Individual Differences, 33,* 379–392.

Rose, P. (2007). Mediators of the association between narcissism and compulsive buying: The roles of materialism and impulse control. *Psychology of Addictive Behaviors, 21,* 576–581.

Rose, P. (2008). [An unsurprising positive correlation between impulsive buying and materialism in a sample of sixty-three undergraduates.] Unpublished raw data.

Rose, P., & DeJesus, S. (2007). A model of motivated cognition to account for the link between self-monitoring and materialism. *Psychology and Marketing, 24,* 93–115.

Rose, P., & Segrist, D. (2008). [Captivating correlations between impulsivity and several indices of excessive buying.] Unpublished raw data.

Rosengren, A., Hawken, S., Ounpuu, S., Sliwa, K., Zubaid, M., Almahmeed, W. A, et al. (2004). Association of psychosocial risk factors with risk of acute myocardial infarction in 11,119 cases and 13,648 controls from 52 countries (the INTERHEART study): Case-control study. *The Lancet, 364,* 953–962.

Schafer, J., & Brown, S. A. (1991). Marijuana and cocaine effect expectancies and drug use patterns. *Journal of Consulting and Clinical Psychology, 59,* 558–565.

Scherhorn. G., Reisch, L. A., & Raab, G. (1990). Addictive buying in West Germany: An empirical study. *Journal of Consumer Policy, 13,* 355–387.

Schuman, A., Christian, M., Rumpf, H. J., Hanover, W., Hapke, U., & John, U. (2005). Stages of change transitions and processes of change, decisional balance, and self-efficacy in smokers: A transtheoretical model validation using longitudinal data. *Psychology of Addictive Behaviors, 19,* 3–9.

Segrist, D. J., Corcoran, K. J., Jordan-Fleming, M. K., & Rose, P. (2007). Yeah, I drink . . . but not as much as other guys: The majority fallacy among male adolescents. *North American Journal of Psychology, 9,* 307–320.

Shaffer, H. J. (1996). Understanding the means and objects of addiction: Technology, the Internet, and gambling. *Journal of Gambling Studies, 12,* 461–469.

Shaffer, H. J., & Freed, C. R. (2005). Assessment of gambling-related disorders. In G .A. Marlatt & D. M. Donovan (Eds.), *Assessment of addictive behaviors* (pp. 334–391). New York: Guilford Press.

Shaffer, H. J., & LaPlante, D. A. (2005). Treatment of gambling disorders. In G. A. Marlatt & D. M. Donovan (Eds.), *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors* (pp. 276–332). New York: Guilford Press.

Shizgal, P., & Arvanitogiannis, A. (2003). Gambling on dopamine. *Science, 299,* 1856–1858.

Siahpush, M., Borland, R., & Scollo, M. (2003). Smoking and financial stress. *Tobacco Control, 12,* 60–66.

Smith, D. E., & Seymour, R. B. (2004). The nature of addiction. In R. H. Coombs (Ed.), *Handbook of addictive behaviors: A practical guide to diagnosis and treatment*. Hoboken, NJ: Wiley.

Smith, G. T., & Goldman, M. S. (1994). Alcohol expectancy theory and the identification of high-risk adolescents. *Journal of Research on Adolescence, 4,* 229–247.

Spinella, M. (2004). Neurobehavioral correlates of impulsivity: Evidence of prefrontal involvement. *International Journal of Neuroscience, 114,* 95–104.

Spinella, M., Yang, B., & Lester, D. (2007). Development of the Executive Personal Finance Scale. *International Journal of Neuroscience, 117,* 301–313.

Stone, J.C.I. (2002). Compulsive buying: Review and relevance to consumer debt management. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 62,* 3839. Abstract obtained from PsycINFO, 2003.

Swan-Kremeier, L. A., Mitchell, J. E., & Faber, R. J. (2005). Compulsive buying: A disorder of compulsivity or impulsivity? In J. S. Abramowitz, & A. C. Houts (Eds.), *Concepts and controversies in obsessive-compulsive disorder* (pp. 185–190). New York: Springer.

Takeuchi, D. T., Williams, D. R., & Adair, R. K. (1991). Economic stress in the family and children's emotional and behavioral problems. *Journal of Marriage and the Family, 53,* 1031–1041.

Tangney, J. P. & Fischer, K. W. (1995). *Self-conscious emotions: Shame, guilt, embarrassment and pride*. New York: Guilford Press.

Tangney, J. P., Wagner, P., & Gramzow, R. (1992). Proneness to shame, proneness to guilt, and psychopathology. *Journal of Abnormal Psychology, 101,* 469–478.

Thom, B. (1986). Sex differences in help seeking for alcohol problems. *British Journal of Addiction, 81,* 777–788.

Troisi, J. D., Christopher, A. N., & Marek, P. (2006). Materialism and money spending disposition as predictors of economic and personality variables. *North American Journal of Psychology, 8,* 421–436.

Valence, G., d'Astous, A., and Fortier, L. (1988). Compulsive buying: Concept and measurement. *Journal of Consumer Policy, 11,* 419–433.

Velicer, W. F., Fava, J. L., Prochaska, J. O., Abrams, D. B., Emmons, K. M., & Pierce, J. (1995). Distribution of smokers by stage in three representative samples. *Preventive Medicine, 24,* 401–411.

Verplanken, B., & Herabadi, A. (2001). Individual differences in impulse buying tendency: Feeling and no thinking. *European Journal of Personality, 15*, 71–83.

Wakefield, K. L., & Baker, J. (1998). Excitement at the mall: Determinants and effects on shopping response. *Journal of Retailing, 74*, 515–539.

Walker, D. D., Roffman, R. A., Stephens, R. S., Berghuis, J., & Kim, W. (2006). Motivational enhancement therapy for adolescent marijuana users: A preliminary randomized controlled trial. *Journal of Consulting and Clinical Psychology, 74*, 628–632.

Walker, M. B. (1989). Some problems with the concept of "gambling addiction": Should theories of addiction be generalized to include excessive gambling? *Journal of Gambling Behavior, 5*, 179–200.

Walters, S. T., & Baer, J. S. (2006). *Talking with college students about alcohol: Motivational strategies for reducing abuse*. New York: Guilford Press.

Weun, S., Jones, M. A., & Beatty, S. E. (August, 1997). *A parsimonious scale to measure impulse buying tendency*. Paper presented at the American Marketing Association Educators' Summer Conference, Chicago.

Wheeler, J. G., George, W. H., & Stephens, K. A. (2005). Assessment of sexual offenders: A model for integrating dynamic risk assessment and relapse prevention approaches. In G. A. Marlatt & D. M. Donovan (Eds.), *Assessment of addictive behaviors* (pp. 392–424). New York: Guilford Press.

Whelan, C. T. (1992). The role of income, life-style deprivation and financial strain in mediating the impact of unemployment on psychological distress: Evidence from the Republic of Ireland. *Journal of Occupational and Organizational Psychology, 65*, 331–344.

Whelan, J. P., Steenbergh, T. A., & Meyers, A. W. (2007). *Problem and pathological gambling*. Cambridge, MA: Hogrefe & Huber.

Wiggins, J. S., & Trapnell, P. (1997). Personality structure: The return of the big five. In R. Hogan, J. Johnson, & S. Briggs (Eds.), *Handbook of Personality Psychology* (pp. 737–765). New York: Academic Press.

World Health Organization (WHO; 1990). *International classification of diseases*. Geneva: World Health Organization.

Young, K. S. (1996). Psychology of computer use: XL. Addictive use of the internet: A case that breaks the stereotype. *Psychological Reports, 79*, 899–902.

Yurchisin, J., & Johnson, K. P. P. (2004). Compulsive buying behavior and its relationship to perceived social status associated with buying, materialism, self-esteem, and apparel-product involvement. *Family and Consumer Sciences Research Journal, 32*, 291–314.

This page intentionally left blank

Chapter **4**

Compulsive Buying Disorder

Mamta Sood, MD, and Meera Vaswani, PhD

Buying is a universal, everyday human experience and has been practiced since ancient times. Besides being recognized for its primary function of acquiring an essential item, it is also recognized as a leisure activity. The ability to buy saw the emergence of monetary power as a significant determinant of social status. Excessive buying is sometimes not uncommon, especially in situations such as those of holidays, festivals, marriage, and so on. However, such occasional buying sprees are not recognized as a problem. In some individuals, indulgence in buying behavior is excessive and extreme, so much so that it results in significant psychological, interpersonal, financial, and legal difficulties. Since the beginning of the twentieth century, a clinically significant problem of buying behavior has been recognized. Kraepelin (1915) referred to it as *oniomania*, a term that is derived from the Greek words *onios*—for sale, and *mania*—insanity. Kraepelin based this on Esquirol's concept of monomania (Esquirol, 1838). Bleuler (1924) recognized impulsiveness as its core feature and classified it with pyromania and kleptomania. Subsequently, interest in this disorder remained limited to psychoanalysts and consumer behaviorists. The situation changed in the early 1990s, when independent research workers published three case series and interest in this condition was rekindled. Data on the demographics and phenomenology of 24 compulsive buyers in comparison to normal buyers was reported (Christenson, Faber, & de Zwann, 1994). For the first time, diagnostic criteria for compulsive buying disorder (CBD) were proposed, and data on 20 consecutive diagnosed patients with problematic buying behavior were published (McElroy, Keck, Pope, Smith, & Strakowski, 1994). In

addition to clinical features, overall lifestyle, comorbidity, and other problems in 46 compulsive buyers were studied (Schlosser, Black, Repertinger, & Freet, 1994). Since then, reports of compulsive buying have started to emerge from all parts of the world. "Uncontrolled problematic buying behavior has been referred to as uncontrolled buying, compulsive buying, compulsive shopping, addictive buying, excessive buying, and spendaholism" (Koran, Faber, Aboujaoude, Large, & Serpe, 2006, p. 1806).

Research in this field in the last decade and a half has witnessed progress from descriptions of compulsive buying behavior to the proposal of diagnostic criteria for compulsive buying disorder. Robins and Guze (1970) proposed five phases for establishing the diagnostic reliability and validity of a psychiatric disorder: clinical description, exclusion of other disorders, follow-up, family studies, and laboratory study. The focus of research has been on identifying and delineating these phases so as to establish the reliability and validity of diagnosis of CBD. Based on all this, there is a proposal to include compulsive buying disorder as a diagnostic category in future revisions of classificatory systems. Currently, it is not included in the *Diagnostic and Statistical Manual of Mental Disorders*. fourth edition, text revision (*DSM–IV–TR;* American Psychiatric Association, 2004) or the *International Classification of Diseases*, 10th edition (*ICD-10;* World Health Organization [WHO], 1992) as a separate diagnostic category.

The researchers in the field have conceptualized compulsive buying disorder as being linked to impulse control disorders, obsessive-compulsive spectrum disorders, mood disorders, and substance and behavioral addiction disorders. It is linked to impulse control disorders (Black, 2007), based on similar clinical features like failure to resist an impulse to perform an act of buying, an increasing sense of arousal or tension prior to the act, and an experience of pleasure, gratification, or release of tension at the time of committing the act. It is also considered close to the obsessive-compulsive disorders (Hollander et al., 1996). The repeated idea of buying is conceptualized as an obsession. When this idea is resisted, it results in anxiety. which is relieved by a compulsive act in the form of buying. In fact, some researchers consider it to be a compulsive-impulsive disorder (Dell'Osso, Altamura, Allen, Marazziti, & Hollander, 2006), as it is proposed that impulsive features initiate the behaviors, and the compulsive drive causes the behaviors to persist over time. Its relationship to mood disorders, mainly depression (Lejoyeux, Ades, Tassain, & Solomon, 1996), is suggested by elevated scores on depression rating scales. Also, the concept of behavioral addiction (Krych, 1989) is applied to compulsive buying disorder because buying produces the same sequence of events as those produced by addictive substances. Buying behavior gains increasing significance despite

adverse consequences. Attempts to cut down the behavior result in restlessness. The conceptualized link to one of these disorders has an important role in deciding under which section of classificatory systems compulsive buying disorder will be included. Conversely, attempts to categorize compulsive buying as an illness are seen as a part of a trend to medicalize behavioral problems that may be better understood within the wider social context of the consumption-driven economy (Lee & Mysyk, 2004).

This chapter aims to present an overview of compulsive buying disorder based on research carried out in the last 15 years, in epidemiology, etiology, clinical features, diagnosis, comorbidity, and treatment.

EPIDEMIOLOGY

Prevalence

Faber and O'Guinn (1992) estimated the prevalence of CBD to be 2–8 percent in the adult general population by administering Compulsive Buying Scale (CBS) to 292 respondents in Illinois. Recently, in a nationwide random telephone survey in the United States, (using a large general population sample), CBS embedded in a computer assisted telephone interview was administered to 2,513 adult respondents. The point prevalence was calculated by using scores on CBS as two standard deviations below the mean and was found to be 5.8 percent. Using a stricter criterion, (three standard deviations below the mean), 1.4 percent of individuals were reported to have this disorder (Koran et al., 2006).

A hospital-based study examined the frequency of co-occurring impulse control disorders including CBD in 204 consecutively admitted psychiatric inpatients. Using the Minnesota Impulsive Disorders Interview, it was found that 30.9 percent had at least one current impulse control disorder. A total of 9.3 percent of patients with impulse control disorders had CBD, which was also the commonest diagnosis (Grant, Levine, Kim, & Potenza, 2005).

An interesting study (Lejoyeux, Mathieu, Embouazza, Huet, & Lequen, 2007) on women consumers in a famous department store found that 32.5 percent exhibited compulsive buying. Further analysis revealed that in comparison to controls, they considered purchases as opportunities not to be missed, used purchased items less often, made purchases to impress others, considered purchases as personally gratifying, and stayed connected to online shopping sites longer.

Gender

Compulsive buying is seen mostly in women and it is ascribed to the appeal of risky and exciting situations by some of them (Bleuler, 1924; Kraepelin,

1915). In the clinical case series referred to above, 80–94 percent of compulsive buyers are reported to be women (Christenson et al., 1994; McElroy et al., 1994; Schlosser et al., 1994). However, the nationwide community survey (Koran et al., 2006) found that point prevalence is almost equal in both sexes; 6.0 percent for women and 5.5 percent for men, thereby refuting the notion that it is more common in women. The difference in clinical and community settings is probably due to the willingness of women to seek treatment. It is also possible that the recruitment strategies, for example, daytime television ads or contact with self-help groups, might have resulted in a larger number of women in the clinical cases.

Age of Onset

The onset of compulsive buying is reported to be in the late teens or early twenties, which correlates with the time when the individual is establishing separation from the nuclear family. Also, this is the age when a person can first establish credit. By the time the behavior is recognized as problematic, individuals are in their thirties (Black, 2007a). Compulsive buying in adolescents is likely to be associated with several risky behaviors like cigarette smoking, alcohol use, drug use, and early sex (Roberts & Tanner, 2000), as seen in a survey conducted among 111 adolescents between the ages of 12 and 19 years.

ETIOLOGY

There is no single cause of compulsive buying disorder. However, psychological, sociocultural, neurobiological, and genetic factors may contribute toward its causation.

Psychological and Sociocultural Perspective

From a psychodynamic perspective, compulsive buying forms a specific complex with common developmental precursors of pathological narcissism (Kruger, 1988). Positive associations emerge between narcissism, materialism, and compulsive buying in young graduates. Personal values and impulse control are important correlates of addictive buying. Relatively narcissistic persons are poor self-regulators and may be at risk of developing a variety of addictive behaviors (Rose, 2007).

Dittmar (2005) found endorsement of materialistic values to be the strongest predictor of individuals' compulsive buying; such endorsement significantly mediated the observed age differences. In-depth interviews with persons

exhibiting compulsive buying suggest that the behavior serves a variety of functions for them; it improves their mood and increases their ability to match their perceptions of socially desirable appearances. This behavior is located in the context of postmodern fragmentation, where personal identity is manifested in a reliance on emotionally charged experiences (Elliott, 1994).

Black (2007) hypothesized that sociocultural mechanisms are necessary for the development of CBD, as evidenced by the fact that the disorder largely occurs in developed countries. A market-based economy, the availability of a wide variety of goods, a disposable income, and significant leisure time are necessary for the development of compulsive buying.

Neurobiological Perspective

Disturbed neurotransmission, specifically of the serotonergic, dopaminergic, and opioid systems has been hypothesized to have a role in the causation of CBD. This causation is presumed because the drugs used in the treatment of CBD act on these neurotransmitter systems. However, there are no studies that have directly examined these hypotheses. The serotonergic system is implicated because selective serotonin reuptake inhibitors (SSRIs) have been used in its treatment based on its closeness to obsessive-compulsive disorder (Ninan et al., 2000). In behavioral addictions like CBD and pathological gambling, the changes in dopaminergic pathways have been implicated. Reward-seeking such as buying in CBD, triggers the release of dopamine and produces feelings of pleasure (Grant, 2003).

Limited research on genetics suggests that compulsive buying may run in families. McElroy et al. (1994) studied family history data on 18 compulsive buyers; 17 had one or more first-degree relatives with a mood disorder, 11 had alcohol or substance abuse, three had an anxiety disorder and three exhibited compulsive buying. In a larger study (Black, Repertinger, Gaffney, & Gabel, 1998), 137 first-degree relatives of 31 compulsive buyers were interviewed; 9.5 percent of them had CBD. In comparison with controls, first-degree relatives of compulsive buyers reported significantly more depression, alcoholism, and drug abuse. However, relatively small sample sizes remain an important limitation of these studies.

Two genetic studies have also been reported. Significant correlation between a polymorphism in the promoter region of the D1 receptor gene and the association of Tourette's disorder with compulsive buying disorder have been reported. However, no association was reported with the serotonin transporter gene promoter polymorphisms (Comings et al., 1997). In another study, 21 patients diagnosed with compulsive buying were compared with 38 normal

controls with regard to two DNA sequence polymorphisms in the gene that encodes the serotonin transport (5-HTT); no significant difference was seen (Devor, Magee, Dill-Devor, Gabel, & Black, 1999).

CLINICAL FEATURES

The onset of compulsive buying disorder is mostly in the teens. By the time the behavior is recognized as problematic, individuals are in their twenties or thirties (see Box 1).

BOX 1

Clinical Features

- Onset is in teens.
- Seen more commonly in women.
- Preoccupation with thoughts about shopping.
- Rising levels of anxiety and tension followed by act of shopping, which leads to tension relief.
- Clothing, shoes, jewelry, makeup, and compact discs are usual items purchased.
- Leads to significant psychological, interpersonal, financial, and legal difficulties.
- Course is either chronic or recurrent.

Compulsive buyers are preoccupied with repetitive and intrusive thoughts like shopping and spending, which they try to resist, usually without much success (Bernik, Akerman, Amaral, & Braun, 1996; Christenson et al., 1994). Compulsive buyers are supposed to have high urge combined with low control (Nataraajan & Goff, 1991). Typically they engage in compulsive buying behaviors by spending many hours in a week buying, year round or in binges.

There is an increasing level of anxiety and tension. The actual shopping experience is intense. The act is completed with the purchase, which leads to tension relief. Attempts to resist urges to buy are present in the majority of subjects with compulsive buying disorder, but invariably they are unsuccessful, as is illustrated by the fact that, most often, 1–5 hours pass between the urge to buy and the purchase (Christenson et al., 1994; Schlosser et al., 1994).

Black (2007) described four phases of compulsive buying disorder: anticipation, preparation, shopping, and spending. In the first phase, a person with compulsive buying disorder is preoccupied with the thought of a specific item

and the act of shopping. Then the preparation for shopping and spending starts. This involves deciding on the time and venue for shopping, the outfit to be worn, and so on. This phase is followed by the actual shopping experience. Negative emotions (Miltenberger et al., 2003) such as anger, anxiety, boredom, and self-critical thoughts are the most common antecedents to shopping binges in individuals, while euphoria or relief from the negative emotions is the most common consequence.

Shopping is generally done alone; it may be in upscale department stores, consignment shops, a garage sales. Internet and catalog shopping are also used. Generally, clothing, shoes, jewelry, makeup, and compact discs are purchased. These items seem to address personal and social identity needs. Shopping provides a sense of recognition and acceptance for people with low self-esteem. Once a purchase is made, its outcome varies; the item may be returned or sold, given away, or not even removed from the package (Schlosser et al., 1994).

Income does not seem to be a factor in developing CBD. A person with low income preoccupied with shopping and spending will tend to shop at a smaller store. However, the presence of CBD may result in interpersonal problems in both groups (Black, 2007).

Adverse consequences include guilt or remorse, excessive debt, bankruptcy, family conflict, divorce, illegal activities, such as writing bad checks and embezzlement, and even suicide attempts.

Compulsive buying occurs along a spectrum of severity. Greater severity is seen in low the income group; they tend to purchase nonsale items, spend a lower percentage of income on sale items (Black, Monahan, Schlosser, & Repertinger, 2001), and give maladaptive responses regarding their consumer behavior (Koran et al., 2006).

In a transnational study, treatment-seeking female compulsive buyers were compared (38 Germans from Bavaria and 39 Americans from North Dakota) by using CBS and the Yale-Brown Obsessive Compulsive Scale-Shopping Version (YBOCS-SV). However, with regard to age and scores on the above scales, the researchers did not find statistically significant differences (Mueller et al., 2007).

Although, there are no long-term follow-up studies, the disorder is either chronic or recurrent (Schlosser et al., 1994; Christenson et al., 1994). In one study, treatment with citalopram in CBD was followed up for a year, which resulted in a good response in 71 percent of subjects. However, an acute response predicted a greater likelihood of continued remission (Aboujaoude, Gamel, & Koran, 2003). This study highlights the impact of treatment on the course of CBD in terms of initial and maintained improvement.

DIAGNOSIS AND ASSESSMENT

Diagnosis

While making a diagnosis, questions regarding individuals' attitudes toward shopping and their specific shopping behaviors and patterns are generally asked. Preoccupation with buying, time spent in desires, urges, fantasies, or behaviors related to buying should be looked into. To judge the dysfunction, feelings of distress and guilt, and financial, legal or interpersonal problems need to be looked into. A history of past psychiatric and medical illness, surgical procedures, and past treatment needs to be incorporated. However, bipolar disorder should be ruled out as a cause of excessive buying.

The diagnostic criteria for CBD proposed by McElroy et al (1994) have been used for research (see Box 2). These include (1) uncontrollable problematic buying behavior characterized as being frequently preoccupied with buying or subject to irresistible, intrusive, and/or senseless impulses to buy; (2) shopping for periods longer than intended; (3) experiencing adverse consequences such as markedly distressing, time consuming, and/or resulting in family, social, vocational, and/or financial difficulties; and (4) not occurring only in the context of hypomanic or manic symptoms.

BOX 2

Diagnostic Criteria for Compulsive Buying Disorder
(McElroy et al., 1994)

1. Uncontrollable problematic buying behavior
2. Shopping for periods longer than intended
3. Experiencing adverse consequences
4. Not occurring in the context of hypomanic or manic symptoms

Assessment Instruments

1. Compulsive Buying Scale
2. Yale Brown Obsessive-Compulsive Scale—Shopping Version
3. Minnesota Impulsive Disorders Interview

Differential Diagnosis

CBD needs to be distinguished from normal buying behavior. Frequent shopping in itself does not warrant a diagnosis of compulsive buying disorder. Normal buying can also sometimes be compulsive and episodic, specifi-

cally around special holidays or birthdays. The distinction is not made on the grounds of the amount of money spent or the income level, but on the extent of the preoccupation with buying behavior, the level of personal distress, and the presence of adverse consequences. Typically, excessive buying in a manic patient arises out of euphoric and cheerful mood, grandiosity, and unrealistic plans, and the duration of the shopping invariably corresponds to the duration of the manic episode. Although a preoccupation with buying behavior is present most of the time in compulsive buying disorder, it lacks the periodicity seen in bipolar disorder.

Assessment Instruments

Several rating instruments have been used for identifying as well as for rating the severity of CBD (see Box 2).

The Compulsive Behavior Scale (CBS) consists of seven items representing specific behaviors, motivations, and feelings associated with compulsive buying disorder. The seven scale items assess the need to spend money, awareness that spending behavior is aberrant, loss of control, and buying things to improve mood and financial problems (Faber & O'Guinn, 1989). Studies have established that subjects meeting the two standard deviation criterion on this scale always meet the clinical diagnostic criteria (Black et al., 1998; Christensen et al., 1994).

The Yale Brown Obsessive-Compulsive Scale has been modified to form the Yale Brown Obsessive-Compulsive Scale-Shopping Version (YBOCS-SV). The 10-item scale rates time spent, interference, distress, resistance, degree of control for cognitions, and behaviors typical of compulsive buying disorder, yielding scores ranging from 0 to 40 (Monahan, Black, & Gabel, 1995).

The Minnesota Impulsive Disorders Interview is a semistructured interview to assess the presence of compulsive buying disorder, kleptomania, trichotillomania, intermittent explosive disorder, compulsive sexual behavior, pathological gambling, and compulsive exercising. However, an 82-item module has been developed specifically for those screening positive for compulsive buying disorder (Christenson et al., 1994).

COMORBIDITY

The presence of comorbid disorders has far-ranging implications for the understanding of CBD. The initial presentation may be changed or may be more severe. There may be shared underlying etiological factors, treatment may need modification, and the future course may be altered. High rates of psychi-

atric comorbidity have been reported in persons having CBD. Various studies have reported higher lifetime prevalence of mood disorders, obsessive-compulsive disorders, anxiety disorders, alcohol abuse, eating disorders, and impulse control disorders on Axis I, and obsessive-compulsive, borderline, avoidant, antisocial, and narcissistic personality disorders on Axis II (Black, 2007).

In Christenson's case series, compulsive buyers had a higher lifetime prevalence of anxiety disorders, substance use disorders, and eating disorders as compared to controls. It was found that buying behavior resembled obsessive compulsive disorder in 66.7 percent of subjects, whereas impulse control disorder was seen in 95.8 percent. In McElroy's case series, 95 percent of the compulsive buyers had lifetime diagnoses of major mood disorders, 80 percent had anxiety disorders, 40 percent had impulse control disorders, and 35 percent had eating disorders. In Schlosser's case series, more than two-thirds met lifetime criteria for Axis I psychiatric disorder, anxiety disorders, substance abuse, and mood disorders were common. Nearly 60 percent met the criteria for personality disorder on Axis II, most commonly the obsessive-compulsive, borderline, and avoidant types. Lejoyeux, Haberman, Solomon, and Ades (1999) studied the effect of the concomitant presence of compulsive buying disorder in depressed subjects. Their results indicated that these patients had significantly more recurrent depression, bipolar disorder, kleptomania, bulimia, suicide attempts, and benzodiazepine abuse as compared to control subjects with depression only.

It is reported (Mueller et al., 2007) that many compulsive buyers also suffer from compulsive hoarding. Hoarding compulsive buyers report severe buying symptoms and obsessive-compulsive symptoms with a higher psychiatric comorbidity, especially with regard to affective, anxiety, and eating disorders. The prevalence of compulsive buying among patients presenting with obsessive-compulsive disorder was 23 percent. Subjects having both disorders also showed more depression and drank more alcohol (Lejoyeux, Bailly, Moula, Loi, & Ades, 2005).

In the transnational study comparing German and American female compulsive buyers, nearly all participants met the criteria for at least one lifetime Axis I disorder. However, the German compulsive buyers showed higher rates of affective disorder, anxiety disorder, and somatoform disorder. In addition, the German compulsive buyers were more likely to have more than one Axis I disorder (Mueller et al., 2007).

TREATMENT

There are no guidelines for treatment; multiple modalities of therapies have been tried in clinical settings. Pharmacotherapy, psychotherapies, and some other treatments are also available (see Box 3).

BOX 3

Treatment

- No treatment guidelines
- Selective serotonin reuptake inhibitors and partial opiate antagonists
- Cognitive-behavioral techniques of cue exposure and response prevention
- Group cognitive-behavioral intervention
- Self-help books
- Debtors Anonymous
- Marital and financial counseling

Pharmacotherapy

Selective serotonin reuptake inhibitors (SSRIs) and partial opiate antagonists have been tried in the treatment of CBD on the basis of its conceptualized closeness to obsessive-compulsive spectrum disorder, mood disorders, and addictive disorders.

The results of open label studies using SSRIs are promising. In McElroy's cases, almost 50 percent experienced full or partial remission to SSRIs or in combination with a mood stabilizer. In most cases, the observation period was limited to a few weeks or months. Treatment with fluvoxamine showed benefit, suggesting that improvement is independent of the treatment of mood symptoms in 9 out of 10 nondepressed subjects with CBD (Black, Monahan, & Gabel, 1997). In an open-label trial with citalopram, it was reported that 17/24 subjects improved in specific measures of buying behavior and global functioning (Koran, Chuang, Bullock, & Smith, 2003). However, treatment with escitalopram showed little effect in an identically designed trial carried out by the same group.

Two randomized-controlled trials using fluvoxamine have been conducted. The results have not been promising. A 12-week trial with 37 subjects found no difference between fluvoxamine and placebo in an intent-to-treat analysis (Ninan et al., 2000). A nine-week trial of 23 nondepressed subjects with compulsive buying disorder reported 50 percent of subjects in the fluvoxamine group and 64 percent in the placebo group being rated as improved (Black, Gabel, Hansen, & Schlosser, 2000). However, in these studies, subjects kept a log of their shopping. Keeping logs is a therapeutic intervention in itself, which may have led to these findings (Dell'Osso et al., 2006). Grant (2003) described cases in which persons with CBD improved with naltrexone, suggesting that opiate antagonists might play a role in the treatment.

Psychotherapy

Psychoanalytic treatment based on early experiences has been used (Kruger, 1988). Recently, Cognitive-behavioral models using techniques of cue exposure and response prevention have been found to be effective in treatment (Bernik et al., 1996; Lejoyeux et al., 1996). The efficacy of a group cognitive-behavioral intervention designed for the treatment of 28 subjects with compulsive buying was compared with 11 subjects on waiting list control. At the end of treatment, the results indicated significant advantages for cognitive-behavioral therapy over the waiting list in terms of reductions in the number of compulsive buying episodes and time spent in buying, as well as in scores on YBOCS-SV and the CBS. Improvement was well maintained at six-month follow-up (Mitchell, Burgard, Faber, Crosby, & de Zwaan, 2006).

Other Therapies

Self-help books are available and have been reported to be beneficial. Debtors Anonymous is patterned after Alcoholics Anonymous. It is a voluntary group that provides mutual support and encouragement for those with substantial debts. Simplicity circles are available in some U.S. cities; voluntary groups encourage people to adopt a simple lifestyle. Marital counseling may be helpful in cases where interpersonal relations are strained. Persons with financial difficulties may benefit from financial counseling (Black, 2007).

CONCLUSION

In the past decade, the focus of research in the field of compulsive buying disorder has been on identifying and delineating its characteristic features. This has resulted in a better understanding of its epidemiology, phenomenology, family history, and treatment. The disorder is not uncommon and is associated with important comorbid psychiatric disorders. Proposals to include this as a diagnostic subcategory in *DSM–V* are the subject of debate. Various researchers have argued for its inclusion under impulse control disorders, substance use disorders, mood disorders, and obsessive compulsive disorders in *DSM–V*. At present, opinion favors its inclusion under impulse control disorders. Its inclusion in classificatory systems will help in routine screening for compulsive buying disorder by mental health professionals and in community-based incidence prevalence studies. It will also help in the identification of vulnerability, precipitating and perpetuating factors, and in the development of effective psychosocial and pharmacological therapies.

At present, there are conflicting reports regarding gender-related differences. Different underlying diatheses have been proposed in the etiology of compulsive buying disorder. Neurobiological studies employing brain imaging and other techniques need to be conducted to clarify these issues. Cognitive-behavioral strategies and medications have been used for treatment with partial success.

Future research needs to focus on establishing the reliability and validity of diagnostic criteria. So, there is a need for large-scale community studies in order to understand the epidemiology. Studies need to be planned for identifying efficacious treatments, type of treatment, short- and long-term outcome, and duration of treatment. Long-term follow-up studies are needed for charting the course of the disorder, identifying various prognostic factors, and identifying their relationship to other psychiatric disorders.

REFERENCES

Aboujaoude, E., Gamel, N., & Koran, L. M. (2003). A one-year naturalistic following of patients with compulsive shopping disorder. *Journal of Clinical Psychiatry, 64,* 946–950.

American Psychiatric Association. (2004). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Bernik, M. A., Akerman, D., Amaral, J. A., & Braun, R. C. (1996). Cue exposure in compulsive buying. *Journal of Clinical Psychiatry, 57,* 90.

Black, D. W. (2007a). Compulsive buying disorder: A review of the evidence. *CNS spectrums, 12,* 124–132.

Black, D. W. (2007b). A review of compulsive buying disorder. *World Psychiatry, 6,* 14–18.

Black, D. W., Gabel, J., Hansen, J., & Schlosser, S. (2000). A double-blind comparison of fluvoxamine versus placebo in the treatment of compulsive buying disorders. *Annals of Clinical Psychiatry, 12,* 205–211.

Black, D. W., Monahan, P., & Gabel, J. (1997). Fluvoxamine in the treatment of compulsive buying. *Journal of Clinical Psychiatry, 58,* 159–163.

Black, D. W., Monahan, P., Schlosser, S., & Repertinger, S. (2001). Compulsive buying severity: An analysis of Compulsive Buying Scale results in 44 subjects. *Journal of Nervous and Mental Disease, 189,* 123–126.

Black, D. W., Repertinger, S., Gaffney, G. R., & Gabel J. (1998). Family history and psychiatric comorbidity in persons with compulsive buying: preliminary findings. *American Journal of Psychiatry, 155,* 960–963.

Bleuler, E. (1924). *Textbook of psychiatry*. New York: McMillan.

Christenson, G. A., Faber, J. R., & de Zwann, M. (1994). Compulsive buying: Descriptive characteristics and psychiatric comorbidity. *Journal of Clinical Psychiatry, 55,* 5–11.

Comings, D. E., Gade, R., Wu, S., Chiu, C., Dietz, G., Muhleman, D., et al. (1997). Studies of the potential role of the dopamine D1 receptor gene in addictive behaviors. *Molecular Psychiatry, 2,* 44–56.

Dell'Osso, B., Altamura, A. C., Allen, A., Marazziti, D., & Hollander E. (2006). Epidemiologic and clinical updates on impulse control disorders: A critical review. *European Archives of Psychiatry and Clinical Neuroscience, 256,* 464–475.

Devor, E. J., Magee, H. J., Dill-Devor, R. M., Gabel, J., & Black, D. W. (1999). Serotonin transporter gene (5-HTT) polymorphisms and compulsive buying. *American Journal of Medical Genetics, 88,* 123–125.

Dittmar, H. (2005). Compulsive buying—a growing concern? An examination of gender, age, and endorsement of materialistic values as predictors. *British Journal of Clinical Psychology, 96,* 467–491.

Elliott, R. (1994). Addictive consumption: Function and fragmentation in postmodernity. *Journal of Consumer Policy, 17,* 159–179.

Esquirol, J. E. (1838). *Des maladies mentales.* Paris: Baillière.

Faber, R. J., & O'Guinn, T. C. (1989). Classifying compulsive consumers: Advances in the development of a diagnostic tool. *Advances in Consumer Research, 16,* 147–157.

Faber, R. J., & O'Guinn, T. C. (1992). A clinical screener for compulsive buying. *Journal of Consumer Research, 19,* 459–469.

Grant, J. E. (2003). Three cases of compulsive buying treated with naltrexone. *International Journal of Psychiatry in Clinical Practice, 7,* 223–225.

Grant, J. E., Levine, L., Kim, D., & Potenza, M. N. (2005). Impulse control disorders in adult psychiatric inpatients. *American Journal of Psychiatry, 162,* 2184–2188.

Hollander, E., Kwon, J. H., Stein, D. J., Broatch, J., Rowland, C. T., & Himelein, C. A. (1996). Obsessive-compulsive and spectrum disorders: Overview and quality of life issues. *Journal of Clinical Psychiatry, 57,* 3–6.

Koran, L. M., Chuang, H. W., Bullock, K. D., & Smith, S. C. (2003). Citalopram for compulsive shopping disorder: An open-label study followed by a double-blind discontinuation. *Journal of Clinical Psychiatry, 64,* 793–798.

Koran, L. M., Faber, R. J., Aboujaoude, E., Large, M. D., & Serpe, R. T. (2006). Estimated prevalence of compulsive buying in the United States. *American Journal of Psychiatry, 163,* 1806–1812.

Kraepelin, E. (1915). *Psychiatrie.* Leipzig, Germany: Verlag Von Johann Ambrosius. (Original work published 1883)

Krueger, D. W. (1988). On compulsive shopping and spending: A psychodynamic inquiry. *American Journal of Psychotherapy, 42,* 574–584.

Krych, R. (1989). Abnormal consumer behavior: A model of addictive behaviors. *Advances in Consumer Research, 16,* 745–748.

Lee, S., & Mysyk, A. (2004). The medicalization of compulsive buying. *Social Science and Medicine, 58,* 1709–1718.

Lejoyeux, M., Ades, J., Tassain, V., & Solomon, J. (1996). Phenomenology and psychopathology of uncontrolled buying. *American Journal of Psychiatry, 153,* 1524–1529.

Lejoyeux, M., Bailly, F., Moula, H., Loi, S., & Ades, J. (2005). Study of compulsive buying in patients presenting obsessive-compulsive disorder. *Comprehensive Psychiatry, 46,* 105–110.

Lejoyeux, M., Haberman, N., Solomon J, & Ades J. (1999). Comparison of buying behavior in depressed patients presenting with or without compulsive buying. *Comprehensive Psychiatry, 40,* 51–56.

Lejoyeux, M., Mathieu, K., Embouazza, H., Huet, F., & Lequen, V. (2007). Prevalence of compulsive buying among customers of a Parisian general store. *Comprehensive Psychiatry, 48,* 42–46.

McElroy, S. L., Keck, P. E., Pope, H. G., Smith, J. M., & Strakowski, S. M. (1994). Compulsive buying: A report of 20 cases. *Journal of Clinical Psychiatry, 55,* 242–248.

Miltenberger, R. G., Redlin, J., Crosby, R., Stickney, M., Mitchell, J., Wonderlich, S., et al. (2003). Direct and retrospective assessment of factors contributing to compulsive buying. *Journal of Behavior Therapy and Experimental Psychiatry, 34,* 1–9.

Mitchell, J. E., Burgard, M., Faber, R., Crosby, R. D., & de Zwaan, M. (2006). Cognitive behavioral therapy for compulsive buying disorder. *Behaviour Research and Therapy, 44,* 1859–1865.

Monahan, P., Black, D. W., & Gabel, J. (1995). Reliability and validity of a scale to measure change in persons with compulsive buying. *Psychiatry Research, 64,* 59–67.

Mueller, A., Mitchell, J. E., Mertens, C., Mueller, U., Silbermann, A., Burgard, M., et al. (2007). Comparison of treatment seeking compulsive buyers in Germany and the United States. *Behaviour Research and Therapy, 45,* 1629–1638.

Mueller, A., Mueller, U., Albert, P., Mertens, C., Silbermann, A., Mitchell, J. E., et al. (2007). Hoarding in a compulsive buying sample. *Behaviour Research and Therapy,* 45, 2754–2763.

Nataraajan, R., & Goff, B. G. (1991). Compulsive buying: Toward a reconceptualization. *Journal of Social Behavior and Personality, 6,* 307–328.

Ninan, P. T., McElroy, S. L., Kane, C. P., Knight, B. T., Casuto, L. S., Rose, S. E., et al. (2000). Placebo controlled study of fluvoxamine in the treatment of patients with compulsive buying. *Journal of Clinical Psychopharmacology, 20,* 362–366.

Roberts, J. A., & Tanner, J. F. (2000). Compulsive buying and risky behavior among adolescents. *Psychological Reports, 86,* 763–770.

Robins, E., & Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry, 126,* 983–987.

Rose, P. (2007). Mediators of the association between narcissism and compulsive buying: The roles of materialism and impulse control. *Psychology of* Addictive Behaviors, *21,* 576–581.

Schlosser, S., Black, D. W., Repertinger, S., & Freet, D. (1994). Compulsive buying: Demography, phenomenology, and comorbidity in 46 subjects. *General Hospital Psychiatry, 16,* 205–212.

World Health Organization. (1992).The ICD-10 classification of mental and behavioral disorders: Clinical descriptions and diagnostic guidelines. Geneva: Author.

This page intentionally left blank

Chapter **5**

Association with Criminality of Habit and Impulse-Control Disorders

Ian H. Treasaden, MB, LRCP, MRCS, FRCPsych, LLM, and
Basant K. Puri, MA, PhD, MB, MRCPsych, MMath

Impulse-control disorders are disorders in which a person acts on an impulse that is potentially harmful and which he or she fails to resist. The impulses are usually perceived as pleasurable (egosyntonic). There is an increasing sense of wishing to commit the act with a sense of pleasure occurring once the act has been committed. These disorders have also been conceptualized as non–substance-related addictions. They do not represent personality disorders. They are described in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (fourth edition, text revision, 2000; *DSM–IV–TR*) as impulse-control disorders, and in the World Health Organization's *International Classification of Diseases and Related Health Problems* (1992; *ICD-10*) as habit and impulse disorders.

In *DSM–IV–TR*, their essential features include the following:

- The failure to resist an impulse, drive, or temptation to perform an act that is harmful to the person or to others.
- For most disorders, the individual feels an increasing sense of tension or arousal before committing the act and then experiences pleasure, gratification, or relief at the time of committing the act.
- Following the act there may or may not be regret, self-reproach, or guilt.

Included are the conditions of pathological gambling, pyromania, kleptomania, and tricotillomania.

ICD-10 (World Health Organization, 1992) has a similar definition for this group of disorders, which it terms "Habit and Impulse Disorders," but also includes intermittent explosive (behavior) disorder. *ICD-10* points out that these disorders have no clear rational motivation.

Pathological gambling, pyromania, and intermittent explosive (behavior) disorder are more common in men, while kleptomania and tricotillomania are more common in women. There is an absence of epidemiological studies of the prevalence of these disorders, but rates among psychiatric inpatients may be higher than in the general population. Using the Minnesota Impulsive Disorders Interview (Christenson et al., 1994), Grant, Levine, Kim, and Potenza (2005) found the following rates, mainly comorbid with depression, among inpatients:

- Kleptomania 8 percent
- Pathological gambling 7 percent
- Intermittent explosive (behavior) disorder 6.4 percent
- Tricotillomania 3.4 percent.

Overall, such impulse control disorders are probably underdiagnosed.

Other disorders, such as pathological buying (oniomania or shopaholism), characterized by buying items that are not needed and often storing them unopened, and workaholism, have also been considered to be impulse-control disorders, but are not classified as such in *DSM–IV–TR* or *ICD-10*. Such behaviors may be motivated by a need for compensation or as a substitute for something missing in life or as a depressive equivalent.

Crime is law-breaking behavior. Impulse-control disorders may lead to offending either directly, for instance, in pyromania, or indirectly, for instance, for financial gain in a pathological gambler. Individuals who commit crimes due to such a disorder have, however, usually been deemed legally culpable for their actions, even though their propensity is psychiatrically considered to be irresistible. It has been argued, however, that this may be a legal injustice, as such individuals clinically apparently have little or no control over their actions.

The interface between impulse-control disorders and offending raises philosophical questions, including questions about the nature of free will and whether all behaviors are determined by the effects of genes, environment, and background. Are those with impulse-control disorders less responsible for their behavior and should they therefore be punished less than those of normal responsibility? Even if impulse-control disorders have a biological basis, in clinical practice the aim is, however, to encourage the sufferer to take responsibility for his or her actions.

HISTORY

During the eighteenth century, the concept of "monomania with propensity" developed, referring to the fact that apparently insane, incomprehensible

actions did not always appear to be the result of delusional thinking. This and subsequent historical developments have been discussed by Gibbens and Prins (1962), who cite the following historical landmarks:

- Philippe Pinel (1745–1826), in the eighteenth century, refer to "mania without delirium" as being a disease of the willpower.
- Esquirol referred in 1885 to instinctive monomanias, including homicide, fire-setting and alcoholism, that is, respectively, homicidal monomania, pyromania, and dypsomania, where the individual acts "without passion or motive but only under involuntary instinctive impulse."
- Referring to some cases of theft and homicide, Rush in 1810 described them as an illness of the moral willpower and equated this illness with the involuntary movements of convulsions.
- Mathey coined the term klepemnia (theft) to add to the list of other manias including dipsomania and pyromania.

As noted by Topp (1973), it was the Frenchman Marc in 1833 who first used the term kleptomania when describing a number of wealthy individuals who carried out bizarre, worthless thefts in which they had little intrinsic interest and to which they confessed spontaneously when challenged.

Subsequently it has been considered that instinctive monomanias such as kleptomania are very rare and, indeed, it has been questioned whether they, in fact, exist. Terms such as pyromania and kleptomania have indeed tended to be increasingly discarded. Neustatter (1953) doubted whether kleptomania existed as an entity, but, if it did, he suggested that it was part of a psychopathic personality that gives way to impulses.

Differentiation from Obsessive-Compulsive Disorder

An important differentiation in this area is between compulsions and impulses. Compulsions, as seen in obsessive-compulsive disorder, are characterized by nonsituational preoccupation with subjective compulsion despite conscious resistance, such preoccupations being thoughts (ruminations or obsessions) or acts (rituals or compulsions). Where there is poor impulse control, impulses are poorly resisted, and this is much more common than compulsions. In the case of obsessive-compulsive disorder, sufferers in general do not act on their ruminations, unlike those with poor impulse control. The key difference between impulse-control disorders and obsessive-compulsive disorder is that while both may lead to relief of anxiety and tension, in obsessive-compulsive disorder the thought of carrying out the act must not in itself be pleasurable, that is the thought must be egodystonic.

PATHOLOGICAL GAMBLING

This is defined in *ICD-10* as persistently repeated gambling that continues and often increases despite social consequences. Gambling involves risking something of value, not necessarily money, in a game or other uncertain event, with the aim of achieving greater value. There is a range of gambling behaviors from the culturally normal to hazardous, professional, problematic, and pathological gambling. In contrast to professional gamblers, who may carefully plan their gambling and base it on information to decrease the risk, pathological gamblers will myopically gamble despite repeated and heavy losses resulting in adverse family and social consequences and financial ruin, to which they appear hyposensitive. Four phases may be distinguished: winning, losing, desperation, and eventual giving up. Problems do not arise from the gambling itself but from the consequences, as seen in alcoholism. Indeed, pathological gambling shows features characteristic of an addiction, with loss of control, extremes of emotions reflecting autonomic nervous system changes, and withdrawal phenomena when not gambling.

DSM–IV–TR defines the essential feature of pathological gambling as a chronic and progressive failure to resist impulses to gamble, with behavior leading to much damage to personal and family life. Evidence suggests this to be a valid and reliable diagnosis (Stinchfield, 2003). Efforts to control, resist, or stop gambling generally fail, and the behavior has been equated to an addiction, with withdrawal symptoms of irritability and restlessness if the person is unable to gamble and an escalation in the size and frequency of bets or other stakes to achieve a desired level of excitement. Such individuals will tend to respond to repeated losses by gambling further to "chase" their losses, in spite of increasing debts, marital breakdown, and law involvement. They anticipate losses as shown in fMRI studies (reduced activity in the ventromedial prefrontal cortex), even while their appetite for gambling and their impulsivity increase, and they will continue gambling until they have lost their available resources. Psychodynamically, they appear unconsciously to aim to lose their money. They tend to focus on their winnings, disavow or deny their losses, lack the courage to own up to losses, and gamble more to break even. This is a pattern also seen in stock market "rogue" traders.

Pathological gambling may also lead to disturbances in eating and sleeping and in sexual relationships, as well as to difficulties in sustaining employment. Lying to and relying financially on friends is also characteristic. Some gamblers steal to finance their habit, and pathological gambling may only come to light following a court case regarding an acquisitive offense such as theft, fraud, or embezzlement.

Some individuals present following an overdose or self-harm, which occurs in 10 percent of pathological gamblers, or with depression. Suicide occurs in 2 percent of attendants of Gamblers Anonymous. Legg-England and Gotestam (1991) and Raylu and Oei (2002) have reviewed pathological gambling in detail.

Epidemiology

Gambling itself is common, with estimates of prevalence around 40 percent of the British population and 60 percent of the United States population (Moran, 1983). Even these figures may now be underestimates, given the current availability of national television and other lotteries. Wardle et al. (2007) found the prevalence of gambling in the United Kingdom (UK) to be 68 percent, but 48 percent when the UK National Lottery was excluded. It is said to be more common among Chinese and less common among Scandinavians. It is more common in men, those with a past history of psychiatric disorder, and criminals. Problem gamblers cause themselves or others to suffer. It is of course possible to lose control and bankrupt oneself through gambling in just one day. Pathological gambling is associated with tolerance and withdrawal phenomena and has been found to have a prevalence of 0.25 percent in Australia (Dickerson, 1988) and 0.77 percent in the United States. Shaffer and Korn (2002) reviewed 120 studies and suggested a lifetime rate of 1.6 percent, a figure not dissimilar to that for schizophrenia. Wardle et al. (2007) in the UK estimated that 0.5–0.6 percent of the population were problem gamblers. The Royal College of Psychiatrists in the UK in 1977 described around 10 percent of prisoners as suffering from pathological gambling.

Pathological gambling is certainly more obvious but probably also more common among those, especially men, who indulge in horse and dog racing, in which losses soon become apparent. Women pathological gamblers, on the other hand, have been reported to be more likely to make use of specialized helplines (Potenza, et al., 2001). In the UK, it is said that women prefer bingo, which may lead to less pathological gambling and in which losses tend to be smaller.

Etiology

The predominant motivation for pathological gambling is the sense of thrill and pleasure at the risk taking, as reflected in changes of heart rate demonstrated during gambling. Winning produces euphoria, said to be comparable to the effects of amphetamines, and helps individuals switch from negative inter-

nal mood states, including despondency and loneliness. Dostoyevsky, in his autobiographical novel *The Gambler*, described the reward of a sense of power obtained from gambling. Freud (1928), commenting on this, considered that Dostoyevsky did his best writing after a big loss from gambling as he was then freed from unconscious guilt feelings concerning patricidal urges that inhibited his creativity. Gambling has also been described as serving to gratify oedipal wishes, for example, wishing to defeat a tyrannical father or woo a mother (Frosch, Frosch, & Frosch, 1985; Greenson, 1947). Moran (1983) cited social pressures, early exposure to gambling, and a father who gambled or drank alcohol as etiological factors among male gamblers, while having an alcoholic spouse who was often absent was characteristic more of women who gamble. Gambling increases with the number of gamblers in one's social network, being initially a social activity, but when pathological, it is usually undertaken alone.

Learning theory has suggested that the pattern of intermittent (variable ratio) reinforcement, the most potent schedule for conditioning, particularly applies to gambling, where repeated losses with frequent near misses are combined with occasional random wins with immediate payouts. The prospect of small but immediate rewards is preferred to higher but delayed rewards. Evidence of psychological dependence may become manifest by the appearance of what can be considered withdrawal symptoms and craving following the stopping of such activity.

Biological factors may also be important (Sharpe, 2002). The orbitofrontal cortex and anterior cingulate gyrus are involved in reward mechanisms. Potenza et al. (2003) undertook an fMRI study of pathological gamblers that showed, compared with controls, decreased activation of the orbitofrontal cortex, basal ganglia, and thalamus, which have been linked to impulsivity and disinhibition.

Pathological gambling has been suggested to be associated with low central serotonin levels, as seen in other impulse-control disorders, and low central dopamine activity, as seen in other addiction disorders. Serotonin is involved in mood and impulse control and dopamine in reward, pleasure, and motivation. Of note is the increasingly recognized phenomenon of individuals with Parkinson's disease, who have low levels of the neurotransmitter dopamine, presenting for treatment of gambling after having been treated with pro-dopaminergic agents such as L-dopa (Driver-Dunkley, Samanta, & Stacy, 2008). Norepinephrine (noradrenalin), which is involved in arousal and excitement, and opioids, involved in urges and pleasure, may also be important.

Comorbidity

While pathological gambling often arises in the absence of other psychiatric disorders, personality disorder, especially antisocial, narcissistic, and borderline

types, and depression may also be present. The presence of personality disorder will, however, only explain part of the excess of impulsivity. Hypomanic or manic episodes of bipolar disorder, which may be associated with general overspending and grandiose beliefs about one's wealth and ability to make money, including the ability to counter losses, may also lead to excessive gambling. Problem gambling is associated with attention deficit hyperactivity disorder (ADHD), delinquency, and recidivist offenders, and is especially high among young offenders.

Risk Factors

These include being male, over 45 years of age, cigarette smoking, alcohol abuse, low income, having debts, being a foreign national, depressive disorder, flat affect, having the metabolic syndrome, and sleep difficulties in females. A history of previous treatment for gambling is self-evidently an important risk factor.

Assessment

This should include eliciting when gambling started and when it became regular, who introduced the individual to gambling or encouraged the individual to gamble, the circle of gambling friends, and the history of escalation in patterns of gambling. Is gambling increased when the individual is despondent and/or by alcohol consumption?

The onset and presence of symptoms of pathological gambling should be noted, for example, inability to stop despite debts, withdrawal symptoms such as restlessness and irritability when not gambling, chasing losses, and raising stakes for the thrill.

The motivation to change should also be assessed. Stages of change include the following:

- Precontemplation, when the need to change is recognized
- Contemplation, when the problem is acknowledged and the individual is willing to change
- Action taken to change
- Maintenance to sustain control of or abstinence from gambling.

Screening instruments such as the South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987) can be useful, especially in screening populations at risk (a score of 3 to 5 out of 10 indicates problem gambling, while a score of over 5 indicates pathological gambling). The SOGS may produce excessive false-positives compared with the National Opinion Research Center DSM Screen

for Gambling Problems (NODS; Gerstein et al. 1999) which was developed as a population-based telephone screening tool to identify gambling problems according to *DSM–IV* criteria (Hodgins, 2004). Stinchfield (2002) found that the SOGS demonstrated good to excellent classification accuracy in his large gambling treatment sample, but had poorer accuracy in the general population sample with a 50 percent false positive rate; the SOGS overestimated the number of pathological gamblers in the general population, compared with *DSM-IV* diagnostic criteria.

A further tool is the Canadian Problem Gambling Severity Index (Ferris & Wynne, 2001), which contains 9 items of the 30-item Canadian Problem Gambling Inventory (CPGI).

Management

Health promotion to populations at risk as identified by screening to counter excessive gambling before problems develop is, of course, ideal. However, pathological gambling may not always follow a chronic and persisting course. Slutske (2006) reported that among individuals with a lifetime history of pathological gambling, 36 to 39 percent did not experience any gambling-related problems in the past year, even though only 7 to 12 percent had ever sought either formal treatment or attended meetings of Gamblers Anonymous. About one-third of the individuals with pathological gambling disorder in her study of two nationally representative U.S. samples were characterized by natural recovery.

Comorbid psychiatric disorders should be excluded or treated, for example, depression. As with other addictive behaviors, selective serotonin reuptake inhibitor (SSRI) antidepressants at high doses have been recommended (Grant & Kim, 2002), and the opioid antagonist naltrexone has been recommended. The selective norepinephric (noradrenergic) reuptake inhibitor (SNRI) venlafaxine, the 5-HT1A partial agonist buspirone, the stimulant cognitive enhancer modafinil, and the psychostimulant methylphenidate have all been used.

Cognitive-behavioral therapy, which concentrates on reducing the preoccupation with gambling and can involve motivational interviewing and risk/harm reduction strategies, has been successfully used and has also been combined with a 12-step group program (Petry & Roll, 2001). There is no real evidence for the efficacy of psychodynamic psychotherapy or aversive behavioral therapy in this disorder. Dickerson (1989) described 22 uncontrolled studies offering a variety of management approaches, including most forms of psychotherapy. Support and counseling for the family, which can include brief focal marital counseling. may also be required.

Help is often only sought as a result of the consequences of gambling, such as debt, deteriorating marital and other relationships, and law involvement, rather than as a result of a primary desire to stop gambling itself. Most gamblers cannot contemplate complete abstinence, though some may consider as a reasonable goal stopping gambling for a number of months with a view to continuing controlled gambling thereafter.

One approach is for the family income to be paid into an account over which only the spouse has control (Moran, 1983). Gamblers Anonymous adopts the approach used by Alcoholics Anonymous and may be more helpful than standard traditional psychiatric approaches. For relatives, Gam-Anon is available for mutual support, akin to Al-Anon for relatives of those suffering from alcoholism. Local citizens advice bureaus and money advisory services may assist with resulting financial difficulties.

Few pathological gamblers will consider a goal of total abstinence. In spite of management approaches, the prognosis is generally considered poor, although 10 percent stop spontaneously and progression and chronicity are not inevitable. Relapse rates after treatment vary from two-thirds to 70 percent. Duration of the disorder and neurocognitive measures of disinhibition and decision-making are powerful predictors of relapse in pathological gambling (Goudriaan, et al., 2008).

Clearly, as the ease of availability of gambling increases, so does the risk of developing pathological gambling. Legislation, for example, controlling casinos and society's hedonistic attitudes, for instance, to national lotteries and so on, may be important in prevention, although a study by Bondolfi, Jermann, Ferroero, Zullino, and Oseic (2008) in Switzerland showed no increase in pathological gambling following an increase in the opening of casinos.

PATHOLOGICAL FIRE-SETTING (PYROMANIA)

ICD-10 defines this as repeated fire setting without any obvious motive. There is an intense interest in watching fires burn and feelings of increasing tension before the act and intense excitement immediately after it.

In *DSM–IV–TR*, it is also classified as an impulse disorder. There is deliberate and purposeful fire-setting on more than one occasion. Tension or affective arousal is present before the act. There is intense pleasure, gratification, and/or relief when setting fires or when witnessing or participating in their aftermath. Such individuals, also referred to as fire bugs, are fascinated with, curious about, and attracted to fire.

This group of fire-setters includes those who are described as having an irresistible impulse and a repeated urge to set fires, which they do not fully

understand and about which they are often inarticulate. They are often isolated and inadequate people who set a number of fires impulsively and who may escalate the seriousness of their fire-setting. This group also overlaps with those who set fires for tension or depression reduction, that is, as an anxiolytic or an antidepressive act. Such individuals discover that fire-setting relieves feelings of despondency or tension. An analogy can be made with the calming effect normal individuals report when observing and sitting in front of a glowing coal fire.

In the past, fire-setting by men was considered to be frequently associated with direct sexual arousal by such an act, that is, the use of fire as a fetish, and there was considerable psychodynamic interest in the symbolism of fire, for example, flames of passion, burning desire, blazing rows, and so on. Freud (1932) described the glow of fires as reflecting sexual excitement and the motion of flames as symbolic of the phallus in action. However, while a number of fire-setters may indeed obtain a sense of excitement from their actions, those who are specifically sexually aroused and who may even masturbate after setting fires are rare.

Pathological fire-setters are a subset of those who tend to set more fires and to whom the fire is a thing of interest in itself. Individuals have a fascination with fire and hence the arson appears outwardly motiveless. There may also be an associated fascination with fire engines and calling out the fire service. The making of false telephone calls to the emergency services can result in a charge of "wasting electricity" in the UK.

Other clinical features include evidence of advanced preparation and indifference to the consequences of fire-setting to property or life.

It is rare in children, but more common in male adolescents, particularly those with poor social skills and learning difficulties.

The Offense of Arson

Arson is the offense associated with fire-setting and is the unlawful and malicious (willful) destruction of or damage to property by setting a fire. Legally, the more serious charge is arson with intent to endanger life or being reckless as to whether life was endangered. Owing to problems of detection, only 5 percent of cases of arson end in successful prosecution in the UK. In the UK, one school in eight is subject to arson each year.

If an individual is charged with arson, it is important to reconstruct in detail what happened at the time of the offense, for example, reading witness statements related to the case, and not just depending on the actual legal offense category. For instance, arson may be the setting fire to a waste paper bin in a busy

hospital ward in front of observing staff and fellow patients, or an impulsive or planned serious fire, in circumstances unlikely to be detected, with intent to kill. Of psychodynamic note is the fact that fire almost uniquely can make things disappear, including evidence. Historically, one reason individuals were burnt at the stake was to avoid spilling blood.

Epidemiology of Arson

Approximately 40 percent of all serious fires are started deliberately. Six percent of fires in the UK are recorded as arson. Arson is responsible for 1 percent of all serious crimes in the UK. However, as the evidence is often burnt, only about a quarter of arson offenses result in conviction. The peak age for arson is 17 years for men and 45 years for women. Eighty percent of those convicted are men. There is increased incidence of arson among those with learning disabilities and those who suffer from alcohol dependence syndrome. Fifty percent of cases of arson follow alcohol abuse, especially binge drinking of alcohol.

Clinical Classification of Arson

Numerous attempts have been made to classify arson clinically, for example, by Puri, Baxter, and Cordess (1995).

There is no typical arsonist. Psychiatric difficulties are common, but the most common diagnoses are personality disorder and substance abuse, in up to two-thirds of cases, with about 8 percent suffering from a psychosis. Pure pyromania appears rare (1 percent) among convicted arsonists (Ritchie & Huff, 1999).

A classification based on these studies is as follows:

1. Fire as a means to an end (motivated). This includes the following:

   (a) Those who set few fires:

   - *Psychosis*, such as schizophrenia. Such individuals may set fires, for instance, to burn out the devil or evil, or in response to hallucinatory voices.
   - *Displaced revenge, anger, or jealousy*. Rather than overt direct aggression against an individual, aggression may be displaced into setting fire to that individual's property. For instance, an employee of a warehouse or supermarket is told off by his boss, but rather than retaliate directly, physically, aggressively, the employee returns after business hours to set fire to his boss's property. This is the commonest reason (in almost 50 percent cases) found by psychiatrists among arsonists referred to them.

- *Cover-up of other crimes*, for example homicide. Modern forensic science, however, usually overcomes such attempts at a cover-up.
- *For insurance*. This has become increasingly common in recent years; for instance, french-fry fires to finance the redecoration of a kitchen.
- *Political motivation*. For example, to further their rise to power, Nazi storm troopers set fire to the Reichstag in 1932 in Berlin.
- *Adolescent gangs*. Individuals are generally more likely to be disinhibited and behave antisocially in a group than when alone. This group is associated with a low rate of recidivism, except among gang leaders.

(b) Those who set more fires:

- *Desire to be powerful or a hero*. Members of this group often have inadequate personalities. Their low self-esteem is bolstered by the sense of power they feel at the results of their having set fires, for example, the panic and the emergency services with flashing lights rushing to the scene. Sometimes this is combined with a desire to be a hero, so that after setting a fire the individual may rush into the premises and rescue pets or the elderly or infirm. On occasions they are caught owing to being seen repeatedly or even photographed, for instance, seen in a local newspaper at the scenes of the fires.
- *To earn money*. This occurs when part-time firemen on call-out rates set a fire. It is a particular problem in rural areas and in some countries such as France. Some individuals may be drawn to the fire service because of their fascination with fire, and psychodynamic associations have been made regarding the phallic symbolism of hoses. Anthropologists and evolutionists have suggested that females may have been impressed by the ability of males to put fires out by urinating.
- *As a cry for help*. To bring attention to a distressed emotional state.

2. Fire as a thing of interest

   This includes the following:

   - Pathological fire-setting (pyromania).

Differential Diagnosis of Pyromania

This includes conduct, adjustment, affective, and psychotic disorders.

Comorbidity of Pyromania

This may include substance misuse, past history of sexual or physical abuse, and personality disorder, especially antisocial personality disorder. High rates

of previous sexual abuse in women who set fires have been frequently described in clinical practice.

Assessment of Arsonists

This depends not only on a careful, detailed history and mental state examination but also on the gathering and study of objective information such as witness statements related to a case. It is important to determine the presence or absence of psychiatric abnormality, especially at the time of the offense, and its relationship with the offense itself. It is clearly important to determine whether there is a history of previous fire-setting and to examine precipitants.

It should be noted that suicide by fire is particularly associated with schizophrenia, perhaps explaining the choice of this most painful means of suicide. Historically, it has been described in the early nineteenth century among Hindu widows in India (suttee) and among monks protesting in Vietnam during the mid-twentieth-century war.

Management

This should clearly address any underlying or comorbid psychiatric disorders. Psychological intervention, for example, with cognitive-behavioral therapy, may be helpful.

The potential dangers of fire-raising must always be borne in mind. The fire service view is that a large fire is merely a small fire not brought under control.

In cases where an individual is charged with arson, the courts will be particularly concerned with the protection of the public and it is likely to be unwilling in serious cases of arson to consider outpatient care or placement in an open psychiatric ward. Ordinary psychiatric hospitals are also inevitably reluctant to admit those who have set fires, so if hospital treatment is required it is frequently undertaken under conditions of medium or maximum security. In the absence of a psychiatric disposal, the courts usually impose a custodial sentence. In England and Wales, under the Criminal Damage Act 1971, a maximum sentence of life imprisonment can be imposed for arson (Section 1) or arson endangering life (Section 2).

Prognosis

Further offenses of arson are increasingly likely if there has been a history of previous arson and if the offender continues to have an irresistible impulse to set fires or to relieve tension or obtain pleasure or sexual excitement from such fire-setting. Increased risk of further fire-setting is seen in individuals who

suffer from psychosis, learning disability, or dementia. However, in an individual case, it may be difficult to tell whether that individual will reoffend. The risk of further serious offending after a period in prison or hospital is low; however, the risk of reoffending may not be apparent in the short term but only on longer follow-up. For example, Soothill and Pope (1973) found a 4 percent recidivism rate over a 20-year period, and Sapsford, Banks and Smith (1978) reported a range over time of 2 to 20 percent.

PATHOLOGICAL STEALING (KLEPTOMANIA)

In *ICD-10*, this is defined as repeated failure to resist impulses to steal objects that are not required for personal use or monetary gain. Objects may be discarded, given away, or hoarded. The person may even later offer to pay for items stolen. An increasing sense of tension before and a sense of gratification during and immediately after the act are seen.

Epidemiology

This is usually seen in women of a mean age of 36 years with a mean duration of illness of 16 years (often after an onset in childhood) (McElroy, Pope, & Hudson, 1991).

The term kleptomania comes from the Greek for stealing madness. The disorder is generally said to be rare, with fewer than 5 percent, according to *DSM–IV–TR*, of arrested shoplifters giving a history consistent with kleptomania. However, such individuals rarely seek psychiatric help and often avoid detection, so that estimates of kleptomania's prevalence have been variable, even up to a quarter of all shoplifters. Up to a quarter of those suffering from bulimia nervosa are said also to meet the diagnostic criteria for kleptomania. This condition is certainly more prevalent in females than males, unlike other impulse-control disorders such as intermittent explosive disorder and pyromania, where males predominate. Onset is around 20 years of age. However, diagnosis is usually made one or two decades later. The individuals concerned are typically married. Males may be underrepresented because they are more likely to receive custodial sentences precluding reoffending in the community during periods of imprisonment.

Clinical Features

Stealing is perpetrated without much planning and without the assistance of others. The objects taken are not needed for personal use or for their monetary

value and may be given away, discarded, or returned surreptitiously, or kept and hidden. The individual invariably has enough money to pay for the stolen objects, but the theft is not committed to express anger or vengeance. Typically, when diagnosed, such individuals have appeared in court several times, feel guilt or remorse, but have not sought psychiatric treatment. There is often a history of a number of years of chronic dysphoric mood, and of the display of signs of depression and anxiety. Individuals' relationships and marriages are often unhappy. There is frequently a history of sexual difficulties and dysfunction and a past history of a turbulent childhood. Individuals often show poor impulse control generally and evidence of personality disorder, but the stealing is not primarily the result of conduct disorder or antisocial personality disorder. They share similarities, therefore, with those who have a past history of childhood sexual abuse.

Differential Diagnosis of Kleptomania

In ordinary shoplifting the act is usually well planned, although it may be impulsive, but it is motivated by need or monetary gain and the objects taken are for individuals' use or monetary gain. Some individuals who shoplift may attempt upon arrest to simulate kleptomania; they are then referred to as malingering.

Shoplifting may also occur in conduct disorder, antisocial personality disorder, depression, manic episodes, schizophrenia, or organic mental disorders, but in such circumstances the act is related to the primary diagnosis.

Comorbidity of Kleptomania

This includes eating disorders or substance abuse disorders. Kleptomania may be precipitated by major stressors such as life events. Depression is common and bipolar disorder may not be infrequent (Lejoyeux, Arbarataz, McLoughlin, and Adès, 2002).

Etiology

There is no definite evidence of a specific genetic or inherited predisposition, although a biological basis has been suspected; for example, Grant, Correia, & Brennan-Krohn (2006) found decreased white matter integrity in the inferior frontal brain regions in women with kleptomania.

Kleptomania has been viewed as a variant of depressive disorder. Those suffering from kleptomania often have depressive symptoms, and the thieving

itself may produce a stimulating excitement that has an antidepressive effect. It has also been viewed as a variant of obsessive-compulsive disorder, but only about half of cases experience with stealing the relief or tension characteristic of that disorder, and in obsessive-compulsive disorder there is not typically the sense of gratification seen in kleptomania.

Psychodynamic Theories

These have included the following:

- *Loss substitution*, in which kleptomania provides symbolic compensation for threatened or actual loss (Cupchik & Atcheson, 1983).
- *Drive theory*, which considers kleptomania in terms of a forbidden activity, engaged in secret and thus having a sexual basis.
- *A perversion*, in which stolen objects represent fetishes as defined by Fenichel.
- *A defensive strategy*. used by females, for example, to acquire a symbolic penis to counter castration fears.
- *Self-psychological theory*, in which kleptomania is seen as a response to narcissistic injuries and a means to counter fragmentation of self.

Management

In keeping with a view that kleptomania may be an equivalent of depressive or obsessive-compulsive disorder, kleptomaniac individuals often respond well to antidepressant medication, especially SSRIs such as fluoxetine. Cognitive-behavioral therapy has also been found to be effective (Gudjonsson, 1990). Psychotherapeutic approaches, including family therapy, have also been reported. A self-imposed ban on shopping may, however, be required where treatment fails.

Prognosis

The condition tends to be chronic but waxes and wanes.

The Offense of Shoplifting

The technical offense is theft, that is, from shops, an offense that, as with all offenses of theft, requires the intent permanently to deprive, as well as the act, for the offense to be proved in court. Intent would clearly be indicated if an individual were seen to be hiding an object in his coat and to be looking around

to make sure he was not being observed. In absent-minded shop-lifting, there would theoretically be no intention to deprive.

Epidemiology

In the UK, about 5 percent of all shoppers shoplift (Buckle & Farrington, 1984). However, up to 50 percent of goods taken from shops may be taken by the staff of those shops, as is the case with many thefts from businesses. Sociologists have viewed shoplifting as a social disorder created by a consumer society and precipitated by the visual provocation of shop displays. Open shelves increase sales and reduce the requirement for staff, as in supermarkets, but they are associated with increased shoplifting, with such businesses having to take this into account in their business planning. Some items are left near the checkout till for impulse buying and, in addition, provide easy but inexpensive objects to be shoplifted. Objects are often taken suddenly on impulse and are of trivial value or useless. Some individuals appear to regard shoplifting as an accepted perk of shopping and may pay for other items.

Up to the early 1970s, most shoplifters in the UK were women, who then undertook more of the shopping than now, and 50 percent showed evidence of psychiatric disorder. Ninety percent did not reoffend after conviction (Gibbens & Prins, 1962). However, the majority of shoplifters in the UK are now male and between the ages of 10 and 18 years, as reflected in signs on shops limiting the number of children allowed in at one time. Males are now more likely than females to have previous convictions. The incidence of psychiatric disorder has been reduced to about 5 percent, and it is questionable now whether shoplifting deserves more psychiatric attention than other thefts (90 percent of all offenses are acquisitive). The previous predominance of female offenders coincided with the view that female offenders tended to be psychiatrically disordered, which may explain the courts' requests for psychiatric reports more often in shoplifting offenses than in other, male-dominated, offenses.

Classification of Shoplifters

Shoplifters have been subject to lay and legal stereotyping as needy, greedy, or seedy. Bluglass (1990) distinguished three groups of shoplifters: professional, amateur, and associated with psychiatric disorder. Building on Bluglass' work, a more detailed classification is as follows:

1. Shoplifting for simple gain, plus excitement with or without associated marked antisocial attitudes:

The principal motivation is excitement, and such individuals are responsible for a significant proportion of shoplifting in large cities. Individuals often feel less constrained by another country's laws when abroad. This category also includes organized gangs and those with chaotic lives who steal impulsively and commit other offenses. They may come from antisocial families and be subject to relative poverty. Such shoplifting may be associated with resentment and feelings of bitterness associated with individuals' lifestyles.

2. Shoplifting associated with psychiatric disturbance:

   The commonest association in this group is with depression in people of previous law-abiding personality. These may include isolated younger women with children, but they may also include middle-aged women isolated from their families, who have lost children, who have experienced the loss of a husband (including loss owing to his career), and who also may have significant physical complaints or ill-health and/or be chronically depressed. Shoplifting may be an early symptom of depression. The depression may also be associated with acute losses. Law involvement, including court appearances and associated publicity, can precipitate self-harm or suicide where offenders are depressed.

   In cases of shoplifting and depression, the motivation may arise from feelings of guilt, a desire to be caught and punished, a cry for help, or represent an act of self-comfort or a treat. Other dynamics include secondary gain, in the newly poor to keep up appearances and to steal something for oneself that is not purchased with money from parents or a husband. In married female offenders, there may particularly be sexual difficulties or rejection and marital problems. Shoplifting may be an act of revenge on a husband or a partner to induce shame or punishment. For instance, it may result in the female having to be accompanied by her husband when shopping in future or alternatively in the husband having to undertake the shopping from which the wife can then opt out. For such individuals, a prison sentence may at one level be a relief from their marital or family situation.

   Other psychiatric disorders associated with shoplifting include anorexia and bulimia nervosa, which may reflect both hunger for food and impulsivity, and early dementia, which is associated with disinhibited behavior, lower resistance to temptation, poor judgment, and late onset offending. Shoplifting may also occur on occasion in association with other psychotic mental illnesses, alcoholism, and learning disability.

3. Absent-minded shoplifting:

   This implies no intent permanently to deprive and, if successfully argued in court, a not guilty verdict will result. Such shoplifting may result from undue preoccupation, distractions or harassment, for example, caused by the shopper's own accompanying children. Other causes cited include

claustrophobia in shops and various medical or psychiatric drugs that impair concentration or cause confusion. It is the prescribing doctor's responsibility to warn of such side effects from medication. Although a defense based on medication side effects, including the effects of benzodiazepines, is not infrequently put forward in court by shoplifters, in reality it is rarely a primary cause.

4. Shoplifting in children:

   This peaks around 14 to 15 years, with boys being predominant. Boys steal candy and books. Girls tend to steal cosmetics and clothes. The items stolen are usually of little value. The commonest group is in fact that of "normal" children stealing for excitement. However, child shoplifting may occur owing to subcultural standards or as an expression of emotional disturbance, for example, as an act of defiance against parents, as a cry for help, or in association with feelings of depression, worthlessness, and a sense of guilt.

Assessment of Shoplifters

An examination of the history and mental state of the individual should elucidate motives and detect any evidence of formal psychiatric disorder. The motive may often initially appear obscure, with useless objects or objects of trivial value taken suddenly on impulse, sometimes as a treat or arising from concealed resentment. Alcohol or drug abuse is often associated with shoplifting. Additional information should be obtained if possible, for example, from the arresting police officer. It is often useful to discuss the case with the probation officer if one has been requested by the court to prepare a social enquiry report, which should also be read. It is essential to establish whether there is a history of previous convictions for shoplifting and any past psychiatric history and its relationship to offending.

Management

If it is argued on psychiatric grounds that there was no intent to shoplift and the patient pleads this successfully, a finding of not guilty will result. However, individuals are often deterred from such a defense, for example, a defense involving absent-minded shoplifting, as it will often require a number of court appearances and considerable legal expense, including payments to lawyers, to plead this successfully, and it may well involve local publicity.

Where the court accepts that intent permanently to deprive was present, the individual is legally convicted of theft. If the individual does suffer from a psychiatric disorder, including kleptomania, requiring treatment, psychiatric

evidence may be used in mitigation with a view to altering the sentence: for example, a psychiatric recommendation of outpatient psychiatric treatment may be made as part of a probation order.

TRICOTILLOMANIA

This is a habit and impulse disorder characterized by noticeable hair loss resulting from recurrent failure to resist impulses to pull out the hair. Hair-pulling is usually preceded by mounting tension and followed by a sense of relief or gratification. It is not itself directly associated with criminality, although it can be associated with personality disorder, which in turn may be associated with offending. It has been well reviewed by Walsh and McDougall (2001).

INTERMITTENT EXPLOSIVE (BEHAVIOR) DISORDER OR EPISODIC DYSCONTROL SYNDROME

This is included in *ICD-10* under habit and impulse disorders, but not in *DSM–IV–TR,* and is characterized by episodes of sudden unprovoked violence. Onset is in adolescence, and males outnumber females in a ratio of 4:1. It was originally conceptualized as a form of limbic epilepsy, but this has not been borne out. The syndrome may, however, be associated with soft neurological signs and temporal lobe electroencephalogram (EEG) abnormalities, and may be helped by anticonvulsants such as carbamazepine and sodium valproate. Mood stabilizers may also be used, and lithium and SSRI antidepressants may also help, suggesting a link to mood (affective) disorder. This disorder, in fact, usually occurs in those with a severe, often explosive, personality disorder with a propensity under stress to intemperate outbursts of anger and impulsive violence when frustrated, which equates to the emotionally unstable impulsive-type personality disorder of *ICD-10* and falls within the antisocial personality disorder of *DSM–IV–TR*. It is of note that half of persistently aggressive offenders in general are said to have an abnormal EEG record, often an immature record (persistence of excess posterior slow-wave activity), characteristic of those with psychopathic disorder and not diagnostic of epilepsy.

NONPARAPHILIC SEXUAL ADDICTIONS

These are culturally acceptable sexual interests and behaviors so intense or frequent that they interfere with sustained intimate relationships. They include, for example, compulsive masturbation, repetitive promiscuity (using people as sex objects and often involving prostitutes), and dependence upon anonymous

sexual outlets such as pornography or telephone sex. These have been conceptualized as addictions, compulsions, or hypersexuality, or as disorders of impulse control (hence their inclusion here).

Nonparaphilic sexual addictions may be depressive equivalents, especially in the presence of affective disorder that causes disorder of sexual regulation and that, in turn, increases nonconventional sexual interests. There are similarities to bulimia nervosa, which also shares comorbidity with depression and responds to antidepressants. Such sexual addiction has also been viewed as an obsessive-compulsive disorder variant. It has been criticized as a concept for being used too loosely and defensively and as including those merely with a high sex drive or those who are prone to marital infidelity. The concept has gained increasing public awareness through celebrities who have cited such a diagnosis and been admitted to private institutions for treatment. Successful treatment has been undertaken with the SSRI fluoxetine at 20–60 mg a day, which would be in keeping with such sexual addiction's being either a depressive equivalent or an obsessive-compulsive disorder variant.

Comparison of Nonparaphilic Sexual Addiction and Paraphilias

Nonparaphilic sexual addictions should be differentiated from paraphilias or disorders of sexual preference, in which there is a persistence of and preference for such behavior over normal adult sexual behavior, which may then lead to sexual offending, although in both there may be feelings in keeping with addiction, for example, a sexual "high" and tolerance with an escalation of the stimulus intensity needed to attain the same level of pleasure.

Those with paraphilias often feel they are addicted to such behaviors or view such behaviors as compulsions. They experience their paraphilic urges as instant, demanded, and fixated. They describe feeling in an altered state during paraphilic acts and unable to stop such behavior unless others intervene. Once caught, they may say they will never behave so again but they do, in spite of law involvement and risk to self or others. This may reflect dissociation due to high sexual arousal. Those with paraphilias often want acceptance from professionals rather than increased self-understanding or change.

The Internet

The majority of sites on the Internet are sexual in content and estimates suggest that up to 1 in 200 of such sites concern child pornography. Such sites may be used by those with nonparaphilic sexual addiction and those with paraphilias.

However, most adults are sexually curious. Among those who access the Internet for sexual reasons, some may be predisposed to use such sites, some may have lifelong paraphilias, and some may discover nonparaphilic or paraphilic interests owing to images seen on Internet sites being etched in the mind. The Internet allows easy access and apparent anonymity, and most sites are now free or affordable. It is unclear whether the Internet has increased the prevalence of paraphilias, but it has certainly led to an increase in the detection and conviction of those viewing child pornography there, in comparison with the past, when such images were viewed in private in magazines. The Internet also makes it easier omnipotently to summon at will and find rare sexual fantasies being carried out in reality, with the illusion of a real object relationship with another; this distances the individual from associated feelings of guilt.

CONCLUSIONS

Impulse-control disorders are a disparate group of conditions with different characteristics and epidemiologies. Whether the urges and impulses and resulting criminality are irresistible is open to question. Perhaps no impulse is irresistible, if an individual is motivated to try hard enough to resist. Certainly in practice, impulse-control disorders appear to be controllable at times but uncontrollable at others, when momentary excitement leading an individual to act on the impulse appears to overwhelm control. A disordered function of control may better describe the situation than an irresistible impulse. Indeed, the impulse to act is often combined with a desire not to act.

The conditions included in habit and impulse-control disorders do not real an identical psychopathology. Pathological gambling is a more complex condition, requiring attention to the whole person, than an impulse-control disorder such as tricotillomania. A pathological gambler shows features akin to substance addiction, with characteristic histories of escalation from use, abuse, and then addiction with tolerance and withdrawal symptoms, with gambling becoming the center of a sufferer's life, unlike the situation in pyromania or tricotillomania.

Impulse-control disorders are at least as prevalent as schizophrenia, but the research interest in such disorders and the evidence base for treatments are limited. Current treatments demonstrated to be effective include cognitive-behavioral therapy and SSRIs. Other treatments for impulsivity with a weaker evidence base include an SNRI, anticonvulsants, stimulants such as methylphenidate, and the cognitive enhancer modafinil. Other approaches being currently considered and researched include biofeedback, repetitive transcranial magnetic stimulation, deep-brain stimulation, and stererotactic neurosurgery.

REFERENCES

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

Bluglass, R. (1990). Shoplifting. In R. Bluglass & P. Bowden (Eds.), *Principles and practice of forensic psychiatry* (pp. 787–795). Edinburgh, Scotland: Churchill Livingstone.

Bondolfi, G., Jermann, F., Ferroero, F., Zullino, D., & Oseic, C. H. (2008). Prevalence of pathological gambling in Switzerland after the opening of casinos and the introduction of new preventative legislation. *Acta Psychiatrica Scandinavica,* 117, 236–239.

Buckle, A., & Farrington, D. P. (1984). An observational study of shoplifting. *British Journal of Criminology, 24,* 63–73.

Christenson, G. A., Faber, R. J., de Zwaan, M., Raymond, N. C., Specker, S. M., Ekern, M. D., et al. (1994). Compulsive buying: Descriptive characteristics and psychiatric comorbidity. *Journal of Clinical Psychiatry, 55,* 5–11.

Cupchik, W., & Atcheson, J. D. (1983). An occasional crime of the moral majority. *Bulletin of the American Academy of Psychiatry and the Law, 11,* 343–352.

Dickerson, M. G. (1988). The prevalence of excessive and pathological gambling in Australia. *Journal of Gambling Behaviour, 4,* 125–151.

Dickerson, M. G. (1989). Gambling: Dependence without a drug. *International Review of Psychiatry, 1,* 157–172.

Driver-Dunkley, E., Samanta, J., & Stacy, M. (2003). Pathological gambling associated with dopamine antagonist therapy in Parkinson's disease. *Neurology, 61,* 422–423.

Esquirol, J.E.D. (1985). *Mental maladies: Treatise on insanity*. London: Hafner.

Ferris, J. & Wynne, H. (2001). *The Canadian problem gambling index: Final report*. Ottawa: Canadian Centre on Substance Abuse.

Freud, S. (1928). Dosteyevsky and parricide. In J. Strachey (Ed.), *The complete works of Sigmund Freud* (Standard Edition 1961, Vol. 21, p. 177). London: Hogarth Press.

Freud, S. (1932). The acquisition of power over fire**.** *International Journal of Psycho-Analysis, 13,* 405–410.

Frosch, W. A., Frosch, J. P., & Frosch, J. (1985). The impulse disorders. In R. Michels & J. O. Cavenar (Eds.), *Psychiatry* (Vol. 1). Philadelphia: J. B. Lippincott.

Gerstein, D., Murphy, S., Toce, M., Hoffman, J., Palmer, A., Johnson, R., et al. (1999). *Gambling impact and behavior study: Report to the national gambling impact study commission*. Chicago: National Opinion Research Center.

Gibbens, T.C.N., & Prins, J. (1962). *Shoplifting.* London: Institute for the Study and Treatment of Delinquency.

Goudriaan, A. E., Oosterlaan, J., De Beurs, E., & Van Den Brinic, W. (2008). The role of self-reported impulsivity and reward sensitivity versus neurocognitive measures of disinhibition and decision-making in the prediction of relapse in pathological gamblers. *Pathological Medicine, 38,* 41–50.

Grant, J. E., Correia, S., & Brennan-Krohn, T. (2006). White matter integrity in kleptomania: A pilot study. *Psychiatric Research, 147,* 233–237.

Grant, J. E., & Kim, S.W. (2002). Effectiveness of pharmacotherapy for pathological gambling: a chart review. *Annals of Clinical Psychiatry, 14,* 155–161.

Grant, J. E., Levine, L., Kim, D., & Potenza, M. N. (2005). Impulse control disorders in adult psychiatric inpatients. *American Journal of Psychiatry, 162,* 2184–2188.

Greenson, R. (1947). On gambling. *American Imago, 4,* 61.

Gudjonsson, G. H. (1990). Psychological and psychiatric aspects of shoplifting. *Medicine, Science and the Law, 30,* 45–51.

Hodgins, D. C. (2004). Using the NORC DSM Screen for gambling problems as an outcome measure for pathological gambling: Psychometric evaluation. *Addictive Behaviors, 29,* 1685–1690.

Legg-England, S., & Gotestam, K. G. (1991). The nature and treatment of excessive gambling. *Acta Psychiatrica Scandinavica, 84,* 113–120.

Lejoyeux, M., Arbarataz, M., McLoughlin, M., & Adès, J. (2002). Impulse control disorders and depression. *Journal of Nervous and Mental Diseases, 190,* 310–314.

Lesieur, H., & Blume, S. (1987). The South Oaks Gambling Screen (SOGS): A new instrument for the identification of pathological gamblers. *American Journal of Psychiatry, 144,* 1184–1188.

McElroy, E. S., Pope, H. J., Jr., & Hudson, N. (1991). Kleptomania: A report of 20 cases. *American Journal of Psychiatry, 148,* 652–657.

Moran, E. (1983). Gambling. In G.F.M. Russell & L.A. Hersov (Eds.), *Handbook of psychiatry 4. The Neuroses and personality disorders* (pp. 385–390). Cambridge, England: Cambridge University Press.

Neustatter, W. L. (1953). *Psychological disorder and crime.* London: Christopher Johnson.

Petry, N. M., & Roll, J. M. (2001). A behavioral approach to understanding and treating pathological gambling. *Seminars in Clinical Neuropsychiatry, 6,* 177–183.

Potenza, M. N., Leung, H. C., Blumberg, H. P., Peterson, B. S., Fulbright, R. K., Lacadie, C. M., et al. (2003). An fMRI Stroop task study of ventromedial prefrontal cortical function in pathological gamblers. *American Journal of Psychiatry, 160,* 1990–1994.

Potenza, M. N., Steinberg, M. A., McLaughlin, S. D., Wu, R., Rounsaville, B. J., O'Malley, S. S. (2001). Gender-related differences in the characteristics of problem gamblers using a gambling helpline. *American Journal of Psychiatry, 158,* 1500–1505.

Puri, B. K., Baxter, R., & Cordess, C. C. (1995). Characteristics of firesetters: A study and proposed multiaxial psychiatric classification. *British Journal of Psychiatry, 166,* 393–396.

Raylu, N., & Oei, T. P. (2002). Pathological gambling: A comprehensive review. *Clinical Psychology Review, 22,* 1009–1061.

Ritchie, E. C., & Huff, T. G. (1999). Psychiatric aspects of arsonists. *Journal of Forensic Science, 44,* 733–740.

Royal College of Psychiatrists. (1977). Submission of evidence to the Royal Commission on Gambling. London: Author.

Sapsford, R. J., Banks, C., & Smith, D. D. (1978). Arsonists in prison. *Medicine, Science and the Law, 13,* 127–138.

Shaffer, H. J., & Korn, D. A. (2002). Gambling and related mental disorders: A public health analysis. *Annual Review of Public Health, 23,* 171–212.

Sharpe, L. (2002). A reformulated cognitive-behavioural model of problem gambling: A biopsychosocial perspective. *Clinical Psychology Review, 22,* 1–22.

Slutske, W. S. (2006). Natural recovery and treatment-seeking in pathological gambling: results of two U.S. national surveys. *American Journal of Psychiatry, 163,* 297–302.

Soothill, K., & Pope, P. J. (1973). Arson: A twenty year cohort study. *Medicine, Science and the Law, 13,* 127–138.

Stinchfield, R. (2002). Reliability, validity, and classification accuracy of the South Oaks Gambling Screen (SOGS). *Addictive Behaviors 27,* 1–19.

Stinchfield, R. (2003). Reliability, validity and classification accuracy of a measure of DSM-IV diagnostic criteria for pathological gambling. *American Journal of Psychiatry, 160,* 180–182.

Topp, D. O. (1973). Fire as a symbol and weapon of death. *Medicine, Science and the Law, 13,* 79–86.

Walsh, K. H., & McDougall, C. J. (2001). Tricotillomania: Presentation, etiology, diagnosis and therapy. *American Journal of Clinical Dermatology, 2,* 327–333.

Wardle, H., Sproston, K., Orford, J., Erens, B., Griffiths, M., Constantine, R., & Piggott, S. (2007). *British gambling prevalence survey 2007.* London: National Centre for Social Research.

World Health Organization [WHO]. (1992). International classification of diseases and related health problems (ICD-10). Geneva: Author.

This page intentionally left blank

Chapter **6**

Troubled IPR Addiction: Habitual Attraction, Abuse, and Violence in Intimate Partner Relationships[1]

Angela Browne-Miller, PhD, DSW, MPH

The intimate partner relationship (what I will herein call the IPR) is an intricate, wondrous system of processes and functions between people who come together largely by way of two highly "rewarding" and "reinforcing" processes—various forms of attraction and of social norm fulfillment. Sadly, a large subset of the population of IPRs is that of troubled IPRs, which are too frequently undetected, or unadmitted via denial functions—or accepted as "normal" (the norm), or worse, simply ignored. Within the domain of the troubled IPR reside habitual detrimental attractions, and various abuses and violences—potentially addictive patterns of attraction, abuse, and violence. These patterns offer insights into behavioral addiction tendencies, functioning on multiple macro and micro levels ranging from overarching species and sociocultural to micro level interpersonal and interactive psychological, psychoneurological and biochemical processes. The psychological, psychoneurological, and biochemical components of patterns of intimate partner abuse and violence parallel patterns of substance abuse and addiction.

Habitual attraction itself, founded upon attraction and/or social norm, is not in itself problematic, and may in fact be some of the glue that keeps well-functioning couples and their families together. While eventually the relationship can become almost a habit, this can be a good habit. It is when this function runs awry that patterns of troubles, risks, and dangers common to runaway addictions emerge (Henry, 2006; Sethi, Marais, Seedat, Nurse, & Butchart, 2004; Weitsman, 2000).

A substance addiction involves continued use of the substance in the face of harm to self or others. Similarly, ongoing habitual attraction-interaction can devolve into detrimental forms including but not limited to ongoing emotional abuse, obsession, masochism, sadomasochism, violence against the self or the other, and co-occurring disorders including substance addiction. (Berliner, 1947; Browne-Miller, 2007; van der Kolk, 1989; see also Dingfelder, 2007 and Gorman, 2007). Troubled habitual attraction—what we might better describe as *mal*attraction—reaches past emotional and physical states of healthy love to the realm of addiction. Here, the triggering of addictive emotional and physical swings, with the wide-swinging roller coaster ride of emotional and physical highs and lows, can go runaway and spin to extremes. Or it can spin until it reaches a relatively stable destructive state where there are no further highs and lows, only increasingly lower lows. In these realms lurk the patterns of self and interpersonal abuse and violence to which too many people become addicted, and at the hands of which too many lives are damaged.

The troubled relationship is addressed by researchers and practitioners in several fields, although rarely addressed as an addiction (Family Violence Prevention Fund, 2007; Hendy, Eggen, Gustitus, McLeod, and Ng, 2003). Many intimate partners, as well as professionals treating and researchers studying them, do not choose to say that destructive relationship patterns such as intimate partner abuse and violence can exhibit habitual and addiction-like characteristics. Still, the IPR itself—or its dynamics—may be a habit that under certain circumstances can turn not only sour but immensely detrimental to one or both partners, the family around them including children, the surrounding community including the workplace, and beyond to the economy, in the form of not only lost productivity and lost work days but also ongoing, even long-term, mental and physical health care consequences and costs (Salber & Taliferro, 2006; Scaer, 2005, Tjaden & Thoennes, 2000; Tjaden & Thoennes, 2000; UNICEF, 2000; USDHHS, 1999). And, at its extreme, one possible outcome of a troubled IPR is that of extreme intimate partner violence (extreme IPV), which can be quite dangerous, even lethal (Rennison & Welchans, 2000; Rivara, Mueller, Somes, Mendoza, Rushforth, & Kellerman, 1997).

As troubled IPR addiction can be so damaging and dangerous, it is only responsible behavior for all who are, will be, or have been in and around IPRs (everyone), and for helping professionals and the researchers who guide them, to consider what positive and also what negative elements of IPR behavior may indeed be habitual, even addicting. I come to this work from a long background in several fields including but not limited to clinical aspects of substance abuse, domestic violence (working with persons abused, persons abusing, and their family members), family dynamics, trauma, family systems theory, and related social policies. This work has converged to bring me to make the following

commentary (addressed to professionals as well as to lay persons, thus written in language aimed at being accessible to a broad range of readers[2]) on IPR pattern addiction and what is best labeled intimate partner *abuse and* violence (IPAV) (Browne-Miller, 2007). The frequently applied acronym for intimate partner violence, IPV, is quite relevant here, although perhaps limited when discussing the full range of abuse and violence patterns, whether or not these are physical. Other terms for IPV may include domestic violence (DV) and gender-based violence (GBV), which can be but are not always subsets of IPV (or in my schema, IPAV).

ELEMENTS OF TROUBLED IPR ADDICTION

Troubled IPR patterning addiction has many faces, and it is perhaps best summarized as falling into three inextricably linked categories: deep neuropsychological elements; powerful behavioral patterning; and overarching social norms to which entire subpopulations and populations are not only committed but are themselves virtually and even actually addicted (applying the broader definition of addiction being used herein).

Deep Neuropsychological Programming.

As is true for substance addiction and other behavioral addiction, troubled IPR addiction contains a powerful biological—neuropsychological—component. Of course, even without the running awry found in an addictive pattern, a healthy love (and or sexual) relationship can parallel a drug "high," especially in its early stages. As Louis Cozolino explains in *The Neuroscience of Human Relationships*, "Love is a drug . . . that includes endorphins and dopamine and results in similar patterns of brain activation as taking cocaine" (Cozolino, 2006, p. 316). This addictive rush of attachment chemicals brings with it the distress at their withdrawal.

Building on Cozolino's observation, we can add that the search for, and the actual or presumed reinstatement of, this or another replacement love sensation poses the risk of the high-low roller coaster ride, of the repetition of highs and lows of love, and in runaway situations, of the extreme highs and lows of love. Desperation in, and distortion of, the search may misinterpret replacement sensations as love when these are not love. This distortion is an all too common component of the troubled IPR addiction pattern (which can even leap from relationship to relationship), and can be said to be self-sustaining. self-fueling, *auto-perpetuating*.

The risk of neuropsychological addiction to such patterns is emotional-biochemical and it is also a factor of present-time social experience. As Cozolino

points out, "we never experience a person as totally new but as some blend of our expectations, implicit schema, and who he or she really is. [Note however that reaction to this experience] . . . results from the fact that implicit memory processes are faster, automatic, and guide explicit memory and conscious experience. . . . Although it takes our brain 400–500 milliseconds to bring sensations to conscious awareness, it takes only 14 milliseconds to implicitly react to, and categorize visual information" (Cozolino, 2006, p. 133).

Again building on Cozolino's viewpoint, we can formulate the notion of a sort of *neuropsychological investment* in a relationship pattern, even one of abuse: We respond to what our implicit memories are responding to, until we have time to reason with ourselves—to see what is happening in the now—if we get a chance at all to do this before impulsively reacting to a stimulus or trigger, as by then we may be neuropsychologically invested in the process. We may also be interpersonally committed to the process, as action has already been taken, without advance review of its outcome. Implicit memory has, in a sense, been socialized (and even traumatized) to the point where it reacts to present-day triggers as if these were harbingers of past events. *Past comfort may seem to be present comfort; past safety may seem to be present safety. Past danger may seem to be present danger; past pain may seem to be present pain.* For example, take a person who has been hit in the face many times as a child, and has grown to cower and fear these repeated assaults. Many years later, in adulthood, there is nothing like this in that person's life. One day someone reaches to adjust the cap on that person's head and that person flinches and raises a fist, almost hitting the hat-adjusting friend. Had the person not been so rapidly placed on automatic by the implicit memory, then the explicit memory could have warded off this behavior.

The effect of this difference in speed of access to implicit and speed of access to explicit memory is powerful, as both memory pools are being accessed almost (in real world—everyday—time) simultaneously. For my purposes here, I will call this the *reaction differential factor*, or RDF. It is in the realm of this RDF that the IPR is vulnerable to on-the-spot, immediate responses, reactions—without the benefit of conscious integration and review of the stimulus or trigger. And almost concurrently the IPR is vulnerable to the next wave of responses, somewhat more conscious responses to the actions taken on impulse.

In is in the first, brief 14-millisecond interval that hurtful and even threatening verbal and physical outbursts, actions, can be initiated. Once in process, implicit triggers work like an avalanche. Where intimate partner violence, physical IPV, takes place, this 14-millisecond response zone is loaded with physical risk. This RDF characterizes much of extreme IPAV. I add the *A* in here, as nonphysical abuses such as impulsive emotional abuse can also arise out of, be triggered by, the implicit memory (Browne-Miller, 2007; Loring, 1998).

Stretching the definition of implicit memory somewhat, we can see that implicit memory reaches back in time—into the memory of the individual, and of the IPR, as well as of the species. The latter is where instinct lives. Here is where the instinct known as the fight or flight response arises. When the individual is threatened, species-honed instinct works in the millisecond space to initiate a rapid impulse response to danger: either fight or take flight.

Powerful Behavioral Patterning

Implicit memory calling back, right into the present, responses to old events and traumas is a driving force in many troubled IPR reactions. However, we need not reach only into the far past to find powerful behavioral patterning. We can search the term, the span, of the RDF, look into the space between reaction out of implicit memory response and reaction out of explicit memory response. We can also search more recently formed implicit memory responses. A long- and even short-term IPR may repeat the same pattern often enough that its characteristics, actions-reactions and triggers-responses, move into implicit memory enough to put certain behaviors onto automatic. This process can also take place in a healthy IPR and likely does. (Do we wish our partner's and our own taking out the garbage and cleaning up in general were guaranteed implicit memory–directed automatic behaviors?) However, in a troubled IPR, troubled patterns are established, maintained, protected, and often even amplified, with repetition. Reinforcement of a problem behavior takes place (even in "the now").

Addiction to these learned patterns is common, and works on both the neuropsychological level and the level of current social interaction and expectation. All sorts of present-time reinforcements of these patterns occur, and we have the addiction scenario arising again and again. The discussion here focuses on this category, powerful behavioral patterning in the troubled IPR, not to the elimination of the other two categories (instinct-driven neuropsych and culturally-driven social norm), but as the locus of interaction between them.

Powerful Social Norms

I add this third layer to my discussion of troubled IPR addiction: the social norm. Where much of the following discussion focuses on the individual's and the couple's potential for addiction to a troubled relationship pattern, everything discussed here takes place within the larger sociocultural context. The social norm as a concept is accepted as a phenomenon found in broad, overarching social and cultural settings and their subpopulation subsets. We tend not to think of the social norm as addictive but rather as an overarching

contextual dictum under which all else takes place. Here I do refer to *addiction* to social norms, and use this reference both lightly and as profoundly as I can. My assertion here is not only that the individual can become addicted to something or some behavior but that an entire population or subpopulation can as well. While some social norms are explicit as givens, others are implicit, virtually invisible while they do their work on all levels including the neuropsychological and behavioral.

The power of the social norm is in its characteristic transferring of expectations regarding values, beliefs, behaviors and attitudes to a broad population. The weakness of the social norm is that is has evolved over a long period of time and that it does not change quickly even when such a change is needed. Where social norms are misinterpreted and resistant to change while being abused or at least misinterpreted, behaviors of individuals can be negatively affected. As an example, the condoning of, or at least the minimizing of the gravity of, violence against women, is still prevalent in many cultures and subcultures around the world. While this chapter does not focus on social-norm-supported, and even social-norm-promoted, violence against women or GBV per se (I choose to do this elsewhere), the role of powerful underlying social norms in interpersonal and relationship violence is the context in which much of the IPAV discussed herein occurs.

EXTENT OF THE PROBLEM

Data-established pieces of the puzzle are available; however, we have no measure of the extent of addiction to troubled IPR patterns or of addiction to IPV or IPAV patterns. The prevalence of such *pattern addiction* would be difficult to measure. Certainly, we cannot prove that all IPV is the result of addiction to individual and social IPAV patterns, as many instances of assault upon an intimate partner may not be the product of addiction to a pattern. The data that are available do tell us that intimate partner violence is significant in scope and impact worldwide, suggesting the relevance of all three factors: social norm, behavioral patterning, and neuropsychology. We pause here to note some basic IPV incidence-prevalence data.

In terms of what is being reported, which is truly only the tip of the iceberg, counted intimate partner violence results in well over a million injuries and a thousand deaths each year in the United States alone, and this is without adding data regarding rape and other forms of sexual molestation and assault, which are not always considered events taking place between "intimate" partners (Centers for Disease Control [CDC], 2002; see also Bugarin, 2002). Given abused persons' general reluctance to report the abuse and violence, especially

when the context is a formal or semiformal marital or pair-bond situation, these are likely vast understatements. And, given what we know from hotlines and shelters (Campbell, Sullivan, and Davidson II, 1995; Rennison, & Welchans, 2000), these are surely vast underestimates. Note that this data just represent rates in the United States; global data echo all this and more.

The World Health Organization (WHO) "Multi-Country Study on Women's Health and Domestic Violence against Women" (WHO, 2005) found that in 10 countries it studied, at least half of all women reported that they had been physically or sexually assaulted since the age of 15, and at least half of these assaults had been inflicted upon them by a "male intimate partner." Of the ten countries in this WHO study (Bangladesh, Brazil, Ethiopia, Japan, Namibia, Peru, Samoa, Serbia, Montenegro, Thailand, and the United Republic of Tanzania), 10 percent of women reported *current* violence by a current intimate partner. In terms of IPV during their lifetimes, reports ranged from "15% in Japan city to 71% in Ethiopia province, with prevalence estimates in most countries ranging from 30% to 60" (WHO, 2005, WHO, 2004).

Again, these data alone say nothing about addictive *patterns*. Much of these violence events are crimes—assault, rape, homicide—that may or may not have occurred as part of a pattern (Coker, Smith, McKeown, & King, 2000; Rivara, et al., 1997; Simon, 2003). Where women are the victims of such violence, this is frequently described as the gender-based violence (GBV) referred to earlier. (Note that IPV is not always violence against women.) When this violence is embedded within overarching cultural norms, including tolerance of violence against women, this violence is not only tolerated and even accepted but in some cases expected. Here, the addiction may be that of a population or subpopulation to a general social norm of toleration (of tolerance of, and programming for, this form of violence) rather than to an individual's addiction to a pattern. Similar social or population phenomena are seen in substance addictions. For example, a subculture or subpopulation may accept, encourage, and virtually require heavy alcohol consumption. The line between a social pressure and a social given as a population addiction blurs.

TERMINOLOGY AND CONCEPTUAL CAVEATS

A few caveats here. First and foremost, nothing about this discussion is in any way a blame the victim approach to intimate partner abuse and violence. All too often, the victim is somehow construed as the cause of the violence of which the victim is the victim—the clearly injured party, clearly at another's hands. Without entering into any contemporary (and quite common in the United States) "who is really the victim" debate here, it is important that the

following material not be used to serve one side or the other of the debate. And where this material may indicate that both partners who are involved in ongoing IPAV are either at risk for, or already are, addicted to IPAV patterns, this in no way says that this addiction to patterning excuses the abuse and violence. Similarly, even addiction to abuse and violence itself does not excuse this abuse and violence. Persons committing the abuse and violence must be accountable for their actions, to their victims, to their families, and to their communities.

Another important note. Clearly, substance abuse (SA) and IPAV co-occur at an alarming rate (Saint-Jacques, Brown, Caplan, & Werk, 2006). Nevertheless, there is no definite and entirely predictable causal direction; neither one nor the other is certain to always come first:

Neither the rule, SA → IPAV,
nor the rule, IPAV → SA,
can be held gospel.

Instead this co-occurrence itself takes myriad forms and directions, and it never takes place in isolation from countless other factors. Nevertheless, with far too great a frequency, SA is often said to be the whole cause of IPAVs, especially domestic violence. This risks a transfer of focus entirely onto the SA element in lieu of delving into the complexities of the IPR itself and of the IPAV addiction where this is present. Calling SA the cause of IPAV also risks absolving the members of the relationship, especially the perpetrators of the abuse and violence, of direct responsibility for their actions.

The definition of the intimate partner relationship, or what here is called the IPR, has itself been debated with regard to the requisite degree of formality of the relationship. Some will say that the sanction of marriage permits sexual intimacy and therefore is the official IPR. Others will disagree, of course. However, whether or not the relationship is official or sanctioned, or long term, troubled patterns of relating and of abuse and violence can emerge. Also note that there are many forms of casual relationships that involve intimacy—thus, intimate partners. We will not differentiate here as we are looking at IPR interaction patterns wherever they may emerge.

A word here about the use of terms such as *domestic violence survivor* and *batterer* or *perpetrator*. Various terms are used for these and similar roles in this dance of pain seen in too many intimate partner relationships. In fact, different professions tend to select different labels for these roles (as in Catalano, 2006; Chu, 2006; Dahlberg & Krug, 2002; McCormick, & Sacks, 2007; NCIPC, 2006; Roberts, Hegarty, & Feder, 2006; see also Meyers, 2007; DeWolfe, & U.S. Department of Mental Health Services, 2004; Rosen, 2007; Simon. 2003). For example, law enforcement tends to respond to what are called vic-

tims of violence, while persons who work with those who have managed to triumph over being abused in their intimate partner relationships may tend to prefer other terms, such as survivor, for the same persons. And the person seen as having committed the act or acts of violence is often described as the batterer or perpetrator, in keeping with the law enforcement and judicial labels for this person. Nevertheless, simplifying the role of this individual to that of batterer is erroneously collapsing the complex role of this person in the troubled IPR or its IPAV. Labeling one party as batterer reduces the role of both parties in the pattern to the point that one is the good guy or girl victim and one is the bad guy or girl batterer. (That there is no gender preference in these labels is purposeful here. Gender is a very important overlaying factor that must be studied separately.) While in many instances, one is the victim and the other is the batterer, for the analysis of addiction to patterns of relating and abusing, these distinctions are at times secondary.

Rather than take a stand on these terms and their utility, we focus here on the likely to be seen as controversial condition and experience of being habitually "stuck" in these roles—*addicted to them*—however they may be labeled. Therefore, here, in about half the instances in which I describe the roles found in intimate partner violence, "people first language" is used, to some extent allowing us to think past labels—to take one step back from a final label. The "abuser" can be the "*person* who is abusing" and the "abusee" can be the "*person* who is being abused." (Note that this also allows for role switching, for role matching, and also for the implicit self abuse that all too frequently attends the IPAV scenario.) Terms such as abuser and abusee are also used, and more rarely are terms such as perpetrator and victim used. Looking past these roles allows honest consideration of mutual abuse, plus self-abuse, plus dominant abuser roles in destructive addictive patterns. And this allows us to look deeply at addictive IPR patterning.

The patterns of hurt we are talking about have many complex dimensions. These dimensions include nonphysical types of abuse and violence that can be precursors to physical violence or can be extremely hurtful, miserable, and destructive even in the absence of physical violence. These nonphysical forms of violence are easily mislabeled and can be missed altogether. For example, emotional abuse can be so subtle that even the participants do not consciously realize it is taking place. And, where they do realize it, they may not recognize this as emotional abuse or emotional violence, let alone as a pattern of such, let alone addiction to such. Another form of non-violent abuse, that is, financial abuse, can seem so natural to the participants that it remains unlabeled. These and other nonphysical abuses—*hidden violences*—must be recognized and addressed to halt them. So must the patterns—all too often addictive patterns—in which these abuses and violences occur. We will return to addictive patterns of emotional abuse and of self abuse later.

Additionally, there are other dimensions of IPAV addiction and patterning that include but are not limited to the earlier mentioned co-occurring substance abuse, as well as stress, psychological and health disorders, and the partners' separate personal histories of substance, self, relationship, and child abuse prior to the marriage or coupling. These conditions, only some of which can be described as co-occurring disorders, or even causal preceding disorders, can be so intertwined with relationship addiction (including any sex addiction or obsession, any abuse and violence), that it is unrealistic to see and to treat one of these to the exclusion of the other. We return to particular elements of co-occurring IPAV and SA at the close of this discussion.

There are those who prefer to separate these conditions out, not to link them in any way. This is understandable because they want to prevent persons who are abusing from passing the buck for their abuse and violence onto conditions such as stress, mental illness, having been abused as a child, and as noted above, alcohol or drug abuse and addiction (of note where etiology of adult problems is relevant are Burgess, Hartman, & Clements Jr., 1995; ICAN, 2000; Schwartz, Hage, Bush, & Burns, 2006). This point of view is taken to help persons doing the abusing take responsibility for their actions no matter what has contributed to them. While this is a noble approach, and persons abusing should be helped to take this responsibility no matter what contributes to their violence, treating their violence without treating the co-occurring conditions is limiting the power of the work being done with and for persons who wish to stop their detrimental and abusive behaviors.

PATTERNS OF ABUSE AND VIOLENCE IN RELATIONSHIPS

That IPAV may be addicting becomes clear when its various levels are included in the delineation of its patterns. The many faces of relationship abuse and violence are frequently categorized along these lines: emotional abuse; verbal abuse; physical abuse. These types of abuse and violence can be placed along a spectrum like this, depicting the pattern found in much relationship abuse and violence, in that it flows from one end of this spectrum to the other, working its way through perceived levels of escalation from nonphysical to physical:

emotional abuse → verbal abuse → physical abuse.

We should add in threats of physical abuse. These can be threatening words or threatening gestures, or implied, hidden but very real threats, and should have their own category (as underlined here):

emotional abuse → verbal abuse
→ *threats of physical abuse* → physical abuse.

As these patterns tend to be cyclic, they are perhaps best diagrammed around a circle, or perhaps as an oscillating or roller coaster sort of line, with the extremes of no violence and then of physical violence at the highest and lowest parts of the "ride." Many other patterns form around IPR abuse and violence, and some patterns devolve to a single ongoing flow of abuse and violence with no, even temporary, de-escalation or relief:

abuse and violence → abuse and violence → abuse and violence → etc.

A great deal of emotional and verbal abuse is very subtle, and not seen for what it is by its recipients or by its perpetrators. In some relationships, there is never physical violence but there is a great deal of other violence taking place. Sadly, these often damaging patterns can be entirely overlooked because there is no physical violence taking place.

VIOLENCE CAN BE HABITUAL, ADDICTIVE

And while there is surely growing understanding that sometimes emotional and verbal abuse are signs of physical abuse, or signs that physical abuse is coming, there is less understanding of the matter of addiction to abuse and violence. The truly addictive nature of actual patterns of attraction, abuse, and violence in relationships remains largely hidden to us, perhaps because we shy away from this understanding. Understanding could be disturbing, could call far too many behaviors into question. Bottom line: we must admit that people can become addicted to intimate partner abuse and violence in the way they can become addicted to just about anything else repeated over time—anything that has positive and negative sensations and reinforcements associated with it. Even where this pattern is experienced on a frequent basis

emotional abuse → verbal abuse
→ *threats of physical abuse* → physical abuse.

it has its built-in pleasurable sensations (such as relief of built-up tension). These built-in sensations can function as rewards—things that may seem to feel good—positive reinforcements that are typical in addictive patterning. These so-called rewards are easy to reap, in that they are natural parts of the cycle.

An example of positive reinforcement of a dangerous habit is the habit of make-up sex after a dangerous level of intimate partner violence has taken place. Some couples have even been known to engage in their habit of make-up sex after extreme physical violence instead of going to the doctor or the hospital for stitches (and then to return to the violence after the sex). How very much like the picture we have of severe drug addiction—craving the drug during withdrawals—then using and getting high and using again despite the damage and injury this is causing (even using while needing to get medical attention for a wound inflicted while high or while desperately seeking the drug). The general script reads like this: undergo suffering, feel relief and/or pleasure, undergo suffering, feel relief and or pleasure, undergo suffering, feel relief and/or pleasure, and so on.

Habitual Rewards

People go for rewards, and for cycles that include rewards, for many reasons. When it comes to intimate partner violence, the reward can tie deeply into the addiction to the pattern, even becoming central. First, there is a potentially addictive *pain—no pain—pain* cycle. As noted above, the cycle of abuse and violence often (but not always) ebbs and flows, and when it does, may bring with it the simple reward that there will at least be breaks—whether moments or hours or weeks long—from cyclic extremes such as physical violence and possibly from the physical pain it brings (although sometimes it is only after the violence stops that the physical injury and pain it causes is felt). Suddenly, the person abusing stops hurting the person being abused and there is new relief—the beating has ceased. This fleeting so-called relief is a form of reward (albeit a cheap reward), a positive reinforcement for this pattern. It is also a shift through the range of the RDF, to the somewhat slower reaction time end of the spectrum.

Let's be very clear here: there is nothing in these words that says the person being abused likes being abused. Instead, these words say, first, that even momentary relief from abuse can be looked forward to or longed for; that tolerating abuse from which there seems to be no escape is facilitated by the anticipation of even brief relief; that when this situation becomes a pattern, both the person abusing and the person being abused can become programmed to it.

Second, there is potential addiction to a specific reward. Where patterns of abuse include what has been called the hearts and flowers or make-up stage, this stage may serve as the reward, the reinforcement, and itself be craved as a drug high would be craved. During this time, no matter how long- or short-lived it may be, there can be politeness, or emotional caring, or gift giving, or

make-up sex. Each of these reinforcing reliefs brings with it not only the positive effect of the respite from the violence but also the positive experience of the make-up activity.

Third, addiction to the highs and lows themselves—to the very behavioral pattern—is quite natural. IPAV addiction's highs and lows are experienced as something like the experience of drug addiction—a roller coaster ride. This ride itself can become addictive, as it can produce sets of biochemical shifts that in themselves produce something similar to these common cycles:

- Seeking stimulation—then relief by excitement, adrenalin rush;
- Tension building—then relief from tension in some form;
- Pain building—then relief from pain in some form;

and even

- Longing for, craving, the sense of contact with someone or something—then contact with something or someone in some form.
- Longing for, craving, relief from discomfort—then comfort in some form;
- Longing for, craving, relief from pain—then relief from pain in some form.

In an abusive relationship (abusive to the other member of the relationship and/or abusive to the self while in the relationship), addiction to a pattern of highs and lows has distinctly detrimental effects. Prolonged addiction to roller coaster rides of stress and violence increases the probability of desensitization to the danger and to the pain that the violence presents. Increasingly severe violence may not be experienced as such. Prolonged addiction to these roller coaster rides also increases the probability of more and more damage and more and more severe instances of damage and injury, with the potential of these *additional* last phases being added onto the spectrum above:

physical abuse → physically damaging abuse
→ *physically disabling abuse* → *death*

Checkpoints Along the Path to Violence

Somewhere along the line, usually from the very start when attraction and/or love is intensely biochemical, we may unwittingly cross checkpoints on a path that could be traveling from *interaction*, to *habitual non-physical abuse*, to *habitual physical violence*, to *addiction to the highs and lows of the pattern of relationship abuse and violence*. This is not to say that all or even a majority of relationships follow this path. This is to emphasize that everyone in a past, present, or future relationship can benefit by knowing about these paths, warning signs, and

checkpoints. To say that anyone having a relationship should not look at this issue is to say we support denial.

Tolerance Can be Dangerous

Sometimes people experiencing patterns of relationship violences—whether these be emotional or physical or both—grow numb to the pain. This numbing to pain takes place because the emotional and physical pain is too much to bear, and also as a result of developed tolerance. This numbing behavior, whether tolerance or a coping skill, is terribly dangerous. This numbing makes all involved miss protective, even life-saving cues. They may not see how serious the situation is, and/or not feel the intensity of the pain—even while in great distress or being maimed or nearly being killed (or being killed). Contrary to programmed safety instincts (pain aversion or pain avoidance), safety is compromised by numbing to the very pain that would allow the sensing of the true level of danger. The IPAV addict moves in surrender of a basic survival instinct for what is in this instance an acquired, maladapted, survival instinct: tolerance. (Again note, this discussion does not stem from a blame-the-victim approach. Rather, this is an *alert and educate* the victim, and all players in the process, stance.)

When our moral, emotional, or basic survival responses to inflicting or receiving abuse and violence diminish, we have developed a *tolerance* to the abuse and violence. This tolerance can emerge much as a person addicted to a drug may eventually take in more and more of the drug to feel or achieve the same effects, and also much as a child who has watched thousands of hours of violence on television may grow accustomed to witnessing violence and may be less and less shocked or morally taken aback by it. The former example raises another matter. We must dare to ask here whether some instances of tolerance to IPAV pain include subconsciously requiring increasingly painful and dangerous violence in order to feel by contrast the longed for relief when the seemingly safe part of the roller coaster ride, the hearts and flowers stage as it is sometimes called, comes around—if it does.

What tolerance can look like in intimate partner abuse and violence is a numbing to the experience of being abused as well as to the experience of abusing. Again, we must remember that numbing to violence does not prevent the damage it causes; rather, it can allow the violence, danger, and damage to continue and even get worse. Addiction to a neurostimulant (such as cocaine, methamphetamine, or even caffeine) offers a standard example of tolerance. A person addicted to a drug with stimulating effects will crave the stimulation and energy increase when not high on the drug. Over time, when not high,

the energy drop will become increasingly low and misery inducing. Each time the stimulant is taken to relieve the low, brain cells may open more receptor sites that fit the stimulant. Tolerance emerges. These sites eventually expect the stimulant, and during phases when there is no stimulant available, the brain cells are hungry for the stimulant, more and more of the stimulant, while the individual is very tired, more and more tired, without it. Tolerance means, in this case, that the highs get lower and the lows get lower. The brain cells require more and more of the same stimulant to feel the high. Even with more and more and more of the same drug, the highs eventually get lower.

Conflicting Experience

We might suggest that while the intensity of the violence may increase, the conscious sensation of receiving the violence (or of giving the violence) decreases over time. The word "conscious" is used here as the person being abused may turn off to conscious awareness of the pain, grow more and more numb to it, while suffering immensely but very deep inside. The suffering is taking place whether or not it is consciously recognized.

People can give themselves such conflicting messages. And this applies to both persons abusing and persons being abused, and of course to situations of mutual abuse and violence. Being either the cause of the pain or the recipient of the pain is painful. Again, this does not in any way say that an abuser who on some level suffers as much as the abusee is therefore off the hook for the damage and injury caused.

Establishing and Maintaining Healthy Patterns

With the great risk of deteriorating into troubled patterns, can an IPR avoid these? Certainly, most relationships can establish a healthy holding pattern, a way of life that stabilizes and promotes not only stability for the relationship but also safety and healthy living for its members and for the people, including the children, around them. However, some relationships stabilize in holding patterns that on the surface work but are laden with risks lingering like time bombs waiting to go off. For example, a "little bit of hitting," or "getting mad and throwing things sometimes," may be alright for partners and may work for quite a while. Still, if behind this there are time bombs lurking, *predispositions to detrimental patterns*, it is generally best to detect and defuse these before they become more damaging and dangerous. Members of IPRs can be assisted or assist themselves in preventing the emergence of troubled patterns. They can be encouraged or encourage themselves to do the following:

- Choose to protect the relationship from deteriorating into problem patterns that once in place are more difficult to change.
- Try to spot, in the early stages of their formation, inherited or acquired, learned, patterns that could grow into problems.
- Weigh the risks of doing nothing preventive about patterns that may eventually become problem patterns.
- See the risk of certain potentially abusive behaviors and patterns, spotting these before they exacerbate into clear abuse and violence.
- When spotting abusive behaviors, even very subtle ones, be ready to call them—admit they are—abusive.
- Recognize the importance of impulse control, even of the time out, and of counting to 10 to go off automatic before speaking or acting.
- Understand that even emotional abuse is violence.
- Direct and change patterns and behaviors in a direction away from potential and/or actual abuse and violence.
- Be highly alert to the process of numbing to pain, and of avoiding, not seeing, and not feeling pain. Pain is a signal that must be addressed.
- Do not let the relationship tolerate certain levels of abuse and violence, as there is no appropriate level.
- If consciously exiting each other's company, temporarily or permanently, have a clear plan and agreement about a clear plan for so doing.
- If a clear plan for so doing is not possible, the individual member or members of the relationship and the children, if any, whose safety is in question or peril must get away.
- Do not sacrifice personal or children's safety for the preservation of the relationship or of a relationship pattern. While ongoing harm may be part of the pattern, this harm does not justify the pattern.

PROGRESSIONS

Warding off the incursions into love of negative psycho-neuro, behavioral, and social programming requires being able to recognize any progression toward detrimental pattern addiction. Most important to see is the simple addictive progression so well recognized in the substance addiction treatment field. An addiction to a behavior frequently begins with *casual behavior.* Casual behavior is brief, occasional, a sort of emotional experiment, seemingly without deep consequences. For example, where there is stress in one's intimate partner relationship, one of the partners may on occasion get into bed with someone else outside the relationship, thinking that this is coping. Of course, this and other casual (and casual escape) behavior can become regular behavior:

Casual Behavior → Regular Behavior

Where there are detrimental present-time or future consequences of this repeated behavior, it at some point may become troubled behavior, a pattern of negative or detrimental behavior:

Regular Behavior → Troubled Behavior

People who are exhibiting troubled behavior continue to do so in the face of adverse effects to themselves (their health, their mind, their work), their families, their businesses, their communities, and/or their societies. It is easy to slip from regular behavior to troubled behavior because the early signs of troubled behavior are subtle and often go undetected.

Again, consider drug use the example. Someone who snorts lines of cocaine to experiment, and then moves into casual social use, and then moves into regular social use, then into increased regular use, likely enters addiction without recognizing this. This is a simple progression, and quite common:

Casual → Regular → Troubled → Addicted Behavior

Fortunately, not everyone who tries a behavior that might be addictive travels this tragic path. Some of these persons, these *casual behav*ORS, (behavORS being the word here for the people exhibiting behavIORS), try a behavior once or a few times and then consider the experiment completed. Or they engage in the casual behavior very rarely and feel this works. But all too commonly, casual behavORS unwittingly slip into regular behavior. We hear people confidently tell themselves, "It can't happen to me. I'm too much in control of my life to develop an addiction to any behavior." However, some do go from casual to addicted behavior in the blink of an eye.

BLEEDING EYE STORY

In the midst of a loud verbal argument, as S yelled at J, "You stupid idiot, you know I'm right!" S raised one arm as if S would hit J if J did not agree.

J jumped back and yelled, "Don't you dare hit me; you look so stupid doing that!"

In that moment, as J was doing and saying this, S leapt forward, right at J, S swinging an arm, this time hitting J in the face, tearing the skin of the side of the head near the eye, and leaving a red mark that would later become a black eye.

Stunned and flinching in pain, J fell to the ground, cowering, saying, "Stop! That's the last time you'll ever hit me!"

S grumbled back, "Oh yeah? Who's going to stop me?"

"I'll call the police."

"No, you won't. You stupid idiot, get up and go wash your face, it's bleeding all over the carpet. And then clean this mess up."

"You clean it up; you hit me."

"This is your fault; you do it," J said, but J finally cleaned it up anyway, as was the pattern.

THE WAY IT SHOULD BE (THE NORM) PATTERN

Shifting the unit of analysis away from the patterns of the individual and the couple, the larger context in which a troubled IPR addiction occurs is highly determinative of the path of that addiction. Given that I am suggesting that entire populations and subpopulations are addicted to the particular social norms in which they are immersed, these norms are here cast as elements of IPRs including troubled IPRs. Social norms themselves, particularly those norms relating to intimate partner relationships, may not only be guiding and comforting but may also be confining and, either way, addicting. Clearly, relationship building and maintenance is not a simple undertaking. It is easier for many to just do it, to rely on the norm for just being involved, and avoid delving into what is happening while they are doing it. And there is a strong social directive to just do it, have a relationship, don't look too closely, don't question the process. There are those who will say. "That is how it is supposed to be" and "That's life." Recall the old rhyme: "[So and so], sitting in a tree, K-I-S-S-I-N-G. First comes love, then comes marriage. Then comes the baby in the baby carriage." This picture—love then marriage then baby—slips into many young minds as early as the nursery rhyme stage of life, forming a sort of directive, a norm—or "normal" pattern—that young people come to feel is most acceptable and most normal.

This basic norm is itself so complex that while it is evolved, adapted, and transmitted from generation to generation via nursery rhymes, fairy tales, spoken expectations, laws and religious codes, media of the times, and modeling done by older generations, no one can say what all goes into it. No one can say for sure that this is truly how it is supposed to be now (today if ever), or what it is that is supposed to be. No one can promise that good and wonderful experiences are normal in and can be expected of this arrangement, nor whether the abuse and violence that can take place in some of these arrangements is alright, part of the plan. However, what becomes clear is that this pattern, instilled via the K-I-S-S-I-N-G message and other traditions, can go awry. Norms work to resist this. Embedded within the nature of the norm is what sustains it, protects it, and wards off threats to its dominance over behavior. The norm reserves for itself the role of dominant, unquestioned imperative—a hypnotic,

addictive, given—*reality* so ubiquitous and self-preserving it is frequently invisible—even when flawed in its intended implementation. While social norms can and do change over time, deviation from a norm prior to its full change can bring ostracism, suffering, and harm to the deviator (Browne-Miller, 2007; Chandler, 2005).

Transaction Habits

There are many hidden trades that take place almost every day, and sometimes almost every minute of a relationship. Each time there is an interaction, a choice to do or not do something, a passage in time in which one or the other puts either the relationship first or the self first, there is a tiny transaction taking place. There is also memory of the pattern of this interaction and its rewards and reinforcements.

These transactions—what we can describe as compromises and trades—are not in themselves sources of pain; they can be wonderfully convenient. And they can be navigated with sensitivity and appreciation for the process of balancing the give-and-take required to keep a relationship functional. Many couples achieve this sort of balance without working hard to do so, and this is ultimately the goal where possible: perceived stability, safety. and sanity. At the same time, too many couples never reach this balance. Then, as time goes by, they trap themselves or at least one of their members into ongoing sacrifice of self and compromise of at least one of their senses of stability, sanity, and safety, all within the comfortable bounds of social norms or (subnorms). It is then that the vows some couples take can become troubled patterns—traps, licenses to cause pain, hurt, and damage.

Progress of Bonds

IPRs form bonds and frequently pursue preservation of these bonds. The pattern of intense bonding, even in a very new love relationship, is natural. Love can form a beautiful bond and a deep emotional engagement and commitment, a truly rich experience making life all the more meaningful. Of course, entering into and then existing as an individual while in an intimate partner relationship is a never-ending process. The relationship changes, just as its members do, as time goes by. The relationship develops a history of its own, a deeper meaning and identity of its own. It truly can take on a personality of its own—not only in the eyes of outsiders who may even come to call the partners in the relationship the "Smiths" or the "couple next door" or "those two"—but in the eyes of its members. Now the relationship is itself a pattern.

When the members of the couple both feel the evolution of their relationship is generally positive, then the progression or pattern moves something like this although not always precisely in this order: from *initial attraction*, to *deeper connection*, to *intersection of lives*, to *identification with the relationship*, to *formalization of the relationship*, to *perpetuation of the relationship*, to *preservation of the relationship*, to *ongoing deepening of the relationship and of the commitment to it.*

Some Bonds Progress Negatively

Of course, there are other less than desirable paths a bond may take. It is important for persons in intimate partner relationships to track, or at least be aware of, the progression of their relationships, the evolution of their bonds, over time. This way, the relationship and its members can maintain an awareness of the direction (or directions) their relationship is taking. They can even influence the direction, if paying close attention—relating consciously and recognizing signs such as those suggesting there may be a need for work on the relationship.

For example, a watchful eye early in a new relationship, when both love hormones and sexual passion can run very high, may help to prevent a relationship from taking the path of a negative progression, which can take many forms. The deterioration of a bond may fluctuate between periods of positive progression and periods of negative progression, as well as experience reversals.

Then, denial finds its way in through every crack in the wall, chink in the armor, of the troubled relationship. Denial creeps in and wants to stay in. Denial itself becomes the glue holding things together. The composite, the whole picture, is a façade not a relationship—an *act* covering over a lack of awareness . . . buttressed by denial upon denial . . . facilitating the *not seeing* of the steps toward participating in, allowing . . . anything needed to preserve the relationship. Frequently this transition into denial is relatively innocuous.

THE COMPLEX MATTER OF EMOTIONAL ABUSE PATTERNING

Patterns of emotional abuse are underway all around us, affecting almost everyone of us, at least in a minor form, at some time in our lives. Emotional abuse patterns are so very common that they are taken for granted, as normal and acceptable. Emotional abuse tends to take a back seat to physical abuse, as physical abuse is seen as more damaging, more dangerous, and more specific. In fact, many persons who are being abused emotionally but not physically do not recognize this abuse as there is no distinct physical sign of it. And of those

being abused physically, many do not include emotional abuse in their descriptions of the abuse they are experiencing. However, the effects of emotional abuse can be as powerful as the effects of physical abuse.

This discussion deals only with emotional abuse of adults by adults. When children experience abuse, and far too many do, a host of highly critical factors not addressed in this chapter are present and require specific attention. Here the discussion focuses on what is, or is supposed to be, behavior taking place between two adults.

Signs

Even adults experiencing emotional abuse may not see that this is taking place. Feelings of discomfort such as those listed here may not be attributed to emotional abuse, even in instances of high levels of emotional abuse; however, these are quite common responses to emotional abuse in intimate partner relationships: embarrassment; confusion; instability; identity doubts, not feeling like oneself; worthlessness, low self-esteem; no level or low level of confidence; sense of complete or extreme failure; depression; isolation; no sense of control over what happens; all-encompassing self-blame—for every problem; humiliation; and pessimism, a negative outlook on the future. And eventually, persons being emotionally abused by a partner over long periods of time can add to this list: feeling that the criticisms of oneself being made by a significant other are correct, believing (or buying into) them; and even . . . defending these criticisms to others. And sadly, sometimes these self-abusing behaviors arise: joining in on the emotional abuse of oneself, hurting oneself emotionally or physically; amplifying the abuse being experienced by working to hurt oneself even more than the abuser does; hiding the pain in substance abuse or other detrimental habitual behavior; possible tendencies to suicidality; bottling up of rage. And of course, what deserves entire volumes and is reported in depth elsewhere, is the risk that the abuse being experienced is then transferred onto others such as children.

DENIAL PRESERVES A PROBLEM RELATIONSHIP

Does denial about harmful patterns preserve a troubled relationship? Yes. Does denial about being in denial further help to preserve this troubled relationship? Yes, for a time. Sometimes too long a time. Relationships can establish an uneasy form of stability by living in denial. However, when the denial is actually camouflaging or burying ongoing suffering, the damage festers and grows. The damage may grow for years, even decades, before it reveals itself

in unusual ways—utilizes unexpected outlets—or simply becomes so extreme that it is finally addressed. Some people are even willing to take their denial to their graves! This is how very scary truth may seem to some.

Sometimes we engage in this denial out of lack of information about the signs of these patterns, sometimes out of insecurity and fear of change, sometimes out of actual fear for our safety. Recognizing and addressing the truth can be unsettling, especially in an environment where truth is repressed and punished if told. We see this scenario many times around us, where someone speaks up about abuse or other interpersonal problems that were expected to remain secret, and the person speaking up is the one who suffers and pays! The role of what we can call the truth teller, perhaps better described as truth revealer, may be a dangerous role to play in a relationship, a family, or a community where abuse and violence is not supposed to be revealed. Powerful unspoken agreements "not to let outsiders know things" are made and can even be carried from generation to generation. Rocking the boat can be risky for the boat rocker. The boat, the troubled relationship system, will fight to stay afloat, to resist change. The sea of social norms in which the boat is immersed may even keep the boat from sinking, supporting the troubled relationship itself.

In that denial serves to preserve many a relationship, even many wonderful, high-functioning relationships, perhaps denial is not in itself always so bad. If the only challenge in a relationship is that one partner wears a plaid shirt, or maybe a particular color of nail polish, that the other partner thinks is a little out of style, it may be that letting this matter go helps. However, far greater issues can be ignored in relationships. Ignoring, not seeing problems so as not to rock the boat, may feel like the only option, the only thing to do. And again, the matter of remaining in denial out of fear of the truth steps forward.

Denial can indeed have a stabilizing effect, preserving what is—the relationship and the patterns that have become the relationship. Denial can also prioritize issues and postpone, back-burner, or simply eliminate attention to problems. Again (and again), when these problems are serious and potentially damaging problems, denial itself is a problem. People can see the black eyes and bruises on their own or their partner's faces and say nothing about these for years and years! When asked, even when looking right at these wounds, people (both persons being abused and those doing the abusing) say they do not see, or actually do not see, them! How much of this is a conscious lie and how much of this is a subconscious lie?

Rocking the boat rocks the boat. And the tendency is to avoid shaking things up, to preserve the status quo, good or bad, whatever this may be. Lies keep the boat from rocking, yes, until the boat springs a leak or sinks, and one or more of its passengers drown.

NUMB TO PAIN STORY

Having been beaten many times, eyes now closed, the burning slaps to the face and the hard knocks to the side of the head feel like more of the same; almost all the hits and slaps and kicks feel the same now. And here comes more and more of the same. And then a fist to the stomach, and a slug to the shoulder, then a hit so hard somewhere—where?

Eyes flicker open and shut. Things start to spin and get darker. Down, falling to the floor. Being kicked now, again and again and again. Each impact making every cell in the body shudder, each cell echoing the thud. Every cell takes each hit and kick, every cell takes it in.

Feeling like nothing but a slab of meat, a beaten, bruised slab of meat. But then, wait, I'm not here, I'm not in here. This doesn't hurt. Hurt is too much to take now. No pain. This isn't happening, I don't feel it. I don't feel anything but the jarring impacts through and through. . . .

Hit, hit, hit. Wait, I'm being beaten as I curl into a ball on the floor. I can make it through this, I can. Again.

HOLLOWING TO PATTERNS

Patterns abound. Patterns are normal. Patterns are a way of life. We need to establish patterns in order to live. Similarly, relationships need to establish patterns in order to live. Relationships build patterns of relating and eventually become dictated by these patterns. In fact, some relationships hollow to nothing but their patterns, losing all defining characteristics and identities outside these patterns, functioning on automatic. Some degree of patterning can work for partners, especially partners in and for life. Establishing healthy patterns can be healthy, so it is good we have this pattern-establishing instinct and capability. But there is a fine line between preserving what is good and preserving what is. Preserving something just to preserve it (no matter what it is) takes a particular mind set, especially when what is being preserved is detrimental or dangerous to the self or others. This is how much we can resist change. This is how continued participation in a troubled IPR can cross into addiction—continued behavior, excessive IPAV behavior, in the face of harm to self or others.

Conflict of Interests

We all engage in some not seeing of patterns, whether desirable or less than desirable patterns, because instinct tells us to go onto automatic. Now another conflict of instincts looms: the powerful instinct to establish and then to pre-

serve patterns may override the instinct to avoid danger! Basically, when we are receiving two conflicting messages from ourselves, we might let one override the other. Amazingly enough, we can endure exposure to a negative experience better when this negative experience is part of an ongoing pattern. When the negative experience is part of a pattern we have grown used to, we are less shocked by it.

Hollowing to Deteriorating Patterns

This function—enduring something simply because it is part of a pattern—allows us to slip unaware into deteriorating patterns, patterns that start out positive and deteriorate into destructive ones, even dangerous ones. For example, if the pattern is one of intimate partner violence in which one partner hits the other repeatedly and continues to do so over time, the intensity and danger of the violence may increase. Little by little, or maybe abruptly, the situation can become dangerous, perhaps even life threatening. Neither party sees the danger of serious injury increasing, even when the danger is there right before their very eyes.

Hollowing to Patterns of Dominance

It is similarly quite easy to slip into relationship arrangements where one partner dominates certain decisions or processes. This dominance may feel quite natural to both members of the partnership, as one may be more of a leader or more extrovert than the other, or may have more knowledge about something than the other, or perhaps be more experienced in certain decision areas than the other. There is nothing wrong with a person who has knowledge or skill in a particular area offering to take the lead there. This can be both efficient and logical.

What can take place, though, when partners are not aware of the permission being given and the power being allocated in this process, is a general transfer of overall power from two people to one of them. This is quite a subtle step, and one which is frequently not seen. A pattern of dominance can grow into an addictive pattern of dominance and control.

Unlike the situation in a town meeting, there are fewer people, usually only two, in the intimate partner relationship. Here, if the overall transfer of power becomes an overall transfer of power and control, we have a potentially dangerous situation. Of course, intimate partner relationships are run like democracies where everyone is supposed to have equal respect, voice, value, and say, right? Wrong. There are no guarantees that intimate partner relationships guarantee

anything close to equal respect, voice, value, and say to the members of this relationship. This is up to each participant in the relationship and is something that deserves conscious monitoring as patterns are being established.

DANGERMOUS DENIAL STORY

High-powered advertising couple, a real alpha or power couple, Vitz and Kat, had been married 15 high-speed years. By the time Kat realized how serious their situation was, both Vitz and Kat were testing HIV-positive. Kat had been entirely monogamous, and also, by the way, drug-free all 15 years. Vitz, on the other hand, had been, for several years, exploring bisexuality —mostly on the sly when high, but not keeping this activity entirely from best friend and partner in civil marriage, Kat. Indeed, Kat knew about Vitz' activities to some extent, and felt Vitz was behaving like this as a result of a tendency to overuse certain drugs rather than out of a desire to cause pain or threaten their relationship. However, Kat just trusted that Vitz would practice safe sex extramaritally. Oh, and by the way, Kat also just assumed Vitz would use sterile needles when shooting up.

EMOTIONAL SADOMASOCHISM

A special discussion of the emotionally sadomasochistic relationship is useful at this point in this discussion of emotional and physical abuse. A sadomasochistic relationship may or may not be one that includes physical-sexual sadomasochism, but it does include elements of emotional sadism and or masochism. Emotional sadomasochism is a relationship pattern that is often hidden although existing right before our eyes, with major components of the sadomasochistic process themselves invisible, nonphysical, emotional, and even non- or pre-emotional (still buried deeply enough in the subconsciousness that they are not registering consciously with any emotional or recognizable impact). In fact, these unseen elements play powerful roles, far more powerful than we give them credit for. These hidden patterns are composed of intricate and often quite subtle energy exchange processes.

Quicksand

Immediately, we step into the quicksand of definition, a sort of now you see safe ground, now you don't, situation. There are several understandings of this concept. Among these are the special definitions of hurt and of the overlap between pleasure and pain. First, the definition of *hurt*. Many readers have heard

the phrase "but it hurts so good" used either jokingly or seriously or both. Here the shifty overlap between the sensation of pain and the sensation of pleasure is identified. Basically, for some, feeling anything, anything at all, is preferred to feeling nothing. Therefore, whether pain or pleasure, both of these experiences fall into the category of feeling something, and thus are thrown together.

Second, sexual relationships—upon and around which many intimate partner relationships are built—can generate both pleasure and pain: at the same time; and/or in such close sequence that they are felt to go together; and/or, in a sequence that places one as a threshold to the other; frequently with some degree of pain being the precursor to some degree of pleasure, and or the reverse.

This is an especially troubled area, as many find discussion of this sort of thing difficult and even offensive. Yet, whether the pain-pleasure linkage is played out physically, emotionally, or in some combination of both, it is safe to say that many emotional relationships play out these sequences in their own, varied ways.

The perpetual compromise and trade process found even in healthy intimate partner relationships can color entire experiences for each member of the couple. Even the way an individual chooses to register an experience as pleasurable or painful can be influenced or even dictated by the compromise and trade process. How frequently these internal decisions are quietly made: "That's not so good for me, but it's great for the other person, and that is good, so it's great for me." "That hurts some, and even when it doesn't it's not fun, even boring, but it gives my partner so much pleasure, so why not?"

What sheer irony it is that while we are compromising and trading—interacting in any way—with our intimate partners, we are actually living in our own worlds, experiencing our own perceptions—not anyone else's—of what is taking place. Hence, we travel through our own personal emotional cycles all alone, even when keeping company with another who may or may not be on the same emotional ride! Take, for example, the comfort-discomfort cycle and the longing for contact cycle (referred to earlier). Clearly, an individual can be taking these cyclic rides—addicted to these patterns—virtually alone. The longing for contact and discomfort-comfort relationship experiences may not be anything like what the other member of the relationship would know or say is taking place.

Like is Too Simple a Word

Another reason why discussion of sadomasochism is touchy is that, quite rightly, there is a concern that some will say persons who are being abused by their intimate partners "like it," and therefore stay. This is not the case. "Like"

is too simple a word here. For example, longing for contact is not liking abuse; taking any form of contact as a form of comfort is not liking abuse.

Emotional sadomasochism involves the overlapping of emotional abuse with perceived consent. "Permission to hurt me, even to break my heart, to destroy me," may seem to have been granted although it has not. Hearing that consent has been given is just thinking that it has, or pretending that it has. This confusion or distortion of reality can become quite perilous. Content and intent blur, their very meanings abused, in these patterns (Atkinson, 2005; Blackwell, 2004; Browne-Miller, 2007; Coker, Smith, McKeown, & King, 2000; Corbett, 2007; see also Asad, 1996).

When Relationships Kill

The majority of abusive, violent relationships do not end in death by accident, murder, or suicide. Yet, ultimately, there is always a risk of actual physical harm once an abusive relationship becomes physical. By degrees, some relationships become so out of control that murder is an actual risk, and where it is not, death by accident during violence is. And where severe depression and/or other psychological problems result from exposure to abuse and violence, there may be risk of suicide.

Swimming in the murky waters where intent and consent are blurred by those who are engaged in not seeing what these are, some people drown. Being repeatedly beaten over time, with the risk of serious injury increasing, involves risk of death. To deny this risk is to not see the problem. To be in a violent relationship where this risk is not acknowledged is to be in a dangerous mix of denial and physical danger. There is no guarantee of accurate prediction regarding when out of control abuse may go too far. (In the extreme, some persons who are suicidal could tend to prefer to kill themselves rather than be murdered, or rather than be wondering when they might be killed during IPV.)

Stages of Change

Models such as the Stages of Change (SOC) model have been proposed to describe the stages people experience as they recover from conditions, frequently moving quite gradually from engaging in harmful behaviors to engaging and maintaining engagement in healthy behaviors. These stages of change, most frequently focused on stages of overcoming and or recovering from addictive behaviors such as drinking, smoking and drug use, are labeled as precontemplation, contemplation, preparation, action, and maintenance (Dunn,

Hungerford, Field, & McCann, 2005; Prochaska, Velcier, Rossi, et. al., 1994; see also Scaer, 2005).

Although there are indeed significant differences between intimate partner abuse (for both its recipients and its perpetrators) and substance addiction, these stages of change can be useful in understanding the process of overcoming and/or recovering from this abuse and violence, especially given that abuse and violence cycles themselves tend to be habitual. This model tells clinicians and others working with those experiencing intimate partner abuse that behavior change occurs in steps, and an abrupt intervention into a habitual situation including ongoing intimate partner abuse is not necessarily effective and in fact is often countereffective. To move as quickly and effectively as possible toward behavioral change away from habitual or compulsive behaviors, we must first slow down to help people explore the why of changing before trying to impress them with the how. (Of course there are situations so severe that life-saving intervention must preempt this approach.)

Trapped in an Unfinished Experience

Ideally, there will be a deeper understanding of therapies that help recognize and release trauma in safe settings, allowing persons who have experienced the trauma to understand its ongoing impact and know the signs and triggers of this impact. Too many victims of trauma experience a deep lack of closure (which is often unseen for years) (Cozolino, 2006; Gazmararian, Spitz, Goodwin, Saltzman, & Marks, 2000; Hammer, Finkelhor, & Sedlak, 2002; NRC, 1996). Even when victims have been treated for the trauma, there can be a lingering sense of being stranded in a space that suggests closure—says that closure has taken place—but in which actual closure has never actually occurred.

Too many persons who have experienced trauma, including traumatic intimate partner abuse and violence, remain profoundly affected for life, with the effects reappearing or taking new forms sometimes years later (Asad, 1996; Bower, 2006; Briere, & Scott, 2006). Furthermore, too often, the effects of this sort of trauma surface in forms not identified with partner violence trauma. This makes the effects all the more difficult for persons experiencing them to address. A sense of free-floating anxiety, fear, disconnection from natural emotional sequences, and other lingering emotional conditions can haunt people and be triggered for no clear reason even years later. Hence, when one thinks the memories of intimate partner abuse and violence are healed, and that one is years past the problem, the dead hand of this abuse and violence may reach out and touch that person. *Traces, almost invisible traces, of patterns, can linger on.*

The sense of being trapped in an unfinished experience, stuck in a lack of closure state, troubles many persons who have experienced intimate partner vio-

lence and abuse. The sense that the trauma is not complete, that the impact of the trauma lingers and is relived—cycled back into—at the slightest reminder or trigger, must be addressed. We must see these sometimes vague but nevertheless profound effects as a serious outcome of intimate partner abuse and violence. Certainly, there is a profound posttraumatic stress component here, in this case a component with many faces. The ghosts of intimate partner violence can float through the subconscious forever, trapped in what seems to be a no-exit pattern (like this):

→ NO EXIT ←

Forgetting, Trauma, and Dissociation as Coping

Numbing while enduring abuse and violence is a coping as well as a tolerance mechanism, as is forgetting. Forgetting can indeed be coping; however, this forgetting of the experience is actually burying the experience deep in the subconscious mind. It lingers there, its pattern's sensations blocked but not erased. Forgetting serves as a barrier to remembering what has taken place, a profound form of not seeing, but, again, a not seeing.

The mind deals with traumatic experience in various ways, the processes of numbing and forgetting being two of them. The mind is skilled at internal protective camouflage, and has the ability to convince, in this case, not the outside world but itself, that the camouflaged, distorted, reality it serves up to its consciousness is real. Mental processes such as *dissociation*, in which the mind separates out normally connected mental processes from each other and from the rest of the mind, are a way of not seeing or processing these as connected experiences, as whole events. Painful memories, taken apart and served back up to ourselves fractured, are possibly less painful than when served up as a whole. This memory-fracturing process as a coping skill is then transferred to experience in the here and now, dissociating the mind from the experience of present-time reality. Memories and current experiences become incompletely perceived and reacted to.

While the person traumatized by intimate partner violence may not be formally diagnosed as dissociating, there may likely be a certain degree of dissociation in any storage of the memory of intimate partner violence trauma. When calling up and addressing these fractured memories, the expression of them as a whole is naturally going to be more reflective of fragmented memories of the experience than of whole ones. The traumatized individual, who is perhaps long over the physical pain the violence may have caused (its physical wounds and even visible trauma), may live forever with the lingering and subconscious sense of incomplete expression of something too vague to label. Being, on a

deep, hidden level, frozen in this unfinished state, the person is done with it all only on the surface.

There can be a deeply buried need for a sense of closure when living with a history of intimate partner abuse and violence, even after the visible, conscious sense of closure has been reached. Sometimes the trauma of violent experience lingers, hidden but present, and subtly affects all aspects of one's existence for years, maybe decades. Pretending this is not the case does not make it not the case; rather, it subjects some traumatized people to half-lives, never being entirely themselves. Given that we are indeed forever changed, on a very deep neurological level, by trauma, we help not only to rebuild what can be salvaged of the self, but to construct a new self, is essential.

Trauma Upon Trauma Upon Trauma

In instances of long-term relationships in which abuse and violence is ongoing, there may be trauma upon trauma upon trauma, continuously compounding the effects of trauma-induced neurological change while burying conscious realization that one is actually traumatized. Not only is violence-related trauma in the face of ongoing violence difficult to detect, but the related need for closure (for something suggesting the closing of the shattering experience of violence and its long-term effects) is buried.

Persons who are being abused may even cling to a dangerous pattern of relationship violence, not realizing that it may be the trauma itself perpetuating the pattern and the addiction to the pattern. The hunger for closure can leave the traumatized individual stuck in a pattern whose cycles may fool all those involved and feel somewhat like closure each time the cycle ends, when the individual takes a break from the violence and "enjoys" a sweet moment of relief.

PASSING ON THE PATTERN

The trauma does not reside in the victim and only the victim. Others around are also affected and may also be traumatized.

The Unaddressed Injuries

Volumes can be written about the impact of intimate partner violence on children who witness it, feel that they are to blame for it, may find role-modeling in it, and when caught in the fray are hurt emotionally as well as physically. Although this discussion is in large part not focused on children,

this is by no means a message that what happens to these child witnesses does not matter.

Carriers of Patterns

These children are the carriers into the future of our values and knowledge. They will likely (and fortunately) want to improve upon their parents' values and expression of these, and this is good. So much can be done better. These precious people, their parents' offspring, deserve a great deal of caring and intelligent assistance in identifying, and recovering from, their exposure to adults' intimate partner violence—and their parents' pain. To break the cycle of intimate partner violence—as well as its long-term health and mental health effects, pain, and trauma—we must understand that all of this can spill over from generation to generation in some form unless a conscious and visible effort to stop the violence is made. Breaking pattern addiction can be multigenerational work.

It is also important to see that, when adults repeat patterns of emotional and physical violence and abuse around children, they are including children in these patterns. Any detrimental habits, negative addictions, played out around children bring the children right into these patterns. The compulsive, destructive, abusive cycles of intimate partners include any children who are apparently on the sidelines. They cannot be unaffected. Nor can be they saved from the roller coaster rides of cycles played out by adults who are likely, in some difficult to explain (to a parent let alone a child) way, addicted to the patterns they have established. No matter how much a parent believes a child is insulated from the parent's ongoing involvement in intimate partner violence and abuse, there is little protection from this reality for the child. Children see and hear—*and feel*—even the smallest signs of this problem. They ride the emotional and physical roller coaster ride with their parents.

Teaching Denial

To pretend to children and teens that there is no abuse and violence when there is abuse and violence is not only absurd but cruel. Young people do perceive something, feel it, usually also hear and see it and the injuries. When the feeling, hearing, and seeing is not validated by the parent, this denial of an actual reality is disturbing, confusing, and distressing. Why drive a child into denial-like patterns, teaching that denial of a serious problem is normal, alright, a fine way of life? Why add lessons in denial to a child's pain and roller

coaster experience? Why fuel children's addictions to detrimental patterns such as IPAV?

Harsh and Painful Reality

The harsh and painful reality is that these children are dependent upon adults who are abusing and being abused. They have no way of ending the relationship with these adults, and they have no way of choosing not to need these adults. This dependence upon disturbed adults, coupled with the mixed messages that children in these situations typically receive (such as, it is bad to hit people even though you see this happening here at home), can be highly stressful and emotionally disturbing for these children.

Riding the roller coaster of fight, feel better, fight, feel better, fight, feel better, and so on, children absorb elements of abuse and violence cycle patterning. Moreover, they absorb the ride itself—high low high low high low—fear safety fear safety fear safety—again and again. For some children, especially those who do not know what patterning is, or what is happening to them as witnesses (which is most children in these instances), these unsettling, terrifying (potentially show-stopping in terms of developmental impact) patterns can become deeply buried inside them. These hidden time bombs tick for years, sometimes many years. Much, much later, or maybe not so much later, a trigger may fire the pattern into action, and the child is at risk of continuing the cycle. Or the child is at risk of playing out the cycle of abuse and violence in another way, such as via alcohol and drug addiction, food addiction, or other detrimental and dangerous behavioral patterns.

Adults seeking to rewrite the programming they may have instilled in their children (when they allowed their children to witness emotional and physical violence) must teach their children the same thing they themselves need to learn: how to recognize and change patterns of abuse and violence. Not to teach these things, out of concern that the material to be taught is too much for children's ears and eyes, is illogical. These children have already been exposed to intimate partner violence and abuse. Now they have a right to a recapturing of this information in a way that prepares them to avoid their parents' experiences. The material presented here, for example, is material young people can learn and many indeed have a hunger for. Young adults forming young intimate partner relationships are especially in need of this sort of information. Knowing how relationships work, knowing how to spot compromises and trades as they are being made, knowing the slippery definitions of intent and consent, is all very important. Children can be taught to recognize patterns of malattraction when they see them.

Tolerance

Of course it is not only fighting parents who teach abuse and violence as a way of life, an acceptable medium of exchange. The media and the world around children teach this as well. Yes, we live in a world where violence is virtually normal. Everything they see, and a large part of what they learn in school, tells children that violence works, that violence is part of life and part of history. The tolerance of violence is instilled so deeply in children, and yet so invisibly. Children's nervous systems react to violence and record their reactions to violence. Repeated exposure to violence dulls some of the response, and generates a mental and biochemical system of incorporating this reality, perhaps a desensitization as the brain cells open receptor sites that are hungry for the addictive roller coaster ride the adults' (and the world's) patterns take them on.

Parents can counter this trend by visibly practicing *positive conflict resolution* processes and telling their children this is what they are doing. Rising above violence can be done and can become a way of life, first in the home. No, we cannot turn back the hands of time, but we can start now, and teach our children well—or at least better than we have done so far. IPAV addiction is rampant, yet it can be halted. The stability, sanity, and safety pattern need devolve no further.

COMPOUNDING IPAV WITH SA PATTERNS

This discussion has delved into the nature of troubled IPR behaviors. Of course, little of the above occurs in isolation or free of other influences such as substance use, abuse, and addiction. Clearly there is frequent co-occurrence of domestic violence, substance abuse, and chemical dependence. Data on this co-occurrence are everywhere, with 20 percent to 80 percent of all instances of battering co-occurring with substance use/abuse. Common data find alcohol use involved in 50 percent of instances of violent behavior.

Characteristics of Co-Occurrence

Particular circumstances bring IPR troubles and SA together (Department of Peace, 2006; Lang, 1993; Leonard, 2000; Nace, 2007). These include: the desire to self-medicate to cope with physical and emotional pain; the learned (in childhood) association of IPAV and SA; the practice of disavowal in using one behavior as an excuse for the other (SA excuses IPAV and vice versa). Characteristics associated with substance abuse can fuel IPAV: disinhibition, in which alcohol and or other drugs reduce inhibition of socially unacceptable

behaviors such as violence; distortion, in which there is a blunting or disturbing of cognitive regulators resulting in misinterpretation and/or distortion of the abusee's remarks and behavior; and paranoia, in which distrust and jealousy and other emotions that can trigger abuse and violence are present.

Patterns of SA can parallel and even cross-trigger IPAV. The basic pattern

$$\text{trigger} \rightarrow \text{urge} \rightarrow \text{response}$$

is seen in both SA and IPAV pattern addictions. So representative of each other are these trigger-urge-response cycles that they can even be confused and that confusion can even be acted upon. Triggers for the SA pattern can be triggers for the IPAV pattern and vice versa. Cravings for a drug of "choice" can be cravings for an abusive interaction of "choice." Moreover, triggers for one pattern may be initiated with the conscious or subconscious intent to trigger the other pattern: SA might be engaged in not only as a response to an IPAV pattern but also as an excuse for it, and vice versa.

Intimate partner violence is often blamed on substance abuse. For example, the person who is abusing may blame the abuse on drugs and or alcohol; the person who is abusing may blame the abuse of the victim on the victim's substance abuse; the victim may claim that the battering is excusable because of either the victim's SA or the batterer's SA.

Persons who are being abused often tend to increase their substance use and abuse in response to IPAV, typically calling this "necessary self-medication" of physical and emotional pain. Many persons being abused are pressured into drug and or alcohol use by the persons abusing them as a mechanism of control and/or abuse. Any treatment of either the IPV or the SA must address this dual condition. As noted earlier, alcohol and drug addiction cannot be said to be the primary cause of IPAV. Once SA and IPAV are in process, then providing only substance abuse/substance addiction treatment will not stop IPV. (Nor will only IPV treatment stop SA.) Note that IPV may be likely to increase once abstinence from drugs and alcohol begins, and to increase as abstinence continues. The same is true for IPAV, as the verbal and emotional violence (threats, manipulation, etc.) are likely to increase as well. Therefore, the safety of the person who has been abused can be quite fragile and must be monitored during and after substance addiction treatment. Many victims in addiction treatment are facing forms of abuse typically unlabeled, overlooked. or downgraded in significance (e.g., the abuser preventing the victim of the IPAV abuse from attending or succeeding in substance addiction treatment—and/or using threats of or even violence itself to interfere with the victim's addiction treatment—and/or even offering the drug/alcohol of addiction to the victim. In other words,

abuse here can include working to keep the person being abused from getting better, from receiving and participating in SA treatment.

Key here is the recognition that in treating IPAV, legitimate safety and survival strategies sometimes conflict with SA recovery strategies. For example, addiction treatment professionals are naturally focused on the involvement of family and significant others in addiction treatment. Addiction treatment quite rightly discourages behaviors and attitudes such as dishonesty, resistance, and noncompliance. However, these same behaviors and attitudes may be essential to protect the person who has been emotionally and/or physically abused via IPAVs and who may still be at risk of being abused, attacked, or killed.

EXAMINATION OF IPR *MAL*ATTRACTION PATTERNS: CONCLUSION

This general discussion of addiction to IPAV patterns with and without accompanying SA identifies what is merely the tip of the iceberg. Patterns of malattraction, abuse, and violence wear many faces and mask themselves in countless ways. Revealing to ourselves the multitude of malattractions and their physical and nonphysical abuse and violence patterns—the IPAV addictions in which too many engage—may be immensely disturbing, chillingly honest, perhaps even repulsive. Yet, removing this mask to unveil and examine troubling personal and interpersonal patterns—addictions that can emerge in relationships—may be profoundly illuminating and have far-reaching benefits to the individuals involved and to the children, communities, and worlds around them.

NOTE

1. Some portions of this chapter are adapted with permission from Angela Browne-Miller, *To Have and to Hurt: Seeing, Changing or Escaping Patterns of Abuse in Relationships*, Angela Browne-Miller, Copyright 2007 (Praeger Publishers, 2007). Reproduced with permission of Greenwood Publishing Group, Inc., Westport, CT. Some other portions of this chapter have been drawn from the prepublication version of Angela Browne-Miller, *Rewiring Your Brain to Change Your Behavior* Angela Browne-Miller copyright 2008, (Praeger, in press, 2009). However, most of the material contained herein is written specifically for this chapter.
2. Portions of this chapter are worded in such a way as to model for practitioners and treatment professionals rationales and explanations (and persuasions) regarding the addictive nature of IPV in such a way that, with further adaptation to the audience, clients and patients can absorb the information. All of these portions, and the vignettes provided in these portions, have been written and tested by the

author, Dr. Angela Browne-Miller, in use with hundreds of continuing education and seminar students and also clients. All rights reserved by Dr. Angela Browne-Miller. Please contact Dr. Browne-Miller to discuss applications of such material at www.AngelaBrowne-Miller.com and DoctorAngela@aol.com.

REFERENCES

Asad, T. (1996, Winter). On torture, or cruel, inhuman, and degrading treatment. *Social Research*.

Atkinson, B. (2005) *Emotional intelligence in couples therapy: Advances from neurobiology and the science of intimate relationships*. New York: W. W. Norton and Company.

Berliner, B. (1947). On some psychodynamics of masochism. *Psychoanalytic Quarterly, 16*, 459–471.

Blackwell, A. H. (2004). *The essential dictionary of law*. New York: Barnes & Noble Books.

Bower, B. (2006, May 27). Burden of abuse: Violent partners take their toll on women. *Science News*, 323.

Briere, J., &. Scott, C. (2006). *Principles of trauma therapy: A guide to symptoms, evaluation, and treatment*. Thousand Oaks, CA: Sage.

Browne-Miller, A. (2007). *To have and to hurt: Seeing, changing or escaping patterns of abuse in relationships*. Westport, CT: Praeger.

Bugarin, Alicia. (2002). *The prevalence of domestic violence in California*. Sacramento: California Research Bureau, California State Library.

Burgess, A. W., Hartman, C. R., & Clements Jr., P. T. (1995, March). Biology of memory and childhood trauma. *Journal of Psychosocial Nursing: Mental Health Services, 33*(3), 16–26.

Campbell, R., Sullivan, C. M., & Davidson II, W. S. (1995). Women who use domestic violence shelters: Changes in depression over time. *Psychology of Women Quarterly, 19*(2), 237–255.

Catalano, S. (2006). Bureau of Justice statistics webpage report, intimate partner violence in the United States. Retrieved from http://www.ojp.usdoj.gov/bjs/intimate/ipv.htm

Centers for Disease Control. (2002, June). CDC injury research agenda. Atlanta, Georgia, National Center for Injury Prevention and Control, Centers for Disease Control, Department of Health and Human Services. Retrieved from http://www.cdc.gov/ncipc/pub-res/research_agenda/07_violence.htm

Chandler, J. (2005, May). The explanatory value of social movement theory. *Strategic Insights, 4*(5).

Chu, K. (2006, May 8). Many marriages today are 'til debt do us part. Managing your money. *USA Today*. Retrieved March 14, 2007, from http://www.usatoday.com/money/perfi/basics/2006–04–27-couples-cash-series_x.htm

Coker, A. L., Smith, P. H., McKeown, R. E., & King, M. J. (2000). Frequency and correlates of intimate partner violence by type: Physical, sexual, and psychological battering. *American Journal of Public Health, 90*, 553–559.

Corbett, S. (2007, March 18). The women's war. *The New York Times Magazine*, section 6, pp. 41–55, 62, 71–72.

Cozolino, L. (2006). *The neuroscience of human relationships: Attachment and the developing social brain*. New York: W. W. Norton and Company.

Dahlberg, L. L., & Krug, E. G. (2002). Violence—A global public health problem. In E. G. Krug, L. L. Dahlberg, J. A. Mercy, A. B. Zwi, & R. Lozano (Eds.), *World report on violence and health* (pp. 3–21). Geneva, Switzerland: World Health Organization.

Department of Peace Campaign: The Peace Alliance. (2006). *Statistics on violence fact sheet, 2006*. Retrieved from http://www.thepeacealliance.org

DeWolfe, D., & U.S. Department of Mental Health Services. (2004). *Mental health response to mass violence and terrorism: A training manual*. Rockville, MD: Substance Abuse and Mental Health Services Administration.

Dingfelder, S. F. (2007, February). The love drug: More than a feeling. *Monitor on Psychology, 38*(2), 40–41.

Dunn, C., Hungerford, D. Field, C., & McCann, B. (2005, September). The stages of change: When are trauma patients truly ready to change? *Journal of Trauma Injury, Infection, and Critical Care, 59*(3), S27–S31;

Family Violence Prevention Fund. (2007, January 16). Drop in domestic violence. *Speaking Up, 13*(1), 1.

Gazmararian, J. A., Spitz, A. M., Goodwin, M. M., Saltzman, L. E., & Marks, J. S. (2000). Violence and reproductive health: Current knowledge and future research directions. *Maternal and Child Health Journal, 4*(2), 79–84.

Global Violence Prevention Advocacy. (2005). Making violence preventable, not inevitable. World Health Organization. Retrieved October 10, 2008, from http://www.pahef.org/about/success_stories/global_violence/violence_publications/GlobalVPAdvocacyBrochures02_2006__6_1_1444.pdf at http://www.pahef.org/search/search.aspx?q=Henry+F&x=0&y=0

Gorman, C. (2007, January 29). A user's guide to the brain: Six lessons for handling stress. *Time*, 80–85.

Hammer, H., Finkelhor, D., & Sedlak, A. J. (2002, October). *Children abducted by family members: National estimates and characteristics*. National incidence studies of missing, abducted, runaway, and thrownaway children. U.S. Department of Justice Office of Justice. Retrieved from http://www.ncjrs.gov/pdffiles1/ojjdp/196466.pdf

Hendy, H. M., Eggen, D., Gustitus, C., McLeod, K. C., & Ng, P. (2003). Decision to Leave Scale: Perceived reasons to stay in or leave violent relationships. *Psychology of Women Quarterly, 27*(2), 162–173.

ICAN (Interagency Council on Child Abuse and Neglect). Retrieved October 10, 2008, from http://ican.co.la.ca.us/about.htm

Lang, A. R. (1993). Alcohol-related violence: Psychological perspectives. In S. E. Martin (Ed.), *Alcohol and interpersonal violence* (pp. 121–147). NIAAA Research Monograph No. 24. NIH Pub. No. 93–3496. Rockville, MD: NIAAA.

Leonard, K.(2000). Domestic violence and alcohol—What is known and what do we need to know to encourage environmental interventions. In *Commissioned papers,*

Alcohol Policy XII Conference: Alcohol and Crime, Research and Practice for Prevention (pp. 69–91). Washington, DC: National Crime Prevention Council.

Leone, J. M., Johnson, M. P., Cohan, C. L., & Lloyd, S. E. (2004) Consequences of male partner violence for low income minority women. *Journal of Marriage and Family, 66*(2), 472–490.

Loring, M. T. (1998). *Emotional abuse: The trauma and treatment.* San Francisco: Jossey Bass.

McCormick, M., & Sacks, G. (2007, January 2). New DOJ domestic violence study undercounts male victims. *Baltimore Sun,* p. 15.

Meyers, L. (2007, February). The love drug: The eternal question—does love last? *Monitor on Psychology 38*(2), 44–46.

Nace, E. P. (2007). *Patients with substance abuse problems: Effective identification, diagnosis, and treatment.* New York: W. W. Norton and Company.

National Research Council [NRC]. (1996). *Understanding violence against women.* Washington, DC: National Academy Press.

NCIPC (National Center for Injury Prevention and Control), Centers for Disease Control and Prevention. (2006, February). *Intimate partner violence fact sheet.* Retrieved February 21, 2006, from http://www.cdc.gov/ncipc/factsheets/ipvfacts.htm

Prochaska, J. O., Velcier, W. F., Rossi, J. S., et. al. (1994). Stages of change and decisional balance for twelve problem behaviors. *Health Psychology, 13,* 39–46.

Rennison, C. M., & Welchans, S. (2000, May). *Intimate partner violence.* Bureau of Justice Statistics Special Report. Washington, DC: U.S. Department of Justice. NCJ 178247.

Rivara, F. P., Mueller, B. A., Somes, G., Mendoza, C. T., Rushforth, N. B., & Kellerman, A. L. (1997). Alcohol and illicit drug abuse and the risk of violent death in the home. *Journal of the American Medical Association, 278,* 569–575.

Roberts, G., Hegarty, K., & Feder, G. (Eds.). (2006). *Intimate partner abuse and health professionals: New approaches to domestic violence.* New York: Elsevier.

Rosen, J. (2007, March 11). Brain on the stand: How neuroscience is transforming the legal system. *The New York Times Magazine,* section 6, pp. 48–53, 70, 77, 82.

Saint-Jacques, M., Brown, T., Caplan, T., & Werk, A. (2006). Congruence in reports of partner violence of men in treatment for substance abuse. *Family Violence and Sexual Assault Bulletin, 22*(2), 5–13.

Salber, P. R., & Taliferro, E. (2006). *The physician's guide to intimate partner violence and abuse: A reference for all health care professionals.* Volcano, CA.: Volcano Press.

Scaer, R. (2005) *The trauma spectrum: Hidden wounds and human resiliency.* New York: W. W. Norton and Company.

Schwartz, J. P., Hage, S. M., Bush, I., & Burns, L. K. (2006, July). Unhealthy parenting and potential mediators as contributing factors to future intimate partner violence: A review of the literature, *Trauma, Violence & Abuse 7*(3), pp. 206–21.

Sethi, D., Marais, S., Seedat, M., Nurse, J., & Butchart, A. (2004). *Handbook for the documentation of interpersonal violence prevention programmes.* Department of

Injuries and Violence Prevention. Geneva, Switzerland: World Health Organization.

Simon, L. (2003). *Psychology, psychotherapy, psychoanalysis, and the politics of human relationships*. Portsmouth, NH: Praeger.

Tjaden, P., & Thoennes, N. (2000). Extent, nature, and consequences of intimate partner violence: Findings from the National Violence against Women Survey. Washington DC: U.S. Department of Justice, Bureau of Justice Statistics.

UNICEF (United Nations Children's Fund). (2000, May). *Domestic violence against women and girls*. Innocenti Digest No. 6 (Preliminary Edition). Florence, Italy: Innocenti Research Center.

U.S. Department of Health and Human Services, Public Health Service (USDHHS), National Institutes of Health. (1999). Agenda for research on women's health for the 21st century. A Report of the Task Force on the NIH Women's Health Research Agenda for the 21st Century. Vol. 6. *Differences Among Populations of Women*. Bethesda, MD. NIH Publication No. 99-4390.

van der Kolk, B. A. (1989, June). The compulsion to repeat the trauma. Re-enactment, revictimization, and masochism. *Psychiatric Clinics of North America, 12*(2), 389–411.

Weitsman, Susan. (2000). *Not to people like us: Hidden abuse in upscale marriages*. New York: Basic Books.

World Health Organization [WHO]. (2004). *Preventing violence: A guide to implementing the recommendations of the "World Report on Violence and Health"*. Geneva, Switzerland, pp. 1–92. Retrieved December 1, 2008, fromhttp://whqlibdoc.who.int/publications/2004/9241592079.pdf

World Health Organization [WHO]. (2005). WHO Multi-country study on women's health and domestic violence against women: Initial results on prevalence, health outcomes and women's responses. Geneva, Switzerland, pp, 1–38. Retrieved July 15, 2008, from http://www.who.int/gender/violence/who_multicountry_study/summary_report/summary_report_English2.pdf

This page intentionally left blank

Part II

EATING AS A SPECIAL ISSUE

This page intentionally left blank

Chapter **7**

Craving Pizza? This Is Your Brain on Drugs: Eating Disorders as Addiction

Amanda Ruiz, MD, Hugo Barrera, MD,
and Norman Jackson, MS

The relationship between eating disorders and other more established forms of addiction (i.e., alcoholism, substance abuse) is becoming better understood as the science on the brain—specifically the reward centers—develops. In this chapter, the research on brain physiology is explored to distinguish the areas in the brain that are involved in this addictive process.

There are various forms of eating disorders. The three most common are anorexia nervosa, bulimia, and compulsive eating. Eating disorders are marked by extremes. Eating disorders are present when a person experiences severe disturbances in eating behavior, such as extreme reduction of food intake or extreme overeating, or feelings of extreme distress or concern about body weight or shape.

Anorexia nervosa (AN) is a potentially fatal condition. People with anorexia are so afraid of gaining weight or losing control over their bodies that they diet to the point of starvation. Even when they are very thin, anorexics see themselves as fat (Bulik, et al., 2008). In recent studies, the human life cost of AN is startling. Among young women with AN, the rate of suicide is eight times higher than among young women in general (Bulik et al., 2008).

Bulimia nervosa is a condition characterized by repeated episodes of binge eating followed by attempts to purge food from the system through vomiting, laxative or diuretic abuse, exercising, or fasting. *Compulsive eating* is like bulimia, except that the compulsive eater does not try to purge the food he or she has eaten. The compulsive eater will often consume large quantities of high-calorie

foods in a short period of time, often leading to feelings of shame and an increase in body weight. Eating disorders can affect people from all walks of life regardless of age, race, religion, gender, and so on. As with all addictions, the substance is never the issue; the issue is the underlying causes that people are attempting to cover up or control.

According to the National Institute on Mental Health, the prevalence of eating disorders in the United States has increased steadily over the last 30 to 40 years. Over 90 percent are adolescent girls and young women. Eating disorders has the highest mortality rate of any mental illness—up to 20 percent. Two to five percent of males and females suffer from binge eating disorder. A total of 1.1 to 4.2 percent of females from bulimia nervosa. The first year of college can be especially challenging, with both females (4.5 percent) and males (0.4 percent) reporting bulimia.

In a recent study among gay or bisexual men, the rate of eating disorders has been found to be more than 15 percent higher than among heterosexual men (Columbia University's Mailman School of Public Health, 2007). The cause for this discrepancy is unknown, according to the authors of the study. The higher rate may be attributed to more body-centered focus norms within the gay/bisexual community. Young athletes, particularly gymnasts, runners, bodybuilders, rowers, wrestlers, jockeys, dancers, and swimmers, are especially vulnerable to developing eating disorders. Unfortunately, many of these disorders go underdiagnosed because of the complexity of symptoms and the lack of forthrightness of patients giving their medical histories.

The desire of athletes to be in elite shape may cause heath challenges, but that should not undermine the importance of incorporating exercise into our lives. The benefits of exercise on the human body are well understood, and numerous scientific studies support the notion that regular exercise can prolong life. This is evidenced in numerous chronic conditions (i.e., in the prevention and/or lowering of high blood pressure). Regular exercise boosts high-density lipoprotein (HDL), or "good" cholesterol, while decreasing low-density lipoprotein (LDL), or "bad" cholesterol. In addition, regular exercise can benefit type II diabetes patients and can elevate mood. This health benefit can be attained with a brisk 30-minute daily walk. These types of interventions can be difficult to adapt initially, but once adopted can lead to easier adherence.

The role of diet is very important in maintaining a healthy weight and preventing chronic illnesses. Proper nutrition has become increasingly difficult with higher-stress lifestyles and the common inclusion of readily available processed foods in the Western diet. For adolescents, being adequately nourished is essential to ensure that their growth and development progresses normally and continuously. Unfortunately, this time of our development is the time when most eating disorders develop.

Rapid physical growth and development in adolescence constitute the unique background to the development of eating disorders at this stage of life. For example, self-esteem problems intensify in many normal young women in the process of doubling their body weight, increasing the percentage of body fat, gaining about four inches in height, developing breasts, and acquiring other features of the mature female body, as well as experiencing menarche. Given that this development occurs within a six- to eight-year period, the rapidity of change contributes to the difficulty of the task of acceptance of the change.

The intensity of physical growth and development also accounts for the vulnerability of adolescents to long-term consequences if they experience semi-starvation. All organisms are subject to the greatest harm from food deprivation at periods when they are synthesizing tissue; they need nutrients to build into tissues and food the energy to fuel the process. Human teenagers are no exception to this basic biological rule.

Adapting a mental image of one's unique body—the body image—is basic to adolescent development. Body image distortion is a core characteristic of anorexia and bulimia nervosa. Thus, these disorders are commonly seen in adolescence, the period when young people are vulnerable to body image problems, and the progress of adopting a positive body image is interrupted for the teenager with an eating disorder.

THE BIOLOGY OF ADDICTION MODELS

One of the most hotly contested issues in models of addiction, no matter the type, is the notion that an addiction is a disease. Because society defines disease as a "condition of the living body that impairs normal functioning and is typically manifested by distinguishing signs and symptoms," (Merriam Webster, 2008) many associations come tied in with the definition, including value judgments.

For example, it seems fitting that persons that are medically ill receive assistance with requisite everyday responsibilities. As such, persons in poor health are nursed back to health while they regain strength.

This argument has not held true as ubiquitously for drug addicts (or food addicts) as well as it has, for example, for poststroke patients. Yet, each of these individuals increases their potential for meaningful recovery when she/he accepts responsibility for her/his health. A poststroke patient that actively engages in physical therapy typically improves more rapidly than the patient that is unmotivated to do so. Similarly, the overweight person that exercises generally gains strength and well-being relative to those persons who do not exercise and choose food-restriction alone.

Using the example just briefly outlined, each of the persons mentioned—the drug addict, the food addict, and the stroke patient—have the following in common:

Each person has evidence of disease on fMRI.
Each person has options for medical treatment, which may be beneficial.
Each person should take responsibility for prognosis and treatment, including diet and exercise for optimal recovery.

Ethical issues aside, our argument is not one that is based on wishful thinking or the desire that everyone get a second chance; it is one that is grounded in science. This chapter hopes to explain our point.

PET/MRI AS APPLICABLE TO ADDICTION MODELS

As we launch into an examination of brain imaging, we should first explain what we are looking at and why.

Brain imaging is useful in many specific ways. For example, in individualizing treatment with newer medications or implementing new medication, a knowledge of affected circuitry can point to chemical dysfunction that may be helped by medication (Freese, 2006). The design of behavioral treatments can tell you the types and severity of deficits and dysfunctions in the brain and the possible areas of the person's general strengths and deficits (Freese, 2006). Finally, brain imaging can potentially show how much viable tissue there is to work with; it can show the response of efficacy of treatment (Freese 2006).

IMAGING BEFORE AND AFTER

The brain is a complex organ with many complex circuits running through it. The circuits that coordinate our drive or "pleasure centers" are outlined below:

The main circuits for mediating reward behaviors are as follows:

Fronto-amygdalar pathway. This pathway connects the amygdala and prefrontal cortices *and* the limbic circuit. It integrates the amygdala with the hypothalamus and septal nuclei. The amygdala and cortico-limbic areas have conventionally been the "anatomical substrates" of eating disorders as described in a case report (Cerrato et al., 2004).

Papez circuit. This pathway joins the hypothalamus with the hippocampus and thalamus.

Why is all this important? We explain below.

The limbic circuit is primarily concerned with unconditionally rewarding stimuli like food, water, and sex. This is supported by the fact that the limbic system is connected to ancient, hard-wired structures in the brain. Other important players within your brain include the hypothalamus. Simply speaking, it is the master control of the autonomic function of the body. Stimulation or lesions of the hypothalamus result in systemic responses, such as increased or decreased appetite, including hyperphagia or anorexia, or change in sexual functioning (Goldberg, 1994). The thalamus is a sensory relay center. It is capable of sensing pain, but not pinpointing it. The hippocampus (or hippocampi, since there are two of them) has important functions related to both memory and mood. The nucleus accumbens relates to the motivation properties of food/palatability. The dorsal striatum regulates the caloric requirements necessary for survival.

Dopamine and Hedonism

Positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) have been used in the study of brain activity related to reward processing in both animal as well as human models for years (James, Gold & Liu, 2006; Martin-Soelch, et al., 2001, pp. 139–149; Spiegel, et al., 2005; Wang, Volkow, Thanos, & Fowler, 2004). Most of these studies have indicated that dopamine has a role in reward processing and motivation.

In other words, persons of all shapes and sizes may be driven by a thought or an idea. This thought or idea is then chemically reinforced by a swell of neurotransmitters and neuromodulators, which bathe the brain, making the person feel wonderful. The "craving" is born.

In one study, rodents were used to measure their dopamine output in response to natural incentives. In comparison to basal output, food and sex generated well over 150 percent and 200 percent, respectively, of the resting output of dopamine in the system (Fiorino & Phillips, 1999, 19). This "extra-extra" is nature's way of making sure we return for more.

NATURAL REWARDS AND THEIR IMPACT ON DOPAMINE

As we apply the same concept to substances of abuse, we see that other studies have demonstrated a correlation between the intensity of the "high" induced by various drugs and the level of dopamine that was released (Wang, et al., 2004).

For example, in another study, , the various substances were administered and then the basal rate of release dopamine was measured in the nucleus

accumbens of rodents. Cocaine and methamphetamine had striking peaks, at 350 percent and 1150 percent, respectively, over the baseline dopamine level (DiChiara, Tanda, & Frau, 1993, 112). Based on these findings, it becomes easier to understand why individuals pursue the substance of their addiction despite medical, legal, economic, and other adverse consequences.

NEURO-IMAGING AND ITS APPLICATION TO FOOD

Studies demonstrating that drug abuse involves reward circuitry in the brain have led to interesting research primarily pioneered by Dr. Nora Volkow at the National Institute of Drug Addiction. She was among the first to use imaging technology to investigate neurochemical changes associated with addiction. Similarly, Dr. Volkow has been at the forefront of research that appears to demonstrate that compulsive eating involves many of the same circuits that drug abuse does (Volkow, 2007; Volkow & O'Brien, 2007; Volkow & Wise, 2005).

Further, research has indicated that compulsive eating is reinforced by fat-laden and high-sugar foods. The pizza you love and cannot get enough of, particularly when you have had a long hard day? It is hard wired, believe it or not. The truth is that high-calorie food packs a lot of energy. When viewed from a historical perspective, it likely offered an advantage in terms of survival. In other words, it was palatable, high energy, and reinforcing.

Viewed from this perspective, it should not be shocking, then, to learn that compulsive overeating appears to share many of the same characteristics as drug addiction. In fact, in one study, both methamphetamine abusers and obese subjects were found to have significantly lower measures of striatal dopamine D2 receptors available than did control subjects. Low levels of endorphins have been associated with cravings for fatty foods and chocolates. Further, serotonin, "the happy neurotransmitter," has been linked to sugar cravings; low levels of serotonin lead persons to crave sugar and gain weight as a result. Even more shocking, the newest word related to eating disorders is "drunkorexia," shorthand for a disturbing blend of "self-imposed starvation or bingeing and purging, combined with alcohol abuse." It describes a phenomenon in which persons, typically women, some even middle-aged, refrain from eating throughout the day in order to offset the calories in the alcohol they consume at after-work or college functions including a cocktail hour (CBS News, 2008).

The age of onset of eating disorders, like waistlines, has been steadily increasing, partially in response to mid-life stresses such as divorce, career changes, remarriage, blended families, the empty nest syndrome, slowing metabolisms, and the ever-present pressures of the fashion industry push against us. Two polymorphisms of the "human obesity gene" account for 20 percent of the variance in basal metabolic index (BMI), especially in young women. This finding

is also consistent with frequent binge eaters who have low dopamine metabolite concentrations in their cerebrospinal fluid. Further, a high prevalence of binge eating disorder has been reported among the morbidly obese subjects who have undergone gastric bypass, a subject that is discussed in the following segment on treatment modalities for compulsive overeaters.

KEY POINTS ON BIOLOGY OF ADDICTION MODELS

Enter again our three subject examples: the substance addict, the food addict, and the stroke victim. Each has evidence of brain dysfunction on fMRI. Each has the option of medical treatment modalities. Each is also to some degree responsible for diet and exercise, that is, for making the right choices and taking little steps, one at a time. That is what every person in recovery does.

Some points to note are as follows:

1. Multiple similarities exist between the neurotransmitters that regulate eating disorders and addiction.
2. Although further research needs to be conducted, recent imaging studies indicate that modalities that have been useful in the treatment of compulsive disorders such as substance abuse may also be useful in the treatment of compulsive eating disorders (see Table 7.1).

Table 7.1
Neuromodulators Mediating Response to Cues/Craving

Neurotransmitter or Neuromodulator	Effect	Evidence/ Reference	Comment
Dopamine (D2) receptor antagonist	Enhances meal size Increases meal duration	Study performed on rodents(Clifton, Rusk, & Cooper, 1991, 105)	Dopamine decrease
Long-term administration D2 antagonists	Increased feeding Increased body weight	Study performed on female rats (Baptista, Parada, & Hernandez, 1987, 27)	Dopamine decrease
Patients receive D2 receptor antagonists	Increased weight gain	Human subjects (Allison & DE, 2001; 62 Supplement 7) (Wetterling, 2001, 24)	Ex. Antipsychotic medications
D2 agonists	Anorexigenic effect Weight loss	Human subjects (Scislowski, Tozzo, & Zhang Y, 1999, 23) Anecdotal reports	Dopamine increase

Table 7.1 (continued)

Neurotransmitter or Neuromodulator	Effect	Evidence/ Reference	Comment
Long-term drug addiction & obese persons	Reduced number D2 dopamine receptors in the striatum	Human subjects (Wang et al., 2004) Anecdotal reports	Dopamine increase
Neuropeptide Y (NPY)	Stimulates food intake	Wang et al, 2004	DA inhibits NPY in the hypothalamus
Opioid	Increases food palatability when mod-high	Animal models (Yeomans, 1996, 27) Anecdotal reports	Excessive levels of opioids result in stupor & anorexia
GABA/benzodiazepines	Increases food palatability Increases food consumption	Mammal models (Yeomans, 1996, 27)	GABA is an inhibitory neurotransmitter; the tongue and gustatory zone of the brain also have GABA receptors
Melanocortin	Regulation of food intake through hypothalamic receptors	F:\Eating as Addiction\ NGFN-Science Neuroimaging of hunger and satiety and assessment of sympathetic nerve activity in carriers of a MC4-R gene mutation.mht	Increased activation of limbic/paralimbic areas in obese individuals during apperception of palatable food
Ghrelin	Increases hunger and food intake	Acts on hypothalamus, but there is increasing evidence that it also acts directly on other areas: dopamine neurons, hippocampus (Malik, 2008)	Peripheral peptide
Leptin	Modulates the synthesis & release of dopamine	Wang et al., 2004	Peripheral peptide
Serotonin	Decreases appetite when high; low levels cause sugar cravings	Case report(s)	SSRI

Compiled by Amanda Ruiz, MD.

THE PRACTICAL ASPECTS OF TREATING ADDICTIVE EATING DISORDERS: TREATMENT MODALITIES AND MEDICAL TREATMENT OPTIONS

Ah, that there were a magic bullet for the treatment of exogenous obesity. The diet industry is a multi-billion-dollar market in the United States, and indeed around the world. Medications and herbs that tout the ability to reduce fat and increase weight loss are developed or released weekly. Yet, while millions of dollars are spent annually on weight loss medications, creams, drinks, and other diet elixirs, few if any of the items marketed deliver the promised results.

The medications listed below are currently approved by the United States Food and Drug Administration for the treatment of obesity (FDA, 2008). These medications typically fall into one of two categories: they are either appetite suppressants or they are metabolic stimulants. Similar to the neurotransmitters outlined above, they effectively increase dopamine.

- Dextro amphetamine
- Phentermine
- Sibutramine

Other medications that have not received a specific indication for the treatment of exogenous obesity but have been found to be helpful in the treatment of such disorders, either in case reports or in anecdotal reports of personal experiences with the medications, include the following:

Topiramate: This has been effective in the treatment of alcohol and nicotine dependence (and possibly food abuse?).

- Possible method: decreased impulsive eating/side effect weight loss.
- Increases Gamma-aminobutyric acid (GABA) (using a mechanism different from that used by barbiturates), thereby decreasing anxiety and possibly "comfort eating."

Rimonabant: This has been effective in the treatment of alcohol and nicotine dependence, as well as obesity. There is some evidence of efficacy for both smoking and weight loss in Europe; it is unapproved for use in the United States. Side effects including severe depression have limited its use in the United States.

Other antiepileptic medications and serotonin reuptake inhibitors, either alone or in combination, may also have potential in the treatment of exogenous obesity and other eating disorders related to addictions. However, without further study of these combinations to assess their safety in this population, it is premature to apply them here without a detailed risk-benefit analysis on a case-

by-case basis. Further, the value of medication as treatment is dwarfed when not coupled with an appropriate exercise, diet, and supportive psychotherapy program, as needed.

SURGICAL TREATMENT OPTIONS/METABOLIC SURGERY

With the advent of more efficient, inexpensive food production following World War II, the prevalence rate of obesity in the United States and the world at large began to increase. By the 1950s, surgeons began encountering obese patients with increasing frequency, stimulating, partially surreptitiously, the search for a surgical cure for this condition.

A well-known condition, the short-gut syndrome, results from a catastrophic loss of the nutritional absorptive capacity of the small intestine, usually following vascular compromise. Most patients with this condition become nutritionally impaired, but obese patients, though losing body mass, are better able to cope in the aftermath of the illness. This observation led pioneering surgeons to consider a small bowel bypass, by which a significant portion of the small intestine is functionally excluded from participating in nutritional absorption, as an operation that might result in loss of excess weight in the obese.

A jejunoileal bypass was the first operation performed for the purpose of weight loss in the morbidly obese. And not surprisingly, the operation was very successful in achieving this goal. With long-term follow-up of these patients, however, it became clear that the side effects arising from this rearrangement in the gut anatomy led to life-threatening complications, and within a generation, this resulted in the abandonment of this operation for weight loss.

The search for a surgical cure for obesity was further stimulated by these initial attempts, however. Approximately 20 different operations since have been described for weight loss. Validation and endorsement for surgical treatment for obesity arrived in the National Institutes of Health's (NIH's) Consensus Conference Statement in 1991, which found that in patients deemed morbidly obese, surgical intervention resulted in the only long-term successful treatment for weight loss and maintenance of weight. Morbid obesity, defined as a body mass index (BMI) greater than 40kg/m^2 or a BMI of 35kg/m^2 with associated comorbid conditions, was deemed primarily to arise out of an organic disruption. In the absence of an effective medical cure for the morbidly obese, surgery gained momentum.

From a general perspective, surgical weight loss procedures deal with weight loss from a gross or macro level, resulting in a reduction in absorptive capacity (*malabsorptive procedures*), or a reduction in caloric intake (*restrictive pro-*

cedures), or a combination of both (*restrictive-malabsorptive procedures*). Many of the metabolic effects of the anatomical rearrangements of the particular operations are slowly becoming better understood, surpassing in complexity the mechanisms originally attempted by physicians.

MALABSORPTIVE PROCEDURES

From a simplistic view, the difference between caloric intake and caloric expenditure is the formula that determines weight gain or loss. Obviously, the two terms are affected by a multitude of variables, intrinsic and extrinsic, organic and psychological, genetic and acquired. In the history of bariatric surgery, as we have seen, the initial surgical approaches were directed toward altering absorption, or more accurately, inducing malabsorption.

Absorption of nutrients in the gut occurs primarily in the small intestine, an organ that is 20 feet or more in length. Intestinal villi, finger-like projections of the mucosa or lining of the intestine, greatly expand the absorptive area. As briefly alluded to, short-gut syndrome arises from a situation, as the name implies, in which a significant amount of length and area of the small intestine is lost, usually as a result of vascular compromise or iatrogenic injury. The result is a syndrome of nutritional deterioration in an individual whose remaining intestinal area can no longer support adequate absorption of required calories in the immediate term. With time, some level of compensation can occur in the small intestine in the form of an increase in the height and number of the villi in the mucosa with a resulting increase in the absorptive area. During this transitional period, it was observed that obese individuals, with their excess stores of fat, could better adapt as they lost the excess weight. The connection between short-gut syndrome and obesity is what led to the creation of the first bariatric procedure, an operation that deliberately induced a short gut.

The jejunoileal bypass, performed first by Dr. Richard Varco in 1953 at the University of Minnesota, resulted in an anatomical change in the gut in which a significant portion of the small intestine was excluded from contact with nutrients. As the name implies, the intestine was *bypassed* but not removed, affording a certain level of safety in the form of reversibility. And after extensive study and time, reversal became necessary in the vast majority of patients, as the effects of this form of anatomic alteration became obvious. As a result of physiologic changes in the bypassed segment, including bacterial reflux, overgrowth, and translocation into portal venous flow, the liver became progressively affected in a presumed inflammatory mechanism. The consequence was hepatic failure in approximately 5 percent of patients in the first year following surgery and up to a 50 percent occurrence of cirrhosis in patients within 25 years of surgery

(O'Leary, 1992). For this reason, the jejunoileal bypass has all but been abandoned, with the added recommendation that previously performed bypasses be reversed to avoid the subsequent complications.

In the interim, other varieties of weight loss procedures were being developed, stimulated by the great success of these initial attempts. But this did not mean the end to *malabsorptive* operations. A second generation of such procedures was developed, including the biliopancreatic bypass and the biliopancreatic diversion with duodenal switch, technically more complex surgeries. These operations resulted in effective weight loss without the attendant side effects and complications inherent in the first-generation procedures. Though less common today, these operations have strong adherents who advocate these procedures in the "super morbidly obese" (BMI >60). The common thread is still that decreasing exposed surface area to nutrients leads to less absorption, and, therefore, the intake portion of the caloric formula is decreased, resulting in weight loss.

RESTRICTIVE/MALABSORPTIVE PROCEDURES

From a physiological standpoint, altering absorptive capacity is more complicated, with potentially more complex consequences, than simply decreasing the amount of calories that enter the body. It was with an eye to this concept, that of restricting the quantity of nutrients that enter the body in the first place, that the gastric bypass model of weight loss surgery came into existence. Dr. Edward Mason at the University of Iowa developed the first gastric bypass procedure in 1966, and within 10 years, following some technical modifications, it became the dominant bariatric procedure in the United States.

The initially described procedure involved partitioning the stomach into a small proximal pouch isolated from the remainder of the distal stomach and duodenum. This proximal gastric pouch was then attached to the proximal jejunum. Intake was therefore restricted by the small size of the pouch, which could handle only a certain volume of food before the patient experienced satiety. In addition, as the food exited this proximal pouch, it bypassed the distal part of the stomach and the whole of the duodenum, a situation that results in important hormonal changes that appear to play significant roles not only in weight loss but in the ameliorative effects on certain comorbidities associated with morbid obesity, such as type II diabetes mellitus. Moreover, these effects appear to occur independent of the weight that is lost.

Dr. Ward Griffin at the University of Kentucky modified the original Mason gastric bypass by converting the loop gastrojejunostomy to a Roux-en-Y gastrojejunostomy in order to eliminate bile-induced gastritis and esophagitis. He

also compared this procedure to the original small bowel bypass procedure, finding equivalent weight loss with significantly decreased complications in the gastric bypass group. With the advent of laparoscopy, the Roux-en-Y gastric bypass, performed laparoscopically, has dominated the bariatric landscape in the United States. Change, however, is continuing, and a new generation of purely restrictive procedures is gaining ground, primarily due to the ease of the newer procedures.

RESTRICTIVE PROCEDURES

The concept of a restrictive operation for weight loss arose primarily from the potential simple mechanistic alteration in the gut anatomy that would be required to achieve restriction in caloric intake. In the context of the physiological alterations that resulted from bypassing the small bowel in the first-generation jejunoileal bypass, which led to life-threatening complications, a way to restrict intake became attractive as a manner in which to achieve weight loss.

The Mason gastric bypass had a significant restrictive component but was still a hybrid procedure, with a bypass and malabsorptive component as an integral part for the subsequent weight loss and metabolic effects. But the bypass component also encompassed the creation of connections between different parts of the gut, with the attendant possibility of leaks at these connection sites. Technically, the creation of these connections can be challenging, especially laparoscopically, but also in the traditional open method. And finally, the metabolic changes resulting from bypassing the distal stomach and duodenum were not fully understood early on, but it was suspected that potentially troublesome complications could arise from such rerouting of the nutrient stream.

The purely restrictive procedures address these concerns by essentially simplifying the anatomical changes required. The gastroplasty procedure, described in 1973 by Dr. Mason, is one in which the proximal portion of the stomach was divided horizontally, leaving a narrow channel between the proximal and distal portions of the stomach, slowing the transit time of nutrients between the two areas, yet leaving the nutrient stream directionally intact. This procedure resulted in restriction of flow, but was not consistently effective due to stretching of the proximal gastric pouch or enlarging of the channel between the two areas. Weight loss with this procedure was, therefore, not consistent.

Subsequent experimentation with the concept of gastric restriction led to the development of a modification of the gastroplasty procedure, namely, the vertical banded gastroplasty, using a silastic band or mesh material to maintain the caliber of the narrowed channel. These modifications have led to more stable

weight loss results, though not quite at the levels achieved with the Roux-en-Y gastric bypass."

The implantation of an adjustable gastric band is a relatively new procedure that is purely restrictive in function and technically easy to perform, even laparoscopically. The idea of utilizing a synthetic implant to restrict intake for weight loss arose out of a similar device, the Angelchik ring, a synthetic ring that was employed at the esophagus for prevention of esophageal reflux. Marcel Molina, MD, of the Spring Branch Medical Center in Houston, Texas, first described wrapping a polypropylene mesh around the upper stomach. Adjustability was soon found to be desirable, and obesity surgeons, L. Kusmak, MD, and G. Hellers, MD, developed an adjustable band connected to a port under the skin that could be subsequently accessed in the office with a needle and inflated or deflated as the situation dictated. This procedure is now becoming one of the fastest growing procedures for weight loss in the world and the United States. The speed of the weight loss is slower than with gastric bypass, and follow-up for continued weight loss and maintenance of lost weight is imperative. Nevertheless, the technical ease, speed of recovery, reduction in serious complications, and reversibility has spurred its growing popularity among patients and surgeons alike.

METABOLIC SURGERY

As alluded to earlier, researchers are learning that the bypass portion of the Roux-en-Y gastric bypass procedure appears to contribute to almost immediate improvement in type II diabetes mellitus through complex interactions between "fore-gut "and "hind-gut" hormonal changes. The improvement in this form of diabetes occurs with weight loss and is, therefore, associated with other forms of bariatric surgery, but it is the immediacy of this effect, independent of actual weight lost with gastric bypass, that has led to a new conceptualization of bariatric surgery, in particular gastric bypass, as a form of metabolic surgery. This concept has even been extended to the point where in the near future it may not be unheard of to consider metabolic surgery for nonobese patients suffering from type II diabetes mellitus.

SELECTION PROCESS

The surgical treatment for morbid obesity is not a panacea, and the results can be dramatically affected by the behavior of the patient postoperatively. Surgery, therefore, needs to be viewed as a tool that the patient needs to use effectively in order to achieve weight loss and maintenance of weight. This tool can easily be misused and does not work in an automatic fashion.

Examples of behaviors that negatively impact the postbariatric patient include "grazing" (eating small quantities of food throughout the day), not adhering to the high protein/low carbohydrate diet (usually prescribed for gastric bypass patients), and ingesting high-caloric drinks. It is hoped that psychological evaluation will detect issues of noncompliance or compulsive behavior so as to either address these issues with the patient before surgery or eliminate the patient as a good candidate for surgery.

Unfortunately, failures in detection of important psychological issues result in a great many of the failures of weight loss in patients who have had bariatric surgery.

THE FUTURE

Only a small proportion of the patients who are potential candidates for bariatric surgery are actually availing themselves of this option. The estimates are that 1–2 percent of morbidly obese patients undergo bariatric surgery every year. In part, this is due to availability and insurance coverage for this type of surgery, though this situation is improving as more surgeons become adept at the procedures and coverage for these surgeries is expanded. Part of the problem, however, is educational, on the part of patients as well as referring physicians. This also appears to be improving, as both patients and physicians are realizing the beneficial effects of weight loss surgery, not only in dealing with the issue of excess weight but also in dealing with associated comorbid conditions.

SUMMARY

Our brain is a complex organ that regulates much more than we realize. It seems odd to think that addiction may come in so many various forms, as indicated by the neuro-imaging studies introduced above. Addictions include the traditional vices such as addiction to cocaine and alcohol, and yet may also include compulsive overeating and, its opposite, stringent fasting. For many persons, eating pizza produces a high, just as fasting produces anorectic euphoria. Alarmingly, the idea that certain age groups were out of the typical age of onset no longer holds true: our population is growing more obese; at the same time, more women are crossing the line from being thin social drinkers to binge-alcohol anorexics, regardless of their age.

Medications and metabolic surgery may prove to be an option for many, yet even these advances must be approached with caution. Up to 20 percent of postbariatric surgery patients experience suicidal ideation after the opera-

tion, requiring strict screening beforehand. The reasons for this phenomenon have yet to be studied academically; the possible risk factors are numerous and beyond the scope of this chapter. Thus despite marvelous advances in neuroimaging, medication, surgery, rehabilitative psychotherapy, and diet/exercise, and the implications these advances hold for treatment tomorrow, the best medicine teaches us that it is through a careful coordination of these modalities that we offer our patients and ourselves optimal results on a long-term basis.

REFERENCES

Baptista, T., Parada, M., & Hernandez, L. (1987). Long term administration of some anti-psychotic drugs increases body weight and feeding in rats. Are dopamine D2 receptors involved? *Pharmocology biochemistry and behavior,* 399–405.

Bulik C. M., et al. (2008, April). Suicide attempts in anorexia nervosa. *Psychosomatic medicine, 70*, 78.

CBS News. (2008 January). *CBS News.* Retrieved April 15, 2008, from http://www.cbsnews.com/stories/2008/01/23/entertainment/main3744289.shtml.

Cerrato, P., Grasso, M., Azzaro, C., Baima, C., Giraudo, M., Rizzuto, A., et al. (2004). Transient compulsive hyperphagia in a patient with a thalamic infarct. *Journal of neurology, neurosurgery and psychiatry , 75*(9), 1364–1365.

Clifton, P., Rusk, I., & Cooper, E. (1991). Effects of dopamine D1 and dopamine D2 antagonists on the free feeding and drinking patterns of rats. *Behavioral neuroscience, 105,* 272–281.

Columbia University's Mailman School of Public Health. (2007, April 14). Gay men have higher prevalence of eating disorders. *ScienceDaily.*

Dagher. (2008, May). Why food looks yummy. *Scientific Blogging.* Retrieved May 25, 2008, from http://www.scientificblogging.com/news_releases/ghrelin_gut_hormone_why_food_looks_yummy

DiChiara, G., Tanda, G., & Frau, R. C. (1993). On the preferential release of dopamine in the nucleus accumbens by amphetamine: Further evidence obtained by vertically implanted concentric dialysis probes. *Psychopharmacology, 112* 398–402.

FDA. (2008, January). *FDA approved drug products.* Retrieved January 30, 2009, from http://www.accessdata.fda.gov/scripts/cder/drugsatfda

Fiorino, D., & Phillips, A. (1999). Facilitation of behavior and enhanced dopamine efflux in male rates in the nucleus accumbens after D-amphetamine induced behavior sensitization. *Journal of Neuroscience, 19,* 456–463.

Freese F. P. (2006). *Methamphetamine: What can brain imaging tell us?* UCLA Integrated Substance Abuse Programs. Retrieved April 15, 2008, from http://www.uclaisap.org/slides/presentations-meth-2006.html#pp.

Goldberg, M. (1994). *Clinical Neuro Anatomy.* Miami: MedMaster, Inc.

James, G., Gold, M., & Liu, Y. (2004). Interaction of satiety and reward response to food stimulation. *Journal of Addictive Disorders, 23*(3), 23–37.

Malik, E .A. (2008, May). Ghrelin modulates brain activity in areas that control appetitive behavior. *Cell Metabolism 7*, 400–409.

Martin, L. F. *Obesity surgery*. (2004). New York: McGraw-Hill.

Martin-Soelch, C., Leenders, K., Chevalley, A., Missimer, J., Künig, G., Magyar, S., et al. (2001). Reward mechanisms in the brain and their role in dependence: Evidence from neurophysiological and neuroimaging studies. *Brain research reviews, 36*(2–3), 139–149.

Mason. E. E., & Ito, C. (1967). Gastric bypass in obesity. *Surgical clinics of North America, 47*, 1845–1852.

Merriam Webster. (2008). Disease. *Merriam-Webster Dictionary*. Retrieved December 16, 2008, from http://www.merriam-webster.com/dictionary/disease.

National Institutes of Health [NIH]. (1991, March). *Gastrointestinal surgery for severe obesity*. *9*(1), 1–20.

O'Leary, JP. (1992). Gastrointestinal malabsorptive procedures. *American Journal of Clinical Nutrition, 5S*, 567S–570S.

Scislowski, P., Tozzo, E., & Zhang Y., et al. (1999). Biochemical mechanisms responsible for the attenuation of diabetic and obese conditions in ob/ob mice treated with dopaminergiz agents. *International journal of obesity related metabolic disorders, 23*, 425–431.

Spiegel, A., Nabel, E., Volkow, N., Landis, S., & Li, T. (2005). Obesity on the brain. *Nature neuroscience, 8*(5), 552–553.

Volkow, N. (2007). This is your brain on food. Interview by Kristin Leutwyler-Ozelli. *Scientific American, 297*(3), 84–85.

Volkow, N., & O'Brien, C. (2007). Issues for DSM-V: Should obesity be included as a brain disorder? *American journal of psychiatry, 164*(5), 708–710.

Volkow, N., & Wise, R. (2005). How can drug addiction help us understand obesity? *Nature neuroscience, 8*(5), 555–560.

Wang, G., Volkow, N., Thanos, P., & Fowler, J. (2004). Similarity between obesity and drug addiction as assessed by neurofunctional imaging: A concept review. *Journal of addictive disorders, 23*(3), 39–53.

Wang, G., Yang, J., Volkow, N., Telang, F., Ma, Y., Zhu, W., et al. (2006). Gastric stimulation in obese subjects activates the hippocampus and other regions involved in brain reward circuitry. *Proceedings of the national academy of sciences of the United States of America, 103*(42), 15641–15645.

Wetterling, T. (2001). Bodyweight gain with atypical antipsychotics. A comparative review. *Drug safety, 24*, 59–73.

Yeomans, M. (1996). Palatability and the micro-structure of feeding in humans: the appetizer effect. *Appetite, 27*, 119–133.

This page intentionally left blank

Chapter **8**

Eating Disorders and Disturbances: The Continuum of Eating Disturbances

Cynthia R. Kalodner, PhD

> *I did not think of myself as having an eating disorder, yet I could intimately understand the pain of these women. I was clearly not anorexic or bulimic, yet I definitely used food to soothe myself, was uncomfortable with my body, and shared the same struggle for wholeness.*
>
> (Radcliffe, 1993, p. 138)

Anorexia nervosa and bulimia nervosa are eating disorders, well known in popular culture since the media has identified and publicized them through articles, movies, and books. However, when considering eating disorders, limiting discussion to anorexia and bulimia is inaccurate since there are other eating disorders and problems that do not fit the criteria for anorexia or bulimia. In this book, the term *eating disorders* refers to psychiatric illnesses with specific criteria; these include anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified, which is a special category used for people who have eating disorders that meet most, but not all, of the criteria for anorexia or bulimia.

Eating disorder not otherwise specified is a very heterogeneous category, with six different types. The "not otherwise specified" part of the name might be misunderstood as somehow less serious or not as important as anorexia or bulimia, however, as we will see later in this chapter, eating disorders not otherwise specified are clinically significant eating disorders that require treatment. People with an eating disorder not otherwise specified do not have less body dissatisfaction than do people with anorexia or bulimia. Furthermore, there

may be a progression from eating disorders not otherwise specified to anorexia or bulimia (Herzog & Delinsky, 2001).

In addition to the three major types of eating disorders, there is a large group of people who are dissatisfied with body image and practice unhealthy eating practices, but they may not fit the criteria for any eating disorder. They engage in disordered eating, but do not meet the criteria for an eating disorder. These people have *eating disturbances*. In fact, there are many, many people who are dissatisfied with their body, have a fear of gaining weight, and may be anxious or stressed about weight and body shape. People with eating disturbances may skip meals, restrict food choices to a few acceptable things, and avoid foods that contain fat. They may binge eat occasionally and self-induce vomiting but do not have an eating disorder.

ASYMPTOMATIC, SYMPTOMATIC, AND EATING DISORDERS

One way to think about the definition of various types of eating disorders and disturbances is to use the terms "asymptomatic," "symptomatic," and "eating disordered" (Mintz, O'Halloran, Mulholland, & Schneider, 1997). Those who are asymptomatic do not have any symptoms of any eating disorder. Those who are symptomatic have symptoms of eating disorders, but do not meet the criteria for anorexia, bulimia, or eating disorder not otherwise specified; in the language of this book, they have eating disturbances. Finally, those who have anorexia nervosa, bulimia nervosa, or eating disorders not otherwise specified are considered to have an eating disorder.

THE CONTINUUM OF EATING DISTURBANCES

The eating disorders continuum is a way to think about the various kinds of eating problems. The continuum of eating disorders places normal eating at one end of the spectrum (asymptomatic), eating disorders at the opposite end, and eating disturbances at intermediate points. On the normal end of the continuum, people have normal eating behaviors whereas those on the eating disordered end have significant eating and body image problems. The groups between normal and eating disordered display some eating disordered behaviors such as dieting, binge eating, and various methods of purging. The phrase "continuum" was first used in regard to eating disorders in 1971 (Nylander), and it continues to be a useful way to explain eating disturbances and eating disorders.

BRIDGE—BUILDING THE RELATIONSHIP BETWEEN BODY IMAGE AND DISORDERED EATING GRAPH AND EXPLANATION

BRIDGE (Russell & Ryder, 2001a) is a graphical presentation of the relationship between body image attitudes and disordered eating behaviors that can provide a framework for connecting attitudes (such as body satisfaction or dissatisfaction) and behaviors (such as exercise, healthy eating, binge eating) on the continuum of eating disturbances and disorders. The BRIDGE concept is consistent with the continuum concept since it does not dichotomize between normal and abnormal behaviors and attitudes, but rather presents a continuum of attitudes and behaviors (see Figure 8.1). The developers of BRIDGE highlight the need to be concerned about a wide range of disordered eating behaviors and attitudes and emphasize the importance of discussing eating disorders in the context of a continuum (Russell & Ryder, 2001).

The graph shows how an eating disorder may develop when unhealthy attitudes and behaviors meet. The horizontal axis is a continuum of body image that ranges from healthy to unhealthy. It is the axis of attitudes and feelings about bodies; the healthiest attitudes are on the left near the intersect point on the graph. As you move to the right on the horizontal line, attitudes become increasingly unhealthy. At the extreme, a person may not see himself or herself accurately, seeing the body as larger than it is. The vertical axis ranges from healthy to unhealthy in terms of different behaviors. The healthiest behaviors start at the bottom of the graph near the intersect point and become less healthy as you go up the vertical line. Anorexia and bulimia are at the extreme of this axis.

The shaded areas on the graph represent the intersection of attitudes and behaviors. The "Body Awareness and Acceptance" ellipse holds the part of the horizontal and vertical axes associated with the healthiest attitudes and behaviors. People who fall into this area have healthy attitudes about their bodies and engage in healthy behaviors. They are asymptomatic. They accept their bodies and understand that the way they look is only one part of who they are. Primary prevention programs are designed to promote these healthy attitudes and behaviors are recommended for people who fit into this ellipse (Russell & Ryder, 2001a, 2001b).

The "Body Preoccupation" ellipse is much larger, and it encompasses restrictive dieting/overeating, binge eating, compulsive exercise, and disordered eating. The people who fall into this ellipse are overly concerned about their bodies and are engaging in behavior that is not healthy. This shaded area may include

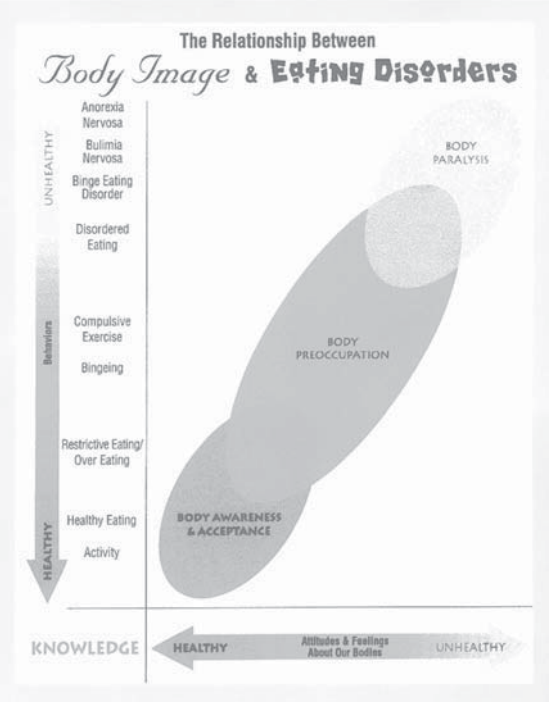

FIGURE 8.1. The Relationship between Body Image and Eating Disorders. Copyright 2001 from Shelly Russell and Sabine Ryder, "BRIDGE: A Tool for Parents and Professionals." Reproduced by permission of Taylor & Francis, Inc., http://www.routledge-ny.com

people who would benefit from secondary prevention or early intervention (such as psycho-education or counseling) to help them change their unhealthy attitudes and behaviors (Russell & Ryder, 2001a, 2001b). Some people in this area have eating disturbances.

The third ellipse is called "Body Paralysis" and is characterized by extremely disordered attitudes and behaviors. People in this ellipse are obsessed with their bodies to such an extent that it becomes the most important thing in life. This ellipse includes people who have anorexia or bulimia, or eating disorders not otherwise specified. These individuals need treatment or specialized services to help them recover from their eating disorders (Russell & Ryder, 2001a, 2001b).

A CLOSER LOOK AT THE EATING DISORDERS CONTINUUM—CROSS-SECTIONAL, RETROSPECTIVE, AND LONGITUDINAL RESEARCH

The continuum conceptualization provides a background for understanding how eating disturbances and disorders may develop and change as time passes. Individuals may progress from a less serious problem to a more severe one over time. Or, it is possible that some people may move from the more severe end of the continuum toward the normal end.

There are three kinds of research that can be used to study this issue: cross-sectional, retrospective, and longitudinal studies. Cross-sectional studies compare different groups of people along the continuum on eating attitudes and behaviors and other variables of interest. Retrospective studies are based on asking people who developed an eating disorder about events that happened to them before the beginning of their eating disorder. Longitudinal studies determine if there is a progression from one point on the continuum to another in the same individual as time passes. Different types of information can be obtained from studies of these sorts.

A Survey of the Cross-Sectional Research

Most of the cross-sectional studies were published in the late 1980s and early 1990s when the field was just beginning to explore the idea of the continuum of eating disorders. The older studies (i.e., Mintz & Betz, 1988; Scarano & Kalodner-Martin, 1994) are cross-sectional studies, since they involved collecting data from a large group, assigning the individuals to a group on the continuum based on their responses to a questionnaire, and then making comparisons between the groups on the continuum. Cross-sectional studies are a way to determine if there are differences between the groups on the continuum.

In one continuum from normal eating to bulimia nervosa (anorexia was not included in this work), six groups were used (Scarano & Kalodner-Martin, 1994). These include normal eaters, weight preoccupied, chronic dieters, purgers, subthreshold bulimia, and clinically diagnosed bulimia. In this continuum, the groups were defined as follows:

- Normal eaters do not binge eat, restrict eating, or use any method of purging.
- Weight preoccupied individuals express significant concern about body weight and shape, but they do not engage in any kind of binge eating, restricting, or purging.

- Chronic dieters engage in repeated dieting behavior at least once a week, including the use of diet pills, fasting, or overexercising.
- Purgers self-induce vomiting, fast, or use diuretics at least once a month.
- Individuals with subthreshold bulimia or bulimia are characterized by both binge eating and purging behaviors, but differ in the frequency of these behaviors; bulimia nervosa is defined by binge episodes at least eight times a month, while those with subthreshold bulimia do this less than eight times a month (Scarano & Kalodner-Martin, 1994).

In several studies based on this continuum with six groups, the groups differed from each other on a variety of eating attitudes and behaviors. For example, a measure of behaviors associated with bulimia showed a linear increase from normal eaters through those with bulimia. A linear increase means that with increasing levels of eating disturbances, there is also an increase in measures of eating related problems, such as binge eating or body image dissatisfaction (Mintz & Betz, 1988; Scarano, 1991; Scarano & Kalodner-Martin, 1994). Other findings that support the linear increase are (1) individuals who meet the criteria for bulimia were more weight preoccupied than those who meet the criteria for subthreshold bulimia, (2) purgers were more weight preoccupied than normal eaters and chronic dieters, and (3) body dissatisfaction increased from normal eaters through the group with bulimia in an incremental way. Similar patterns exist for thinking about appearance and food, feeling fat, and fearing becoming fat.

In another more recent cross-sectional study of the continuum, the three groups of asymptomatic, symptomatic, and eating disordered were used (Mintz, O'Halloran, Mulholland, & Schneider, 1997). A study of college students using this three-group continuum indicated that a continuum existed for the issues most closely associated with eating disorders, such as body dissatisfaction. Scores on a measure of body dissatisfaction increased from 37.9 to 75.9 to 76.2 for asymptomatic, symptomatic, and eating disordered groups respectively (Tylka & Subich, 1999). This kind of linear increase supports the idea that a continuum exists. However, Tylka and Subich also found that some variables did not differentiate between the three groups in this way. Some measures of psychological and behavioral variables (such as extraversion, openness to experience, agreeableness, and conscientiousness) did not distinguish the three groups in any meaningful way. These variables may be less relevant to the eating disorders continuum.

A Survey of the Retrospective Research

Shisslak and Crago (2001) reviewed the studies that used a retrospective model and summarized the findings by saying that people who develop eating

disorders are more likely than other psychiatric patients to have experienced greater parental pressure, high expectations or abuse, more health problems, childhood obesity, more familial criticism about their weight, shape, or eating habits, and a more negative self-evaluation. In contrast, the death of someone close, a loss of relationship with a friend or family member, and work or school problems were not factors that were reported more often by those who developed an eating disorder than those who had another psychiatric problem.

An example of retrospective research is provided by Fairburn, Welch, Doll, Davies, & O'Connor (1997). In this study, 102 people with bulimia were compared with 204 control group participants, and 102 psychiatric patients with disorders other than eating disorders. All participants were British females between 16 and 35 years, and they were matched for age and social class. The participants with bulimia reported exposure to twenty-nine risk factors, but only 4 of the 58 were at greater levels than those who developed another psychiatric problem. This led the researchers to conclude that risk factors for bulimia are similar to those factors that lead to the development of psychiatric problems in general.

There has been no study of the groups on the continuum using a retrospective approach, thus it is unclear if asymptomatic, symptomatic, and eating disordered groups would provide different data on the factors that preceded the development of eating problems. It is also worth noting that retrospective studies are subject to the bias of recall of stressful events. For example, some events that participants may report occurred before their eating problems began, may actually have developed at the same time, or even as a result of the eating disorder. In addition, individuals vary in their perception of what is stressful; that is, what creates a great deal of stress for one person may not be as stressful to another. Even in light of these limitations, retrospective research adds to our understanding of the differences between groups of people who develop a problem and those who do not.

A Survey of the Longitudinal Research

Longitudinal studies can be used to study how individuals change from less severe to more severe eating disturbances in the same individual over time (Shisslak & Crago, 2001). This type of study is based on collecting data from a large sample of individuals and following their development to see who develops eating problems and what precedes or is associated with the development of eating disorders. According to Shisslak and Crago, twenty-six studies have been published that fit into this category; the first one was published in 1989 (Attie & Brooks-Gunn). Since longitudinal studies take much more time to

complete (because you have to wait for people to get older and see how their attitudes and behavior change), and there can be problems with attrition (participants drop out of the study or cannot be reached to provide data), they are quite expensive to conduct.

Longitudinal research suggests that low self-esteem, weight concerns, dietary restraint, body dissatisfaction, depression, negative emotionality, early maturation and being overweight are risk factors for the development of eating disorders and disturbances (Shisslak & Crago, 2001). The four factors that are associated most strongly with the development of eating disorders are weight concerns, dietary restraint, body dissatisfaction, and early maturation, while the other factors are associated with the development of other psychiatric disorders as well. This is consistent with a two-track approach to the continuum that is discussed below.

An example of a longitudinal study of 800 children and their mothers is provided by Kotler, Cohen, Davies, Pine, & Walsh (2001). The procedures involved interviewing mothers and children between the ages of 1 and 10 (childhood), then again in adolescence (mean age 13.9), late adolescence (mean age 16.3), and early adulthood (mean age 22.1). It can be expected, at childhood, there were no children who met the criteria for anorexia or bulimia. In early adolescence, 1 (.2 percent) male and no females met the criteria for anorexia, and 7 (1.4 percent) females met the criteria for eating disorder not otherwise specified Type 1 (anorexia nervosa without the criteria of amenorrhea). There were 6 (1.2 percent) females and 1 male (.2 percent) with bulimia. In late adolescence, 4 (1 percent) males and no females met the criteria for anorexia, and 4 (1.1 percent) females met the criteria for eating disorder not otherwise specified as Type 1. There were 12 (3.2 percent) females and 2 (.5 percent) males with bulimia. In early adulthood, no males or females met the criteria for anorexia, and 2 (.5 percent) females met the criteria for eating disorder not otherwise specified as Type 1. There were 4 (1.1 percent) females and 4 (1.1 percent) males with bulimia. These data fit with the data that suggest there is a low prevalence of anorexia and bulimia.

Relevant to the notion of the continuum and understanding what factors may lead to the development of these eating disorders, Kotler et al. (2001) indicated that the adolescents who scored highest on a measure of symptoms of bulimia developed bulimia at a much higher rate than those with no symptoms when they were younger (7.9 percent more likely); having severe symptoms of anorexia or bulimia in early or late adolescence predicted severe symptoms of these disorders in young adulthood. In addition, certain childhood eating problems, such as conflicts over eating, struggles with meals, and unpleasant meals (as rated by mother) increased the risk for the later diagnosis of anorexia

nervosa. Additional research of this type that includes more psychological assessments may add considerably to our understanding of the risk factors for the development of eating disorders and help understand how eating disturbances develop into eating disorders.

A longitudinal study based on twenty-one female college students who were symptomatic of eating disorders during their college years was conducted to see what happened to their eating attitudes and behaviors after they graduated from college (Hesse-Biber, Marina, & Watts-Roy, 1999). In this interview-based study, it was noted that 11 women experienced reductions in their eating problems, whereas 10 other women remained at risk of developing an eating disorder. The group that reduced eating problems reported better interpersonal relationships and more adaptive means for coping with stress, whereas the group that continued to experience eating-related difficulties described feelings of isolation and discontent with relationships with family and friends and had less satisfying relationships with men.

The results of a longitudinal study involving a ten-year long follow-up of 509 women and 206 men who completed a questionnaire about eating attitudes and behaviors when they were in college has implications for the continuum of eating disturbance and the natural progression among the continuum as people mature and become adults (Heatherton, Mahamedi, Striepe, Field, & Keel, 1997). The first questionnaire was collected in 1982, and the follow-up was in 1992. The ten years after college are ones in which people generally settle down, get married, have children, and establish careers. The researchers wanted to know how their eating attitudes and behaviors would change during that time. A large percentage of the initial respondents returned the follow-up questionnaire (82 percent of the women and 76 percent of the men). During the ten years, the women gained an average of 4 pounds, while men gained 12 pounds.

The groups on the continuum used in this research included nondieters, dieters, problem dieters, and those with subclinical eating disorders and clinical eating disorders. The percentage of women classified as having any sort of eating problem (problem dieter, subclinical, or clinical) dropped from more than 40 percent in 1982 to just over 15 percent in 1992. Thus, it was found that 46 percent of the women moved to a lower category of eating disorder, while 41 percent stayed in the same category and 14 percent moved to a more disordered category. Overall, this study found that body dissatisfaction, chronic dieting, and eating disorder symptoms declined for women in the 10 years after college. Rates of eating disorders dropped by more than half and the prevalence of binge eating and purging declined as well. Maturing into adulthood seems to help women stop dieting and abnormal eating. Some participants wrote notes

to the researchers saying that dieting was much less important to them as they gained some distance from the college experience. So even though it may be normative for women to have some degree of problem eating while in college, it may also be normative for the problems to diminish as the person moves into adulthood. Unfortunately, some of the women continued to have eating related problems after college. About 1 person in 5 who met the clinical criteria for an eating disorder in college still met the criteria ten years later.

For men, the data suggest something quite different. Men gained more than ten pounds from 1982 to 1992, and they also reported increases in body weight concerns, desire to lose weight, and dieting behavior. These changes were associated with increases in attitudes consistent with eating problems. The data suggest that it was the men who were most concerned about being thin who actually got heavier in the ten years.

DOES THE CONTINUUM REALLY EXIST?

Whether eating disorders actually exist on a continuum has been the subject of a great deal of study. Based on many studies, it appears that there is a continuum of food and body image issues, but there may not be a continuum of the psychological aspects of eating disorders (Connors, 1996). There may be meaningful differences on psychological variables between those who have eating disorders and those who do not. In other words, there may be a continuum for some aspects of eating disorders, but not for others. The eating disorders continuum holds true for variables that are more closely related to eating and body image, rather than the psychological issues that may be associated with anorexia and bulimia.

The continuum of food and body image includes body dissatisfaction and negative body image, weight preoccupation, and dieting. The food and body image issues (which include intense concern with weight, appearance, and body shape) may be common among people who diet and those who have eating disorders. The second set of concerns refers to psychological issues such as problems with affect (mood), low self-esteem, and insecure relationships with parents. The second set of issues may be deeper and more complex and may be issues that affect those with the more serious eating disorders—not those who do not have a diagnosis of any eating disorder.

A summary of the state of the art of the continuum follows:

> The data suggest that the normative levels of body dissatisfaction and dieting so prevalent in the current sociocultural context may be differentiated from clinically significant eating disorders on the basis of emotional disturbance.

> Body dissatisfaction and dieting behaviors could be viewed as spanning a continuum from slight to very intense. Individuals may have mild to moderate levels without other life impairment. Women with more symptoms of eating disorders seem to have high levels of body dissatisfaction and disturbed eating attitudes and behaviors in conjunction with other psychological problems, including greater levels of depression, feelings of ineffectiveness, self-criticism, impulsivity, emotional reactivity, and life impairment. (Connors, 1996, pp. 289–290)
>
> This quote means that it may take eating and body image problems along with other psychological problems to lead to the development of eating disorders. Connors (1996) also indicates that when a person has both body dissatisfaction and certain psychological issues (such as depression or a high degree of self-criticism), an eating disorder may develop, but that when body dissatisfaction occurs without psychological issues, an outcome might be normative discontent dieting. The term normative discontent is a classic phrase because it is used so often in the literature. It was first used by Rodin, Silberstein, and Striegel-Moore in 1985 to describe the pervasiveness of women and their dissatisfaction with appearance. Authors continue to describe the problem differentiating between pathological concern associated with eating disorders and cultural norms of thinness. (Herzog & Delinsky, 2001)

THE CONTINUUM AND IMPLICATIONS FOR TREATMENT OF EATING DISTURBANCES

The two aspects of the continuum (food/body issues and psychological concerns) are consistent with a two-track approach for counseling clients who have eating disturbances and disorders (Garner, Vitousek, & Pike, 1997). The first component to treatment is concerned with weight and body image, while the second track involves the psychological and emotional disturbances. Track one issues refer to weight preoccupation, body image, and eating, including binge eating and methods of purging. Track two refers to the psychological issues such as self-esteem, anxiety, depression, and family issues.

These two tracks can be used to describe treatment for all groups on the eating disorders continuum. For example, people who fall into the category of weight preoccupied may benefit from track one interventions to target unhealthy attitudes toward weight and body image. If these attitudes are not modified, individuals are likely to begin to diet. Social pressures to be thin may be addressed in a media literacy kind of intervention for people who are in the weight-preoccupied group. The two tracks of treatment may also be applied to repeat or chronic dieters. Treatment for repeat dieters should include psychoeducation about the negative effects of chronic diets.

DO NORMAL EATERS NEED TREATMENT?

In the first article to address this topic, Polivy and Herman (1987) raised the idea that treatment may be necessary for "normal eaters"; they wrote about this in an article with the provocative title, "The Diagnosis and Treatment of Normal Eating." This highlights the fact that what is societally normal eating may be quite abnormal, depending on the definition of normal. Does normal mean normative, in that it is what most people are doing? If that is true, then what is normal may include concern with weight and use of repeated diets. People who are of normal weight may feel as if they are overweight. Chronic dieting is normal in some groups. The important point of this article is that just because behavior is normal does not mean that it is okay; in fact, chronic dieting is not healthy. Dieting is the single most common factor in developing an eating disorder (Ghaderi, 2001). Many professionals in the eating disorders field have addressed the problems associated with dieting as a risk factor for the development of eating disorders.

Since dieting is a common eating style, it may be that this seems like normal eating, though it is quite a bit like disordered eating and therefore it may require treatment. Physiologically normal eating requires eating in response to hunger, which means that people must be able to accurately determine if they are truly hungry. Normal eating, simply put, is eating when you are hungry and stopping when you are full and satisfied. To learn (or relearn) to respond to hunger and satiety (being full), one must perceive them accurately. Dieters learn to ignore these normal cues; as part of their diets, they learn not to eat when hungry and to stop eating before they are full. Ignoring hunger and satiety cues creates a kind of distorted regulation of eating. Dieters fear that when they eat naturally the result will be uncontrollable binge eating and weight gain. However, uninhibited eating does not lead to overeating and binge eating. Sometimes dieters who stop dieting actually lose weight by stopping their diets. The reversion to physiologically (rather than cognitively) controlled eating ends obsessions with dieting and allows dieters to recognize that normal eating is not a threat to well-being. In *Full Lives*, Hutchinson (1993) said, "It was dieting, and not some intrinsic neurosis, that made me into a compulsive overeater. Therefore, it was dieting, not compulsive overeating, from which I really needed to recover" (p. 97).

Dieting behaviors are associated with a drive for thinness and body dissatisfaction. Reduction in dietary fat is one way in which people may attempt to lose weight. This is a strategy that is viewed as positive; there is an inherent assumption that fat avoidance is desirable and consistent with improved health. However, fat avoidance behaviors have also been associated with high levels

of eating pathology and psychosocial problems (Liebman, Cameron, Carson, Brown, & Meyer, 2001).

DOES DIETING LEAD TO BINGE EATING?

Polivy and Herman (1985) were among the first to study the relationship between dieting and binge eating. Their initial work provided a great deal of evidence that suggests that dieting causes binge eating. Recently, Stice (2001) summarized a great deal of research on the temporal relationship between dieting and binge eating, indicating that in prospective longitudinal studies dieting does seem to predate bulimic behaviors. This may be due to the "abstinence-violation effect," which means that although someone may create a set of rules about eating and restrict intake of food, when these rules are broken, it may lead to overeating or binge eating. The abstinence-violation effect means going on a diet, breaking a rule of the diet, and then binge eating.

Several experiments in which subjects were put on diets and then developed binge eating behaviors provide additional support for the idea that dieting causes binge eating. The famous Keys study (of starvation in male volunteers) showed that when people are placed on a restrictive diet, they "exhibited a persistent tendency to binge, gorging at meals to the limit of their physical capacity" (Polivy & Herman, 1985, p. 195). Successful dieting demands that physiological controls, which by themselves are conducive to a "desirable" weight level, be replaced with cognitive controls designed specifically to achieve a lower weight in line with the dieter's personal aspirations (Polivy & Herman, 1985, p. 198).

Why does dieting precede binge eating? A physiological reason is that binge eating might be the body's attempt to restore weight to a more appropriate level. This relates to the idea of set point, which holds that a person has a range of weight that is determined for them biologically. When a person gets much lower than the set point, the body may respond by developing a binge eating style. However, there may be other reasons to explain this relationship. As indicated earlier, dieting is a cognitive (thinking) kind of activity. Cognitive factors may be more important determinants of intake on a given occasion than are physical factors. In the final comment of the article, Polivy and Herman (1985) suggest that dieting is the disorder that we should be attempting to cure.

EATING DISORDER NOT OTHERWISE SPECIFIED

Serious eating problems exist in individuals who do not meet the criteria of anorexia or bulimia. In addition to understanding anorexia and bulimia, it is important to attend to those eating disorders that are assigned to the category

eating disorder not otherwise specified. This is a poorly defined large "catch-all" category (Striegel-Moore & Smolak, 2001) with 25 percent–60 percent of people presenting for treatment fitting in the "not otherwise specified" group (Andersen, Bowers, & Watson, 2001). Individuals with an eating disorder not otherwise specified can be quite distressed and need attention to the eating issues and associated psychological concerns.

As you can see, the types are defined by "falling just short of full criteria" (Herzog & Delinsky, 2001, p. 36). Type One includes females who meet all the criteria for anorexia except the individual has regular menses. Type Two is for people who meet all criteria for anorexia except that, despite significant weight loss, the individual's weight is in the normal range. Type Three includes people who meet all criteria for bulimia except that binge eating and purging or other ways to control weight gain occur at a frequency of less than twice a week or for less than three months. In Type Four, individuals of normal weight vomit after eating a small amount of food or use other inappropriate compensatory behavior. Type Five is for people who repeatedly chew and spit out food (they do not swallow it). Type Six is called binge eating disorder and is described below.

People may move from one type of eating disorder not otherwise specified to another, and from an eating disorder not otherwise specified to anorexia or bulimia. For example, in Marya Hornbacher's (1998) book *Wasted*, about her personal struggle with eating, she wrote, "I became bulimic at the age of nine, anorexic at the age of fifteen. I couldn't decide between the two and veered back and forth from one to the other until I was twenty, and now, at twenty-three, I am an interesting creature, an eating disorder not otherwise specified" (p. 2).

Eating disorder not otherwise specified is a category that concerns researchers and clinicians for several reasons. First, the large number of people who are diagnosed with eating disorders not otherwise specified makes one wonder if the criteria for anorexia and bulimia may be too restrictive. Second, insurance companies may restrict coverage for people with this diagnosis, assuming that it is less serious than anorexia or bulimia (Andersen et al., 2001). Third, some clinicians who treat eating disorders have expressed uncertainty of the methods that they should use with eating disorders not otherwise specified since the research is based on anorexia and bulimia, but rarely mentions eating disorders not otherwise specified. In fact, the Practice Guidelines for the Treatment of Patients with Eating Disorders does not make specific treatment recommendations for people with eating disorders not otherwise specified (APA, 2000b).

If there were changes that broadened the diagnostic criteria for anorexia and bulimia, there could be a significant reduction in the number of people assigned to an eating disorder not otherwise specified. Redefining anorexia and bulimia would result in an increase in the cases of both of these disorders. For

example, one criterion for the diagnosis for anorexia requires an absence of menstruation for three months. Forty-seven percent of a group of eating disorders not otherwise specified fit into this category, thus with a revision in the criteria for anorexia, they would no longer be considered eating disorders not otherwise specified. When adjustments to the criteria for anorexia and bulimia were made, only 18 percent of people with a diagnosis of eating disorder not otherwise specified remained in this category. Since the majority of influence of reducing eating disorders not otherwise specified is due to changes in the criteria for anorexia nervosa, this is described in the chapter on anorexia.

INCIDENCE AND PREVALENCE OF EATING DISTURBANCES AND DISORDERS

Epidemiology concerns the number of people who are diagnosed with a specific disorder. Here, we are concerned with the number of people who have eating disorders or disturbances of various types. Both incidence and prevalence data are available to provide estimates of the number of people who have eating disorders or disturbances. Incidence is defined as the frequency of the occurrence of a disorder; it may refer to the number of new cases of a disorder. Prevalence is defined as the number of cases of a disorder in a specific population at a specific point in time.

Eating disturbances and disorders occur in children, adolescents, adults, and the elderly, but the majority of the research has focused on people between the ages of twelve and twenty-two. In the sections s that follow, data are presented to demonstrate the number of children, adolescents, and young adults with eating disturbances. In chapters 3 and 4, data are provided on the frequency of occurrence of anorexia and bulimia. Briefly, anorexia has a lifetime prevalence of .05 percent and the prevalence reported for bulimia ranges from 1 percent to 3 percent (APA, 2000a). For bulimia nervosa, the prevalence rate is 1 to 3 percent (APA, 2000a).

Eating disorders not otherwise specified occurs in 4 percent to 6 percent of the general population (Herzog & Delinsky, 2001), thus, the prevalence of eating disorders not otherwise specified is approximately twice that of anorexia and bulimia. In addition, eating disorder not otherwise specified is the appropriate diagnosis for more than 50 percent of patients with eating disorders who present for treatment (APA, 2000b). Major epidemiological studies of eating disorders have shown that by adopting subthreshold criteria, defined as meeting all but one of the diagnostic criteria for anorexia or bulimia, the number of cases of anorexia or bulimia would more than double (Garfinkel, 1996; Garfinkel, Lin, Goering, Spegg, Goldbloom, Kennedy, Kaplan, & Woodside,

1995). Those who meet all but one of the diagnostic criteria for bulimia do not differ from those who have been diagnosed with bulimia in terms of demographic characteristics, psychiatric comorbidity, family history, or early childhood experiences (Garfinkel, 1996; Garfinkel et al., 1995). Since those with an eating disorder not otherwise specified may engage in all the same disturbed eating behaviors as those with the diagnosis of anorexia or bulimia, it is an important group that requires attention.

As for eating disturbances, studies suggest that unhealthy eating and weight-related behaviors and body image dissatisfaction exists in vast numbers of young females, as well as college students and adults. In addition, there is also increasing emphasis on eating disturbances in males (Andersen, Cohn, & Holbrook, 2000).

COLLEGE STUDENTS

College students have been the subjects in a great deal of prevalence research. For example, a group of researchers using the same instrument in four different studies of the prevalence of eating disorders in college students revealed that the prevalence of bulimia ranged from 0 percent to 3 percent, eating disorder not otherwise specified ranged from 2 percent to 5 percent and symptomatic eating issues ranged from 19 percent to 23 percent (Mintz, O'Halloran, Mulholland, & Schneider, 1997; Mulholland & Mintz, 2001). In a prevalence study of African American women enrolled in a predominately Caucasian university, 2 percent met criteria for eating disorders, 23 percent were symptomatic, and 75 percent were asymptomatic (Mulholland & Mintz, 2001). In a sample of 330 female undergraduates enrolled in psychology classes at a public university, Tripp and Petrie (2001) reported 7.6 percent met criteria for an eating disorder, 72.7 percent were symptomatic, and only 19.7 percent were asymptomatic. Likewise, using a different measure, Franko and Omori (1999) reported that in their sample of 207 female students enrolled in psychology classes, 2.4 percent fell into a group they called "probable bulimic," 6.7 percent were dieters, 23 percent were called intense dieters, 17 percent were termed casual dieters, and 51 percent were not dieters.

CHILDREN AND ADOLESCENTS

In the introduction to a book entitled *Body Image, Eating Disorders and Obesity in Youth* published in 2001, Thompson and Smolak reviewed the most recent prevalence data for children and adolescents. Three recent large surveys

of children have been conducted to assess the number of children and adolescents who have symptoms of eating disturbances.

Dieting prevalence was studied by the Heart, Lung and Blood Institute (Schreiber, Robins, Striegel-Moore, Obarzanek, Morrison, & Wright, 1996) in a study of over 2,000 black and white 9- and 10-year-old girls.

Among the 9-year-olds, 42 percent of the black girls and 37 percent of the white girls reported that they were trying to lose weight. For the 10 year olds, the corresponding percentages are 44 percent and 37 percent.

Another study that is cited often because of the large number of participants and the data available from adolescents of varying ethnic backgrounds yielded higher percentages of adolescents who report weight loss attempts of varying kinds (Serdula, Collins, Williamson, Anda, Pamuk, & Byers, 1993). Of Caucasian female adolescents, 47.4 percent were trying to lose weight, while 30.4 percent of African American and 39.1 percent of Hispanic American adolescents were also trying to lose weight. For males, the percentages were Caucasian 16.2 percent, African American 10 percent, and Hispanic 16.7 percent. Interestingly, this survey also assessed desire to gain weight and found that 26 percent of boys wanted to gain weight, along with 6.6 percent of the girls.

Field and colleagues (1999) studied a very large sample of more than 16,000 9- to 14-year-old boys and girls (93 percent of the sample was white) and found that the 44 percent of the older girls were trying to lose weight. However, they also found that 20 percent of the 9-year-old girls were trying to lose weight. Girls reported that they exercised to lose weight rather than dieted. Of the boys in this study, 17 percent of the 9year-olds and 19 percent of the 14-year-olds were trying to lose weight.

These studies suggest that there are a large number of children and adolescents who are dissatisfied with their bodies and attempting to lose (or, in some cases, gain) weight. It is important to remember that these high rates of dieting may not indicate that these individuals have eating disorders. Rather, the data suggest that many adolescents and some children have weight concerns that may be associated with unhealthy behaviors. Some of these individuals may develop symptoms of eating disorders.

After reviewing the information on prevalence on the various types of eating disorders, one might ask the question: Why is it so difficult to provide precise figures that represent the prevalence of these problems? It is impossible to come up with exact percentages for several reasons. First of all, the current percentages provided by the American Psychiatric Association are based on documented, or reported, cases of eating disorders. These numbers represent people who are receiving treatment for their eating disorder. It is very likely that many

cases go unreported or undiagnosed, which could make this number inaccurate. Second, oftentimes, young women are asked to fill out questionnaires to indicate whether they meet criteria for an eating disorder. As with any kind of self-report questionnaire, this may not be the most accurate method of determining who has an eating disorder. For example, people who have an eating disorder may be uncomfortable filling out questionnaires or may be ashamed to answer the questions honestly. Another reason is that the numbers may not accurately reflect the prevalence of the disorder in different populations. Whenever we read about percentages or prevalence rates, we should ask the question: What group did they use to get this information? For example, if we know that a group of athletes were used, with an equal percentage of African American, Hispanic, Native American, Asian American, and white females, we may have a pretty good idea that the results represent athletes from various racial backgrounds. If, on the other hand, a group of white swimmers were used, we can say that those numbers represent that population only. Some groups may be more at-risk than other groups. For example, sorority members and athletes may be more prone to develop eating problems than other populations. For this reason, we must always be aware of the group upon which results are based.

BINGE EATING DISORDER

Binge eating disorder, in the eating disorder not otherwise specified category, involves recurrent episodes of binge eating in the absence of regular use of purging, fasting, or excessive exercise, which are also characteristic of bulimia (see criteria). Binges are characterized by some of the following: rapid eating, eating until uncomfortably full, eating when not hungry, eating alone to avoid embarrassment about how much food is eaten, and feeling disgusted, depressed, or guilty when overeating. People with binge eating disorder are concerned about their binge eating, including concern about the long-term effects of binge eating on the body. In addition, in order to have binge eating disorder, the binges must occur an average of two times a week for at least six months. Finally, binge eating disorder is not diagnosed when the person meets the criteria for anorexia or bulimia.

Binge eating disorder is also more prevalent than anorexia or bulimia. The overall prevalence of binge eating disorder taken from weight-control programs is 15 percent to 50 percent (with a mean of 30 percent; APA, 2000a). Less is known about the prevalence of this disorder in the general population. In samples taken from the general community, the prevalence of binge eating disorder ranged from .7 percent to 4 percent (APA, 2000a), though some researchers believe that the number is much greater. Some researchers say that there is no

doubt that binge eating will be increasingly recognized as a clinical problem and will be the object of additional research.

There has been more attention to binge eating disorder than any of the other types of eating disorder not otherwise specified. Although binge eating disorder is not common in adolescents or college students, a few paragraphs are included to define and describe this eating disorder. Binge eating disorder may be associated with depression and anxiety (APA, 2000a). It appears that binge eating disorder often begins following a significant weight loss from dieting. Some people report that they feel numb or spaced out by the binge episodes. Binge eating disorder is associated with obesity; this makes sense when you remember that this is a disorder of binge eating without any kind of purging or other way to compensate for the calories consumed in the binges. Many people with binge eating disorder have been struggling with weight issues for many years and have repeatedly dieted and experienced failure in their ability to lose weight and keep the weight off. Females are 1.5 times as likely to have binge eating disorder than males but note that the female/male ratio in binge eating disorder is much closer to even than in either anorexia or bulimia. The onset of binge eating disorder occurs more frequently in adults than in adolescents (APA, 2000a). Binge eating disorder appears to be a chronic kind of disorder, which means that it recurs in the lifetime.

Case of Binge Eating Disorder

Mr. Cohen is a thirty-seven-year-old man who weighed 272 pounds at 5 feet 9 inches tall. He sought therapy for weight loss related to a job promotion. Mr. Cohen indicated that he had gained 60 pounds in a year and he "ate all the time." He explained that he dieted and could lose weight in the past, but now he could not generate the willpower. Mr. Cohen thought that having someone weigh him each week would help him to start to lose some weight. Mr. Cohen had some interesting things to say about guilt. "My guilt drives me here, but why do I feel so guilty? Why is it so out of proportion to what I have done? It is not that terrible to overeat and yet I feel it is." Through counseling, it was difficult to discover what triggered Mr. Cohen's overeating. It seemed as though he ate when he was frustrated and also ate when he felt like he had made a significant achievement. He did manage to lose some weight and began to feel better. The weekly weigh-in and attention in psychotherapy may have been helping. But, after New Year's Day, Mr. Cohen reported a food binge—after he cashed his paycheck, he kept $100 and "everything just seemed to go blank . . . all of my good intentions just seemed to fade away I . . . just said 'what the hell' and started eating and what I did then was an absolute sin." He ate a

cake, several pieces of pie, and several boxes of cookies, which he ate while he drove his car around town. He ate quickly, in a kind of frenzy. Then he visited a series of restaurants, eating a little bit in each. When he described this binge, he said that he didn't enjoy it but that he couldn't stop it. He said that a part of him just blacked out (Stunkard, 1993, pp. 18–21).

These food binges described in this case were part of Mr. Cohen's food problems. They also became a part of the history of binge eating disorder, since the therapist that he saw was Dr. Albert Stunkard, who is now well known for his work in binge eating disorder.

SUMMARY

The eating disorders continuum is a way to think about the various kinds of eating problems. The continuum of eating disorders and disturbances places normal eating at one end of the spectrum (asymptomatic), eating disorders at the opposite end, and eating disturbances at intermediate points. The continuum continues to be a useful way to explain the difference between eating disturbances and eating disorders and how people may move from eating disturbances to disorders. BRIDGE (Building the Relationship between Body Image and Disordered Eating Graph and Explanation) is a graphical presentation that links attitudes and behaviors on the continuum of eating disturbances and disorders. In addition to understanding these well-known eating disorders, it is important to attend to those eating disorders that are assigned to the category called eating disorder not otherwise specified. Anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified (including binge eating disorder) are all disorders of eating.

DSM-IV-TR Criteria for Eating Disorder Not Otherwise Specified

The Eating Disorder Not Otherwise Specified category is for disorders of eating that do not meet the criteria for any specific eating disorder. Examples include:

1. For females, all of the criteria for anorexia nervosa are met except that the individual has regular menses.
2. All of the criteria for anorexia nervosa are met except that, despite significant weight loss, the individual's weight is in the normal range.
3. All of the criteria for bulimia nervosa are met except that the binge eating and inappropriate compensatory mechanisms occur at a frequency of less than twice a week or for a duration of less than 3 months.

4. The regular use of and inappropriate compensatory behavior by an individual of normal body weight after eating small amounts of food (e.g., self-induced vomiting after the consumption of two cookies).
5. Repeatedly chewing and spitting out, but not swallowing large amounts of food.
6. Binge eating disorder: recurrent episodes of binge eating in the absence of the regular use of and inappropriate compensatory behaviors characteristic of bulimia nervosa. (APA, 2000, p. 594 Reprinted with permission from the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision.* Copyright 2000 American Psychiatric Association.)

DSM-IV-TR Criteria for Binge Eating Disorder

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

   (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 during a similar period of time and under similar circumstances
   (2) a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)

B. The binge-eating episodes are associated with three (or more) of the following:

   (1) eating much more rapidly than normal
   (2) eating until feeling uncomfortably full
   (3) eating large amounts of food when not feeling physically 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 is present.
D. The binge eating occurs, on average, at least 2 days a week for 6 months.
E. The binge eating is not associated with regular use of inappropriate compensatory behavior (e.g., purging, fasting, excessive exercise) and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa. (APA, 2000, p. 787 Reprinted with permission from the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision.* Copyright 2000 American Psychiatric Association.)

NOTE

1. From *Too Fat or Too Thin?* Cynthia R. Kalodner. Copyright 2003 by Cynthia R. Kalodner. Reproduced with permission of Greenwood Publishing Group, Inc., Westport, CT.

REFERENCES

American Psychiatric Association (APA). (2000a). *Diagnostic and statistical manual of mental disorders* (4th ed.—TR.). Washington, DC: Author.

American Psychiatric Association (APA). (2000b). Practice guidelines for the treatment of patients with eating disorders (revision). *American Journal of Psychiatry, 157* (1), 1–39.

Andersen, A. E., Bowers, W. A., & Watson, T. (2001). A slimming program for eating disorders not otherwise specified. *The Psychiatric Clinics of North America, 24*(2), 271–280.

Andersen, A., Cohn, L., & Holbrook, T. (2000). *Making weight: Men's conflicts with food, weight, shape, and appearance.* Carlsbad, CA: Gurze.

Attie, I., & Brooks-Gunn, J. (1989). The development of eating problems in adolescent girls: A longitudinal study. *Developmental Psychopathology, 25,* 70–79.

Connors, M. E. (1996). Developmental vulnerabilities for eating disorders. In L. Smolak, M. P. Levine, & R. H. Striegel-Moore (Eds.), *The developmental psychopathology of eating disorders* (pp. 285–310). Mahwah, NJ: Lawrence Erlbaum.

Fairburn, C. G., Welch, S. L., Doll, H. A., Davies, B. A., & O'Connor, M. E. (1997). Risk factors for bulimia nervosa: A community-based case-control study. *Archives of General Psychiatry, 54,* 509–517.

Field, A., Camargo, C, Taylor, C. B., Berkey, C., Frazier, L., Gillman, M., & Colditz, G. (1999). Overweight, weight concerns, and bulimic behaviors among girls and boys. *Journal of the American Academy of Adolescent Psychiatry, 38,* 754–760.

Franko, D. L., & Omori, M. (1999). Subclinical eating disorders in adolescent women: A test of the continuity hypothesis and its psychological correlates. *Journal of Adolescence, 22,* 389–396.

Garfinkel, P. E. (1996). Should amenorrhoea be necessary for the diagnosis of anorexia nervosa? *British Journal of Psychiatry, 152,* 1052–1058.

Garfinkel, P. E., Lin, E., Goering, P., Spegg, C., Goldbloom, D. S., Kennedy, S., Kaplan, A. S., & Woodside, D. B. (1995). Bulimia nervosa in a Canadian community sample: Prevalence and comparison of subgroups. *American Journal of Psychiatry, 152*(7), 1052–1058.

Garner, D. M., Vitousek, K. M., & Pike, K. M. (1997). Cognitive-behavioral therapy for anorexia nervosa. In D. M. Garner & P. E. Garfinkel (Eds.), *Handbook of treatment for eating disorders* (2nd ed., pp. 94–144). New York: Guilford Press.

Ghaderi, A. (2001). Review of risk factors for eating disorders: Implications for primary prevention and cognitive behavioural therapy. *Scandinavian Journal of Behaviour Therapy, 30,* 57–74.

Heatherton, T. F., Mahamedi, F., Striepe, M., Field, A. E., & Keel, P. (1997). A 10-year longitudinal study of body weight, dieting, and eating disorder symptoms. *Journal of Abnormal Psychology, 106*(1), 117–125.

Herzog, D. B., & Delinsky, S. S. (2001). Classification of eating disorders. In R. H. Striegel-Moore & L. Smolak (Eds.), *Eating disorders: Innovative directions in*

research and practice (pp. 31–50). Washington, DC: American Psychological Association

Hesse-Biber, S., Marino, M, & Watts-Roy, D. (1999). A longitudinal study of eating disorders among college women: Factors that influence recovery. *Gender & Society, 13*, 385–408.

Hornbacher, M. (1998). *Wasted: A memoir of anorexia and bulimia*. New York: Harper Collins.

Hutchinson, M. G. (1993). To be recovered and fat. In L. Hall (Ed.), *Full lives: Women who have freed themselves from food and weight obsession* (pp. 95–106). Carlsbad, CA: Gurze Books.

Kotler, L. A., Cohen, P., Davies, M, Pine, D. S., & Walsh, B. T. (2001). Longitudinal relationships between childhood, adolescent, and adult eating disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*, 1434–1440.

Liebman, M., Cameron, B. A., Carson, D. K., Brown, D. M., & Meyer, S. S. (2001). Dietary fat reduction behaviors in college students: Relationships to dieting status, gender, and key psychosocial variables. *Appetite, 36*, 51–56.

Mintz, L. B., & Betz, N. E. (1988). Prevalence and correlates of eating disordered behaviors among undergraduate women. *Journal of Counseling Psychology, 35*(4), 463–471.

Mintz, L. B., O'Halloran, M. S., Mulholland, A. M., & Schneider, P. A. (1997). The questionnaire for eating disorder diagnoses: Reliability and validity of operationalizing *DSM-IV* criteria into a self-report format. *Journal of Counseling Psychology, 44*, 63–79.

Mulholland, A. M., & Mintz, L. B. (2001). Prevalence of eating disorders among African American women. *Journal of Counseling Psychology, 48*, 111–116.

Nylander, J. (1971). The feeling of being fat and dieting in a school population: Epidemiologic interview investigation. *Acta Sociomedica Scandinavica, 3*, 17–26.

Polivy, J., & Herman, C. P. (1985). Dieting and binging: A causal analysis. *American Psychologist, 40*(2), 193–201.

Polivy, J., & Herman, C. P. (1987). Diagnosis and treatment of normal eating. *Journal of Consulting and Clinical Psychology, 55*(5), 635–644.

Radcliffe, R. R. (1993). Hunger for more. In L. Hall (Ed.), *Full lives: Women who have freed themselves from food and weight obsession* (pp. 132–143), Carlsbad, CA: Gurze Books.

Rodin, J., Silberstein, L. R., & Striegel-Moore, R. H. (1985). Women and weight: A normative discontent. In T. B. Sonderegger (Ed.), *Psychology and Gender: Nebraska Symposium on Motivation, 1984* (pp. 267–307). Lincoln: University of Nebraska Press.

Russell, S., & Ryder, S. (2001a). BRIDGE (Building the Relationship Between Body Image and Disordered Eating Graph and Explanation): A tool for parents and professionals. *Eating Disorders: The Journal of Treatment and Prevention, 9*, 1–14.

Russell, S., & Ryder, S. (2001b). BRIDGE 2 (Building the Relationship Between Body Image and Disordered Eating Graph and Explanation): Interventions and transitions. *Eating Disorders: The Journal of Treatment and Prevention, 9,* 15–27.

Scarano, G. M. (1991). Self-worth determinations and self-efficacy expectations: Their relations to eating disorders among college women. Unpublished master's thesis, The University of Akron, Ohio.

Scarano, G. M., & Kalodner-Martin, C. R. (1994). A description of the continuum of eating disorders: Implications for intervention and research. *Journal of Counseling and Development, 72,* 356–361.

Schreiber, G. B., Robins, M., Striegel-Moore, R., Obarzanek, E., Morrison, J. A., & Wright, D. J. (1996). Weight modification efforts reported by Black and White preadolescent girls: National Heart, Lung, and Blood Institute Growth and Health Study, *Pediatrics, 98,* 63–70.

Serdula, M. K., Collins, M. E., Williamson, D. F., Anda, R. F., Pamuk, E. & Byers, T. E. (1993). Weight control practices of U.S. adolescents and adults. *Annals of Internal Medicine, 119,* 667–671.

Shisslak, C. M., & Crago, M. (2001). Risk and protective factors in the development of eating disorders. In J. K. Thompson & L. Smolak (Eds.), *Body image, eating disorders, and obesity in youth: Assessment, prevention and treatment* (pp. 103–125). Washington, DC: APA.

Stice, E. (2001). Risk factors for eating pathology: Recent advances and future directions. In R. H. Striegel-Moore & L. Smolak (Eds.), *Eating disorders: Innovative directions in research and practice* (pp. 51–74). Washington, DC: American Psychological Association.

Striegel-Moore, R. H., & Smolak, L. (2001). Introduction. In R. H. Striegel-Moore & L. Smolak (Eds.), *Eating disorders: Innovative directions in research and practice* (pp. 3–7). Washington, DC: American Psychological Association.

Stunkard, A. J. (1993). A history of binge eating. In C. G. Fairburn & G. T. Wilson (Eds.), *Binge Eating: Nature, assessment and treatment* (pp. 15–34). New York: Guilford.

Thompson, J. K. & Smolak, L. (2001). Introduction: Body image, eating disorders and obesity in youth—The future is now. In J. K. Thompson & L. Smolak (Eds.), *Body image, eating disorders and obesity in youth: Assessment, prevention, and treatment* (pp. 1–18). Washington, DC: American Psychological Association.

Tripp, M. M., & Petrie, T. A. (2001). Sexual abuse and eating disorders: A test of a conceptual model. *Sex Roles, 44,* 17–32.

Tylka, T. L., & Subich, L. M. (1999). Exploring the construct validity of the eating disorder continuum. *Journal of Counseling Psychology, 46*(2), 268–276.

Part III

PORNOGRAPHY, INTERNET, GAMING, AND GAMBLING

This page intentionally left blank

Chapter **9**

Addiction to Pornography: Its Psychological and Behavioral Implications[1]

Robert W. Kubey, PhD

In addition to the Internet and video games another form of media content often claimed to be addictive is pornography. Nonprint pornography has experienced an enormous growth in recent decades, and the opportunities for serious habits involving these materials have clearly increased. Where once only a small minority of interested parties could afford to buy or rent films, or venture into public theaters of dubious quality and atmosphere, for nearly three decades there has been an explosion in the availability of pornography. Here, the use of the word "explosion" is hardly hyperbolic. This is now a huge, multi-billion-dollar growth industry, and even a five-year old child staying with his parents in a hotel room can see signs that it exists from the ads sitting atop the TV, unless a vigilant parent puts them in a drawer the minute the family checks into the room.

With the advent of interactive pornography and live, visual, sexual chat rooms and live video sites, the debate over the value or harm of pornography has heated up over and over again in the last decades, yet it is questionable whether the research literature has been able to keep pace. When we get to the quality of research, the issue becomes more problematic, as at least in the opinion of this observer, some researchers appear to have found it difficult to steer clear of the political controversies surrounding this highly volatile topic.

It can also be very hard to obtain good, unbiased, and honest data from people who use pornography, who potentially have a problem with it, or who have committed sex crimes. Sexuality is always a challenging area for researchers, and clearly determining whether someone is addicted to pornography may not

be so simple. As I have done in applying the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV–R)* to understanding so-called television addiction (see chapter 2 in this volume), readers might wish to consult Irons and Schneider (1996), who have adapted the *DSM* for the analysis of sexual addictions, though they have little to say about pornography.

The federal government sometimes funds studies in order to inform lawmakers and set policy on pornography, but going back at least to the Nixon administration, the government or party in power, at least in the U.S. situation, sometimes appears to wants to see certain results emphasized in research or official government studies, and in some instances certain findings that disagree with administration policy may be disowned or eschewed and, some believe, even suppressed. This politicization of media effects issues has been evident for an even longer period in the debate over the impact of violence in film and television, and in many of the same ways.

That said, I will review some of the alleged effects of pornography and the ways in which an addiction to pornography may develop. This chapter will focus as well on concerns that I believe are raised by the delivery of new media forms and interactive erotica.

As with other media effects debates, it is very difficult to disentangle cause from effect. Still, a number of researchers and clinicians report both negative effects *and* evidence for dependence, or in their words, *addiction*, with regard to the use of pornography (Oddone-Paolucci, Genuis, & Violato, 2000; Zillmann, 2004). As will be seen, some of the negative effects are thought to be intertwined with pornography addiction. Those interested in a dissenting research view, one that holds that pornography might be a much more benign influence in people's lives, might consult Daniel Linz's (2004) work.

Now to some of the claims. One claim is that large private pornography collections are often found by authorities in the residences of persons arrested for sexual crimes (Cline, 1994; Reed, 1994), especially pedophiles (Lanning & Burgess, 1989). There is also evidence indicating that some rapists and child molesters use sexually explicit materials both before and during sexual assaults (Marshall, 1988). At a minimum, it does seem that there is a relationship between the frequent use of pornography and problematic sexual disorders for some individuals. Whether the pornography is merely symptomatic of the disorder, or plays a causal role, is much more difficult to establish.

Still, for some, there is little doubt that both negative effects and pornography addiction do indeed occur. Reed (1994), a practicing psychiatrist, is explicit in his presentation of specific criteria that he believes would constitute an addiction to pornography. He notes that the *DSM* itself recognizes that

many *paraphilias* (compulsive sexual deviances) frequently involve the use and collection of pornography. Reed lists 13 paraphilias and the ways in which they are related to the use of pornography.

Cline (1994), a clinical psychologist who has treated hundreds of people with sexual disorders, describes a four-step process in the involvement of his patients with pornography. First described is an "addiction effect," in which the person comes back repeatedly for more material because it provides "a very powerful sexual stimulant or aphrodisiac effect followed by sexual release most often through masturbation" (p. 233).

Cline goes on to describe an "escalation effect," in which there is an "increasing need for more of the stimulant to get the same effect" obtained initially (p. 233). Third, he observes "desensitization," in which things that might have once seemed shocking become less so and are thereby legitimized. Fourth, Cline claims that there is an "increasing tendency to act out sexually the behaviors viewed in the pornography" (p. 234).

A number of psychological and physiological mechanisms have been posited to explain how pornography addiction might develop. Among the most common is sexual gratification as a powerful reinforcer (Lyons, Anderson, & Larson, 1994). This is the "addiction effect" described by Cline, in which learning is made all the more powerful by virtue of the sexual release that attends pornography's use. Here, Cline draws on McGaugh's (1983) memory research suggesting that experiences that co-occur with high emotional and physical arousal may be better remembered. Reed (1994) suggests the possibility that some such learning might be occurring on the biological as well as the psychological level when he points out that "the neurotransmitters that are activated by pornography use may trigger similar neural pathways as cocaine or heroin" (p. 265).

Of note is a trade book by Dodge (2007), arguing that the neuroplasticity of the brain can enable an addiction to Internet- or other-mediated pornography that can leave the user with a typical pattern of less enjoyment over time, as a result of which the user looks for new forms of excitement (pp. 102–112).

In my chapter on television addiction, I applied an operant conditioning approach to the role relaxation plays in the development of the television viewing habit. It certainly makes sense that the pleasure accompanying orgasm may increase the potential for a habit to develop for some users of pornography, especially those who have few other outlets for sexual gratification. The early literature on sexual behavior points to strong associations developing between the particular ways in which first or early sexual gratifications were obtained and the object or means of that gratification (Ellis, 1906/1936). According to Ellis and the research he cites in this early, seminal work, well before Kinsey's,

if one's primary means of sexual gratification at an early and impressionable age is via a particular technique or a particular object of desire, then there may be a kind of fixation on that technique and/or object.

Cline argues further that if sexual problems can be alleviated in sex-counseling clinics with the use of sexual films, books, and videos as tools in therapy, then one must suspect that exposure to pornography can also have an effect. For Cline, and for many other observers, pornography provides powerful occasions on which modeling and imitative learning can occur.

Zillmann and Bryant (1988b) have made an important experimental contribution to the addiction hypothesis by showing that prolonged exposure to pornography can decrease some people's level of satisfaction with their partners and with the quality of their sex lives. Zillmann (1994) has gone on to propose that in many instances, "initial sexual dissatisfaction drives exposure to pornography" and a vicious circle ensues. With the consumption of pornography, the dissatisfaction grows stronger and draws the person into further consumption. For Zillmann, consumption of pornography invites comparisons that help drive dissatisfaction: "consumers compare what they have, by way of sexual intimacy, with what pornography tells them they might and should have" (1994, p. 210).

I have proposed, similarly, that the frequent presentation of highly romanticized and sexually arousing material on television, and elsewhere in our mainstream contemporary media, may fuel similar dissatisfactions and a propensity toward invidious comparison in a much broader spectrum of the population than was previously the case (Kubey, 1994, 1996; see also Bryant & Rockwell, 1994).

Other effects of pornography, aside from dependence—or addiction—and modeling, have .been studied and merit comment. Weaver (1994) has reviewed evidence indicating that exposure to pornography increases sexual callousness toward women. This callousness includes increased aggressivity toward women as well as a desensitization to the injury that violence or sexual assault causes.

Zillmann and Bryant have been interested in the degree to which so-called family values may be on a collision course with pornography, and they again offer experimental evidence. These studies (see Zillmann, 1994, for a review) typically expose an experimental group of adults to pornographic videos over a number of weeks (often six). Then, a week after the exposure, the group's answers to survey questions are compared with those of a control group that was not exposed.

The researchers' studies show that experimentally produced prolonged exposure to pornography results in a greater acceptance of both male and female promiscuity, and that as promiscuity is presumed to be more natural, adults also begin to assume that faithfulness among sexual intimates is less

common than is assumed by those in the control group. The participants in the experiment also report being more accepting of nonexclusive sexual intimacy for themselves.

In one study, when asked, "Do you feel that the institution of marriage is essential to the well-functioning of society?" 60 percent of the control group answered in the affirmative, but this was true for only 38.8 percent of the group exposed to pornography. Zillmann and Bryant (1988a) have also reported that exposure to pornography reduced the desire of their research participants, male and female, student and nonstudent, to want to have children. Zillmann (1994) suggests that this finding may

> Support the contention that prolonged consumption of pornography makes having children and raising a family appear an unnecessary inconvenience—presumably because pornography continually projects easy access to superlative sexual gratification, these gratifications being attainable without emotional investment, without social confinements, without economic obligations, and without sacrifices of time and effort. (p. 208)

In this regard, the immediate gratification that standard commercial television drama and film so frequently offers and promotes may in its own right be in conflict with the values of constancy and commitment so necessary to the healthy functioning of family life (Kubey, 1994). While this research is intriguing, I can't help but editorialize and wonder how internal review boards at universities permit research to be conducted on undergraduate populations if a propensity toward promiscuity is the known, and expected (from prior research trials), result in the experimental group. This work has been replicated; it is interesting to ponder the ethics of such work, and yet its importance, and the challenge of how to do this work without exposing people to the very material that one might hypothesize will be injurious.

As noted, new technologies have led to an explosion in pornography. And, if a pornography habit—or addiction—can indeed develop, it would seem more likely to develop if pornographic materials can be easily obtained.

It is not difficult to imagine how young people can come into contact with such materials. Even if a 12-year-old boy cannot rent a pornographic video on his own, it may well be that his friend's older brother, who is 16 but looks 18, can. And, of course, an increasing number of parents own such materials and keep them in their homes. In addition, such materials are available on many cable channels, if parents do not block them or if the child wishes to order material and suffer the consequences, if there are any, later.

While I do not personally frown on all uses of pornography, I do believe that it is not to be recommended for certain audiences. I believe it is unwise for

a 12-year-old boy to experience hard core pornography, especially as it is likely to be one of his very first exposures to sexual intimacy and such intense sexual experiences at a young age may constitute powerful early occasions for learning and impression formation.

Zillmann and Bryant's work suggests that such materials might also prove detrimental in the formation of a boy's impressions of female sexuality, since most such pornography depicts women as sexual objects whose primary goal is to serve the sexual desires of men. An occasional viewing of such materials by a pubescent or prepubescent boy might not have any strong or deleterious effects. But when we recognize that some boys may view such material every few days, or even more often, and if we add that some boys typically seek and obtain sexual release upon viewing, I believe we raise the possibility not only that a strong habit or "addiction" may develop, but also that it may be one we would not want to encourage, especially when we consider that this same boy is likely to begin having his first real sexual experiences with a girl or young woman in the not so distant future.

These concerns are multiplied when we consider the arrival of interactive erotica. An early interactive offering was called Virtual Valerie. Here, computer-generated, movie-quality images of young women take their clothes off at the command of the viewer. Women on the screen can also be programmed at the touch of a button to say arousing things to the viewer, as well as to perform a variety of sexually suggestive acts before the viewer's eyes.

Let's return again to our 12-year-old boy. Imagine that he has obtained some interactive video products like Virtual Valerie. Imagine that he interacts with them while masturbating several times a week, typically spending 10 to 30 minutes in each encounter, off and on, for a few years before, at age 15, he has his first intimate encounter with an *actual, real-life* young female of the same age. Might his expectations of how she will act and how he should act if they become intimate have been altered by the many hours spent with his interactive pornography disks?

I don't believe we know the answer. Conceivably, this form of pornography may have positive effects. Perhaps it will help some people fantasize and obtain sexual release in such a way that there is a reduction in the commission of sexual crimes (see Linz and Malamuth, 1993, for a review of research on the positive, cathartic effect of traditional pornography). Still, combining common sense with what we know about the learning of sexual behavior, I must say that I am concerned about young people, as well as some adults, overusing, and perhaps becoming dependent on or "addicted" to, such a form of entertainment.

NOTE

1. Portions of this article were originally presented in, or have been adapted from, R. Kubey, Television Dependence, Diagnosis, and Prevention: With Commentary on Video Games, Pornography, and Media Education, in Tuning in to Young Viewers: Social Science Perspectives on Television, ed. T. MacBeth, pp. 221–260 (1996: Newbury Park, CA: Sage).

REFERENCES

Bryant, J., & Rockwell, S. C. (1994). Effects of massive exposure to sexually oriented prime-time television programming on adolescent moral judgment. In D. Zillmann, J. Bryant, & A. C. Huston (Eds.), *Media, children, and the family: Social scientific, psychodynamic, and clinical perspectives* (pp. 183–195). Hillsdale, NJ: Erlbaum.

Cline, V. B. (1994). Pornography effects: Empirical and clinical evidence. In D. Zillmann, J. Bryant, & A. C. Huston (Eds.), *Media, children, and the family: Social scientific, psychodynamic, and clinical perspectives* (pp. 229–247). Hillsdale, NJ: Erlbaum.

Dodge, N. (2007). *The brain that changes itself: Stories of personal triumph from the frontiers of brain science*. New York: Penguin.

Ellis, H. (1906/1936). *Studies in the psychology of sex* (Vol. 2). New York: Random House.

Irons, R., & Schneider, J. P. (1996). Differential diagnosis of addictive sexual disorders using the DSM–IV. *Sexual addiction and compulsivity, 3*, 7–21.

Kubey, R. (1994). Media implications for the quality of family life. In D. Zillmann, J. Bryant, & A. C. Huston (Eds.), *Media, children, and the family: Social scientific, psychodynamic, and clinical perspectives* (pp. 183–195). Hillsdale, NJ: Erlbaum.

Kubey, R. (1996). Television dependence, diagnosis, and prevention: With commentary on video games, pornography, and media education. In T. MacBeth (Ed.), *Tuning in to young viewers: Social science perspectives on television* (pp. 221–260). Newbury Park, CA: Sage.

Lanning, K., & Burgess, A. (1989). Child pornography and sex rings. In D. Zillmann & J. Bryant (Eds.), *Pornography: Research advances and policy considerations* (pp. 235–255). Hillsdale, NJ: Erlbaum.

Linz., D. (2004). The science behind pornography addiction. Response to testimony before the U.S. Senate Subcommittee on Science, Technology, and Space. Retrieved December 23, 2008, from http://www.freespeechcoalition.com/dan_linz.htm

Linz, D., & Malamuth, N. (1993). *Pornography*. Newbury Park, CA: Sage.

Lyons, J. S., Anderson, R. L., & Larson, D. B. (1994). A systematic review of the effects of aggressive and nonaggressive pornography. In D. Zillmann, J. Bryant, & A. C.

Huston (Eds.), *Media, children, and the family: Social scientific, psychodynamic, and clinical perspectives* (pp. 271–310). Hillsdale, NJ: Erlbaum.

Marshall, W. L. (1988). The use of explicit sexual stimuli by rapists, child molesters, and nonoffender males. *Journal of Sex Research, 25,* 267–288.

McGaugh, J. L. (1983). Preserving the presence of the past. *American Psychologist, 38,* 161.

Oddone-Paolucci, E., Genuis, M., & Violato, C. (2000). A meta-analysis of the published research on the effects of pornography. In C. Violato, E. Oddone-Paolucci, & M. Genuis (Eds.), *The changing family and child development* (pp. 48–59). Aldershot, England: Ashgate Publishing.

Reed, M. D. (1994). Pornography addiction and compulsive sexual behavior. In D. Zillmann, J. Bryant, & A. C. Huston (Eds.), *Media, children, and the family: Social scientific, psychodynamic, and clinical perspectives* (pp. 249–269). Hillsdale, NJ: Erlbaum.

Weaver, J. B. (1994). Pornography and sexual callousness: The perceptual and behavioral consequences of exposure to pornography. In D. Zillmann, J. Bryant, & A. C. Huston (Eds.), *Media, children, and the family: Social scientific, psychodynamic, and clinical perspectives* (pp. 215–228). Hillsdale, NJ: Erlbaum.

Zillmann, D. (1994). Erotica and family values. In D. Zillmann, J. Bryant, & A. C. Huston (Eds.), *Media, children, and the family: Social scientific, psychodynamic, and clinical perspectives* (pp. 199–213). Hillsdale, NJ: Erlbaum.

Zillmann, D. (2004). Pornografie. In R. Mangold, P. Vorderer, & G. Bente (Eds.), *Lehrbuch der Medienpsychologie* (pp. 565–585). Göttingen, Germany: Hogrefe Verlag.

Zillmann, D., & Bryant, J. (1988a). Effects of prolonged consumption of pornography on family values. *Journal of Family Issues, 9,* 518–544.

Zillmann, D., & Bryant, J. (1988b). Pornography's impact on sexual satisfaction. *Journal of Applied Social Psychology, 18,* 438–453.

Chapter **10**

Assessment and Treatment of Internet Addiction

Kimberly S. Young, PhD

Notions of technological addictions (Griffiths, 1996) and computer addiction (Shotton, 1991) have previously been studied in England. However, when Internet addiction was first introduced in 1996 at the American Psychological Association meeting (Young, 1996), it sparked a controversy among clinicians and academicians alike.

In contrast to chemical dependency, the Internet offers several direct benefits as a technological advancement in our society, rather than a device to be criticized as addictive (Levy, 1997). These benefits range from practical applications including conducting research, performing business transactions, accessing libraries, communicating with colleagues, and making vacation plans. Books have been written outlining the psychological as well as functional benefits of the Internet in our daily lives (Rheingold, 1993; Turkle, 1995). By comparison, chemical dependence is not an integral part of our professional lives, nor does it offer any direct benefit.

Furthermore, many researchers argued that the term *addiction* should be applied only to cases involving the ingestion of a drug (e.g., Rachlin, 1990; Walker, 1989). However, defining addiction has moved beyond this to include a number of behaviors that do not involve an intoxicant: these include compulsive gambling (Griffiths, 1990), video game playing (Keepers, 1990), overeating (Lesuire & Blume, 1993), exercise (Morgan, 1979), love relationships (Peele & Brodsky, 1975), and television viewing (Winn, 1983). Over the past decade, a growing body of peer-reviewed literature has adopted the term *Internet addiction,* and its acceptance as a legitimate disorder has grown (e.g., Ferris, 2001; Greenfield, 1999; Hansen, 2002).

DIAGNOSIS

The Internet is a highly promoted technological tool, making the detection and diagnosis of addiction difficult. Therefore, it is essential to understand the criteria that differentiate normal from pathological Internet use. Proper diagnosis is often complicated by the fact that there is currently no accepted set of criteria for addiction listed in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (*DSM–IV*, 1994). Of all the diagnoses referenced, pathological gambling was viewed as most akin to compulsive Internet use, being defined as an impulse-control disorder that does not involve an intoxicant. In what is known as the Internet Addiction Diagnostic Questionnaire (IADQ), Young (1998a) developed the first screening instrument, which outlined the following criteria:

1. Do you feel preoccupied with the Internet (think about previous online activity or anticipate next online session)?
2. Do you feel the need to use the Internet with increasing amounts of time in order to achieve satisfaction?
3. Have you repeatedly made unsuccessful efforts to control, cut back, or stop Internet use?
4. Do you feel restless, moody, depressed, or irritable when attempting to cut down or stop Internet use?
5. Do you stay online longer than originally intended?
6. Have you jeopardized or risked the loss of significant relationship, job, educational or career opportunity because of the Internet?
7. Have you lied to family members, therapist, or others to conceal the extent of involvement with the Internet?
8. Do you use the Internet as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)?

When using the IADQ, only nonessential computer/Internet use (i.e., use that is not for business purposes or academically related) should be evaluated, and addiction is present when clients answer "yes" to five (or more) of the questions over a six-month period. This list of questions offers a workable definition of Internet addiction to help us differentiate normal from compulsive Internet use, but the warning signs can often be masked by cultural norms that encourage and reinforce Internet use. Thus, even if a person meets all eight criteria, signs of abuse can be rationalized away as "I need this for my job" or "It's just a machine," when in reality the Internet is causing significant problems in a user's life.

While time is not a function in diagnosing Internet addiction, addicts are generally are excessive in their online usage, spending anywhere from 40 to

80 hours per week on the Internet, with sessions that could last up to 20 hours at a time. Sleep patterns are disrupted due to late night log-ins, and addicts generally stay up surfing until two, three, or four in the morning despite having to wake up early for work or school. In extreme cases, caffeine pills are used to facilitate longer Internet sessions. Such sleep depravation causes excessive fatigue, impairing academic or occupational performance, and may affect the immune system, leaving the addict vulnerable to disease. Sitting at the computer for such prolonged periods also means that addicts aren't getting the proper exercise, and sometimes addicts are at increased risk for carpal tunnel syndrome.

Like alcoholics who need to consume greater levels of alcohol in order to achieve satisfaction, addicts routinely spend significant amounts of time online. Furthermore, addicts will go to great lengths to mask the nature of their online activities, primarily to conceal the extent and nature of the behavior. In most cases of impulse-control disorder, an individual's compulsion is often associated with increasingly painful states of tension and agitation that is relieved through the completion of the act. For example, an alcoholic is driven to drink or an overeater is driven to binge on food during moments of tension. In each case, the compulsive behavior serves to reduce the underlying emotional tension and serves to reward behavior. In the same manner, an Internet addict's use of the computer is less about using it as an information tool and more about finding a psychological means to cope with life's problems.

SUBTYPES OF ONLINE ABUSE

The Internet is a term denoting a variety of functions accessible online, such as the World Wide Web (WWW), chat rooms, instant messaging systems, interactive games, virtual casinos, online auction houses, news groups, and databases. Internet addicts typically become addicted to a particular application that acts as a trigger for excessive Internet use. Internet use becomes focused on a particular chat room, a particular online game, a particular virtual casino, or a particular set of adult sites. Early attempts to categorize the behavior suggested that the patterns of use fall into four major subtypes: online abuse, cybersex addiction, online affairs, online gaming, and Internet gambling. An overview of each is provided below.

Cybersex Addiction

Individuals who suffer from cybersex addiction are typically engaged in viewing, downloading, and trading online pornography or are involved in

adult fantasy role-play rooms. Adult Web sites comprise the largest segment of online development and electronic commerce, catering to a wide variety of sexual interests. Given the extensive availability of sexually explicit material online, cybersex addiction has been the most common form of problem online behavior among new users (70%) and has a high relapse rate among prior sexual compulsives (55%) (Young, 2001).

Young (2001, p. 36) stated that "computer-enabled fantasies are highly reinforcing and the addict's preoccupation with sexual arousal stems from his own imagination and fantasy history." The association of the Internet with sexual arousal is so potent that it transforms the Internet from a practical business or research device into a modern-day sex toy. Sometimes, just recalling the potent images of one's last online episode triggers arousal and reinforces the notion that cyberspace is an open gateway to immediate sexual fulfillment; as one addict explained, "I get a major erection just clicking on my computer." Based upon her studies, Young describes five interdependent stages of cybersex addiction that highlight how users utilize the Internet as a progressive means of escape as part of an addiction cycle: discovery, experimentation, escalation, compulsivity, and hopelessness.

In the discovery stage, the discovery that adult Web sites and chat rooms exist is made by the user. A man doing research online may accidentally bump into a pornographic Web site or a woman may enter a social chat room and meet a man who entices her to have cybersex. In either case, the person discovers the sexual thrill of the act, which opens the door for further exploration. In the experimentation stage, the user may secretly begin to explore and experiment online without the fear of being caught. Users feel encouraged and validated by acceptance of the cyberspace culture, especially when, cloaked in the anonymity of the computer screen, they may feel less accountable for their actions over the Internet (Cooper, Putnam, Planchon, & Boies, 1999). Within the anonymous context of cyberspace, conventional messages about sex are eliminated, allowing users to play out hidden or repressed sexual fantasies in a private lab.

In the experimentation stage, a curious person may be completely unprepared when she steps into one of many sites specifically designed for the purposes of facilitating sexual experimentation. Web site names such as the "Hot Sex Room," the "Fetish Room," or the "Bisexual Room" may intrigue the casual browser, who is initially shocked but at the same time titillated by the permissiveness of others engaged in virtual sex. Such virtual environments may be more seductive than most users anticipate, providing short-term comfort, distraction, and/or excitement. Users begin to dabble in darker or more deviant types of sexual material online. Furthermore, online experiences occur in the privacy of one's home, office, or bedroom, facilitating the perception of anonymity and the idea that Internet use is personal and untraceable.

In the escalation stage, the behavior becomes more chronic and pronounced, such that the addict becomes saturated with a continuous stream of sexual content that can take on riskier and riskier forms. Most people do not yet realize that there is any risk involved in engaging in online sexual pursuits. While in some ways such engagement may seem like a journey into foreign territory, online sexual behaviors occur in the familiar and comfortable environment of home or office, thus reducing the feeling of risk and allowing even more adventurous behaviors (Young, 2001).

In the compulsivity stage, the user engages in more constant sexual behavior online, downloading hundreds or thousands of pornographic images, or meeting people for the sole purpose of having cybersex in online sex chat rooms. Just as the alcoholic requires larger doses of the drug to achieve the same sensation and pleasure from the experience, the online addict now looks for the next big virtual thrill, perhaps engaging in more extreme sexual fantasies online.

In the hopelessness stage, in order to deal with the double life that develops, the addict often rationalizes the behavior and disowns what he says or does online with self-statements as, "It's just a computer fantasy" or "This isn't who I really am." Addicts detach themselves from the online sexual experience and perceive their secret fantasy world as a parallel life that is completely separate from their identity in real life. They may also progress into more sexually deviant topics that they would normally find reprehensible but that over time become acceptable as they become increasingly desensitized to the experience. The addict becomes preoccupied with the computer, attempts to conceal the nature of his online activities, and continues to engage in the activity despite its known potential risks, including possible job loss, divorce, or arrest. In one such case, a 34-year-old minister arrested for possession of child pornography obtained from the Internet explained, "I soon discovered the vast array of pornography, including child pornography, available on the Internet. My attraction to pornography on the computer was born of sheer amazement at the volume of available material and this amazement turned to fascination and ultimately to obsession. I knew it was wrong. My life became a lonely, isolated mess. I realized that I could lose my job, my marriage, and the respect of everyone I love if I was caught. I have two daughters and would never think about doing anything inappropriate with them, but I could not bring myself to stop, despite knowing the consequences of my actions."

Online Affairs

Individuals who become overinvolved in online relationships through instant messaging, chat rooms, voice-over Internet, and e-mail are at risk for developing an addictive pattern in these relationships. Online friends quickly become

more important to an individual, often at the expense of real-life relationships with family and friends. Among married couples, this can lead to online affairs. Married users may utilize interactive online environments to seek out support, comfort, and acceptance that provide them with a sense of belonging in a non-threatening manner.

Online affairs are among the most common consequences of online addiction. At an alarming rate, long-term and previously stable marriages are destroyed by intimate words shared over the computer. An online affair can be defined as a romantic and/or sexual relationship that is initiated via online contact and maintained predominantly through electronic conversations that occur through e-mail, chat rooms, or interactive games (Young, 1999). These virtual communities allow strangers from all over the world to meet instantly 24 hours per day, seven days a week, creating a breeding ground for online affairs. Young (1998b) found that serious relationship problems were reported by 53 percent of Internet addicts surveyed that led to marital discord, separation, and even divorce. However, the scope of the relationship problems caused by the Internet can be undermined by its popularity and advanced utility.

Online affairs differ dynamically from real-life affairs and are potentially more seductive. Given the global nature of the Internet, online affairs can be culturally diverse and consequently seem more glamorous than the people one already knows in day-to-day living (Greenfield, 1999). Electronic communication allows individuals to feel less inhibited, which accelerates perceived intimacy. Online, people are more likely to be open, honest, and forthright, revealing personal truths, and the intimacy that might take months or years to develop in an offline relationship may only take days or weeks online (Cooper & Sportolari, 1997). A friendly online conversation can quickly turn erotic, allowing users to share private sexual fantasies with one another; this can be accompanied by self-stimulation to heighten the sexual experience (Young, 1998a).

Seemingly harmless online relationships can easily progress to secret phone calls, letters, and offline meetings, and getting one's needs met through an online affair can adversely impact one's marriage. Clinicians are increasingly seeing cases of couples seeking counseling to deal with an online affair (Young, 2004), and online infidelity has accounted for a growing number of divorce cases. According to the American Academy of Matrimonial Lawyers (Quittner, 1997), 63 percent of lawyers surveyed reported that the Internet has played a significant role in the divorces they have handled during the past year (AAML, 2002). Unlike affairs that happen outside the home, online affairs occur in the home, often while an unsuspecting spouse is sitting in the next room. The warning signs of an online affair are often masked with the claim of legitimate

or necessary use of the computer. For instance, a spouse may begin to come to bed in the early morning hours or they leap out of bed an hour or two early to use the computer for a prework e-mail exchange with a new romantic partner. If a spouse begins an affair, whether online or offline, that spouse will usually go to great lengths to hide the truth from a partner. In the case of an online affair, the attempt to hide the truth usually leads to a need for greater privacy and secrecy surrounding computer use. The computer may be moved from a visible den to a secluded corner of a locked study, or the password may be changed. If disturbed or interrupted when online, unfaithful spouses may react with anger or defensiveness to conceal the extent of their online involvement (Young, 1998a).

Some online affairs can evolve into phone sex or real-life meetings, but even the process of sharing one's sexual fantasies online can alter patterns of sexual interest. If chats with an online lover also include masturbation, a person may suddenly show a reduced interest in sex with a real-life partner; this may be one of the signs that an individual has found another sexual outlet online. Often, people who engage in online affairs are less enthusiastic, energetic, and responsive with regard to lovemaking with a real-life partner and prefer the newness and excitement of virtual sexual stimulation (Young, 2004).

Online Gaming

Massive multiuser online role-playing games, or MMORPGs as they are often called, are one of the fastest growing sub-areas of Internet addiction among children and teens. Parents across the globe are increasingly concerned about their sons' and daughters' online gaming habits (Young, 2004). They are sure that there is a problem, but counselors unfamiliar with online gaming addiction often do not understand how seductive such games can be. As one parent explained, "I had gone to my son's guidance counselor, the school psychologist, and two local addiction counselors. None of them ever heard of World of Warcraft, much less someone becoming addicted to it. They told me it was a phase and that I should limit my son's game playing. They didn't understand that I couldn't. He had lost touch with reality. He lost interest in everything else. He didn't eat, sleep, or go to school. The game was the only thing that mattered to him."

Parents often feel alone and scared as their children become hooked on something that no one seems to understand. "I couldn't believe it when my therapist told me to just turn off the computer. That was like telling the parent of an alcoholic son to tell him to just stop drinking. It wasn't that simple. We felt like no one was taking us seriously, that our son had a real problem."

Like those addicted to alcohol or drugs, gamers show several classic signs of addiction. They become preoccupied with gaming, lie about their gaming use, lose interest in other activities just to game, withdraw from family and friends to game, and use gaming as a means of psychological escape (Young, 1998b). Because of their addiction, gamers become defensive about their need to play the game and angry when forced to go without it. Parents who try to put time limits on the game describe how their sons and daughters become angry, irrational, and even violent. Gamers who can't access the game experience a loss. They want to be on the game and they miss playing the game. This feeling can become so intense that they become irritable, anxious, or depressed when they are forced to go without the game (Young, 1998b). They can't concentrate on anything except going back online to play. Their minds become so fixated on the game that they can experience a psychological withdrawal from the game. As their feelings intensify, they stop thinking rationally and begin to act out toward other people in their lives, especially a parent or anyone who threatens to take the game away.

Internet Gambling

Online casinos have practically overnight sprung up and turned into a multi-million-dollar business, attracting a large number of gamblers worldwide. Compulsive gambling has been around for decades, but now access and opportunity have greatly increased with the invention of Internet gambling, bringing with it a new form of addictive behavior. The global nature of the Internet, combined with the limited, if not nonexistent, ability of local governments to effectively regulate or ban online gambling, will have profound psychological and social consequences. "It's fun. It's exciting. It's glamorized on TV and in the media in a way that other addictions are not," states the National Council on Problem Gambling (2008), referring to the traditional gambling that takes places in casino-rich places like Las Vegas and Atlantic City. Today, all anyone needs is a computer and the Internet to access the thousands of online casinos.

There are now an estimated 1,700 gambling Web sites on the Internet. As well as using the Internet, users can gamble through interactive television and mobile phones. The convenience of gambling at home, the ease of setting up an online gambling account, and the variety of Internet gambling opportunities, ranging from traditional betting to casino gambling and lotteries, makes online gambling very appealing (Petry, 2006).

However, while many people gamble online without developing any problems, several factors make online gambling more seductive for teens, increasing their risk of developing a gambling problem:

- The ability to gamble 24 hours a day.
- The access underage children have to gambling sites.
- The absorbing nature of computers, leading teens to online casinos in the first place.
- The decrease in perception of the value of cash—i.e., players forget that they are spending money.

According to the National Gambling Impact Commission (1998), the national lifetime gambling population is no less than 1.2 percent of the total population (2.5 million). That would make gambling twice as prevalent as cancer among Americans. In mature gambling markets such as Nevada, more than 5 percent of the population will develop some problem with gambling, a prevalence rate about five times that of schizophrenia and more than twice that of cocaine addiction. According to the National Coalition against Legalized Gambling, young children and teenagers are at the greatest risk of developing a problem with Internet gambling. Some recent estimates they suggest are as follows:

- 16–24-year-old males comprise 4% of Internet gamblers; and
- 11–18-year-old males comprise 4–7% of Internet gamblers

Teen gambling has a special appeal as television shows promote the fun associated with poker or Texas Hold Um. Teenagers' access to these televised poker shows makes them believe that gambling is harmless fun and not potentially addictive.

Teens experiencing a problem with Internet gambling start to become preoccupied with gambling, causing disruptions in their personal, family, and social lives. Petry (2006) found that teen Internet gamblers were more likely to have a serious problem with gambling than other gamblers. Furthermore, teen Internet gamblers were more likely to suffer from health and emotional problems such as substance abuse, circulatory disease, depression, and risky sexual behaviors. As teens and preteens go online with greater frequency, the risk of addiction in various forms becomes greater. Old favorites such as sports betting and casino games still dominate the Internet, but in the future there will be more opportunities that could draw new gamblers into the fold. People can go online and bet about whether Brad Pitt and Angelina Jolie will get married or whether Tom Cruise and Katie Holmes's marriage will last. They can bet on the outcome of the Oscars or who will win on *Survivor*. These are the new kinds of bets that are placed by people who might not normally visit a gambling site.

RISK FACTORS

While most agree that the Internet is a productive tool, research findings document serious negative consequences when it is used in an addictive

manner. In particular, recent research suggests that compulsive use of the Internet is associated with increased levels of social isolation, increased depression, familial discord, divorce, academic failure, job loss, or significant financial debt as a result of obsessive online gambling, shopping, or gaming (e.g., Cooper, Putnam, Planchon, & Boies, 1999; Cooper, Scherer, Boies, & Gordon, 1999, Orzack, 1999).

Despite the consequences, addictions accomplish something for the person, however illusory or momentary the benefits may actually be. Underlying Internet addiction is the anonymity of electronic transactions, which provide a virtual context that cultivates a subjective escape from emotional difficulties (e.g., stress, depression, anxiety) or problematic situations or personal hardships (e.g., job burnout, academic troubles, sudden unemployment, marital discord).

As the addiction cycle grows, the Internet becomes a way for the addict to self-medicate in order to temporarily run away from life's problems. Over time, however, this coping mechanism proves to be unproductive and potentially harmful, as the issues hidden by the addictive behavior develop into larger and larger problems. While not everyone becomes addicted to the Internet in the same way for the same reason, some general patterns have emerged with regard to why people become hooked and the ways in which they use the Internet to escape from or cope with underlying problems in their lives.

Emotional Problems

Like a craving for a cigarette or the desire to have a drink, emotions such as stress, depression, loneliness, anxiety, or burnout can lead to an addict's need to go online, which serves as a temporary distraction to fill an emotional void (Peele & Brodsky, 1979). Consistently, Internet addicts explain that they feel a difference between their online and offline emotions. They feel frustrated, worried, angry, anxious, and depressed when offline. When online, they feel excited, thrilled, uninhibited, attractive, supported, and more desirable. These strong positive emotions reinforce the compulsive behavior (Young, 1999). The addictive behavior itself acts as a way to temporarily avoid negative or unpleasant feelings. Under the influence, the alcoholic feels as if all the other problems in his life disappear, and when eating, the overeater experiences a sense of peace and relaxation, lessening the overwhelming stress and frustration he feels.

Interpersonal Difficulties

Individuals who suffer from low self-esteem and feel lonely, restless, or withdrawn can use cyberspace connections with others to make them feel better

about themselves and their circumstances. Internet sex offers a fantasy world in which there are endless numbers of people who appear to be interesting to—and interested in—the individuals concerned. Young, socially awkward, or emotionally troubled individuals may find it easier to engage in Internet "relationships" than risk face-to-face rejection by a real person. As the addict becomes more immersed in this shadow world, denial takes hold and she comes to view these "friends" and "partners" as more real than an actual spouse or family member.

The anonymity associated with electronic communication and the general milieu of the Internet often facilitates more frank and open communication with other users (Cooper and Sportolari, 1997). Anonymity can also increase the online user's feeling of comfort, since it is less easy for the user to detect signs of insincerity, disapproval, or judgment than it is in face-to-face interactions. The distance afforded by cyberspace enables a person to share intimate feelings often reserved for a significant other, thus opening the door for bonding and an accelerated sense of intimacy, which in turn can disrupt real-life relationships. This can in turn lead to greater problems in the addict's marriage or family (Young, 2004). Partners often feel hurt, betrayed, rejected, abandoned, devastated, jealous, and angry, as well as experiencing a loss of self-esteem at the discovery of the user's activities. Being lied to repeatedly is a major source of distress. Partners will feel angry and jealous of the computer and view it as a source of conflict, pain, and stress in the relationship as the addict withdraws deeper into the virtual life.

Relapse from Prior Addictions

For those in recovery from prior addictions, the Internet becomes another way to engage in compulsive or addictive behavior (Greenfield, 1999). Recovering addicts who feel overwhelmed, experience work or money problems, or experience life-changing events such as divorce, relocation, or a death in the family can absorb themselves in a virtual world full of fantasy and intrigue. They can lose themselves in online pornography, Internet gambling, or online gaming, and once they are online, the difficulties of their lives fade into the background as their attention becomes completely focused on the Internet. Those in recovery from prior addictions look to the Internet as a new way of escaping without really dealing with the underlying problems causing their addictive behavior. Without the crutch of their prior addiction, say, to alcohol or drugs, the stress that comes from a job or a marriage or relationships in general can trigger sexually addictive online behavior. Using the Internet becomes a quick fix and an instant way to wash away troubling feelings or difficult situations that they really don't know how to deal with (Young, 2004).

Students Most At Risk

The Internet has been touted as a premier educational tool driving schools to integrate Internet services into their classroom environments. However, one survey revealed that 86 percent of responding teachers, librarians, and computer coordinators believe that Internet use by children does not improve performance (Barber, 1997). They argue that information on the Internet is too disorganized and unrelated to the school curriculum to help students, and can even serve as a distraction. Young (1998b) found that 58 percent of students suffered from poor study habits or poor grades, or failed school due to excessive Internet use. Increasingly, college administrators are recognizing that they have put a great deal of money into an educational tool that can easily be abused.

Colleges are starting to see the potential impact of student Internet use. At Alfred University in Alfred, New York, Provost W. Richard Ott investigated why normally successful students with scores of 1200 to 1300 in the SATs had recently been dismissed. His investigation found that 43 percent of these students failed school due to extensive patterns of late-night log-ins to the university computer system (Brady, 1997). Counselors at the University of Texas at Austin began seeing students whose primary problem was an inability to control their Internet use, and in one of the first campus studies of Internet abuse they found that 14 percent met the criteria for Internet addiction (Scherer, 1997).

College counselors have argued that students are the most population most at risk of developing an addiction to the Internet because campuses, computer labs, wired dorms, and mobile Internet devices access possible anytime, day or night (Scherer, 1997). The University of Maryland even started an Internet addiction support group to help students who abused Internet use (Murphey, 1996), and gradually more such support groups are developing across campuses.

TREATMENT ISSUES

Our computerized society makes it difficult to simply go "cold turkey" from Internet sex. Today, most people need to use the computer every day for work, making abstinence from Internet sex more complex than abstinence from drink or drugs. As more jobs involve computers and as more homes have computers, complete abstinence from the Internet may be impossible, forcing the Internet addict to use self-control to achieve corrective action and abstinence from problematic Internet applications. In food addiction, recovery can be objectively measured through caloric intake and weight loss; in the same way, online addicts can objectively measure success through maintaining abstinence from problematic online applications and increasing meaningful offline activities.

But how can addicts learn that kind of willpower and self-discipline when any contact with the computer feels like a temptation?

In *Caught in the Net*, Young (1998a) provides an integrated recovery approach that combines cognitive-behavioral and insight-oriented therapies. She postulates that recovery from Internet addiction is most akin to recovery from food addiction. Food addicts cannot simply abstain from food as part of their recovery; instead they must discover healthier ways to live with food in their lives. Similarly, online addicts must discover healthier ways to live with the Internet in their lives. To make this discovery, the addict must be willing to identify and understand the underlying emotional, cognitive, or situational factors that trigger the addictive behavior, such as depression, anxiety, loneliness, stress, marital troubles, divorce, or career problems, and learn to cope with those underlying issues in a more adaptive manner.

Cognitive-Behavioral Therapy

Researchers have likened Internet addiction to addictive impulse-control disorders on the Axis I scale in the *DSM* (Beard & Wolf, 2001; Orzack, 1999), and they have utilized various forms of *DSM–IV*-based criteria to define Internet addiction. Cognitive Behavioral Therapy (CBT) has been shown to be an effective treatment for compulsive disorders such as intermittent explosive disorder, pathological gambling, and trichotillomania (Hucker, 2004). CBT has also been effective in treating substance abuse, emotional disorders, and eating disorders (Beck, 1979; Beck, Wright, Newman, & Liese, 1993).

CBT is a familiar treatment based on the premise that thoughts determine feelings. Patients are taught to monitor their thoughts and identify those that trigger addictive feelings and actions while they learn new coping skills and ways to prevent a relapse. CBT usually requires three months of treatment or approximately 12 weekly sessions. The early stage of therapy is behavioral, focusing on specific behaviors and situations in which the impulse control disorder causes the greatest difficulty. As therapy progresses, there is more of a focus on the cognitive assumptions and distortions that have developed and the effects of these on behavior. This involves assessment of the type of distortion, training in problem-solving skills and coping strategies, modeling in therapy, use of support groups, and keeping thought journals (Beck, 1979).

Behavior Change

In cases of Internet addiction, abstinence recovery models are not practical, as computers have become such a salient part of our daily lives. Therefore, clinicians have generally agreed that moderated and controlled use of the Internet

is most appropriate as a means to treat Internet addiction. Behavior therapy is the initial focus of recovery, examining both computer behavior and noncomputer behavior (Hall & Parsons, 2001). Computer behavior deals with actual online use, with a primary goal of abstinence from problematic applications while retaining controlled use of the computer for legitimate purposes. For example, a lawyer addicted to Internet pornography would need to learn to abstain from adult Web sites, while still being able to access the Internet to conduct legal research and to e-mail clients. Noncomputer behavior focuses on helping clients develop positive lifestyle changes for life without the Internet. Life activities that do not involve the computer are evaluated and may include relationship function, social function, or occupational function.

Young (2001) suggests using a daily internet log to evaluate computer behavior and establish a baseline for clinical treatment. Once a baseline has been established, behavior therapy is used to relearn how to use the Internet to achieve specific outcomes, such as moderated online use and more specifically abstinence from problematic online applications and controlled use for legitimate purposes. Behavior management for both computer usage and adaptive noncomputer behavior focuses on present and overt behavior. The techniques involved may include assertion training, behavioral rehearsal, coaching, cognitive restructuring, desensitization, modeling, reinforcement, relaxation methods, self-management, or new social skills.

Cognitive Restructuring

Addictive thinkers, for no logical reason, will feel apprehensive when anticipating disaster (Twerski, 1990). While addicts are not the only people who worry and anticipate negative happenings, they tend to do this more often than other people. Young (1998a) suggested that this type of catastrophic thinking might contribute to compulsive Internet use by providing a psychological escape mechanism to avoid real or perceived problems. Subsequent studies hypothesized that other maladaptive cognitions, such as overgeneralizing or catastrophizing, negative core beliefs, and cognitive distortions also contribute to compulsive use of the Internet (Caplan, 2002; Davis, 2001; LaRose, Mastro, & Easton, 2001). Young hypothesized that those who suffer from negative core beliefs may be the ones who are drawn the most to the anonymous interactive capabilities of the Internet in order to overcome their perceived inadequacies. She suggested that cognitive restructuring should be used to address underlying negative core beliefs, cognitive distortions, and rationalizations such as "Just a few more minutes won't hurt" for effective management of the patient's primary symptoms.

Developing Support Systems

Young suggests that addicts must first assess their current use of the Internet to examine the extent of Internet use and identify high-risk situations, feelings, or events that trigger the behavior. Most importantly, for continued recovery and relapse prevention, they need to avoid high-risk situations that can lead to relapse and they need to repair relationships hurt by their addiction.

Due to their addiction, addicts often hurt or lose significant real-life relationships, such as relationships with a spouse, a parent, or a close friend. Often, these were individuals who provided the addict with support, love, and acceptance before the Internet addiction, and their absence only makes the addict feel worthless and reinforces past notions of being unlovable. The addict must amend and reestablish these broken relationships to achieve recovery and find the support necessary to fight the addiction. Young (1998a) emphasizes that the recovery process is an ongoing self-exploration that must separate the behavior from the person, relieve shame about the behavior, correct maladaptive cognitions, and promote opportunities to learn from mistakes. The recovery process must also build relationships, provide new ways to relate to others, and allow amends to be made. Involving loved ones in the recovery process can be a rich source of the nurturing and sponsorship needed to help a client maintain sobriety and abstinence. Finally, the recovery process should provide continuous support and affirmation that creates a positive self-image.

FUTURE AREAS OF RESEARCH AND PRACTICE

Over the last decade, acceptance of Internet addiction has grown in the mental health field, and new journals such as *CyberPsychology and Behavior,* which focus on Internet behavior and addiction, have emerged. It is difficult to predict the results of these early endeavors. However, it is quite possible that with years of collective effort, Internet addiction may be recognized as a legitimate impulse-control disorder worthy of its own classification in future revisions of the *Diagnostic and Statistical Manual of Mental Disorders*. Until then, there is a need for the professional community to recognize and respond to the threat of its rapid expansion.

With the growing popularity of the Internet, the mental health field needs to develop an infrastructure of treatment programs specifically designed to care for the Internet addicted. Since this is a new and often laughed at addiction, individuals are reluctant to seek out treatment, fearing that clinicians may not take their complaints seriously. Drug and alcohol rehabilitation centers, community mental health clinics, and clinicians in private practice should avoid

minimizing the impact to addicts whose complaint involves Internet addiction and offer effective recovery programs. Advertisement of such programs both online and within the local community may encourage timid individuals to come forward to seek the help they need.

Finally, to pursue such effective recovery programs, continued research is needed to better understand the underlying motivations of Internet addiction. Future research should focus on how psychiatric illness such as depression or obsessive-compulsive disorder plays a role in the development of compulsive Internet use. Longitudinal studies may reveal how personality traits, family dynamics, or interpersonal skills influence the way people utilize the Internet. Outcome studies are needed to determine the efficacy of specialized therapy approaches to treat Internet addiction and to compare these outcomes with traditional recovery modalities.

REFERENCES

American Academy of Matrimonial Lawyers [AAML]. (2002, November 14). *Is Internet bad for your marriage?* Press release published by Jonathan Dedmon.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders*. (4th ed.). Washington, DC: Author.

Barber, A. (1997, March 11). Net's educational value questioned. *USA Today*, p. 4D.

Beard, K. W., & Wolf, E. M. (2001). Modification in the proposed diagnostic criteria for Internet addiction. *CyberPsychology and Behavior, 4*, 377–383.

Beck, A. T. (1979). *Cognitive therapy and the emotional disorders*. New York: Plume.

Beck, A. T., Wright, F. D., Newman, C. F., & Liese, B. S. (1993). *Cognitive therapy of substance abuse*. New York: Guilford Press.

Brady, K. (1997, April 21). Dropout rise a net result of computers. *The Buffalo News*, p. A1.

Caplan, S. E. (2002). Problematic Internet use and psychosocial well-being: Development of a theory-based cognitive-behavioral measurement instrument. *Computers in Human Behavior, 18*, 553–575.

Cooper, A., Putnam, D. E., Planchon, L. A. & Boies, S. C. (1999). Online sexual compulsivity: Getting tangled in the net. *Sexual Addiction and Compulsivity: The Journal of Treatment and Prevention, 6*(2), 79–104.

Cooper, A., Scherer, C., Boies, S. C., & Gordon, B. (1999). Sexuality on the internet: From sexual exploration to pathological expression. *Professional Psychology: Research and Practice, 30*(2), 154–164.

Cooper, A. & Sportolari, L. (1997). Romance in cyberspace: Understanding online attraction. *Journal of Sex Education and Therapy, 22*(1), 7–14.

Davis, R. A. (2001). A cognitive behavioral model of pathological Internet use. *Computers in Human Behavior, 17*, 187–195.

Ferris, J. (2001). Social ramifications of excessive Internet use among college-age males. *Journal of Technology and Culture, 20*(1), 44–53.

Greenfield, D. (1999). *Virtual Addiction: Help for netheads, cyberfreaks, and those who love them.* Oakland, CA: New Harbinger Publication.

Griffiths, M. (1990). The cognitive psychology of gambling. *Journal of Gambling Studies, 6,* 31–42.

Griffiths, M. (1996). Technological addictions. *Clinical Psychology Forum. 76,* 14–19.

Hall, A. S., & Parsons, J. (2001). Internet addiction: College students case study using best practices in behavior therapy. *Journal of Mental Health Counseling, 23,* 312.

Hansen, S. (2002). Excessive Internet usage or "Internet addiction"? Diagnostic categories for student users. *Journal of Computer Assisted Learning, 18*(2), 235–239.

Hucker, S. J. (2004). Disorders of impulse control In W. O'Donohue and E. Levensky (Eds.), *Forensic Psychology.* New York: Academic Press, 72.

Keepers, G. A. (1990). Pathological preoccupation with video games. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*(1), 49–50.

LaRose, R., Mastro, D., & Easton, M. S (2001). Understanding Internet usage: A social-cognitive approach to uses and gratifications. *Social Science Computer Review, 19*(4), 395–413.

Lesieur, H. R., & Blume, S. B. (1993). Pathological gambling, eating disorders, and the psychoactive substance use disorders. *Comorbidity of Addictive and Psychiatric Disorders, 12,* 89–102.

Levy, S. (1997, December 30–January 6). Breathing is also addictive, *Newsweek,* pp. 52–53.

Morgan, W. (1979). Negative addiction in runners. *Physician and Sportsmedicine, 7,* 56–69.

Murphey, B. (1996, June). Computer addictions entangle students. *The APA Monitor,* p. 38.

National Council on Problem Gambling. (2008). *What is problem gambling?* Retrieved November 25, 2008, from www.ncpgambling.org.

National Gambling Impact Commission. (1998). *Research.* Retrieved November 25, 2008, from http://govinfo.library.unt.edu/ngisc/research/research.html.

Orzack, M. (1999). Computer addiction: Is it real or is it virtual? *Harvard Mental Health Letter, 15*(7), 8.

Peele, S., & Brodsky, A. (1979). *Love and addiction.* Scarborough, Ontario, Canada: New American Library of Canada.

Petry, N. (2006). Internet gambling: An emerging concern in family practice medicine? *Family Practice,* 23(4), 421–426.

Quittner, J. (1997, April 14). Divorce Internet style, *Time,* p. 72.

Rachlin, H. (1990). Why do people gamble and keep gambling despite heavy losses? *Psychological Science, 1,* 294–297.

Rheingold, H. (1993). *The virtual community: Homesteading on the electronic frontier.* Reading, MA: Addison-Wesley.

Scherer, K. (1997, August 18). *College life online: Healthy and unhealthy Internet use.* Paper presented at the 104th annual meeting of the American Psychological Association, Chicago.

Shotton, M. (1991). The costs and benefits of "computer addiction." *Behaviour and Information Technology, 10*(3), 219–230.

Turkle, S. (1995). *Life behind the screen: Identity in the age of the Internet.* New York: Simon & Schuster.

Twerski, A. (1990). *Addictive thinking: Understanding self-deception.* New York: HarperCollins.

Walker, M. B. (1989). Some problems with the concept of "gambling addiction": Should theories of addiction be generalized to include excessive gambling? *Journal of Gambling Behavior, 5,* 179–200.

Winn, M. (1983). *The plug-in drug.* New York: Viking Penguin.

Young, K. (1996). Internet addiction: The emergence of a new disorder. Paper presented at the 104th annual convention of the American Psychological Association, Toronto, Canada, August 15, 1996.

Young, K. S. (1998a). *Caught in the Net: How to recognize the signs of Internet addiction and a winning strategy for recovery.* New York: Wiley.

Young, K. S. (1998b) Internet addiction: The emergence of a new clinical disorder. *CyberPsychology and Behavior, 1,* 237–244.

Young, K. S. (1999) *The evaluation and treatment of Internet addiction.* In L. VandeCreek & T. Jackson (Eds.), *Innovations in clinical practice: A source book* (17, 19–31). Sarasota, FL: Professional Resource Press.

Young, K. S. (2001). *Tangled in the Web: Understanding cybersex from fantasy to addiction.* Bloomington, IN: Authorhouse.

Young, K. S. (2004) Internet addiction: The consequences of a new clinical phenomenon. In: K. Doyle (Ed.), *Psychology and the new media* (pp. 1–14). Thousand Oaks, CA: American Behavioral Scientist.

Young K. & Klausing, P. (2007). *Breaking free of the Web: Catholics and Internet addiction.* Cincinnati, OH: St. Anthony's Messenger Press.

Chapter **11**

Gambling Addictions

Mark Griffiths, PhD

Gambling is an activity that is popular across many cultures. Surveys of gambling on a national level have tended to conclude that there are more gamblers than nongamblers, but that most participants gamble infrequently (Abbott, Volberg, Bellringer, & Reith, 2004; Wardle et al., 2007). Estimates based upon survey data from countries all over the world indicate that the majority of people have gambled at some time in their life (Griffiths, 2007; Orford, Sproston, Erens, & Mitchell, 2003).

The introduction of national lotteries and new casinos, the proliferation of electronic gaming machines, and the introduction of remote gambling (e.g., Internet gambling, mobile phone gambling, interactive television gambling) have greatly increased the accessibility and popularity of gambling all over the world. As a consequence, the number of people seeking assistance with gambling-related problems has increased (Abbott et al., 2004). Government-commissioned studies in a number of countries including the United States, the United Kingdom (UK), Australia, and New Zealand have all concluded that (in general) increased gambling availability has led to an increase in problem gambling, although the relationship is complex and nonlinear (Abbott, 2007).

Despite the recognition of the complexity of gambling behavior, most research in the area has been confined to narrow areas of specialization. Singular theoretical perspectives tend to be adhered to (e.g., behaviorism, cognitivism, addiction theory), with few attempts to establish links or draw contrasts with other research programs (Griffiths & Delfabbro, 2001). Singular perspectives assume that a single explanation or theory is sufficient to explain every aspect

of gambling behavior and that rival perspectives are thereby misguided. Yet, as a number of authors assert, this may not be so (e.g., Griffiths, 2005; Griffiths & Larkin, 2004; Shaffer et al., 2004).

A number of authors have noted that gambling is a multifaceted rather than a unitary phenomenon (Griffiths & Delfabbro, 2001; Shaffer et al., 2004). Consequently, many factors may come into play in various ways and at different levels of analysis (e.g., biological, social, or psychological) (Griffiths, 2008). Theories may be complementary rather than mutually exclusive, which suggests that the limitations of individual theories might be overcome through the combination of ideas from different perspectives. This has often been discussed before in terms of recommendations for an eclectic approach to gambling (Griffiths, 1995) or a distinction between proximal and distal influences upon gambling (Walker, 1992). However, for the most part, such discussions have been descriptive rather than analytical, and so far, few attempts have been made to explain why an adherence to singular perspectives is untenable. There are generally three specific levels of analysis: social, psychological, and biological. This chapter will focus on the psychological aspects.

Central to this eclectic view, no single level of analysis is considered sufficient to explain either the etiology or the maintenance of gambling behavior. Moreover, this view asserts that all research is context bound and should be analyzed from a combined, or biopsychosocial, perspective (Griffiths, 2005, 2008). Variations in the motivations and characteristics of gamblers and in gambling activities themselves mean that findings obtained in one context are unlikely to be relevant or valid in another. In each of the following sections, broad details of each level of analysis are provided, followed by discussions of the limitations and interdependence of each theoretical approach and the implications for research and clinical interventions.

PATHOLOGICAL GAMBLING: PREVALENCE AND HISTORY

Estimates of the number of probable adult pathological gamblers vary from just under 1 percent in the UK to 1.1–1.9 percent in the United States and 2.3 percent in Australia (Wardle et al., 2007). These surveys have also indicated that pathological gambling is twice as common among males as it is among females, that nonwhites have higher rates than whites, and that those with low levels of education are more likely to be pathological gamblers (Abbott et al., 2004; Griffiths, 2007). In 1980, pathological gambling was recognized as a mental disorder in the third edition of the *Diagnostic and Statistical Manual* (*DSM–III*) under the section titled "Disorders of Impulse Control," along

with other illnesses such as kleptomania and pyromania (American Psychiatric Association, 1980). Adopting a medical model of pathological gambling in this way displaced the old image that the gambler was a sinner or a criminal.

Before the appearance of *DSM-III* (1980), the subject of pathological gambling had produced an expanding body of literature by psychiatrists, psychologists, psychoanalysts, and social workers, and the subject had appeared under a variety of labels including "neurotic," "compulsive," "addictive," "excessive," and "pathological" (Griffiths, 2007). There now seems to be an increased preference among professionals for the terms "problem" or "pathological gambling," as opposed to terms like "compulsive" or "addictive," which might suggest specific and homogenous etiologies.

In diagnosing the pathological gambler, *DSM–III* states that the individual is chronically and progressively unable to resist impulses to gamble and that gambling compromises, disrupts, or damages family, personal, and vocational pursuits. The behavior increases at times of stress, and associated features include lying to obtain money, committing crimes (forgery, embezzlement, fraud, etc.), and concealment from others of the extent of the individual's gambling activities. In addition, *DSM–III* stated that, for a diagnosis of pathological gambling, the gambling must not be due to antisocial personality disorder (see Table 11.1).

As Lesieur (1988) pointed out, these criteria were criticized for (1) a middle class bias, that is, that the criminal offences like embezzlement and income tax evasion were middle class offences, (2) a lack of recognition that many com-

Table 11.1
***DSM–III* Criteria for Pathological Gambling**

The criteria state that maladaptive gambling is indicated by the following:

A. The individual is chronically and progressively unable to resist impulses to gamble.
B. Gambling compromises, disrupts, or damages family, personal, and vocational pursuits, as indicated by at least three of the following:
   1. Arrest for forgery, fraud, embezzlement, or income tax evasion due to attempts to obtain money for gambling
   2. Default on debts or other financial responsibilities
   3. Disrupted family or spouse relationships due to gambling
   4. Borrowing money from illegal sources (loan sharks)
   5. Inability to account for loss of money or to produce evidence of winning money if this is claimed
   6. Loss of work due to absenteeism in order to pursue gambling activity
   7. Necessity for another person to provide money to relieve a desperate financial situation
C. The gambling is not due to antisocial personality disorder.

Source: American Psychiatric Association, 1980.

Table 11.2
DSM–III–R Criteria for Pathological Gambling

The criteria state that maladaptive gambling is indicated by at least four of the following:

1. Is frequently preoccupied with gambling or obtaining money to gamble.
2. Often gambles larger amounts of money or over a longer period than intended.
3. Needs to increase the size or frequency of bets to achieve the desired excitement.
4. Exhibits restlessness or irritability if unable to gamble.
5. Repeatedly loses money gambling and returns another day to win back losses ("chasing").
6. Makes repeated efforts to cut down or stop gambling.
7. Often gambles when expected to fulfill social, educational, or occupational obligations.
8. Has given up some important social, occupational, or recreational activity in order to gamble.
9. Continues to gamble despite inability to pay mounting debts, or despite other significant social, occupational, or legal problems that the individual knows to be exacerbated by gambling.

Source: American Psychiatric Association, 1987.

pulsive gamblers are self-employed, and (3) the exclusion of individuals with antisocial personality disorder. Lesieur recommended the same custom be followed for pathological gamblers as for substance abusers and alcoholics in the past, that is, to allow for simultaneous diagnosis with no exclusions. In addition, the criteria leave out the "problem gambler," who by self-admission, or by others' testimony, spends a disproportionate amount of time gambling but has yet to produce the serious consequences laid down in *DSM–III*. The new criteria were subsequently changed, in the *Diagnostic and Statistical Manual*, third edition, revised (*DSM–III–R*) (see Table 11.2), taking on board the criticisms and modeled extensively on substance abuse disorders, due to the growing acceptance of gambling as a bona fide addictive behavior (American Psychiatric Association, 1987).

However, Rosenthal (1989) conducted an analysis of the use of the *DSM–III–R* criteria by treatment professionals. It was reported that there was some dissatisfaction with the new criteria and that there was some preference for a compromise between *DSM–III* and *DSM–III–R*. As a consequence, the criteria were changed for the *Diagnostic and Statistical Manual*, fourth edition (*DSM–IV*; American Psychiatric Association, 1994; see Table 11.3). Preparations are already under way for *DSM–V*.

PHASES OF THE PATHOLOGICAL GAMBLER'S CAREER

The acquisition, development, and maintenance of pathological gambling is an area that is continually disputed. The exact causes and reasons for continu-

Table 11.3
DSM–IV–TR Criteria for Pathological Gambling

A. Persistent and recurrent maladaptive gambling behavior is indicated by five (or more) of the following:
   1. Is preoccupied with gambling (e.g., preoccupied with reliving past gambling experiences, handicapping or planning the next venture, or thinking of ways to get money with which to gamble).
   2. Needs to gamble with increasing amounts of money in order to achieve the desired excitement.
   3. Made repeated unsuccessful efforts to control, cut back, or stop gambling.
   4. Is restless or irritable when attempting to cut down or stop gambling.
   5. Gambles as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression).
   6. After losing money gambling, often returns another day to get even ("chasing" one's losses).
   7. Lies to family members, therapist, or others to conceal the extent of involvement with gambling.
   8. Has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling.
   9. Has jeopardized or lost a significant relationship, job, educational, or career opportunity because of gambling
   10. Relies on others to provide money to relieve a desperate financial situation caused by gambling.

B. The gambling behavior is not better accounted for by a manic episode.

Source: American Psychiatric Association, 1994.

ing gambling behavior seem to be dependent upon the individual, but there do seem to be some general underlying factors and recurring themes. Problem gambling generally begins in adolescence and may start following a major life stress, for example the death of a parent or birth of a first child (Griffiths, 2003a). Such events may induce a need to escape from the problems of reality.

Lesieur and Custer (1984) concluded that pathological gambling behavior consists of three stages—the winning phase, the losing phase, and the desperation phase. The winning phase normally begins with small but successful bets in adolescence. Early wins prompt more skillful gambling, which usually leads to larger winnings. Most social gamblers stop at this stage. However, after a considerable win maybe equaling or exceeding the individual's annual salary, the gambler accepts the thought that this can happen again.

The next stage—the losing phase—is characterized by unrealistic optimism on the gambler's part, and all bets placed are in an effort to recoup losses: this has been termed "the chase" by Lesieur (1984) . The result is that instead of cutting their losses, gamblers get deeper into debt, preoccupying themselves

with gambling, certain that a big win will repay their loans and solve all their problems. Family troubles (both marital and with relatives) begin, and illegal borrowing and other criminal activities usually start to occur, in an effort to get money (Lesieur, 1984). At this point in the pathological gambler's career, family and/or friends may bail out the gambler. Alienation from those closest to the pathological gambler characterizes the appearance of the final stage—the desperation phase. In a last-ditch, frenzied effort to repay debts, illegal criminal behavior reaches its height, and when there are finally no more options left, the gambler may suffer severe depression and have suicidal thoughts.

It is, then, usually at the insistence of the family (if not the courts) that the gambler must seek help. Because the pathological gambler is impatient, requiring immediate results, help should be aimed at priority areas, that is, the lessening of legal and financial difficulties, counseling to resolve family and marital problems, and most importantly hospitalization for desperate patients who are depressed and suicidal. In addition to this, Rosenthal (1989) has described a fourth phase called the hopeless or giving up phase. This is a phase in which gamblers know they cannot possibly retrieve their losses and they do not care, leading to play for play's sake.

EXPLANATIONS OF GAMBLING INVOLVEMENT

In general, research has consistently shown a positive relationship between the availability of gambling and both regular and problem gambling (Abbott, 2007; Griffiths, 2003b). Whenever new forms of gambling are introduced, or existing forms become more readily available, there is an increase in gambling, suggesting that the demand for gambling products is closely linked to their supply—although there are exceptions to this (Griffiths, Parke, & Rigbye, 2008). The larger the gambling industry infrastructure that is established (e.g., new venues), the larger the range of gambling products (e.g., through the application of new technologies), and the greater the industry's marketing efforts, the more likely people will be to gamble in the first place.

But why is gambling so popular? According to sociologists, gambling is an inherent component of human society (Goffman, 1967) and human beings have a natural penchant for play, risk, and competition. Gambling, they argue, fits easily with cultural values, virtues, and lifestyles, so that when gambling becomes more accessible and socially acceptable, more people will gamble (Abt, Smith, & McGurrin, 1985). As a form of social interaction, gambling provides a means by which people can escape the boredom of everyday life, adopt new roles, and enjoy the excitement of the action, namely, the suspense, anticipation,

and social reinforcement resulting from taking risks and being rewarded for one's daring (Abt & Smith, 1984).

Almost all surveys of gambling have shown that these broad motivational factors are central to gambling and that attitudes toward gambling are positively related to availability and cultural acceptability. However, this perspective fails to take into account many key findings and observations in gambling research. Research has consistently shown that people often gamble for reasons other than broad social and economic reasons (Griffiths, 1996). These other motivations may vary according to the personal characteristics of the gambler and the type of gambling activity. Finally, broad social and economic theories fail to explain why certain gambling activities are more popular or "addictive" than others.

Demographic variations in gambling participation have been observed since surveys were first administered. Typically, gambling has been more popular in lower socioeconomic groups, among Catholics rather than Protestants, among unmarried people, in younger age groups, and in men (Griffiths, 2007). Consistent with trends observed in overall participation rates, research has found that the incidence of gambling-related problems is considerably higher in lower socioeconomic groups and in younger people, and it is more likely to be associated with slot machines, one of the few activities that attract similar numbers of men and women (Griffiths & Delfabbro, 2001; Griffiths, 2007). Accordingly, understanding demographic variations in overall participation is vital if one is to estimate the likely social effects of expansion or product changes in existing gambling markets.

Variations in gambling preferences are thought to result from differences in both accessibility and motivation. Older people tend to choose activities that minimize the need for complex decision making or concentration (e.g., bingo, slot machines), whereas gender differences have been attributed to a number of factors, including variations in sex-role socialization, cultural differences, and theories of motivation (Delfabbro, 2000; Griffiths, 2007; Griffiths & Delfabbro, 2001).

Variations in motivation are also frequently observed among people who participate in the same gambling activity. For example, slot machine players may gamble to win money, for enjoyment and excitement, to socialize, and to escape negative feelings (Griffiths, 2002). Some people gamble for one reason only, whereas others gamble for a variety of reasons. A further complexity is that people's motivations for gambling have a strong temporal dimension; that is, they do not remain stable over time. As people progress from social to regular and finally to excessive gambling, there are often significant changes in their reasons for gambling. Whereas a person might have initially gambled to

obtain enjoyment, excitement, and socialization, the progression to problem gambling is almost always accompanied by an increased preoccupation with winning money and chasing losses.

STRUCTURAL AND SITUATIONAL CHARACTERISTICS OF GAMBLING ACTIVITIES

Another factor central to understanding gambling behavior is the structure of gambling activities. Griffiths and colleagues (Griffiths, 1993a, 1999; Parke & Griffiths, 2006, 2007) have consistently argued, gambling activities vary considerably in their structural characteristics, including the probability of winning, the amount of gambler involvement, the amount of skill that can be applied, the length of the interval between stake and outcome, and the magnitude of potential winnings. Structural variations are also observed within certain classes of activities, such as slot machines, where differences in reinforcement frequency, colors, sound effects, and machines' features can significantly influence the profitability and attractiveness of machines. Each of these structural features may (and almost certainly does) have implications for gamblers' motivations and the potential "addictiveness" of gambling activities.

For example, skillful activities that offer players the opportunity to use complex systems, study the odds, and apply skill and concentration appeal to many gamblers because their actions can influence the outcomes. Such characteristics attract people who enjoy a challenge when gambling. They may also contribute to excessive gambling if people overestimate the effectiveness of their gambling systems and strategies (see discussion of cognitive theories, below). Chantal and Vallerand (1996) have argued that people who gamble on these activities (e.g., racing punters) tend to be more intrinsically motivated than lottery gamblers in that they gamble for self-determination (to display their competence and to improve their performance). People who gamble on chance activities, such as lotteries, usually do so for external reasons (to win money or escape from problems) (Griffiths, 2007).

Although many slot machine players also overestimate the amount of skill involved in their gambling, other motivational factors (such as the desire to escape worries or to relax) tend to predominate. Thus, excessive gambling on slot machines may be more likely to result from people becoming conditioned to the tranquilizing effect brought about by playing rather than from just the pursuit of money (Griffiths, 2002).

Another vital structural characteristic of gambling is the continuity of the activity: namely, the length of the interval between stake and outcome (Parke & Griffiths, 2007). In nearly all studies, it has been found that continuous activi-

ties (e.g., racing, slot machines, casino games) with a more rapid play-rate are more likely to be associated with gambling problems (Parke & Griffiths, 2006). The ability to make repeated stakes in short time intervals increases the amount of money that can be lost and also increases the likelihood that gamblers will be unable to control their spending. Such problems are rarely observed in noncontinuous activities, such as weekly or biweekly lotteries, in which gambling is undertaken less frequently and in which outcomes are often unknown for days. Consequently, it is important to recognize that the overall social and economic impact of expansion of the gambling industry will be considerably greater if the expanded activities are continuous rather than noncontinuous.

Other factors central to understanding gambling behavior are the situational characteristics of gambling activities. These are the factors that often facilitate and encourage gambling in the first place (Griffiths & Parke, 2003). Situational characteristics are primarily features of the environment (e.g., accessibility factors such as the location of the gambling venue, the number of venues in a specified area, and possible membership requirements) but can also include internal features of the venue itself (e.g., décor, heating, lighting, color, background music, floor layout, refreshment facilities) or facilitating factors that may influence gambling in the first place (e.g., advertising, free travel and/or accommodation to the gambling venue, free bets or gambles on particular games) or influence continued gambling (e.g., the placing of a cash dispenser on the casino floor, free food and/or alcoholic drinks while gambling) (Abbott, 2007; Griffiths & Parke, 2003).

These variables may be important in both the initial decision to gamble and the maintenance of the behavior. Although many of these situational characteristics are thought to influence vulnerable gamblers, there has been very little empirical research into these factors, and more research is needed before any definitive conclusions can be arrived at on the direct or indirect influence on gambling behavior and on whether vulnerable individuals are any more likely to be influenced by these particular types of marketing ploys (Griffiths, 2007).

THEORIES OF GAMBLING BEHAVIOR

Although sociological, situational, and demographic factors can explain why some people are more likely to gamble than others, these theories cannot explain why some people gamble more than others or what factors contribute to behavior maintenance in gambling. Psychological theories become important at this level. Research in this area is remarkably diverse. Almost every major branch of psychology (e.g., cognitivism, behaviorism, Freudian theory, addiction theory) has been utilized in an attempt to understand gambling. Despite this diversity,

it is possible to distinguish two broad, general perspectives: first, theories that attribute ongoing behavior and excessive gambling to habitual processes that are the consequences of gambling; second, theories that state that variations in behavior result from variations in the characteristics or makeup, of individual gamblers (Griffiths & Delfabbro, 2001). In other words, whereas the first places a stronger emphasis upon psychological determinants of gambling, the second emphasizes biological differences between individuals.

Central to psychological explanations is the idea that every person who gambles has the potential to become a problem gambler. This is because gambling activities are difficult to resist by their very nature: excitement, risk taking, and the possibility of monetary gains. The more a person gambles, the more difficult it becomes to resist the temptation to commence a gambling session or stop once gambling has commenced (Dickerson, 1989). Accordingly, it has been suggested that there is no neat distinction between problem gambling and normal gambling; rather, there is a continuum from social gambling to regular gambling to problem gambling.

People who gamble regularly may display many of the same behaviors as people with gambling problems, although to a lesser degree. This view gives rise to conceptualizations of problem gambling that emphasize the developmental and habitual nature of problem gambling behavior rather than individual pathology. This perspective avoids terms such as compulsive, addiction, or pathology, in preference for terms such as impaired control (Dickerson & O'Connor, 2006). Although researchers' views differ concerning the psychological mechanisms behind loss of control, three general classes of theory will be used to illustrate the limitations of psychological accounts. They are behaviorist theories that explain persistent gambling as a conditioned process; need-state models that see gambling as a form of psychological or physiological dependence; and cognitive theories that attribute excessive gambling to erroneous beliefs about the potential profitability of gambling.

BEHAVIORIST APPROACHES

Both classical and operant conditioning principles have been applied to the study of gambling. In operant explanations for problem gambling (Delfabbro & Winefield, 1999), persistent gambling is seen as a conditioned behavior maintained by intermittent schedules of reinforcement, most likely a variable-ratio schedule. This involves the provision of infrequent rewards after varying numbers of responses. On the other hand, proponents of classical conditioning models argue that people continue to gamble as a result of becoming conditioned to the excitement or arousal associated with gambling, so that they feel

bored, unstimulated, and restless when they are not gambling. Both the classical and operant perspectives have been central to the development of measures of impaired control over gambling (Dickerson & O'Connor, 2006) and clinical interventions using desensitization, aversive conditioning, and satiation techniques. In each of these examples, it is assumed that the more a person gambles, the more a person's behavior is dictated by factors beyond the person's control.

Despite evidence supporting both theories, neither is entirely satisfactory on its own. Classical conditioning theory seems useful in explaining people's motivation to commence a gambling session but appears less useful in explaining persistent gambling behavior. Conversely, while operant conditioning might explain ongoing behavior, it appears less useful in explaining why people commence gambling or recommence gambling after a prolonged period of abstinence (Griffiths, 1995). Researchers have also raised questions about the extent to which gambling behavior adheres to operant theory at all, since gamblers lose more than they win and because reinforcement magnitudes are not independent of player responses, for example, stake sizes (Delfabbro & Winefield, 1999). Nevertheless, the importance of subtle variations in machine characteristics in influencing behavior reinforces the role of operant conditioning in the maintenance of behavior, although perhaps in more subtle ways than has been envisaged.

It is important to recognize that these theories cannot stand in isolation. As with other psychological theories, conditioning theories cannot explain why people exposed to similar stimuli respond differently; why some gamble whereas others do not; or why some people gamble more than others. In addition, the effectiveness or strength of the conditioning effect may be a function of motivational factors and type of activity. Some but not all people gamble for excitement or relaxation, and as discussed above, people satisfy these needs by engaging in different activities. Thus, it is unlikely that classical conditioning will affect all types of gambling or gamblers. Similar difficulties plague attempts to develop general operant theories of gambling. Some activities appear to suit this form of explanation more than others. Examples include slot machines and scratch tickets, where there is a short time interval between stake and outcome, and where outcomes are entirely determined by chance. It seems more difficult to apply these principles to skilled gambling games such as blackjack, poker, and sports betting, where player decisions can significantly influence outcomes.

NEED-STATE MODELS AND THEORIES OF ADDICTION

Much of the discussion relating to classical conditioning also applies to need-state theories of gambling, which assume that people gamble to escape unpleasant

feeling states such as anxiety, depression, and boredom. These perspectives have been applied to all facets of gambling, including involvement, ongoing behavior, and excessive gambling. They are incorporated into the *DSM–IV* classification of pathological gambling (i.e., gambling as a way of escaping from problems or intolerable feeling states). Although not all researchers agree that these motivations signify the existence of a physiological addiction (Walker, 1989), most agree that people can become psychologically addicted to gambling.

The concept of arousal has been studied most extensively (e.g., Coventry & Hudson, 2001; Diskin & Hodgins, 2003; Griffiths, 1993b), but the results have not been consistent. Arousal increases have been observed in some studies but not in others, and most increases have been relatively small. Variations in arousal have co-varied reliably neither with the persistence of behavior nor with the onset of gambling sessions. Walker (1992) has questioned the explanatory value of arousal theories, arguing that the excitement of gambling is unlikely to be independent of people's desire to win money.

Similar problems have plagued attempts to associate gambling with anxiety and depression. While a considerable number of studies (Blaszczynski & McConaghy, 1989; Blaszczynski, McConaghy & Frankova, 1990; Dickerson, Cunningham, Legg England, & Hinchy, 1991; Dickerson, Hinchy, Legg England, Fabre, & Cunningham, 1992; Ramirez, McCormick, Russo, & Taber, 1984) have revealed that negative mood states commonly accompany gambling or predict the duration of gambling sessions, most analyses have been confined to problem gamblers and high-frequency gamblers. For this reason, it is unclear whether these mood states are also associated with less frequent gambling. Moreover, it is not possible to determine whether mood states precede gambling or arise as a consequence of gambling. Indeed, as Walker (1992) points out, it may be that gamblers become depressed as a result of losing more money than they can afford.

Again, the temporal dimension suggests that the role of mood states is unlikely to be independent of the gambler's characteristics. As with arousal, it is unlikely that avoidance of negative feeling states will be common to all activities or all gamblers. For example, slot machines appear to reduce anxiety, whereas racing provides arousal and excitement. In addition, variations in gambling motivation among participants involved in the same activity suggest that not all people gamble to satisfy unfulfilled needs. It is also unclear why some people apparently have a greater need for arousal or relaxation than others, and whether this is sufficient to explain differences between normal and excessive gambling. It is important to place behavior in a social context in order to understand how gambling compensates for, or assuages, problems or deficits experienced in other areas of life. Alternatively, as will be suggested later in this

chapter, it may be useful to look for dispositional or biological differences to explain the varying motivations and behavior of individual gamblers.

COGNITIVE THEORIES

Despite the fact that the odds for almost all activities are weighted strongly in favor of the house, gamblers continue to believe they can win money from gambling. This observation leads to the conclusion that gambling may be maintained by irrational or erroneous beliefs. For example, people overestimate the extent to which they can predict or influence gambling outcomes and tend to misjudge how much money they have won or lost. This hypothesis has been confirmed in numerous studies (Langer, 1975; Langer & Roth, 1983) showing that people overestimate the degree of skill or control that can be exerted in chance activities, and also in studies using the so-called thinking aloud method (Griffiths, 1994), which reveal high levels of irrationality in verbalized statements made during gambling sessions. These findings have been confirmed not only under laboratory conditions but also in ecologically valid gambling settings, using regular gamblers (Griffiths, 1994; Ladouceur, Gaboury, Bujold, Lachance, & Tremblay, 1991).

Based upon these findings, it has been suggested that irrational thinking may be related to problematic gambling behavior (Ladouceur & Walker, 1996; Wagenaar, 1988), with persistent behavior thought to be the result of people's overconfidence in their ability to win money (Wagenaar, 1988; Walker, 1992). Evidence suggests that problem gamblers frequently overestimate the amount of control and skill involved in gambling (Griffiths, 1994). Unfortunately, some of these observations have also been made using students with no gambling experience, indicating that irrational beliefs are not positively related to level of gambling involvement (Ladouceur et al., 1991). A further problem is that irrationality does not appear to co-vary with other observable facets of gambling, such as the level of risk taking or reinforcement frequency. Alternatively, where irrationality relates positively to involvement, few differences in behavior have been observed. Consequently, Dickerson and Baron (2000) have concluded that irrational thinking is probably more a reflection of demand characteristics than a rational underlying behavior. A lot of what people say may result only from the difficulty of trying to come up with rational, meaningful statements in chance-determined situations.

In additional to these conceptual difficulties, it is also possible that contextual factors play a role in cognitive research. For example, Griffiths (1994) found that regular players had greater difficulty than occasional players in verbalizing their thoughts while they were gambling. Regular players seemed capable of gambling

without attending to what they were doing, suggesting (a) that cognitive processes did not play a major role in the maintenance of their behavior, or (b) that the original justifications or rationales for behavior were less accessible. In either case, Griffiths's observations suggested that temporal factors (namely, how long a person has been gambling) appear to be important. Therefore, all other things being equal, it appears that valid comparisons cannot be drawn between gamblers with differing levels of gambling experience, because what holds for infrequent gamblers might not hold for regular players, and vice versa.

Finally, it is again important to observe that cognitive theories need to take structural variations in activities into account. Many cognitive processes thought to underlie gambling behavior (e.g., overestimations of control, biased attributions) are more likely to be observed when activities are perceived as having some skill component (Griffiths, 1995). In some activities, there is a genuine possibility for skillful play (e.g., racing, blackjack, table poker). The more people play or know about these activities, the greater is their awareness of the skills involved. Thus, beliefs about control and skill are neither completely irrational nor consistent across players. Instead, in these situations, researchers must examine the quality of play: for example, looking at the extent to which the person adheres to optimal strategies rather than looking for evidence of irrational thinking (Keren & Wagenaar, 1985).

Even in activities where outcomes are chance-determined, there are likely to be variations in the extent to which gamblers perceive that the outcomes are solely chance-determined (e.g., roulette and craps are probably more likely to be perceived as skillful than Australian slot machines, because of the greater complexity of the rules and the possibility for variations in playing strategy). Therefore, it may be ineffective to compare results across studies using different chance activities without controlling for variations in perceived skill.

It should also be noted that social and psychological explanations are insufficient to explain the full complexity of gambling behavior; that there are many other theoretical accounts examining problem gambling and ambling addictions, including those using biological and dispositional theories, sociological theories, and economic theories (Griffiths & Delfabbro, 2001), and that an unified theory of addiction will be complex and biopsychosocial (Griffiths, 2005, 2008). Whether ongoing behavior is explained in terms of behaviorism, need-state models, or cognitive theories, it remains unclear why one person gambles more heavily than another. In other words, while it seems likely that increased involvement with gambling is likely to contribute to loss of control over behavior, development of irrational beliefs, and greater psychological dependence, it is important to determine what makes some gamblers more susceptible to these outcomes than others. It is here that research into biological and personality

factors becomes important. Central to this research is the effort to ascertain whether pathological gamblers possess qualities that would predispose them to excessive gambling.

Biological and dispositional accounts assume that such factors should override environmental or contextual factors and allow for the development of a general theory of gambling addiction. However, this is clearly not so. Apart from the conceptual difficulties associated with determining a causal relationship between characteristics and behavior, dispositional theories are unable to account for the full diversity of gambling patterns and behavior. They fail to explain demographic differences in preference for activities and variations in motivation. Neither can they explain why some activities are more "addictive" than others and why the structural characteristics of specific activities (e.g., slot machines) can influence behavior. Therefore, it appears that excessive gambling is likely to result from both dispositional and psychological factors and the complex interaction between them. Psychological explanations must play a role because of the obvious importance of external factors (e.g., environmental and situational variables) in the development of gambling habits. However, it is also clear that internal factors influence the way in which certain individuals respond to these situations. The implications of this observation for the study and treatment of problem gambling are discussed below.

PATHWAYS INTO GAMBLING ADDICTION

Blaszczynski and Nower (2002) postulated a "pathway" model of the determinants of problem gambling based upon a series of clinical observations with problem gamblers and integration with the literature. They argued that there are common influences that affect all problem gamblers, such as availability and access, classical and operant conditioning reinforcements, arousal effects, and biased cognitive schemas. However, they suggested that there are three distinct pathways into problem gambling, representing three primary motivating forces that drive different problem gamblers to gamble. The members of the first group of gamblers, *behaviorally conditioned problem gamblers*, are not pathologically disturbed but instead gamble excessively as a result of poor decision-making strategies and bad judgments. Any features such as preoccupation with gambling, chasing, depression, anxiety, and related substance abuse are seen as the consequence, not the cause, of their excessive gambling. These gamblers are usually motivated to seek and attend treatment, and to reestablish controlled levels of gambling posttreatment.

The members of the second group, *emotionally vulnerable problem gamblers*, are characterized by a predisposition to be emotionally susceptible. This group

uses gambling as a means of modifying mood states and/or in order to meet specific psychological needs. These gamblers display higher levels of premorbid psychopathology including depression, anxiety, substance dependence, and deficits in coping or managing stress. They tend to engage in avoidant or passive aggressive behavior and to use gambling as a means of emotional relief through dissociation and mood modification. The psychological dysfunction in these gamblers makes them more resistant to treatment and not suitable for controlled gambling. Treatment must focus the underlying vulnerabilities as well as the gambling behavior.

The members of the third group, *antisocial impulsivist' problem gamblers*, have biological dysfunctions, either neurological or neurochemical. They also possess psychosocial vulnerabilities similar to the second group of gamblers mentioned above. However, they are characterized by antisocial personality disorder and impulsivity and/or attention-deficit disorders. It is argued that these gamblers have a propensity to seek out rewarding activities (such as gambling) in order to receive stimulation. They tend to be clinically impulsive and display a broad range of problems independent of their gambling. These problems include substance abuse, low tolerance for boredom, sensation seeking, criminal acts, poor relationship skills, family history of antisocial behavior, and alcoholism. Gambling usually begins at an early age, has a rapid onset, and occurs in binges. The members of this third group are less motivated to seek treatment, have poor compliance rates, and respond poorly to all interventions. All three groups are affected by environmental variables, conditioning effects, and cognitive processes. However, in terms of treatment intervention, each group will have specific needs.

FURTHER IMPLICATIONS FOR RESEARCH AND INTERVENTIONS

In summary, it seems that gamblers are first influenced by sociological factors: for example, the availability of gambling opportunities and the attitudes and habits of parents, friends, and peer groups as well as a lack of alternative activities. During the middle stages of development, there are many factors that heavily influence the maintenance of gambling behavior. Three of these factors are schedules of reinforcement, the escape qualities of gambling, and cognitive biases, all of which have been summarized in this chapter. While it remains unclear exactly how some people come to gamble excessively, it is agreed that persistent gambling eventually leads to a desperate "spiral of options" (Lesieur, 1984), in which gambling is largely maintained by the desire to win money, recover losses, and pay back debts. Gambling is thus a complex, multidimen-

sional activity that is unlikely to be explained by any single theory. Instead, research into this subject is best served by a biopsychosocial model that stresses the individual and idiosyncratic nature of the development of gambling problems and emphasizes the role of contextual factors internal and external to the process of gambling (Griffiths, 2005, 2008).

Recognition of this complexity has important implications for gambling research, in terms of both the selection of samples and the data analysis. First, the existence of structural variations in activities suggests that results obtained using one activity cannot be generalized to other activities that are not structurally equivalent. Existing research suggests that continuity and the element of skill involved are two factors that must be similar in order for valid comparisons to be made. Second, studies of gambling motivation are unlikely to be valid unless both individual and situational factors are taken into account. Since motivations differ across demographic groups (e.g., different genders and ages), across activities, and over time, studies must ensure that these factors are controlled for before conclusions are drawn. Samples should contain equal numbers of men and women of a similar age with similar levels of gambling experience. In situations where this cannot be achieved, gender, age, and experience should be used as co-variants or as the first variables in regression analyses.

Third, in recognition that personality may influence the strength of experimental effects, it is important that researchers match comparison groups in terms of these variables. For example, cognitive experiments investigating the illusion of control should include measures of desirability for control, whereas arousal experiments should include measures of gambling motivation (Griffiths & Delfabbro, 2001). In addition, researchers should not assume that biological differences or psychological factors will explain all gambling behavior. Instead, it may be useful to explore the interaction between these different levels of analysis, for example, by examining whether variations in the structural characteristics of activities (e.g., reinforcement frequency) affect people with, or without, the characteristic under observation.

IMPLICATIONS FOR PREVENTION, INTERVENTION, AND TREATMENT

Since sociological factors appear to be critical in the acquisition of gambling behavior and subsequent gambling addiction, prevention needs to be aimed at the social and situational antecedents. These can be approached from a number of levels (e.g., societal, school, family, individual), some of which may be more practical than others. Since problem gamblers start gambling at a significantly earlier age than nonpathological gamblers, an obvious step would be for

governments to legislate against young people gambling (i.e., below 18 years of age). A blanket ban on gambling would, in most cases, reduce acquisition until at least late adolescence. Both parents and peers may model gambling; therefore, the family's role in maintaining gambling behavior should be addressed in therapy, and prevention plans should aim to increase the gambler's contact with nongambling peers. Also, evidence or knowledge of a gambler's own negative thoughts or feelings about gambling behavior and irrational biases may provide useful cues for behavior modification.

These findings have led to suggestions designed to enhance educational awareness of the dangers of gambling, not only among children and adolescents but also among parents, guardians, and teachers. Although recommendations of this nature typically tend to focus upon the need for greater awareness of the true odds and the nonprofitability of gambling, this approach needs to be applied with caution. It is quite possible for education to have the opposite effect, namely, to increase students' knowledge of how to gamble. In addition, it is questionable whether knowing the true odds has a significant effect upon dissuading people from gambling, given that many problems gamblers are well educated and have, in some cases, some knowledge of basic mathematics. For many, the belief that they are inherently lucky or different from others helps maintain their interest in gambling. Accordingly, educational campaigns that focus upon the negative consequences of gambling and alternatives to it may have greater success. While these sorts of campaigns are unlikely to prevent gambling in all young people, they might reduce (a) the total number of adolescents who start to gamble and (b) the amount of time an adolescent spends gambling.

The fact that some gamblers are socially rewarded for gambling cannot be altered directly, but more adaptive personal and social skills can be taught as responses to stress (i.e., emotional antecedents), for example, relaxation, assertion, and social skills training. Alternatively, where people seek the company of other gamblers as a way to escape from unpleasant feeling states or life stress, the development of alternative interests, hobbies, and social networks should be afforded priority during intervention. This approach could also be extended to people who gamble alone. An essential aspect of treatments should be to identify and address the factors that are antecedents to gambling, those that provide the underlying motivation and social and cultural context in which the behavior has developed. Only when these are addressed can treatment be extended to more specific psychological aspects of the behavior itself. This is because these broader social and structural factors influence a person's exposure to gambling, opportunities to gamble. and ability to recover. Detailed analysis of the person's daily schedule and the nature and extent of available social supports is essential during this phase of treatment.

Viewing problem gambling as a biopsychosocial process recognizes the diversity of psychological factors involved in maintaining the behavior as well as the fact that problem gamblers are not a homogeneous group; in fact, there appear to be a number of subtypes (Griffiths, 2005). This has major treatment implications. For instance, Griffiths (1995) outlined two very different types of gamblers. The first type appeared to be addicted to gambling itself and played to test skill, to gain social rewards, and mostly for excitement (i.e., the "buzz" or "high"). This was termed a primary addiction and appears to be a mixture of subcultural and impulsivist types of gamblers (Moran, 1970; Blaszczynski & Nower, 2002). Identifying the environmental, situational, or emotional factors that precede a gambling session are important to know in administering any therapeutic intervention. Imaginal desensitization, counterconditioning, and situational exposure are methods that have been used to teach people to resist the urge to gamble. Of course, therapists differ in their views concerning the factors underlying this urge. Whereas some emphasize the learned or conditional quality of the behavior and emphasize the role of stimulus control, others may emphasize irrational beliefs or the gambler's desire to obtain physiological stimulation from the activity.

Furthermore, as emphasized by Griffiths (1995), a second type of gambler may gamble for the reasons described earlier, such as escape. These gamblers are usually depressed and socially isolated, and could be described as having a secondary addiction, in that the player uses gambling as an escape from a primary problem (e.g., broken home, relationship crisis, etc.). It seems that this type of escape gambler is not confined to the United Kingdom. This type appears to be a mixture of neurotic and symptomatic"(i.e., emotionally vulnerable) types (Moran, 1970; Blaszczynski & Nower, 2002). If the primary problem is resolved by excessive gambling, then playing should disappear. This distinction between types of gamblers obviously has clinical usefulness and may also help explain conflicting research, some of which states that gambling is a social activity and some of which states that it is a solitary activity. As discussed above, addicted gamblers are likely to benefit from any intervention that tries to find alternative activities to take the place of gambling. Like Griffiths's writing, more recent writings by Blasczcynski and Nower (2002), suggest there are different subtypes of gamblers that follow different pathways.

CONCLUSIONS

Examining gambling and gambling addiction as a biopsychosocial behavior makes it evident that individual differences and broader contextual factors must be considered and not ignored (Griffiths, 2005). This chapter provides

evidence that a narrow focus upon one theoretical perspective in research and clinical interventions may, in many cases, not be justified. Such an approach fails to consider the interrelationships between different levels of analysis. It would be of limited value to many gamblers whose problems have a different etiology, which may be multifaceted. As Gambino and Shaffer (1979) pointed out nearly three decades ago, individuals are self-determining agents, and therefore, a taxonomy of situations must be developed to describe the vast majority of contexts and conditions in which people use substances or engage in habitual behaviors to alter their perceived experience.

Gambino and Shaffer also make the important point that these behaviors are not completely self-developed or understood by the people themselves and should be examined more broadly. This is because gambling becomes a habitual behavior. Since the perceived experience of the individual can change over time, it is possible that focusing upon the self-reported factors currently maintaining the behavior does not provide insights into the factors that led to the development of the behavior. Thus, when one takes a biopsychosocial view, it becomes possible to perceive individual gambling in terms of its broader social and cultural context. This approach also suggests that different perspectives and approaches may be beneficial, as long as they appear to apply to the particular gambler concerned. Moreover, it indicates that a variety of treatments could be beneficial simultaneously.

REFERENCES

Abbott, M. W. (2007). Situational factors that affect gambling behavior. In G. Smith, D. Hodgins, & R. Williams (Eds.), *Research and measurement issues in gambling studies* (pp. 251–278). New York: Elsevier.

Abbott, M. W., Volberg, R. A., Bellringer, M., & Reith, G. (2004). *A review of research aspects of problem gambling.* London: Responsibility in Gambling Trust.

Abt, V., & Smith, J. F. (1984). *Gambling as play.* Annals of the American Academy of Political and Social Sciences, 474, 122–132.

Abt, V., Smith, J. F., & McGurrin, M. C. (1985). Ritual, risk, and reward: A role analysis of race track and casino encounters. *Journal of Gambling Behavior, 1,* 64–75.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., revised). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Blaszczynski, A., & McConaghy, N. (1989). Anxiety and/or depression in the pathogenesis of addictive gambling. *International Journal of the Addictions, 24,* 337–350.

Blaszczynski, A. P., McConaghy, N., & Frankova, A. (1990). Boredom proneness in pathological gamblers. *Psychological Reports, 67,* 35–42.

Blaszczynski, A., & Nower, L. (2002). A pathways model of pathological gambling. *Addiction, 97,* 487–500.

Chantal, Y., & Vallerand, R. J. (1996). Skill versus luck: A motivational analysis of gambling involvement. *Journal of Gambling Studies, 12,* 407–418.

Coventry, K. R., & Hudson, J. (2001). Gender differences, physiological arousal and the role of winning in fruit machine gamblers. *Addiction, 96,* 871–879.

Delfabbro, P. H. (2000). Gender differences in Australian gambling: A critical summary of sociological and psychological research. *Australian Journal of Social Issues, 35,* 145–158.

Delfabbro, P. H., & Winefield, A. H. (1999). Poker machine gambling: An analysis of within session characteristics. *British Journal of Psychology, 90,* 425–439.

Dickerson, M. G. (1989). Gambling: A dependence without a drug. *International Review of Psychiatry, 1,* 157–172.

Dickerson M. G., & Baron, E. (2000). Contemporary issues and future directions for research into pathological gambling. *Addiction, 95,* 1145–1159.

Dickerson, M. G., Cunningham, R., Legg England, S., & Hinchy, J. (1991). On the determinants of persistent gambling: III. Personality, prior mood, and poker machine play. *International Journal of the Addictions, 26,* 531–548.

Dickerson, M. G., Hinchy, J., Legg England, S., Fabre, J., & Cunningham, R. (1992). On the determinants of persistent gambling behaviour. I. High-frequency poker machine players. British *Journal of Psychology, 83,* 237–248.

Dickerson, M. G., & O'Connor, J. (2006). *Gambling as an addictive behaviour: Impaired control and its relationship to other variables.* Cambridge, England: Cambridge University Press.

Diskin, K. M., & Hodgins, D. (2003). Psychophysiological and subjective arousal during gambling in pathological and non-pathological video lottery gamblers. *International Gambling Studies, 3,* 37–51.

Gambino, B., & Shaffer, H. (1979). The concept of paradigm and the treatment of addiction. *Professional Psychology, 10,* 207–223.

Goffman, E. (1967). *Interaction Ritual.* New York: Anchor.

Griffiths, M. D. (1993a). Fruit machine gambling: The importance of structural characteristics. *Journal of Gambling Studies, 9,* 101–120.

Griffiths, M. D. (1993b). Tolerance in gambling: An objective measure using the psychophysiological analysis of male fruit machine gamblers. *Addictive Behaviors, 18,* 365–372.

Griffiths, M. D. (1994). The role of cognitive bias and skill in fruit machine gambling. *British Journal of Psychology, 85,* 351–369.

Griffiths, M. D. (1995). *Adolescent Gambling.* London: Routledge.

Griffiths, M. D. (1996). Pathological gambling: A review of the literature. *Journal of Psychiatric and Mental Health Nursing, 3,* 347–353.

Griffiths, M. D. (1999). Gambling technologies: Prospects for problem gambling. *Journal of Gambling Studies, 15,* 265–283.

Griffiths, M. D. (2002). *Gambling and gaming addictions in adolescence*. Leicester, England: British Psychological Society/Blackwells.

Griffiths, M. D. (2003a). Adolescent gambling: Risk factors and implications for prevention, intervention, and treatment. In D. Romer (Ed.), *Reducing adolescent risk: Toward an integrated approach* (pp. 223–238). London: Sage.

Griffiths, M. D. (2003b). Problem gambling. *The Psychologist: Bulletin of the British Psychological Society, 16*, 582–584.

Griffiths, M. D. (2005). A "components" model of addiction within a biopsychosocial framework. *Journal of Substance Use, 10*, 191–197.

Griffiths, M. D. (2007). *Gambling addiction and its treatment within the NHS*. London: British Medical Association.

Griffiths, M. D. (in press). The biopsychosocial and "complex" systems approach as a unified framework for addiction. *Behavioral and Brain Sciences*.

Griffiths, M. D., & Delfabbro, P. (2001). The biopsychosocial approach to gambling: Contextual factors in research and clinical interventions. *Journal of Gambling Issues, 5*, 1–33. Retrieved December 2, 2008, from http://www.camh.net/egambling/issue5/feature/index.html.

Griffiths, M. D., & Larkin, M. (2004). Conceptualizing addiction: The case for a "complex systems" account. *Addiction Research and Theory, 12*, 99–102.

Griffiths, M. D., & Parke, J. (2003). The environmental psychology of gambling. In G. Reith (Ed.), *Gambling: Who wins? Who loses?* (pp. 277–292). Amherst, New York : Prometheus Books.

Griffiths, M. D., Parke, J., & Rigbye, J. (2008). *Key factors in distinguishing regional casinos from other venues based on potential impact on problem gambling*. Report for Manchester City Council. Manchester: Manchester City Council.

Keren, G., & Wagenaar, W. A. (1985). On the psychology of playing blackjack: Normative and descriptive considerations with implications for decision theory. *Journal of Experimental Psychology, 114*, 133–158.

Ladouceur, R., Gaboury, A., Bujold, A., Lachance, N., & Tremblay, S. (1991). Ecological validity of laboratory studies of videopoker gaming. *Journal of Gambling Studies, 7*, 109–116.

Ladouceur, R., & Walker, M. B. (1996). A cognitive perspective on gambling. In P. M. Salkovkis (Ed.), *Trends in cognitive and behavioural therapies* (pp. 89–120). London: John Wiley and Sons.

Langer, E. J. (1975). The illusion of control. *Journal of Personality and Social Psychology, 32*, 311–328.

Langer, E. J., & Roth, J. (1983). Heads you win, tails it's chance: The illusion of control as a function of the sequence of outcomes in a purely chance task. *Journal of Personality and Social Psychology, 32*, 951–955.

Lesieur, H. (1988). Altering the DSM–III criteria for pathological gambling. *Journal of Gambling Behavior, 4*, 38–47.

Lesieur, H. R. (1984). *The chase: Career of the compulsive gambler* (2nd ed.). Rochester, VT: Schenkman Books.

Lesieur, H. R., & Custer, R. L. (1984). Pathological gambling: Roots, phases and treatment. *Annals of the American Academy of Political and Social Sciences, 474,* 146–156.

Moran, E. (1970). Varieties of pathological gambling. *British Journal of Psychiatry, 116,* 593–597.

Orford, J., Sproston, K., Erens, B., & Mitchell, L. (2003). *Gambling and problem gambling in Britain*. Hove, England: Brunner-Routledge.

Parke, J., & Griffiths, M. D. (2006). The psychology of the fruit machine: The role of structural characteristics (revisited). *International Journal of Mental Health and Addiction, 4,* 151–179.

Parke, J., & Griffiths, M. D. (2007). The role of structural characteristics in gambling. In G. Smith, D. Hodgins, & R. Williams (Eds.), *Research and measurement issues in gambling studies* (pp. 211–243). New York: Elsevier.

Ramirez, L. F., McCormick, R. A., Russo, A. M., & Taber, J. I. (1984). Patterns of substance abuse in pathological gamblers undergoing treatment. *Addictive Behaviors, 8,* 425–428.

Rosenthal, R. J. (1989, November). *Compulsive gambling.* Paper presented at the California Society for the Treatment of Alcoholism and Other Drug Dependencies, San Diego.

Shaffer, H. J., LaPlante, D. A., LaBrie, R. A., Kidman, R. C., Donato, A. N., & Stanton, M. V. (2004). Towards a syndrome model of addiction: Multiple expressions, common etiology. *Harvard Review of Psychiatry, 12,* 1–8.

Wagenaar, W. A. (1988). *Paradoxes of gambling behaviour.* London: Erlbaum.

Walker, M. B. (1989). Some problems with the concept of "gambling addiction": Should theories of addiction be generalised to include excessive gambling? *Journal of Gambling Behavior, 5,* 179–200.

Walker, M. B. (1992). *The psychology of gambling.* Oxford, England: Pergamon Press.

Wardle, H., Sproston, K., Erens, B., Orford, J., Griffiths, M., Constantine, R., & Piggott, S. (2007). *The British gambling prevalence survey*. London: Stationery Office.

This page intentionally left blank

Chapter **12**

Youth Gambling Problems: An International Perspective

Isabelle D. Lussier, MA,
Jeffrey L. Derevensky, PhD, and Rina Gupta, PhD

Gambling involves the wagering of money on games of chance and is a popular pastime for people in most parts of the world. The literature on gambling behavior indicates that children and adolescents enjoy and frequently participate in gambling despite legal restrictions on underage gambling (Derevensky & Gupta, 2000; Gupta & Derevensky, 1998a). Recent reviews suggest that upwards of two-thirds of underage North American youth have gambled in regulated and licensed gambling venues (Jacobs, 2000, 2004), with adolescents having been reported to have pathological gambling prevalence rates two to four times those of adults (Gupta & Derevensky, 1998a; National Research Council [NRC], 1999). In fact, there is growing evidence that a small but identifiable proportion of adolescents in many countries exhibit excessive gambling behavior (Becoña, 1997; Delfabbro & Thrupp, 2003; Fisher, 1993; Johansson & Götestam, 2003; Ólason, Sigurdardottir, & Smari, 2005; Skokauskas, 2007).

Current research supports the notion that problem gambling behavior often begins early, between the ages of nine and eleven years of age (Gupta & Derevensky, 1998a; Jacobs, 2000, 2004), an age of onset that is earlier than that for most illicit substances (Gupta & Derevensky, 1998a). As well, excessive gambling among adolescents has been shown to be positively correlated with participation in increased delinquency and criminal behaviors, substance use, and antisocial behaviors (Derevensky & Gupta, 2004a; Ladouceur, Dubé, & Bujold, 1994). The serious nature of gambling problems is especially disconcerting considering that gambling is perceived to be a highly socially acceptable

activity among adults and adolescents, with little recognition of its inherent risks (Azmier, 2000; Gupta & Derevensky, 1997).

For a long time, adolescent gambling dependency went unnoticed compared with other addictions given that gambling dependence is not always directly observable in youth, that the negative consequences for youth may not be perceived to be as severe as those for adults, and that gambling behavior is prevalent and currently widely accepted (Derevensky, 2007, and Derevensky, in press; Derevensky & Gupta, 2004a; Hardoon & Derevensky, 2002). In fact, gambling is so widely acceptable in our society that unregulated forms of gambling frequently occur in the home and begin as a family activity (Felsher, Derevensky, & Gupta, 2003; Gupta & Derevensky, 1997). Parents are often aware of their children's gambling activities and many children's first gambling experience occurs in the home with a family member. However, as children get older, they tend to gamble less with family members and more with peers (Gupta & Derevensky, 1997).

Recently, a pathways model has been formulated, suggesting that there are several subtypes of adolescents who meet the criteria for probable pathological gambling behavior, but who demonstrate different antecedents and related symptoms (Nower & Blaszczynski, 2004). Further sharpening our understanding of gambling problems, Abbott, Volberg, Bellringer, and Reith (2004) point out that the scientific investigation of gambling problems must address nuances inherent to the broad subject of gambling. For example, the popularity of certain types of gambling appears to vary from country to country, and some forms of gambling are likely more strongly associated with gambling problems (Abbott et al., 2004). However, it is equally important to note that apparently less problematic forms of gambling (for example, the lottery) may be a gateway to more harmful types of gambling (Felsher, Derevensky, & Gupta, 2004).

MEASUREMENT ISSUES

The most widely used categories to represent youth gambling behavior are based upon a continuum and diagnostic screens that include designated cut-off scores for problem and pathological gambling, at-risk gambling behaviors, and social gambling (Derevensky, Gupta, & Winters, 2003). Adolescents who meet the criteria for pathological gambling demonstrate a pervasive pattern of excessive gambling behaviors and experience severe gambling-related problems. Derevensky and Gupta (2004b) contend that the adolescent classification of *pathological gambler* is in itself problematic since pathological behavior, as it is currently operationalized, implies a long history of gambling behavior. As such, they have argued that it may be premature to apply this term to young

populations, and they recommend the use of the term *probable pathological gambler* instead. The category *at-risk gambler* refers to adolescents who do not yet meet sufficient criteria to be classified as probable pathological gamblers on gambling screens, but who remain at risk for the development of severe gambling problems should their behaviors and the negative consequences of excessive gambling escalate (Shaffer & Hall, 1996). In contrast, *social gamblers* most often gamble occasionally, appear to have no difficulty controlling their gambling behavior (in terms of setting money and time limits) and have no negative consequences associated with their gambling.

Despite advances in our understanding of the development, acquisition, and maintenance of youth gambling problems, most adolescent gambling screens are adaptations of adult instruments, with items having been modified to make them more developmentally appropriate. Several of the most commonly used instruments include the South Oaks Gambling Screen—Revised for Adolescents (SOGS-RA) (Winters, Stinchfield, & Fulkerson, 1993), the DSM-IV-J (Fisher, 1992), and its revision the DSM-IV-MR-J (Fisher, 2000) (see Derevensky & Gupta, 2004b for a detailed description of each instrument and its criteria). Currently, the Canadian Centre for Substance Abuse and the Ontario Problem Gambling Research Centre are in the final stages of developing a new adolescent instrument, the Canadian Adolescent Gambling Inventory. Further testing of this instrument will be necessary, but this will in fact be the first instrument specifically designed for adolescents.

Although no gold standard for the measurement of adolescent problem gambling currently exists (Derevensky et al., 2003), concordance between the DSM-IV, DSM-IV-MR-J, and the SOGS-RA is reportedly high, particularly for identifying gambling problems among boys (Derevensky & Gupta, 2000). The DSM-IV-MR-J appears to be a slightly more conservative measure than the SOGS-RA (Derevensky & Gupta, 2000; Ólason et al., 2005). However, variations in cut score criteria, omissions and/or insertion of items, and translation problems have led to serious difficulties in reliably estimating the prevalence rates of adolescent problem gambling and comparing study outcomes (Derevensky & Gupta, 2006). As well, Gambino (2006) has emphasized the need for community-specific validation of youth gambling scales, given the variability in accessibility to different forms of gambling.

PREVALENCE

Gambling and problem gambling have been described as "moving targets" (Abbott et al., 2004), because gambling activities and venues for gambling continue to change (e.g., Internet gambling; mobile gambling), and because the

criteria for classifying individuals with gambling problems continue to evolve with each new release of the classificatory system in the *Diagnostic and Statistical Manual* (*DSM*), thus making comparisons between outcomes in prevalence studies over time difficult. Although little is known regarding best practices in gambling research, increasingly governments are establishing systems to monitor the impact of gambling over time (e.g., in Australia, Canada, New Zealand, South Africa, and the United States) (Abbott et al., 2004).

The fact that the discrepant variability in prevalence rates for youth problem gambling is generally larger than the variability in prevalence rates for adults (e.g., NRC, 1999) has elicited some debate among researchers (Derevensky & Gupta, 2006; Derevensky et al., 2003; Ladouceur et al., 2000). Aside from the possibility that adolescent reports are simply more variable than adult reports (Derevensky & Gupta, 2006), it has been postulated that the larger variability in youth gambling prevalence studies may be due to various situational and measurement variables including sampling procedures, the use of different instruments (including varying cut-point scores, the use of modified instruments, and unverified translations), gender distributions within samples, the age of the population being assessed, cultural and ethnic differences, the availability and accessibility of different forms of gambling, the existing statutes concerning regulated forms of gambling, and the time frame used for assessing gambling behavior (see Derevensky & Gupta, 2000; Derevensky et al., 2003; Shaffer, LaBrie, LaPlante, Nelson, & Stanton, 2004; Stinchfield, 2002). In addition, the variability in terminology used to identify adolescents with gambling problems (e.g., pathological gamblers, probable pathological gamblers, compulsive gamblers, problem gamblers, level 3 gamblers, disordered gamblers) further obscures comparisons among outcomes in prevalence studies and has led to a need for greater standardization in nomenclature and terminology in youth gambling research (Derevensky & Gupta, 2006; Shaffer et al., 2004).

North America

The onset of gambling experiences occurs early among North American youth, with a median age ranging between 11 and 13 years (Jacobs, 2000). This age of onset is younger than the expected age of onset for cigarette use, consumption of alcohol, and substance use (Gupta & Derevensky, 1998a; Jacobs, 2000). Among North American youth, the lottery appears to be the preferred legalized/regulated form of wagering (Jacobs, 2000, 2004), likely due to the lax regulations concerning selling to underage minors and the perception of the lottery as a nonaddictive form of gambling.

Youth gambling behaviors are often dependent upon the availability and accessibility of games and gaming locations, gender and type of game (males more frequently report sports wagering as a preference, whereas girls more frequently report bingo), age (older adolescents are more likely to play video lottery terminals and to engage in casino gambling), and cultural and ethnic background (Chevalier, Deguire, Gupta, & Derevensky, 2003; Derevensky, in press; Ellenbogen, Gupta, & Derevensky, 2007; Gupta & Derevensky, 2004).

Meta-analyses and reviews that look at youth gambling behaviors and problems in North America reveal lifetime gambling rates among adolescents that range from 39 percent to 92 percent, prevalence rates for serious youth gambling problems that range from 4 percent to 8 percent, and prevalence rates for youth at risk for developing or returning to serious gambling problems that range from 10 percent to 15 percent (Jacobs, 2000, 2004; NRC, 1999; Shaffer & Hall, 1996). Gambling participation and problematic gambling behavior among youth appear to have increased substantially between 1984 and 2002 (Jacobs, 2004). Based on findings from an examination of 20 prevalence studies, Jacobs (2000) estimated that 2.2 million North American adolescents experience gambling-related problems. The National Research Council (NRC, 1999), designed to assist the United States National Gambling Impact Study Commission, reported that the proportion of adolescents exhibiting pathological gambling behavior in the United States could be more than three times that of adults (5.0 percent vs. 1.5 percent) (NRC, 1999). Several recent studies conducted in the United States describe findings similar to those of these large reviews and early meta-analyses (Gealt & O'Connell, 2006; Langhinrichsen-Rohling, Rohde, Seeley, & Rohling, 2004).

In Canada, recent studies also demonstrate that approximately 3–7 percent of adolescents surveyed in prevalence studies meet the criteria for pathological gambling using the DSM-IV or DSM-IV-MR-J screens (Derevensky & Gupta, 2000 [3.4 percent], 2001 [3.4 percent]; Gupta & Derevensky, 1998a [4.7 percent], 2000 [6.7 percent]; Hardoon, Gupta, & Derevensky, 2002 [4.9 percent]; Lussier, Derevensky, Gupta, Bergevin, & Ellenbogen, 2007 [3.2 percent]; Ste-Marie, Derevensky, & Gupta, 2002 [4.4 percent]). While most of these surveys have been conducted in Quebec and Ontario, a large-scale prevalence study in four Atlantic provinces of Canada was conducted in 1998. The sample design was a single-stage cluster sample of randomly selected and stratified classes (grades 7, 9, 10, and 12) (Poulin, 2000). Broad definitions of problem gambling and at-risk gambling led to prevalence rates of 6.4 percent and 8.2 percent, respectively, whereas narrow definitions of problem gambling and at-risk gambling revealed prevalence rates of 2.2 percent and 3.8 percent, respectively.

Europe

During the 1960s and 1970s, many European countries legalized various forms of gambling, including electronic gaming machines (Becoña, Labrador, Echeburúa, Ochoa, & Vallejo, 1995). Early studies suggested that per capita spending on gambling in Germany, Holland, and Spain was among the highest in European countries (Becoña et al., 1995). Further, it was estimated that at least half of the people with pathological gambling problems in Holland, Spain, and Germany were under the age of 30 (Becoña et al., 1995). Despite these statistics, there remains a dearth of information regarding gambling behavior and problem gambling estimates among children and adolescents in these countries. Most published research on youth gambling in Europe has been conducted in the United Kingdom (UK), the Nordic countries, Spain, Iceland, and more recently Eastern Europe. The findings of these studies are summarized by region below.

United Kingdom

Research on youth gambling problems in the UK has focused mostly on amusement machines, commonly referred to as fruit machines (Fisher, 1993, 1995; Fisher & Griffiths, 1995; Yeoman & Griffiths, 1996), lottery and scratchcards (Griffiths, 2000; Wood & Griffiths, 1998, 2002, 2004; Wood, Griffiths, Derevensky, & Gupta, 2002; Wood, Griffiths, Stevens, Bartlett, & Pye, 2006), and more recently, video games and the Internet (Chappell, Eatough, Davies, & Griffiths, 2006; Griffiths & Wood, 2000; Wood, Griffiths, & Parke, 2007). Although most forms of commercial gambling are reserved for adults (18+), no age restriction has been placed on low-stakes fruit machines, and the lottery and sports pools have long incorporated an age restriction to the age of 16 years and above (Fisher, 1999). The UK is currently the only country in the world that allows juveniles to legally wager on fruit machines, for which the government has been subject to criticism (Orford, 2003). From 1997 to 1998, more than 82 percent of problem-related calls from adolescents placed to a gambling helpline in Britain were from individuals who gambled on fruit machines (Griffiths, Scarfe, & Bellringer, 1999).

Fruit machines, found in seaside arcades, cafés, and restaurants, are easily accessible to many youths in the UK and represent a serious problem (Griffiths, 2000). As such, it is not surprising that there has been a proliferation of research and proposals for the elimination of fruit machines and for raising the minimum age at which to gamble. Similarly, given the wide variety of gambling opportunities and the social acceptability of gambling, it is not surprising that

the rates of youth problem gambling in the UK remain relatively high. The first national prevalence study, conducted by Fisher (1999), examined youth gambling behaviors among 10,000 adolescents in England and Wales, aged 12–15. The study focused on fruit machines and scratchcards. Based on DSM-IV-MR-J scores, findings revealed an overall problem gambling prevalence rate of 5.6 percent. Males were more likely to be problem gamblers than females, and children identified as problem gamblers were more than three times as likely to report that their parents gambled too much. As well, problem gambling was reported to be more prevalent among seaside residents than among those that lived inland, likely due to the popularity of seaside arcades (Fisher, 1999). Similarly, using an adapted version of the DSM-IV-J, Wood and Griffiths (1998) explored the psychosocial effects of the lottery and scratchcards among 1,195 adolescents (aged 11–15). A positive correlation was found between parental and child gambling, with most lottery tickets and scratchcards being bought for juveniles by their parents. Of the 6 percent of participants who met the criteria for pathological gambling, the majority were male.

More recently, Wood and his colleagues (2006) conducted a large-scale national prevalence study, which included 8,017 adolescents aged 12–15. Overall, lifetime participation in gambling activities in this age group had fallen to 73 percent. Most notably, the prevalence of problem gambling had declined to 3.5 percent (based on DSM-IV-MR-J cut scores). Although prevalence of problem gambling on scratchcards and/or fruit machines in particular had also declined, it remained relatively high at 6.0 percent compared with international rates. As might be expected, boys were more likely to participate in gambling activities and to demonstrate problematic gambling behaviors. As well, youth living in coastal areas were more likely to exhibit problems with fruit machine gambling compared to those that lived inland (4.7 percent vs. 2.9 percent). This last-mentioned finding was attributed to the wide availability and easy accessibility of fruit machines in coastal areas. It is also important to note that this prevalence study did not include older adolescents.

In Scotland, Moodie and Finnigan (2006) conducted a study of the prevalence of gambling behavior and problems in Scotland among 2,043 adolescents aged 11–16. This was the first prevalence study of its kind in the country. Using the DSM-IV-J as a gambling screen, findings revealed a striking 9.0 percent probable pathological gambling rate with an additional 15.1 percent reported as being at risk for developing or returning to a serious gambling problem. The most popular gambling activity was the use of fruit machines. Males were more likely to meet the criteria for probable pathological gambling (3.5:1), and 76 percent of all adolescents reported that they had gambled in the past. Also, youth with gambling problems were more likely to report having friends and

family members who gambled. The age of onset for gambling was young, at 10.3 years of age for nonproblem gambling, 9.5 years for at-risk gambling, and 9.3 years for probable pathological gambling. More than a third of the sample considered gambling a "good" or "very good" way to make money. Despite the high rates of problem gambling in Scotland, treatment options remain scarce (Moodie & Finnigan, 2006).

Nordic Countries

Prevalence estimates indicate that the rate of problem gambling in Nordic countries may be lower than those obtained in North America and the UK (Johansson & Götestam, 2003; Ólason et al., 2005; Ólason, Skarphedinsson, Jonsdottir, Mikaelsson, & Gretarsson, 2006; Volberg, Abbott, Ronnberg, & Munck, 2001). However, further research must be conducted within Nordic countries to confirm these results, particularly in Denmark and Finland where no published studies of adolescent problem gambling were found.

In Iceland, there is no age restriction on scratch cards, lotteries, sport pools, and bingo. However, electronic gaming machines (EGMs) are restricted for youth under the age of 16, and wagering on horse races is illegal for anyone under the age of 18 (Ólason et al., 2005). Two adolescent prevalence studies in Iceland have examined youth gambling rates amongst 13–15-year-old and 16–18-year-old adolescents (Ólason et al., 2005, 2006). The survey for youth aged 13–15 was distributed among 25 primary schools in Reykjavik (N = 3,511). The DSM-IV-MR-J and the SOGS-RA screening items were both administered, with results indicating prevalence rates of 1.9 percent (DSM-IV-MR-J) and 2.8 percent (SOGS-RA) for problematic gambling, and an additional 3.7 percent (DSM-IV-MR-J) and 4.1 percent (SOGS-RA) for being at risk of developing or returning to serious gambling problems (Ólason et al., 2006). Of the full sample, 70 percent of adolescents reported that they had gambled in the past year, and 93 percent reported that they had gambled at least once in their lifetime. The age of onset for gambling was 9.5 years, and boys were again more likely than girls to demonstrate gambling problems. In addition, those classified as exhibiting a gambling problem were more likely to report that their parents and peers gambled. EGMs, which are widely available, were the most popular form of gambling among youth with gambling problems (41 percent). The survey for youth aged 16–18 found similar rates, with 2 percent (DSM-IV-MR-J) and 2.7 percent (SOGS-RA) of youth identified as having gambling problems, and an additional 3.2 percent (DSM-IV-MR-J) and 4.4 percent (SOGS-RA) being at risk for gambling problems (Ólason et al., 2005).

In Norway, a study conducted by Johansson and Götestam (2003) included 3,237 youths aged 12–18 that were solicited via telephone and postal interviews. Using selected DSM-IV criteria to classify problem gamblers, 1.8 percent of adolescents were identified as exhibiting problematic gambling behavior, while another 5.2 percent were at risk of developing or returning to serious gambling problems. Boys were four times as likely as girls to exhibit gambling problems. Slot machines were rated as the most popular form of gambling, and 82 percent of youth reported that they had gambled in the past. Interestingly, the age of onset for gambling in this study was 9 years with an onset age range of 4–18 years of age, indicating that while the age of onset in Norway is low, problematic gambling rates also remain relatively low.

In a study carried out by Statistics Sweden, 2 percent of adults were identified as exhibiting gambling problems (Volberg et al., 2001). Individuals in the sample ranged in age from 15 to 74 years (N = 9,917). Analyses carried out on the younger participants (15–24) revealed that youth had a 151 percent higher risk than those over 25 years of exhibiting lifetime problem gambling behavior. Unfortunately, the prevalence rates for adolescents (15–17) were not reported in this study, and only adult gambling screens were administered.

Spain

Spain is believed to be among the countries in Europe that spends the most money per capita on gambling (Becoña et al., 1995; Hand, 1992). In Spain, type A machines, known as amusement machines or more colloquially as *comecocos,* are available to youth under the age of 18. Although they offer no cash prizes, they do offer free games and publish the winner's name and score (Becoña et al., 1995). A study conducted by Becoña (1997) estimated the prevalence rates to be 2.2 percent and 1.6 percent for problem gambling among adolescents aged 11–16 in two northern regions of Spain (Galicia and Asturias) (N = 2,185). More recently, Becoña and Miguez (2001) surveyed a sample of 2,790 adolescents using the SOGS-RA. Their findings revealed that overall, 5.6 percent of the participants met the criteria for problem gambling and another 8.2 percent were at risk of developing or returning to serious gambling problems.

Eastern Europe

Research conducted in Lithuania and Romania has recently emerged regarding youth gambling behavior. During Soviet control, access to gambling was largely prohibited. However, there appears to have been a rapid expansion of gambling venues and opportunities since that time (Lupu, Onaca, & Lupu,

2002; Skokauskas, 2007). In the early 1990s, slot machines, initially registered as computer games or gaming machines, were made available to the public in Lithuania (Skokauskas, 2007). Due to inadequate controls, youth were free to engage in these forms of wagering. In 2002, gambling was legalized in Lithuania and youth became exposed to a wider variety of gambling activities (Skokauskas, 2007). A recent prevalence study in Lithuania using the DSM-IV-MR-J identified 4.2 percent of youth (aged 10–18) as exhibiting probable pathological gambling behavior. An additional 9.1 percent were classified as being at risk of developing or returning to severe gambling problems. Overall, 82.6 percent of adolescents had engaged in some form of gambling. Six characteristics that were found to be associated with problem gambling included being male, having cognitive distortions regarding gambling, having parents who gambled, using alcohol, and smoking (Skokauskas, 2007). In Romania, a prevalence study of youth gambling based on the Gamblers Anonymous 20 questions in three districts identified 6.8 percent of youth (aged 14–19) as exhibiting gambling problems (Lupu et al., 2002). Males were found to be almost five times more likely to meet the criteria for problem gambling than females. The most frequent forms of gambling were pool (56%), poker (35%), and bingo (32%).

Australia

In Australia, gambling activities are generally available only in licensed establishments and are restricted to adults (Delfabbro & Thrupp, 2003). Most gambling among Australian youth occurs privately (individually or with peers and/or parents), which has been presented as support for the notion that regulatory provisions are working well in the country (Delfabbro, Lahn, & Grabosky, 2005). In terms of prevalence estimates in Australia, early research by Moore and Ohtsuka (1997) revealed that 3 percent of youth aged 14–25 in Melbourne were identified, using a modified version of the SOGS, as having a gambling problem. Approximately 75 percent of the sample had gambled in the past, with higher prevalence estimates among boys. Further research by Moore and Ohtsuka (1999, 2000) supported these findings, with 3.8 percent of youth being classified as having gambling problems based on scores from the SOGS in two studies of 14–25-year-old and 15–18-year-old participants. As well, findings from a sample of 505 adolescents aged 15–17 by Delfabbro and Thrupp (2003), revealed that 62.5 percent had gambled in the previous year, and that 3.5 percent could be classified, based on scores from the DSM-IV-J, as having a gambling problem. Lotteries, scratch-tickets, and sports betting were identified as the most popular gambling activities, and youth with gambling

problems were more likely to have friends and family members who approved of their gambling and/or gambled themselves. Most recently, Delfabbro and his colleagues (2005) identified 4.4 percent of youth aged 11–19 as exhibiting gambling problems (based on DSM-IV-J scores), indicating a slightly higher prevalence rate of gambling problems than was revealed by prior studies in the country.

RISK AND VULNERABILITY FACTORS

Among adolescents, there appears to be a rapid movement from social to problem gambling (Derevensky & Gupta, 1999). There are many factors involved in the acquisition, development, and maintenance of youth gambling problems. Although gambling does not involve the ingestion of a substance, and is unique in its emphasis on attributions of luck, skill, and attitudes about money, pathological gambling and drug dependency share common consequences including tolerance, dissociative states, and physiological arousal (APA, 2000; Felsher, 2007). Stinchfield and Winters (1998) have identified commonalities between risk factors identified in the substance abuse and youth gambling literature, including family history of the respective problem, low self-esteem, depression, family norms (e.g., attitudes that promote the problem), physical or sexual abuse, poor academic performance, delinquency, community norms (e.g., promotion and access related to drug use or gambling), and early onset. Similarly, Dickson, Derevensky, and Gupta (2002) demonstrated a large degree of overlap in risk factors shared by people exhibiting problem gambling and those exhibiting other addictions over a wide breadth of domains including individual, social/familial, and neighborhood/societal (Dickson et al., 2002). The overlap in risk factors identified in the gambling and substance abuse literature has led clinicians to adopt Jacobs' (1986) general theory of addictions as a framework for conceptualizing problem gambling behavior and commonalities across addictions (Gupta & Derevensky, 1998b; Winters & Anderson, 2000). Unique risk factors and overlapping risk factors between youth gambling problems and other youth addictions are described in greater detail below.

Individual Factors

Individual factors such as physiological, personality, emotional, coping, values and attitudes, and chronic problem behaviors have been shown to be associated with excessive youth gambling behavior (Derevensky & Gupta, 2004a; Dickson et al., 2002; Hardoon & Derevensky, 2002). Adolescents meeting the criteria for pathological gambling are also more likely to report difficulty in

school, exhibit truancy, and have poor grades (Hardoon, Gupta, & Derevensky, 2004; Lesieur et al., 1991). Current research regarding individual risk correlates among adolescents with gambling problems suggests that probable pathological gambling is more prevalent in males than females (NRC, 1999), with males being more likely to gamble larger amounts of money (Derevensky, Gupta, & Della-Cioppa, 1996), to begin gambling at an earlier age (Derevensky & Gupta, 2001), to gamble more frequently (Jacobs, 2000, 2004), and to exhibit a wide variety of gambling-related problems (Derevensky & Gupta, 2004a).

Adolescents with gambling problems often report higher levels of risk-taking behaviors (Gupta, Derevensky, & Ellenbogen, 2006), impulsivity (Nower, Derevensky, & Gupta, 2004; Vitaro, Arseneault, & Tremblay, 1999), anxiety (Ste-Marie, Gupta, & Derevensky, 2006), depression, suicide ideation, and suicide attempts (Gupta & Derevensky, 1998b; Kaufman, 2004; Nower, Gupta, Blaszczynski, & Derevensky, 2004). As well, youth with gambling problems are more likely to have a history of delinquency (Magoon, Gupta, & Derevensky, 2005) and/or to engage in other maladaptive behaviors including substance and alcohol abuse (Hardoon et al., 2004; Winters & Anderson, 2000). Adolescents have been reported to be four times more likely to gamble daily or weekly if they also consume drugs on a regular basis (Winters & Anderson, 2000).

Familial/Social Factors

Many youth who exhibit severe gambling problems report that their first gambling experience occurred at home with a family member (Gupta & Derevensky, 1997). They are also more likely to have a parent who struggles with an addiction (Gupta & Derevensky, 1998a; Wood & Griffiths, 1998). Although youth gambling frequency appears to be related to both parents' gambling frequency and gambling problems, youth gambling problems appear to be linked mostly to fathers' severity of gambling problems (Vachon, Vitaro, Wanner, & Tremblay, 2004). In addition, research findings indicate that even after controlling for socioeconomic status (SES), gender, and impulsivity-hyperactivity problems, significant associations remain between youth gambling problems and poor parental monitoring and disciplinary strategies (Vachon et al., 2004).

More recently, the combination of being in a family that interacts infrequently and concurrently feeling a lack of bond to one's family was found to contribute to the prediction of problem gambling (Lussier et al., 2007). Although these findings require replication and further study, they indicate the possibility of a curvilinear relationship between familial interaction and gambling

behavior. A curvilinear relationship for parental control/monitoring has been demonstrated in adolescent sexual behavior research (Miller, McCoy, Olson, & Wallace, 1986). As well, it has been noted that drug-using adolescents often report their parents as being overcontrolling (Meschke & Patterson, 2003).

There is some evidence that peer modeling and social learning are involved in the onset of gambling problems (Gupta & Derevensky, 1997; Hardoon & Derevensky, 2001). Many adolescents report that they gamble because their friends engage in this behavior (Griffiths, 1990). As well, adolescents with gambling problems commonly replace old friends with individuals who share their interests in gambling (Gupta & Derevensky, 2000).

Neighborhood/Societal Factors

Despite the lack of formal prevalence studies for youth problem gambling among various socioeconomic groups, preliminary data demonstrate that low SES youth may be at higher risk for gambling problems compared to other youth (Fisher, 1993; Kaufman, 2004; Schissel, 2001; Vitaro et al., 1999). As well, ethnic/racial minority youth appear to be at greater risk for developing and maintaining gambling problems (Lesieur et al., 1991; Schissel, 2001; Volberg et al., 2001). Interestingly, compared with Francophone (French-speaking) or Anglophone (English-speaking) youth, Allophone (neither English nor French is their mother tongue) adolescents made up the greatest proportion of youth with gambling problems in a community sample of over 1,000 Quebec adolescents (Ellenbogen, Gupta, & Derevensky, 2007).

First Nation youth appear to be at particularly high risk for gambling problems (Delfabbro et al., 2005; Peacock, Day, & Peacock, 1999; Schissel, 2001; Stinchfield, Cassuto, Winters, & Latimer, 1997). In a review of literature concerning aboriginal populations and problem gambling, Wardman, el-Guebaly, and Hodgins (2001) concluded that aboriginal adolescent problem gambling and adult problem and pathological gambling rates were considerably higher, ranging from 2 to 16 times those of non-Aboriginal populations, although they caution that there are concerns regarding the validity and reliability of these rates.

Situational and environmental risk factors may interact with individual risk factors through the availability and accessibility of gambling activities and venues to further augment the possibility of developing and maintaining gambling problems (Felsher et al., 2003, 2004; Fisher, 1999; Gupta & Derevensky, 1998a; Jacobs, 2004). More specifically, game features and technological advances have been associated with problem gambling. Structural characteristics of games that encourage continued, repetitive play such as high event

frequencies and intermittent reinforcement schedules (e.g., electronic gambling machines and scratchcards) as well as qualities that create a favorable ambience for play, such as vivid colors, sounds, music, and lights (e.g., electronic forms of gambling) are believed to enhance the availability, accessibility, and addictive potential of games (Abbott et al., 2004; Derevensky, 2007; Felsher et al., 2003; Griffiths & Wood, 2004). Technological advances continue to provide enticing gambling opportunities in the form of Internet gambling, interactive lotteries, keno and television wagering, mobile gambling (via cell phones), and novel slot machines (Griffiths & Wood, 2000). Recent research indicates that a large number of youth have gambled on Internet sites without money (Byrne, Gupta, & Derevensky, 2004), leading to a growing concern that such sites may be training youth to gamble with money once they obtain their own means to do so (Derevensky, 2007).

YOUTH GAMBLING BEHAVIOR AND RESILIENCY

Clearly, bio-psycho-social factors are involved in the acquisition, development, and maintenance of gambling behavior. Despite the existence of much literature on factors that predispose youth to problem gambling, very little research has addressed the identification of moderating variables that serve to mitigate the development and maintenance of severe gambling behavior (Dickson, Derevensky, & Gupta, 2008; Lussier et al., 2007). Youth gambling researchers have specified a need for effective ways to strengthen resiliency traits in children (Derevensky, Gupta, Dickson, & Deguire, 2004; Dickson et al., 2002, 2008; Winters, Arthur, Leitten, & Botzet, 2004). Educating children to develop the capacity and resources required to resist and overcome adversity is a rational and increasingly effective approach. The research that has focused on adolescent resilience has demonstrated a strong relationship between healthy, resilient behaviors and successful outcomes (Werner & Smith, 1992).

Dickson and colleagues hypothesized that the protective factors that apply to other addictive behaviors would also buffer against the acquisition, development, and maintenance of excessive youth gambling (Dickson et al., 2008). Using a community sample of adolescents, Lussier and colleagues (2007) examined whether youth identified as resilient (high risk exposure/high internalized protection scores) were as likely as those identified as vulnerable (high risk exposure/low internalized protection scores) to engage in excessive gambling behavior. Their findings demonstrated that resilient and vulnerable youth differed significantly in their self-reported gambling severity. As well, youth identified as resilient were not found to be statistically distinguishable from low-risk exposure groups in terms of their gambling severity. Lussier and colleagues also

examined the relative contribution of various risk (family, peers, neighborhood, substance use) and resiliency traits (social bonding, personal competence, social competence) domains in relation to problem gambling behavior and reported that environmental risk and resiliency traits each provided a unique contribution to the prediction model of gambling problems, with low levels of social bonding being the greatest predictor (Lussier et al., 2007).

SUMMARY AND FUTURE DIRECTIONS

While caution is necessary in comparing prevalence rates from different studies, it appears that the prevalence of youth gambling problems in Nordic countries (Johansson & Götestam, 2003; Ólason et al., 2005, 2006) and Australia (Delfabbro & Thrupp, 2003) is lower than in North America (Derevensky & Gupta, 2000; Jacobs, 2000, 2004; NRC, 1999) and the UK. However, in all countries, adolescents and young adults appear to have higher prevalence rates than older adults (Abbott et al., 2004), boys appear to have higher prevalence rates than girls, and youth whose parents endorse gambling activities have higher prevalence rates than youth whose parents do not. Also, a small but identifiable proportion of the adolescents in all countries where prevalence estimates exist reported significant gambling-related problems.

In Europe, electronic gambling machines appear to be more easily accessible and more popular among adolescents than in North America. The fact that they are often widely distributed in public places in Europe has led some policy experts to suggest that their popularity is related in part to easy access, where enforcement of age limits is lax (Ólason et al., 2006). In fact, accessibility and availability of gambling activities and venues are increasingly identified as significant risk factors that may interact with individual risk factors to further augment the chances for certain youth to acquire and maintain problematic gambling behavior (Abbott et al., 2004; Derevensky, 2007). For this reason, it has been suggested that legislation and policies that increase access to gambling activities will likely create higher problem gambling prevalence rates and in turn generate social and economic costs to families and communities (Abbott et al., 2004).

It is estimated that active participation in Internet gambling among youth may continue to rise significantly as accessibility becomes easier and as popularity of and familiarity with such gambling venues increase (Derevensky, 2007). Internationally, there is ample indication that individuals having significant gambling problems do not seek or acquire treatment for their symptoms, indicating a need for better prevention and intervention programs that reduce the barriers for individuals seeking help.

Adolescent problem gambling remains an important social policy issue in need of appropriate prevention and responsible social policies (see chapter 17 in this volume for more information regarding prevention strategies). The study of youth gambling research supports and broadens contemporary theories of typical and atypical development and provides information concerning risk and protective factors that may be applicable to the development of prevention and intervention initiatives for a targeted gender, or for age groups, cultures, or adolescents as a whole.

REFERENCES

Abbott, M. W., Volberg, R. A., Bellringer, M., & Reith, G. (2004). *A review of research on aspects of problem gambling. Final report.* Prepared for the Responsibility in Gambling Trust. Auckland, New Zealand: Auckland University of Technology.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR* (4th ed.). Washington, DC: Author.

Azmier, J. (2000). *Gambling in Canada: Triumph, tragedy, or tradeoff. Canadian gambling behaviour and attitudes.* Calgary, Alberta: Canada West Foundation.

Becoña, E. (1997). Pathological gambling in Spanish children and adolescents: An emerging problem. *Psychological Reports, 81,* 275–287.

Becoña, E., Labrador, F., Echeburúa, E., Ochoa, E., & Vallejo, M. A. (1995). Slot machine gambling in Spain: An important new social problem. *Journal of Gambling Studies, 11,* 265–284.

Becoña E., & Miguez, M. C. (2001). Problem gambling and risky gambling in adolescents: Its relation with tobacco and alcohol consumption. *Adicciones, 13,* 279–288.

Byrne, A., Gupta, R., & Derevensky, J. (2004, June). *Internet gambling in Canadian youth.* Paper presented at the annual meeting of the National Council on Problem Gambling, Phoenix.

Chappell, D., Eatough, V., Davies, M.N.O., & Griffiths, M. (2006). EverQuest—It's just a computer game right? An interpretative phenomenological analysis of online gaming addiction. *International Journal of Mental Health Addiction, 4,* 205–216.

Chevalier, S., Deguire, A.-E., Gupta R., & Derevensky, J. (2003). Jeux de hasard et d'argent. In B. Perron & J. Loiselle (Eds.), *Où en sont les jeunes face au tabac, à l'alcool, aux drogues et au jeu? Enquête québécoise sur le tabagisme chez les élèves du secondaire (2002)* (pp. 175–203). Québec, Canada: Institut de la statistique de Québec.

Delfabbro, P., Lahn, J., & Grabosky, P. (2005). Further evidence concerning the prevalence of adolescent gambling and problem gambling in Australia: A study of the ACT. *International Gambling Studies, 5,* 209–228.

Delfabbro P., & Thrupp, L. (2003). The social determinants of youth gambling in South Australian adolescents. *Journal of Adolescence, 26,* 313–330.

Derevensky, J., (2007). Youth gambling and problem gambling: Another high risk behavior. (Impulsividade: Jogo entre Jovens e Jogo Problematico). In A. Gigliotti & A. Guimarães (Eds.), *Dependencia, compulsão e impulsividade* (Compulsivity, impulsivity and addiction) (pp. 81–90). Rio de Janeiro, Brazil: Editoro Rubio.

Derevensky, J. (in press). Youth gambling: A Canadian perspective. In J. F. Cosgrave & T. R. Klassen (Eds.), *Gambling in 21st century Canada: Citizens, consumers and the state*. Toronto, Ontario, Canada: McGill-Queen's Press.

Derevensky, J. L., & Gupta, R. (1999). Youth gambling problems: A new issue for school psychologists. *Nova Scotia Psychologist, 12*, 8–11.

Derevensky, J. L., & Gupta, R. (2000). Prevalence estimates of adolescent gambling: A comparison of the SOGS-RA, DSM-IV-J, and the GA 20 Questions. *Journal of Gambling Studies, 16*, 227–251.

Derevensky, J., & Gupta, R. (2001). Le problème de jeu touche aussi les jeunes. *Psychologie Québec, 18*, 23–27.

Derevensky, J. L., & Gupta, R. (2004a). Adolescents with gambling problems: A synopsis of our current knowledge. *e-Gambling: The Electronic Journal of Gambling Issues, 10*, 119–140.

Derevensky, J., & Gupta, R. (2004b). The measurement of youth gambling problems: Current instruments, methodological issues and future directions. In J. Derevensky & R. Gupta (Eds.), *Gambling problems in youth: Theoretical and applied perspectives* (pp. 121–144). New York: Kluwer Academic/ Plenum Publishers.

Derevensky, J., & Gupta, R. (2006). Measuring gambling problems amongst adolescents: Current status and future directions. *International Gambling Studies, 6*, 201–215.

Derevensky, J., Gupta, R., & Della-Cioppa, G. (1996). A developmental perspective of gambling behaviour in children and adolescents. *Journal of Gambling Studies 12*, 49–66.

Derevensky, J. Gupta, R., Dickson, L., & Deguire, A.-E. (2004). Prevention efforts toward reducing gambling problems. In J. Derevensky & R. Gupta (Eds.), *Gambling problems in youth: Theoretical and applied perspectives* (pp. 211–230). New York: Kluwer Academic/Plenum Publishers.

Derevensky, J., Gupta, R., & Winters, K. (2003). Prevalence rates of youth gambling problems: Are the current rates inflated? *Journal of Gambling Studies, 19*, 405-425.

Dickson, L., Derevensky, J., & Gupta, R. (2002). The prevention of gambling problems in youth: A conceptual framework. *Journal of Gambling Studies, 18*, 97–159.

Dickson, L., Derevensky, J., & Gupta, R. (2008). Youth gambling problems: An examination of risk and protective factors. *International Gambling Studies, 8*, 25–47.

Ellenbogen, S., Gupta, R., & Derevensky, J. (2007). A cross-cultural study of gambling behavior among adolescents. *Journal of Gambling Studies, 23*, 25–39.

Felsher, J. R. (2007). Etiological factors related to gambling problems: The impact of childhood maltreatment and subsequent stressors. *Dissertation Abstracts International, 68*(3-B). (UMI No. AAINR25142).

Felsher, J. R., Derevensky, J. L., & Gupta, R. (2003). Parental influences and social modeling of youth lottery participation. *Journal of Community and Applied Social Psychology, 13,* 361–377.

Felsher, J. R., Derevensky, J., & Gupta, R. (2004). Lottery playing amongst youth: Implications for prevention and social policy. *Journal of Gambling Studies, 20,* 127–154.

Fisher, S. (1992). Measuring pathological gambling in children: The case of fruit machines in the U.K. *Journal of Gambling Studies, 9,* 277–288.

Fisher, S. (1993). Gambling and pathological gambling in adolescents. *Journal of Gambling Studies, 9,* 277–288.

Fisher, S. (1995). The amusement arcade as a social space for adolescents: An empirical study. *Journal of Adolescence, 18,* 71–86.

Fisher, S. (1999). A prevalence study of gambling and problem gambling in British adolescents. *Addiction Research, 7,* 509–538.

Fisher, S. (2000). Developing the DSM-IV-MR-J criteria to identify adolescent problem gambling in non-clinical populations. *Journal of Gambling Studies, 16,* 253–273.

Fisher, S., & Griffiths, M. (1995). Current trends in slot machine gambling: Research and policy issues. *Journal of Gambling Studies, 11,* 239–247.

Gambino, B. (2006). A comment on the utility of prevalence estimates of pathological gambling. *Journal of Gambling Studies, 22,* 321–328.

Gealt, R., & O'Connell, D. J. (2006). *Gambling with Delaware's kids 2006. The annual report of the Delaware Council on Gambling Problems on Youth Gambling.* Prepared for the Delaware Council on Gambling Problems. Newark, Delaware: The Centre for Drug and Alcohol Studies: University of Delaware.

Griffiths, M. (1990). The acquisition, development, and maintenance of fruit machine gambling in adolescents. *Journal of Gambling Studies, 6,* 193–204.

Griffiths, M. (2000). Scratchcard gambling among adolescent males. *Journal of Gambling Studies, 16,* 79–91.

Griffiths, M., Scarfe, A., & Bellringer, P. (1999). The UK national telephone gambling helpline: Results on the first year of operation. *Journal of Gambling Studies, 15,* 83–90.

Griffiths, M., & Wood, R.T.A. (2000). Risk factors in adolescence: The case of gambling, videogame playing, and the Internet. *Journal of Gambling Studies, 16,* 199–225.

Griffiths, M., & Wood, R.T.A. (2004). Youth and technology: The case of gambling, video-game playing and the Internet. In J. Derevensky & R. Gupta (Eds.), *Gambling problems in youth: Theoretical and applied perspectives* (pp. 101–120). New York: Kluwer Academic/Plenum Publishers.

Gupta, R., & Derevensky, J. L. (1997). Familial and social influences on juvenile gambling behaviour. *Journal of Gambling Studies, 13,* 179–192.

Gupta, R., & Derevensky, J. (1998a). Adolescent gambling behavior: A prevalence study and examination of the correlates associated with problem gambling. *Journal of Gambling Studies, 14,* 319–345.

Gupta, R., & Derevensky, J. (1998b). An empirical examination of Jacobs' general theory of addictions: Do adolescent gamblers fit the theory? *Journal of Gambling Studies, 14,* 17-49.

Gupta, R., & Derevensky, J. (2000). Adolescents with gambling problems: From research to treatment. *Journal of Gambling Studies, 16,* 315–342.

Gupta, R., & Derevensky, J. (2004). A treatment approach for adolescents with gambling problems. In J. Derevensky & R. Gupta (Eds.), *Gambling problems in youth: Theoretical and applied perspectives* (pp. 165–188). New York: Kluwer Academic/Plenum Publishers.

Gupta, R., Derevensky, J., & Ellenbogen, S. (2006). Personality characteristics and risk-taking tendencies among adolescent gamblers. *Canadian Journal of Behavioral Science, 38,* 201–213.

Hand, I. (1992). Gambling in Europe, part I: Germany. *Journal of Gambling Studies, 8,* 1–7.

Hardoon, K. K., & Derevensky, J. L. (2001). Social influences involved in children's gambling behavior. *Journal of Gambling Studies, 17,* 191–215.

Hardoon, K. K., & Derevensky, J. L. (2002). Child and adolescent gambling behavior: Current knowledge. *Clinical Child Psychology and Psychiatry, 7,* 263–281.

Hardoon, K. K., Gupta, R., & Derevensky, J. (2004). Psychosocial variables associated with adolescent gambling: A model for problem gambling. *Psychology of Addictive Behaviors, 18,* 170–179.

Jacobs, D. F. (1986). A general theory of addictions: A new theoretical model. *Journal of Gambling Behavior, 2,* 15–31.

Jacobs, D. F. (2000). Juvenile gambling in North America: An analysis of long term trends and future prospects. *Journal of Gambling Studies, 16,* 119–152.

Jacobs, D. F. (2004). Youth gambling in North America: Long term trends and future prospects. In J. Derevensky & R. Gupta (Eds.), *Gambling problems in youth: Theoretical and applied perspectives* (pp. 1–26). New York: Kluwer Academic/Plenum Publishers.

Johansson, A., & Götestam, K. G. (2003). Gambling and problematic gambling with money among Norwegian youth (12–18 years). *Nordic Journal of Psychiatry, 57,* 317–321.

Kaufman, F. D. (2004). The relationship between gambling activity, the occurrence of life stress, and differential coping styles in an adolescent sample. *Dissertation Abstracts International, 65*(1-B). (UMI No. AAINQ88494)

Ladouceur, R., Bouchard, C., Rhéaume, N., Jacques, C., Ferland, F., Leblond, J., & Walker, M. (2000). Is the SOGS an accurate measure of pathological gambling among children, adolescents and adults? *Journal of Gambling Studies, 16,* 1–21.

Ladouceur, R., Dubé, D., & Bujold, A. (1994). Prevalence of pathological gambling and related problems among college students in the Quebec metropolitan area. *Canadian Journal of Psychiatry, 39,* 289–293.

Langhinrichsen-Rohling, J., Rohde, P., Seeley, J. R., & Rohling, M. L. (2004). Individual, family, and peer correlates of adolescent gambling. *Journal of Gambling Studies, 20,* 23–46.

Lesieur, H. R., Cross, J., Frank, M., Welch, M., White, C. M., Rubenstein, G., et al. (1991). Gambling and pathological gambling among university students. *Addictive Behaviors, 16,* 517–527.

Lupu, V., Onaca, E., & Lupu, D. (2002). The prevalence of pathological gambling in Romanian teenagers. *Minerva Medica, 93,* 413–418.

Lussier, I., Derevensky, J., Gupta, R., Bergevin, T., & Ellenbogen, S. (2007). Youth gambling behaviours: An examination of the role of resilience. *Psychology of Addictive Behaviors, 21,* 165–173.

Magoon, M., Gupta, R., & Derevensky, J. (2005). Juvenile delinquency and adolescent gambling: Implications for the juvenile justice system. *Criminal Justice and Behavior, 32,* 690–713.

Meschke, L. L., & Patterson, J. M. (2003). Resilience as a theoretical basis for substance abuse prevention. *Journal of Primary Prevention, 23,* 483–514.

Miller, B.C., McCoy, J. K., Olson, T. D., & Wallace, C. M. (1986). Parental discipline and control attempts in relation to adolescent sexual attitudes and behaviors. *Journal of Marriage and the Family, 48,* 503–512.

Moodie, C., & Finnigan, F. (2006). Prevalence and correlates of youth gambling in Scotland. *Addiction Research and Theory, 14,* 365–385.

Moore, S., & Ohtsuka, K. (1997). Gambling activities of young Australians: Developing a model of behavior. *Journal of Gambling Studies, 13,* 207–236.

Moore, S., & Ohtsuka, K. (1999). Beliefs about control over gambling among young people, and their relation to problem gambling. *Psychology of Addictive Behaviors, 13,* 339–347.

Moore, S., & Ohtsuka, K. (2000). The structure of young people's leisure and their gambling behaviour. *Behaviour Change, 17,* 167–177.

National Research Council. (1999). *Pathological gambling: A critical review.* Washington, DC: National Academy Press.

Nower, L., & Blaszczynski, A. (2004). A pathways approach to treating youth gamblers. In J. Derevensky and R. Gupta (Eds.), *Gambling problems in youth: Theoretical and applied perspectives* (pp. 189–210). New York: Kluwer Academic/Plenum Publishers.

Nower, L., Derevensky, J., & Gupta, R. (2004). The relationship of impulsivity, sensation seeking, coping and substance use in youth gamblers. *Psychology of Addictive Behaviors, 18,* 49–55.

Nower, L., Gupta, R., Blaszczynski, A., & Derevensky, J. (2004). Suicidality ideation and depression among youth gamblers: A preliminary examination of three studies. *International Gambling Studies, 4,* 69–80.

Ólason, D. T., Sigurdardottir, K. J., & Smari, J. (2005). Prevalence estimates of gambling participation and problem gambling among 16–18-year-old students in Iceland: A comparison of the SOGS-RA and DSM-IV-MR-J. *Journal of Gambling Studies, 22,* 23- 39.

Ólason, D. T., Skarphedinsson, G. A., Jonsdottir, J. E., Mikaelsson, M., & Gretarsson, S. J. (2006). Prevalence estimates of gambling and problem gambling among 13- to 15-year-old adolescents in Reykjavik: An examination of correlates of problem

gambling and different accessibility to electronic gambling machines in Iceland. *Journal of Gambling Issues, 18,* 39–55.

Orford, J. (2003). Why the British government is wrong to continue to allow juvenile gaming machine playing. *Addiction Research and Theory, 11,* 375–382.

Peacock, R. B., Day, P. A., & Peacock, T. D. (1999). Adolescent gambling on a great lakes Indian reservation. *Journal of Human Behavior in the Social Environment, 2,* 5–17.

Poulin, C. (2000). Problem gambling among adolescent students in the Atlantic provinces of Canada. *Journal of Gambling Studies, 16,* 53–78.

Schissel, B. (2001). Betting against youth: The effects of socioeconomic marginality on gambling among young people. *Youth and Society, 32,* 473–491.

Shaffer, H., LaBrie, R., LaPlante, D., Nelson, S., & Stanton, M. (2004). The road less travelled: Moving from distribution to determinants in the study of gambling epidemiology. *Canadian Journal of Psychiatry, 49,* 504–516.

Shaffer, H. J., & Hall, M. N. (1996). Estimating the prevalence of adolescent gambling disorders: A quantitative synthesis and guide toward standard gambling nomenclature. *Journal of Gambling Studies, 12,* 193–214.

Skokauskas, N. (2007). Adolescent pathological gambling in Kaunas, Lithuania. *Nordic Journal of Psychiatry, 61,* 86–91.

Ste-Marie, C., Derevensky, J., & Gupta, R. (2002). Anxiety and adolescent gambling behaviour. *International Gambling Studies, 2,* 123–141.

Ste-Marie, C., Gupta, R., & Derevensky, J. L. (2006). Anxiety and social stress related to adolescent gambling behavior and substance use. *Journal of child and adolescent substance abuse, 15,* 55–74.

Stinchfield, R. (2002). Youth Gambling: How big a problem? *Psychiatry Annals, 32,* 197–202.

Stinchfield, R., Cassuto, N., Winters, K., & Latimer, W. (1997). Prevalence of gambling among Minnesota public school students in 1992 and 1995. *Journal of Gambling Studies, 13,* 25–48.

Stinchfield, R. D., & Winters, K. C. (1998). Gambling and problem gambling among youths. *Annals of the American Academy of Political and Social Sciences, 556,* 172–185.

Vachon, J., Vitaro, F., Wanner, B., & Tremblay, R. (2004). Adolescent gambling: Relationships with parent gambling and parenting practices. *Psychology of Addictive Behaviors, 18,* 398–401.

Vitaro, F., Arseneault, L., & Tremblay, R. E. (1999). Impulsivity predicts problem gambling in low SES adolescent males. *Addiction, 94,* 565–575.

Volberg, R. A., Abbott, M. W., Ronnberg, S., & Munck, I.M.E. (2001). Prevalence and risks of pathological gambling in Sweden. *Acta Psychiatrica Scandinavica, 104,* 250–256.

Wardman, D., el-Guebaly, N., & Hodgins, D. (2001). Problem and pathological gambling in North American Aboriginal populations: A review of the empirical literature. *Journal of Gambling Studies, 17,* 81–100.

Werner, E. E., & Smith, R. S. (1992). *Overcoming the odds: High risk children from birth to adulthood.* Ithaca, NY: Cornell University Press.

Winters, K. C., & Anderson, N. (2000). Gambling involvement and drug use among adolescents. *Journal of Gambling Studies, 16,* 175–198.

Winters, K. C., Arthur, N., Leitten, W., & Botzet, A. (2004). Gambling and drug abuse in adolescence. In J. Derevensky & R. Gupta (Eds.), *Gambling problems in youth: Theoretical and applied perspectives.* (pp. 57–80). New York: Kluwer Academic/Plenum Publishers.

Winters, K. C., Stinchfield, R. D., & Fulkerson, J. (1993). Toward the development of an adolescent gambling problem severity scale. *Journal of Gambling Studies, 9,* 371–386.

Wood, R.T.A., & Griffiths, M. D. (1998). The acquisition, development and maintenance of lottery and scratchcard gambling in adolescence. *Journal of Adolescence, 21,* 265–273.

Wood, R.T.A., & Griffiths, M. D. (2002). Adolescent perceptions of the national lottery and scratchcards: A qualitative study using group interviews. *Journal of Adolescence, 25,* 655–668.

Wood, R.T.A., & Griffiths, M. D. (2004). Adolescent lottery and scratchcard players: Do their attitudes influence their gambling behaviour? *Journal of Adolescence, 27,* 467–475.

Wood, R.T.A., Griffiths, M. D., Derevensky, J. L., & Gupta, R. (2002). Adolescent accounts of the UK national lottery and scratchcards: An analysis using Q-Sorts. *Journal of Gambling Studies, 18,* 161–183.

Wood, R.T.A., Griffiths, M., & Parke, J. (2007). Acquisition, development, and maintenance of online poker playing in a student sample. *CyberPsychology and Behavior, 10,* 354–361.

Wood, R.T.A., Griffiths, M., Stevens, J., Bartlett, H., & Pye, J. (2006). *Under 16s and the national lottery. Final report.* Prepared for the National Lottery Commission. London: National Lottery Commission.

Yeoman T., & Griffiths, M. (1996). Adolescent machine gambling and crime. *Journal of Adolescence, 19,* 183–188.

Chapter **13**

Macau: China's Entry into the World of Gambling[1]

Richard A. McGowan, MA, MDiv, ThM, DBA

Gaming in Macau is not a new phenomenon. Its history traces back to before the Portuguese colonized the island in the sixteenth century. When the Portuguese did arrive, they did not attempt to change the customs of the indigenous people. However, gaming was not officially legalized until the 1850s, and the industry did not start booming until the end of the nineteenth century.

In 1930, the decision to auction off a monopoly on gaming was made by the Portuguese Colonial rulers to the Hou Heng Company, headed by Fok Chi Ting.[2] It was then awarded in 1937 to the Tai Heng Company, which narrowly lost the right to it in 1962 to STDM. The Portuguese government contemplated four special licenses in 1982, which would have given groups a specific geographic region over which they would have a monopoly. However, this proposition did not pass, and STDM's monopoly existed until 2002. In 2002, Portugal handed over control of Macau to the Chinese government. Since 2002, the Chinese government has made many concessions and subconcessions to gaming companies. The first resort to open as a result of these moves was the Sands in May 2004. Another achievement in 2004 for the gaming companies was the new law that gave them the ability to grant credit and enforce gaming debts, a practice previously not allowed because of the cultural ideals of the residents. However, this new law was so important that Steve Wynn (owner of a premier Las Vegas casino) announced that he would not plan on buying property or opening a casino in Macau until the law was in effect.

MACAU'S GAMBLING EXPLOSION

Recently, Macau has been dubbed the "Monte Carlo of the Orient."[3] This nickname is becoming well-deserved. There were only 11 casinos in 2002. As of August 2006, there were 21 casinos operating on the island. This number was expected to more than double, to 45, in the subsequent three years. As of 2006, 60 new hotels were being planned or built to provide rooms for the upcoming influx of gamblers. However, according to locals, only two of the casinos are actually flourishing. Despite these reports, Macau's gross domestic product (GDP) grew 14.2 percent in 2003 and 28.3 percent in 2004. These factors help the younger locals to be optimistic about Macau's future.

This increase in casinos and hotels has resulted in a similarly strong increase in visitors to the island. In 2005 Macau welcomed 10.5 million visitors from China, 2.5 times as many as it had seen in 2002. Chinese visitors accounted for 56 percent of all visitors in 2005, up from 37 percent in the same time frame. Macau has growth potential in a variety of areas. According to the American Gaming Association, the acronym MICE describes the facets on which Macau should concentrate: meetings, incentives, conventions, and exhibitions. Almost every hotel planned for Macau is looking to specialize in at least one of these growth areas. For instance, the Venetian Macau plans on having 35 percent of its rooms filled by conventioneers. The Macau government has stated publicly that it intends to double its number of visitors to approximately 37–38 million by 2010.[4] Although most indicators are positive, many difficulties need to be addressed. First, there are still various issues with the Chinese government, mainly China's strong tendency to have inconsistent policies. Also, locals have some major concerns for Macau. One obvious concern is that they will become, if they have not already, too dependent on casinos. In a similar vein, they are somewhat bothered by their overreliance on gamblers coming from the Chinese mainland. Also, they realize that the rapid growth of the casino industry in Macau in the recent past and near future cannot continue given restrictions on not only demand, but also, and more importantly, land. Last, local authorities have been citing a sharp increase in casino-related crimes, such as money laundering, but others feel that the numbers are inflated because the grouping "casino-related crimes" is vaguely defined. Clearly, there is a fair amount of unease about Macau's long-term development.

Many companies are vying for a strong foothold in Macau, and some have a set strategy already. There are two basic strategies that casino-hotels can follow, embodied by two Hong Kong–listed companies. First, Melco International Development will compete directly with the U.S. gambling heavyweights Wynn

Resorts, Las Vegas Sands, and MGM Mirage on the basis of grandiose luxury.[5] Melco is planning on building Macau's first six-star hotel, the Crown Macau, in 2007, as well as the City of Dreams (a series of casinos that Macau hopes to rival the Las Vegas strip) marketed to both VIP and mass-market gamblers, in 2008, in a joint venture with Australia's Publishing and Broadcasting. Melco has a leg up in the race for supremacy in Macau because of the familiarity of its CEO, Lawrence Ho, the son of the casino monopolist Stanley Ho, with the gaming industry in Macau.

Galaxy Entertainment Group, on the other hand, intends to pursue casual visitors from mainland China who are on a budget. This is the less popular of the two strategies, but it has its advantages because there is less competition with the U.S. companies, and Melco is looking for higher-end customers. With this strategy, Galaxy does not have to invest as much in extravagant architecture and other amenities. However, Galaxy cannot afford to save on real estate, so the company is investing heavily in some of the best real estate on Macau. Either way, any new casino must have a good conception, a good location, and imagination because copycat casinos will suffer.

The growing popularity of the Macau gaming industry is aided in many ways. First, the government is improving the infrastructure in the hope, as noted earlier, of doubling visitation from 18.7 million in 2005 to 37 million in 2010. Second, the most profitable game, slots, is surprisingly popular with the Asian gamblers in Macau. Also, they have taken a liking to electronic table games, which are preferred by casinos over live table games because they take up less space and do not require paying dealer salaries. This is a key point because there are currently major labor shortages, including for dealers, in Macau, and this could signify a shift in Macau into Vegas-style gaming. This shortage of a labor supply exists because Macau's population is less than 500,000, but its residents protest the hiring of Chinese and Philippine workers, slowing the growth ability of the gaming industry in Macau. Many new casinos now poach some of the best employees from competing casinos and start wage wars. All of these developments are creating growing concerns about wage inflation among gaming companies. The average Macau gambler is searching for convenience and value. Most of them will not travel beyond the Macau peninsula center of mass, and despite the cheap hotel rates (US$20–40 per night), a decent percentage will stay just off the island for the slightly cheaper rates and then commute to the casinos, making Macau extremely dependent on gaming revenues. Because this market is geared to a low-end gambler, Macau's government does not want any permanent residents, but would like to see some time shares, second homes, and vacation suites.

COTAI—THE RICH MAN'S ALTERNATIVE

Currently, Macau is generating the majority of buzz in the gaming industry, but much of that attention may soon be shifting to its neighbor island, Cotai. Right now, Cotai mainly attracts "Macau rejecters"[6] and some conventioneers, but the island plans on attracting more developers and gamblers from Macau beginning around 2009. These "Macau rejecters" are affluent gamblers who have already become disinterested in Macau and want Cotai to become a high-end resort. The geographical area that Cotai would target has 120 million residents, giving it a large population from which to draw. This target audience will travel great lengths and pay premium prices (approximately US$200 per night) for a quality product. Also, nearby Hangquin Island is waiting for government approval to begin development of residential and leisure resorts. It is hoped that by 2010, Cotai can reach its full potential with many high-quality casinos and improved access to the area.

However, many pitfalls could arise and lessen Cotai's ability to match or surpass Macau's gaming potential. There is currently a lack of critical mass of casino venues on and access to Cotai, which will not be improved until at least 2008. This means that only "must see" casinos will have any chance to succeed. The most difficult of these problems to improve is the projected extra nine million land visitors coming through already saturated border crossings and congested roadways. Once the critical mass and access issues are resolved, the casinos of Cotai have to convince the gamblers of Macau to change their preferred casino, so that they can add them to the current group of "Macau rejecters." The optimistic view points to the surprising change in Asian preferences toward slots as a possible precedent for gamblers switching from Macau to Cotai. A bad sign for Cotai is that the opening of the Grand Waldo Cotai fell short of all expectations and has spooked investors for other potential Cotai casinos. However, optimists in this case focus on the differences between the unsuccessful opening of the Grand Waldo Cotai and the successful opening of the Venetian Macau. A few of the reasons for these differing results are that the Grand Waldo Cotai did not have as strong of a marketing strategy and it also lags behind the Venetian Macau in size and amenities. Many companies, including Wynn, are taking a wait-and-see approach to get a better feel for the market, its customers, and the progress on Cotai's infrastructure, delaying many openings until 2009 or 2010. The casinos are not the only industry struggling on Cotai. As in Macau, the short supply of land in Cotai has started a land scramble that is sending real estate prices through the roof.

If all of Cotai's needed changes occur in a timely fashion, and the pitfalls are avoided, then Cotai has the ability to become one of the top casino resort locations in Asia. The projections for Cotai predict that it could have double or triple the number of casinos that Macau has, sometime between 2012 and 2015.[7] This could make it the dominant casino resort destination in the world.

Another potential revenue driver is the availability of retail on both Macau and Cotai. Many investors are skeptical because of the difficulty that the Fisherman's Wharf is having. However, this area has some of the lowest rents on the island and attracts a low-end shopper. Two planned casinos will court high-end retailers, in order to be attractive to high-end customers. The Wynn Macau is planning on opening 10,000 square feet of retail space to satisfy its gamblers. The Venetian Macau is pulling out all of the stops to open up a retail mall that will rival its Grand Canal Shoppes in Las Vegas. However, many retailers do have reservations, so they are signing short-term leases, to take a wait-and-see approach toward the viability of retail at the Venetian Macau before signing a long-term lease. Most indicators suggest that both of these retailing endeavors will thrive, and investors should not be concerned with the Fisherman's Wharf struggles.

QUESTIONS

Clearly, Macau has achieved the greatest growth of any recent market for additional gambling. Yet some interesting questions need to be asked about whether this growth can be sustained. The first consideration is, of course, the attitude of Chinese officials. If they fear that they are losing control of these islands, they can easily deny the remaining available land parcels on both Macau and Cotai. This could easily happen, given their fear of being too dependent on one industry—namely, casino gambling. And after all, the Chinese government has made no firm commitment to the concept of private property, especially for casino operations.

Another concern of Chinese officials is the possibility of wage inflation because of the labor shortage on the island. Finally, it will be interesting to see how the governing Communist Party will reconcile its previous ban of all types of gambling in China with this establishment of another Las Vegas. If the party's authority is ever threatened on the mainland, would the Communist Party crack down on gambling on Macau in an effort to reestablish its "purity" and authority? So far, the Chinese government has "tolerated" this gambling exception, but whether or not it would sacrifice Macau as its gambling capital in times of need has yet to be tested.

NOTES

1. From *The Gambling Debate*, Richard A. McGowan. Copyright 2007 by Richard A. McGowan. Reproduced with permission of Greenwood Publishing Group, Inc., Westport, CT.
2. Jorge Godinho, "Macau Gaming Law," paper presented at the 13th International Conference on Gambling and Risk-Taking, Lake Tahoe, May 2006.
3. "Macau," *Antara News*, August 8, 2006.
4. Keri Geiger, "In Macau, It's Time to Bet," *Wall Street Journal*, June 30, 2006.
5. Ibid.
6. J. P. Morgan, *Macau Update*, newsletter, June 12, 2006.
7. Ibid.

Chapter **14**

Native American Gambling: Economic Development or Dependence?

Richard A. McGowan, MDiv, ThM, DBA

There are over four million Native Americans living in the 567 federally recognized Indian Tribes in the United States. Over the past 20 years, the quality of life for American Indians living on reservations has increased tremendously. However, the economic welfare statistics of these tribes still consistently place them far behind the rest of the American population. Although the cultural and historical reasons for this discrepancy are important, they will not be the focus of this chapter. This chapter concentrates purely on the rationale for Native American gambling. More specifically, this chapter concentrates on the effects that the Indian Gaming Regulatory Act (IGRA, 1988) has had on the Native American population in the United States.

This issue is of importance because many financial and political effects must be considered during development of plans for new casinos, creation of tax codes, or drafting of federal legislation, for example. States cannot impose taxes on American Indian casinos, but the IGRA allows states to negotiate compacts for exclusive rights with tribes for a share of their revenues. One-third of the 22 states that permit "Las Vegas–style" games on American Indian land receive significant revenue from the tribes.[1] Although certainly many other aspects must be considered when these political tasks are undertaken, the effects that tribal casinos have had on the welfare of the Native American population, if significant, should be weighed heavily. And if not, then the claims that tribal casinos have been a huge success need to be examined more closely. Although the data available limit the scope of this study, it should be recognized that concerns over the social welfare of Native Americans since the passing of the

IGRA (drafted as a direct result of the decision of the *California v. Cabazon* case) have even served as an impetus for a recent push for Congress to draft further legislation. Some of the proposed bills call for more closely measuring the negative effects that casinos have had on Native Americans, altering the legal uses for Indian casino revenue and changing the way the oversight committee operates. Further, in light of the recent Jack Abramoff lobbying scandals, the political associations of tribal leaders have been called into question, thereby implicating the casinos that they run.[2]

Tribal gaming is a $19.6 billion per year industry, and it is getting larger,[3] as reports from February and June 2005 indicate with headlines such as, "Tribal Casino Takes Are Soaring, Surpassing Those in Nevada."[4] According to excerpts from Dr. Alan Meister's study on Indian gaming, the growth rate of Indian casino revenues from 2003 to 2004 exceeded 15 percent[5] The number of tribes with gaming facilities grew about 3 percent in 2004 (from 221 to 228), and the number of Indian gaming facilities saw growth at about 5 percent in 2004 (from 385 to 405).[6] Although they are highly correlated with the development of new gaming facilities, the numbers of gaming tables and gaming machines have seen tremendous growth as well. In 2004, the number of gaming machines grew by 10.7 percent, and the number of table games grew by 9.4 percent. These numbers are significantly larger than the percentage of growth we see in new gaming facilities; therefore, the currently existing gaming facilities clearly are continuing to grow. In 2004, for instance, in California there were no new tribes with gaming facilities, and there were no additional Indian gaming facilities in the entire state. However, California saw a 4.5 percent growth in gaming machines and saw 16.9 percent growth in the amount of table games within its already existing 54 Indian casinos.[7]

Even nongaming revenue at Indian gaming facilities saw significant growth in 2004. This revenue includes money spent by casino patrons on food, beverages, hotel stays, retail purchases, and other entertainment at the gaming facilities. If it were possible to measure the contribution these patrons made to neighboring facilities as a direct result of their visits to the casino, then this revenue would be included as well. However, because this data is practically nonexistent, the reported nongaming revenues have the effect of underestimating the total economic contribution of expenditures at casinos. Nongaming revenue grew by 7.6 percent in 2004 or from $1.79 billion to $1.93 billion dollars. However, as discussed in the following sections, the stipulations of the IGRA are less strict about nongaming revenue than they are about revenue directly created by and received from gaming expenditures at casinos. Therefore, the effects that these revenues have on Native American welfare are much less than the effects of revenues received from gaming. Also, nongaming revenues make up less than

10 percent of gaming revenue. In general, it can be asserted that noncasino revenue does not play an important role for Native American casinos.

Furthermore, in the economic impact analysis that Dr. Alan Meister performed, he estimated several effects that tribal gaming has had on the overall economy. In total, the claim is that Indian gaming in the year 2004 contributed about $53.1 billion in output, $19.7 billion in wages, and 545,000 total jobs and helped create about $6.3 billion in tax revenue. Indian gaming in the year 2003 saw about $45.3 billion in output, $17.3 billion in wages, 489,000 jobs, and $5.7 billion in tax revenue. When the revenue-sharing agreements that each state has developed are considered, totaling $889 million, the total tax revenue garnered in 2004 reaches $7.2 billion. Of the 545,000 jobs supported by Indian casinos in 2004, 279,000 of those jobs were directly induced, and 266,000 were indirectly supported, by the output of Indian gaming. The $6.3 billion of tax revenue mainly comes from secondary economic activity as estimated by the input-output analysis that Dr. Alan Meister conducted. In his study, Meister used the Impact Analysis for Planning (IMPLAN) method, which has been in use since 1979 and is used by the Federal Emergency Management Agency (FEMA) and the Bureau of Economic Analysis; it is also very similar to the format used by the United Nations. For most of the economic estimations such as tax revenue, jobs created, and wages distributed, IMPLAN came into use.

Figure 14.1 shows the amount of revenue shared by the tribes and states according to their agreements in 2004. As one can see, states such as Connecticut, Wisconsin, and California have reasonably substantial revenue-sharing agreements with their tribes, whereas the governors of states such as Minnesota and Washington often receive flak for failing to negotiate successful compacts for their states. At this time, the IGRA does not explicitly mandate that states receive a portion of the revenue from the tribes. However, it is pervasively assumed that changes to the IGRA will be made in the future and that among those changes will be an additional clause that provides the framework for adopting revenue-sharing agreements in new compacts.[8]

The issue surrounding revenue-sharing agreements is a tricky one. From the states' perspective, they are permitting such activity to go on within their borders, and most likely their residents are the patrons of Indian casinos on reservations in their state. Therefore, as with any other commercial activity, the states should have the right to receive some portion of the profit from these casinos. However, from the Native American viewpoint, they are a sovereign nation and not subject to the laws and taxation principles of the states in which they are located. Also, a fear often expressed by politicians who side with Indian tribes is that the states will basically coerce the tribes rather than meet them

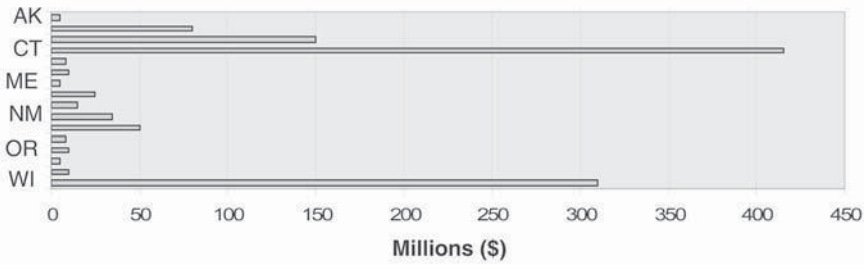

FIGURE 14.1 Revenue sharing between states and Native American casinos. Source: Analysis of the Economic Impact of Indian Gaming in 2004, National Indian Gaming Association (January 2005).

as equals at the bargaining table.[9] Hence the issue is once again whether the tribes are truly sovereign or have become merely wards of state government. Two well-versed political scientists have raised the question of whether compacts are "*compromises*, or are they *compromised?*"[10] The focus of Steven Light and Kathryn Rand's work is the political consideration of the impact of Indian gaming, mainly on sovereignty of the tribes. Light and Rand's work is considered further throughout this chapter and more specifically when we consider the pros and cons of tribal gambling.

Clearly, given the preceding facts and figures, Indian gaming is becoming a significant factor in the U.S. economy, in addition to being the most significant contributor to the Native American economy. Although commercial casinos brought in just under $30 billion in revenue last year, Indian casinos grew to nearly $20 billion. The growth rate of Indian casinos has also significantly outpaced commercial casino development—even since the year 2000—as the establishment of Indian casinos has become more complete. Dr. Alan Meister sees no reason to assume that the growth of Indian casinos will stop and even predicts that they will continue to grow faster than their commercial counterparts. Obviously, as the markets mature and the base of revenue becomes much larger, Indian growth rates will have to decline. However, as more and more states begin to negotiate casino development with the tribes located within their states, continued expansion is to be expected.[11] Another reason it is important to consider the influence of casinos on Indian welfare is that at first glance there appears to be a significant relationship between casinos and expedited improvement in quality of life. Table 14.1 shows a chart created by a group at Harvard when they looked at some of the simple observable results from the 2000 U.S. census.

Table 14.1.
Changes on Reservations Other than Navajo (Changes 1990–2000 presented in points unless indicated as %; OTSAs excluded)

	Nongaming	Gaming	United States
Real per capita income	+21%	+36%	+11%
Median household income	+14%	+35%	+4%
Family poverty	−6.9	−11.8	−0.8
Child poverty	−8.1	−11.6	−1.7
Deep poverty	−1.4	−3.4	−0.4
Public assistance	+0.7	−1.6	+0.3
Unemployment	−1.8	−4.8	−0.5
Labor force participation	−1.6	+1.6	−1.3
Overcrowded homes	−1.3	−0.1	+1.1
Homes lacking complete plumbing	−4.6	−3.3	−0.1
Homes lacking complete kitchen	+1.3	−0.6	+0.2
College graduates	+1.7	+2.6	+4.2
High school or equivalency only	−0.3	+1.8	−1.4
Less than ninth-grade education	−5.5	−6.3	−2.8

Source: Taylor, Jonathan B., and Kalt, Joseph P, *American Indians on Reservations: A Databook of Socioeconomic Change between the 1990 and 2000 Censuses* (Cambridge, MA: Harvard Project on American Indian Economic Development, 2005). See www.ksg.harvard.edu/hpaied/pubs/pub_151.htm.

These numbers suggest that there might be a significant correlation between gaming and a stronger performance of welfare improvement over time. In 13 of the 15 categories, gaming tribes performed better than nongaming tribes. Although at first glance it is apparent that Native Americans' lives have improved since the inception of the IGRA, will advances and growth in Indian gaming result in comparable gains in Native American welfare in the future? Does this improvement in the lives of Native Americans justify the costs of gambling in the communities that surround these casinos? Are states using Native American casinos as agents for revenue while leaving these casinos largely unregulated?

INDIAN GAMING REGULATORY ACT (IGRA)

The impetus to address the Indian gaming situation came from a court case involving a tribe of Native Americans from California called the Cabazon Band of Mission Indians. In 1953 Congress passed a law that authorized states to

extend state criminal laws to Native Americans. This law, known as Public Law 280, was often used by the state to justify regulation of activity on reservations. However, when states began to impede the rights of tribes to operate gaming facilities on their reservations, the Cabazon tribe forced the state of California to take them to the Supreme Court. In *California v. Cabazon* (1987), the Supreme Court ruled that Indian tribes had the inherent right to self-rule and that Public Law 280 applied only to limited circumstances, particularly when criminal activity was taking place between Indians and non-Indians. Specifically, the courts ruled that Public Law 280 was not enough justification for states to impede on tribal sovereignty in civil terms.[12] Noticing this glaring legal omission, Congress quickly acted to draft legislation that would help fill the void of confusion, whence came the Indian Gaming Regulatory Act.

In the time between the *Cabazon* decision and the adoption of the Indian Gaming Regulatory Act (IGRA), gaming sprang up nationwide on tribal reservations. This helped charge the atmosphere surrounding the debate on the IGRA. In drafting the IGRA, Congress sought to balance tribal rights to sovereignty with the right of a state to regulate what sort of activity takes place within its borders, thereby affecting its citizens. One of the major considerations and justifications for federal intervention in this matter was the issue of organized crime. One major fear that came in discussions of gambling was the concern about organized crime gaining a foothold in the casino industry. Because the business involves transactions with large amounts of cash, many spectators were suspicious that Indians, without private-run management interference or federal intervention, would turn to the realm of organized crime to help them run their businesses and gain political influence. This was definitely something that legislators kept in mind when drafting earlier forms of the bill and that influenced legislators' decision to allow private firms to help run Indian casinos.[13]

According to the opening section of U.S. Code Title 25 Chapter 29, Congress had five main things in mind when considering the IGRA: (1) tribes had begun to utilize the revenue drawn from casinos to generate governmental funding; (2) tribes had turned to outside management, but existing law provided no standards by which those management contracts could be regulated or approved; (3) existing federal law did not provide any clear stipulations for regulation of Indian gaming; (4) at that time, the goal of federal policy involving Indians was to "promote tribal economic development, tribal self-sufficiency, and strong tribal government"; and (5) Indian tribes had the right to wholly regulate any gaming activity on their land that was neither prohibited nor strictly regulated by the state or federal governments.

The major provisions of the IGRA sought to create three separate classes of gaming, and a different regulatory scheme for each class, and put stipulations

on the use of casino revenue. The first thing that the IGRA established, the three classes of gaming, are still used today to define various types of gaming in both commercial and Indian casinos. Class I gaming refers to traditional social games with minimal prizes, clearly targeted toward ceremonial Native American forms of gaming. The regulatory authority over these types of games is vested exclusively in tribal governments and is not subject to any of the requirements in the IGRA. Class II gaming refers to bingo and other similar games of interplayer chance, such as lotto, pull-tabs, and punchboards, if played at the same location as bingo. Also included in this are card games where the establishment is not banking any of the money. The regulatory authority over these types of games is vested in the tribal governments insofar as the state in which the tribe is located permits such gaming for any purpose and the tribal government adopts a gaming ordinance that is approved by the National Indian Gaming Commission (which is also established later in the IGRA). Class III gaming refers to every other type of gaming, including slot machines, banked card games, and typical casino games such as blackjack, roulette, craps, any wagering games, and electronic facsimiles of any game of chance. There are three main clauses regarding the regulatory authority of such gaming: the particular form of gaming must already be permitted by the state in which the tribe is located, the tribe must negotiate a contract with the state and have it approved by the Secretary of the Interior, and the tribe must have a tribal gaming ordinance that has been approved by the National Indian Gaming Commission.

Further, the IGRA limits the use of any casino revenue to three major categories: (1) to fund tribal government operations or programs; (2) to provide for the general welfare of the Indian tribe and its members; or (3) to promote tribal economic development. Of course, the tribes are also allowed to make donations to charitable organizations or help fund the operations of local government agencies.

The tribal compacts described in the text of the IGRA are very vaguely outlined. The IGRA does not require the compacts to have any specific terms except that both parties approach the negotiations in good faith—particularly the state, given that the Indian tribe is the party that initiates the negotiations. If this disposition of good faith is in question, the Indian tribe has the right to sue the state in federal court. Many procedures detailed in the IGRA are in place to handle these types of situations as they develop. The procedures are not as important as the implications for tribal and state sovereignties, but in general, government mediators step in and utilize loosely constructed general principles, the goal of which is to establish a well-balanced compromise between both parties. It is through these mediums that states are able to negotiate revenue-sharing agreements and other issues of taxation. Although these

work on a state-by-state basis, as is evident by the disparity among revenues collected by states, in general they provide a means to mitigate the cost that the casinos incur to the state, and they often times go above and beyond this inferred cost.

In particular cases, it is in the Indian tribe's interest to agree to pay more if the state will promise to help keep commercial casinos banned or kept far away from the prime location of the tribal casino. For instance, in 1992, the Mashantucket Pequot tribe negotiated such a contract with the state of Connecticut. In exchange for the tribe's promise to share with the state 25 percent of the revenue generated from the Foxwoods Resort Casino's slot machines, Connecticut effectively guarantees them exclusive rights (along with Mohegan Sun, which now has the same contract) to operate slot machines within the state. Since the precedence of this revenue-sharing agreement, many other tribes have offered similar terms and come to settlement on them. Quite recently, Harrah's Entertainment offered similar commercial revenue-sharing terms with the states of Rhode Island and Pennsylvania if they grant Harrah's exclusive slot machine rights. In Rhode Island, Harrah's has agreed to pay 25 percent of slot revenue up if revenues do not surpass $400 million and a higher percentage to be determined if revenues exceeded $400 million, while in Pennsylvania, Harrah's has agreed to pay a whopping 53 percent excise tax on slot revenue.[14] The justification that this commercial entertainment giant offers to Rhode Island legislators is that the facility to be developed in Rhode Island is more of a resort; therefore, it needs fewer taxes to operate its smaller casino. Harrah's Entertainment assists in the management of as many as nearly 200 Indian casinos in North America.

Also established by the IGRA was the National Indian Gaming Commission (NIGC). As an oversight committee on tribal gaming in the United States, with limited regulatory powers, the NIGC was to be funded by a minuscule tax on the revenues of Indian casinos. With the commission's funding limited to 2.5 percent of the first $1.5 million in revenues and 5 percent thereafter, with a cap of $8 million, the effectiveness of the commission has been questioned at every level of government (U.S. Code Title 25). In 2005, Senator John McCain (R-AZ), then chair of the Senate Indian Affairs Committee, introduced the legislation regarding the NIGC. This bill, S.1295, would have forced the NIGC to be held more accountable and would also provided an increased amount of funding, to the tune of .08 percent of Indian casino revenue without any cap. Under this setup, the revenues received by the commission would have increased to about double what they are now, and then would have proportionally increased relative to the growth of the industry. This bill would have also allow the NIGC to crack down on off-reservation gaming, which is often seen

as a scam that allows the tribes to illegitimately profit by creating and supporting commercial casinos under the guise of tribal sovereignty. In 2006, this bill passed the Senate Indian Affairs Committee but still remains on the agenda of the House Resources Committee awaiting approval for vote.[15]

PRO–INDIAN CASINO ARGUMENTS

There is a lot of literature supporting the propagation of Indian casinos. The study with the most data to back its conclusions was conducted by the Harvard Project for American Indian Economic Development. Authored primarily by Jonathan Taylor, a leading research expert on Indian welfare, *American Indians on Reservations: A Databook of Socioeconomic Change between the 1990 and 2000 Censuses* provides a summary glimpse into the changes in welfare that American Indians experienced during the explosion of tribal casinos. Basically providing two snapshot images of the state of Native American welfare, this study looked at the empirical census data from both 1990 and 2000, breaking it down by every single federally recognized reservation. But aside from the fact that this study neglected to use econometric analysis or examine the revenue-side data, the institution that funded this study receives massive donations from the National Indian Gaming Association (NIGA) to run its research. The NIGA is a nonprofit organization of all the Indian tribes that have casinos—it is clearly in their best interest to promote the expansion and growth of the Indian casino industry. Therefore, the research that the NIGA conducts, though mainly objective, often has suspect results that appear to maintain an optimistic outlook on Indian gaming. The *Cabazon* project, one that specifically focuses on economic development with regard to Indian gaming and of which the Harvard Project study is a part, on the whole manifestly supports the expansion of tribal sovereignty and rights—issues that are not so clearly defined on the national political stage. "Essentially, the research of the Harvard Project finds that poverty in Indian Country is a political problem—not an economic one."[16] Their claim is that the economic development of the tribes, which is bolstered by Indian gaming, is merely the means to the end of achieving full tribal sovereignty and self-reliance. In 2004, the NIGA produced its *Analysis of the Economic Impact of Indian Gaming*. This report details much of the alleged success that tribes have had with casinos. Although much of the evidence is anecdotal or qualitative, the report does provide some firm statistical support for its very optimistic outlook for Indian gaming. The NIGA touts statistics such as the fact that in 2005, 69 percent of Americans thought that Indian nations deserve their help, or that 86 percent of Americans thought that Indian tribes benefit from having casinos.[17] The *Analysis of the Economic Impact*

of Indian Gaming clearly states that the mission of NIGA is to "protect and preserve the general welfare of tribes striving for self-sufficiency through gaming enterprises in Indian Country."[18] Also included in the association's analysis is a mention of every positive outcome that Indian gaming could have ever possibly influenced, from jobs to roads to schools. In order to further garner sympathy and support, the NIGA points out that there are various shortfalls among the Native American community when it comes to keeping up with national norms and includes notations of various education, poverty, health, and crime statistics.

Other published books that may give the reader a clearer picture of the effects of Native American gambling are *Gambling and Survival in Native North America* by Paul Pasquaretta (2003) and *Indian Gaming and Tribal Sovereignty* by Steven Andrew Light and Kathryn R. L. Rand (2005). The latter provides a very thorough analysis of the political implications of casinos through the lens of tribal sovereignty. Again, the researchers behind *Indian Gaming* clearly favor the expansion of tribal sovereignty, and their book is predominately anecdotal and historically based.

ANTI–INDIAN CASINO LITERATURE

Legalized Gambling (2006), edited by David Haugen, provides a collection of abridged articles on various gambling topics, such as Indian gaming, Internet gaming, social consequences, personal stories, and a general overview of gaming in the United States. Although not every article in this collection is anti-casino—in fact, it attempts to provide an equal amount of articles for each side—it is one of the only available sources that clearly try to present this point of view. In these articles, once again, we see that the evidence provided is largely anecdotal and subjective. Although this is clearly an important component of the consideration as to whether casinos are a viable and good option for Indian tribes to gain economic independence, anecdotal evidence is very difficult to include in any quantitative analysis or econometric study. Some of the interesting things for legislatures and concerned citizens to keep in mind about Indian casinos are the alleged social burdens that they place on the public: costs to local communities in the form of upkeep, roadway paving, and police patrols and other implied public costs. There are claims that casinos lead to increased crime in the surrounding areas, that the people who are drawn to visit communities with casinos are not the most upstanding citizens of this country, and finally, that the harmful effects on addicted gamblers and ordinary, but excessive, gamblers are not nearly outweighed by the benefits that Indian casinos receive from the economic independence they gain. And these concerns do not

consider the larger moral question as to whether gambling should be legalized in the first place, which as a democratic society, America has determined it should, viewing it as an acceptable form of business practice.

Finally, there is the story about the famous Mashantucket Pequot founder who, on his marriage license, claimed to be "white." Many people point to this as evidence of Native Americans simply taking advantage of a crooked system that seeks to pay reparations to a people that no longer necessarily deserves them. It is true that there were only four registered members of this tribe in 1990; today, however, there are several hundred members. The Mashantucket Pequots now host the largest Native American casino in the country and hence are flourishing as operators of one of the most successful casinos in the world. There are two sides to every story. The next section gives the reader a chance to ascertain whether the advent of Native American casino gaming has really made a difference in the welfare of tribal members.

COMPARING GAMING AND NONGAMING TRIBES IN 2005

One fundamental question that needs to be addressed is this: has Native American gambling improved the plight of Native Americans? One way to explore this question would be to test different sets of welfare statistics on two groups: those with gaming and those without. The method that was utilized to determine whether or not there was a statistically significant difference exists is called the unpaired (independent) samples "t" test. The categories (which refer to American Indians on reservations, including Navajo, unless otherwise noted) to be tested included:

1. median income,
2. family poverty, unemployment,
3. percentage of houses that lack kitchen facilities,
4. actual number of houses that lack kitchen facilities,
5. aggregate income of tribe,
6. average aggregate income,
7. percentage over the age of 25 who are college graduates,
8. percentage over the age of 25 who have a high school degree,
9. percentage over the age of 25 who have less than a ninth-grade education,
10. the actual numbers for each of the preceding three statistics,
11. percentage of families living in poverty, percentage of population that owns houses,
12. percentage of occupied houses that are actually owned by the Indians occupying them,

13. percentage of homes that are occupied by American Indians that are overcrowded,
14. percentage of all races on reservations that are self-employed, and
15. percentage of American Indians that live in deep poverty.

RESULTS OF INDEPENDENT SAMPLES TESTS

Out of the various categories that were tested, only six of them came out statistically significant (a "t" test value of 2 or greater), with a seventh one reasonably close. Median income produced t-scores of 3.086 and 3.094; percentage of houses lacking kitchen facilities produced t-scores of –2.391 and –2.304; average aggregate income produced t-scores of 1.797 and 1.873; actual number of college graduates came really close, producing a t-score of 1.320; percentage of American Indians over the age of 25 with a less than a ninth-grade education produced t-scores of –2.045 and –1.991; percentage of occupied homes that are owned by American Indian residents produced t-scores of –2.370 and –2.346; and percentage of population of all races on reservations that are self-employed produced t-scores of 2.069 and 2.049. The rest of the statistics produced t-scores that ranged from .344 to .998, none of them being significant at even the 10 percent level.

CONCLUSIONS

From these analyses, it appears as though the only welfare statistic that has been significantly influenced by casino revenue in the past 16 years is average aggregate income of American Indians. When you take this result and compare it with the fact that median income has not seen an equivalent impact, it appears as though a lot of the money could be staying at the top. If some of the wealthiest tribal members were keeping a lot of the profits for themselves, it would explain why the average income is much higher than the median. This is being addressed in various political circles. In particular, Senator John McCain's bill addresses the issue of tribal casinos using profits to reinvest in commercial casinos or build other casinos in off-reservation locations. Although tribal leaders argue that, oftentimes, building a casino away from their reservation can provide opportunities that otherwise would not be economically feasible, opponents of this argue that it also has the potential to cause a great deal of trouble. No one imagined that Indian casinos would have expanded to the extent that they already have, and this is just one more way that they could grow even more—which is undesirable to many.[19]

Whereas commercial casinos grew at a rate of 6.7 percent in 2004, according to the American Gaming Association,[20] Dr. Alan Meister's numbers show us that Indian casino revenue grew at 15.2 percent.[21] This beats even Las Vegas's growth rate of about 10 percent. These significant increases mean that the influence that Indian tribes have on politics and the welfare of their citizens is only going to increase over time. In 1999, Indian tribes donated a total of $2,000 to politicians in the United States. In 2004 that number had surged to $7 million.[22] Although in the wake of the Jack Abramoff scandals, this tremendous increase should slow down, the contributions are not expected to stop any time soon. As their economic well-being increases, Indian tribes are going to become steadily more influential. Real per capita income of gaming tribes increased 36 percent between 1990 and 2000, whereas the income of nongaming tribes and the rest of the nation grew at 21 percent and 11 percent, respectively, during that same time period. This seems significant considering that American Indians received the lowest amount of per capita income assistance out of all Americans.[23] Obviously, it is also possible that the tremendous growth we see is merely a result of the historical setbacks that American Indians have faced over time—that any improvement we see is a result of the relative weakness of their starting point. However, this is not always the case, and if one looks at mere percentage point changes in the census data, there are still significant differences on the surface between the data for Indian reservations and the data for the nation as a whole.

Between the years 1990 and 2000, Indian tribes on the whole increased their overall income at an astoundingly higher rate than the rest of the United States, as described earlier in this chapter. Yet the differences between Indian tribes with gaming and without do not seem to be significant at this time. There are many things to consider when recognizing the lack of influence that gaming has had in 17 years. Many of the effects that are calculated would take much longer than these 17 years to have a massive measurable impact on the Indian population as a whole. Welfare statistics that are related to education and housing, for example, would take a lot longer to be affected by casino income than the aggregate incomenumbers. The time it takes to receive an education must be considered, and additionally, the investment in infrastructure and way of life definitely does not occur successfully over night. Aside from the fact that it took about 8 years for Indian casinos to take off and about 10 years for those tribes to gain any significant political influence, schools have to be built, a culture of education has to be established, and pupils that are of the appropriate age need to pass through the newly established learning systems. For instance, take the Mashantucket Pequot Tribal Nation; this tribe profits so substantially

from its world-renowned Foxwoods Resort Casino that it pays for any tribal member to attend college. Assuming that a person begins high school at the age of 14, that same person probably decides whether he or she is going to attend college by about age 17. Because the education statistics are not measured for anyone under the age of 25, at which age education levels become standardized and typically no longer change, this effect would take anywhere from 8 to 11 years to have any significant effect and much longer to make up a significant portion of the 25-and-older population. Considering this, it is not hard to see why the positive effects that many claim economic independence is having on the tribes might not yet be showing themselves . Either this is the case, or tribal leaders are, to date, making poor investment decisions or restricting the flow of money downward to the rest of the members of their tribes.

Finally, it is always important to look at the big picture when critiquing policy decisions. It is therefore difficult to gauge the specific impact that casinos have had—even more so for the Indian gaming industry because of its perceived isolation on reservations and mysterious reporting practices that are not subject to the same transparency laws as commercial casinos in the rest of the country. More specific to this chapter, it is even more difficult to estimate the negative effects that casinos have had on the Native American populace. Although studies may provide hard statistical evidence of negative effects in the future—one such study is soon due to produce the results of research on the use of methamphetamines by Indians on reservations—currently there is no such evidence.

Therefore, in coming years, we can only hope that the size and power of the Indian gaming industry influences public and private parties to engage in further studies of the effects that Indian gaming has had on the Native American population as well as on the American population at large.

NOTES

1. Swanson, Al, "Indian Casino Revenues Soar," United Press International, Boston Globe, July 7, 2004.
2. National Public Radio, "Indian Casinos and Lobbying, after Abramoff," *Talk of the Nation,* January 17, 2006.
3. Barfield, Chet, "Report Puts Indian Casino Revenue in U.S. at $19 Billion," *Copley News Service*, June 15, 2005.
4. Werner, Erica, "Lawmakers Seek to Limit Off-Reservation Indian Casinos," Ithaca Journal, Associated Press, March 6, 2006.
5. Meister, Alan, *Casino City's Indian Gaming Industry Report* (Newton, MA: Casino City Press, 2005).
6. Ibid.
7. Ibid.

8. Melmer, David, "Tribes Fall Victim to State Deficits," *Global News Wire, Indian Country Today*, June 8, 2005.
9. Light, Steven Andrew, and Kathryn R. L. Rand, *Indian Gaming and Tribal Sovereignty: The Casino Compromise* (Lawrence: University Press of Kansas, 2005).
10. Ibid.
11. Butterfield, Fox, "Indian Casino Revenues Grow or Sizeable Segment of Industry," *New York Times*, June 16, 2005.
12. Light and Rand, *Indian Gaming and Tribal Sovereignty*, 34.
13. Light and Rand, *Indian Gaming and Tribal Sovereignty*, 34.
14. Gregg, Katherine, "In Pennsylvania, Harrah's Offers to Pay Higher Tax Rate," *Providence Journal*, February 14, 2006.
15. Batt, Tony, "Vote Near on Bill," *Las Vegas Review-Journal*, February 9, 2006.
16. National Indian Gaming Association, *Analysis of the Economic Impact of Indian Gaming in 2005*, http://www.indiangaming.org/NIGA_econ_impact_2004.pdf.
17. Ibid.
18. Ibid.
19. Werner, Erica, "Tribal Casino Takes Are Soaring, Surpassing Those in Nevada," *Ithaca Journal*, Associated Press, February 15, 2005.
20. American Gaming Association. "Tribal Gaming: An Issue Overview." http://www.americangaming.org/industry/factsheets/issues.
21. Meister, Alan, Casino City's Indian Gaming Industry Report.
22. American Gaming Association. "Sports Wagering: An Issue Overview." http://www.americangaming.org/industry/factsheets/issues.
23. Taylor, Jonathan, and Joseph Kalt, *American Indian on Reservations: A Data-book of Socioeconomic Change between the 1990 and 2000 Censuses* (Cambridge, MA: Harvard Project on American Indian Economic Development, 2005).

This page intentionally left blank

Chapter **15**

The Current Climate of Gambling in the United States[1]

Richard A. McGowan, MA, MDiv, ThM, DBA

A look at how gambling revenues stack up against revenues from other recreational/leisure time activity sectors reveals not only that the gambling revenues outweigh both music sales and movies combined, but also that the gambling industry is the only one of the industries in this study to have shown consistent growth in each of the last three years.

—Joseph Greff, "U.S. Gambling" (Bear, Stearns, January 2005)

With a few notable exceptions, it is a great time to be in the gambling industry. The year 2005, for example, was a record-breaking one for Las Vegas casinos, which took in $11 billion from gamblers in casino revenues—excluding hotel, restaurant, and bar revenues.[2] In 2004 alone, U.S. gamblers spent $78.6 billion on commercial gambling, a 7.6 percent increase over the year before.[3] Lottery sales increased by an average of 12.5 percent throughout the United States.[4] Tribal gambling and Internet gambling experienced double-digit growth, a rate not seen anywhere else within the gambling industry.

But can discussions about the gambling industry be painted in such broad, sweeping terms? Can one really talk about "the" gambling industry as a whole without generalizing to the point that trends become half-truths and facts and figures become meaningless? What similarities are there between an Internet sports book based on the Isle of Man and the Massachusetts state lottery's daily operations? Given the explosive rate of growth in Internet gambling and the gradual decline of horserace betting, can one ignore the fact that the various segments within the industry are at different stages in their life cycles and

should therefore expect very different opportunities and threats in coming years? It is obvious that each segment deserves (or, more accurately, demands) its own assessment in order for any analysis to be fully developed, relevant, and—most important—meaningful.

There are, however, recurring themes that weave throughout each segment of "the" gambling industry and that merit special attention. The most obvious, and perhaps most important, of these themes is that of regulation. Regardless of what type of gambling is being discussed—be it Internet poker, slot machines in Vegas, or a sports book in the United Kingdom—each and every one of these various business enterprises faces some sort of regulation. In some instances, regulation creates state-held monopolies through special gambling licensing. In other jurisdictions more lax regulations allow for numerous firms to compete for consumer dollars. Finally, in the United States, ambiguous legislation and inconsistent activity by the courts regarding Internet gambling create a legal gray area. In this type of environment, sites enjoy a surplus of consumers and a scarcity of competitors, often resulting in huge profits for the more daring Internet gambling site operators.

Monopolies in the industry, however, are becoming things of the past. A second recurring and universal theme throughout "the" gambling industry is increasing levels of competition. The forms in which this competition manifests itself are as diverse as ever. California's tribal casinos now compete with the Las Vegas Strip. Las Vegas bookmakers compete with Internet sports books based in the United Kingdom. These Internet sports books diversify their product offerings and allow users to play poker online, competing with other Internet poker sites. In many instances, gambling operators compete not only with other operators within the same segment (for example, a casino competing with another casino) but with *every other form of gambling readily available to customers*.

INTERNET GAMBLING

The rise of Internet gambling has been the primary driver of the intensifying levels of competition within the industry. No longer is consumers' ability to place wagers limited by their geographic proximity to gambling operators (nor, in some instances, are they limited by the legislation of their country's jurisdiction—for example, China). Internet gambling operators can serve a truly global customer base, and with the financial barriers to entry relatively low in comparison with the huge profits being reaped through Internet gambling, new entrants can enter at will and attempt to compete with the market leaders. As discussed earlier, these online operators compete not only with each other

but with traditional land-based operators as well. Although both land-based and Internet gambling operators have done fairly well in recent years, given the explosive growth and successful initial public offerings (IPOs) of several online-only firms, it appears that the Internet sites will ultimately come out ahead. Indeed, players in the gambling industry must be ready to compete in the "borderless global marketplace the Internet has created."[5]

Regardless of which segment of the gambling industry a particular firm is involved in, be it market leader or new entrant, no firm can afford to underestimate the impact that the Internet will have on its business. Internet gambling has revolutionized the gambling industry, and what we have seen thus far is only the tip of the iceberg. Estimates vary, but for discussion's sake, one expert estimates that consumers spent over $8 billion on Internet gambling in 2004, and he expects that number to more than triple by the end of the decade.[6] Although this sum represents only a small portion of global expenditures on commercial gambling, certain indicators point toward a bright future for Internet gambling operators. A case in point: PartyGambling plc, operator of PartyPoker, executed a highly successful IPO and listed on the London Stock Exchange with a market cap of almost $8.5 billion. Its shares have appreciated significantly since then, and PartyGambling now has a market capitalization larger than Harrah's Entertainment.[7] In light of the fact that Harrah's Entertainment is the world's largest casino operator, the true potential of Internet gambling has become apparent.

The landscape of the online gambling industry is undoubtedly the most challenging to navigate of any of the gambling industry's segments. In no other segment must an operator navigate such a web of legislative snarls or compete so ferociously for revenues. Unlike for traditional land-based gambling operators, for online casinos, poker tables, and bookmakers, barriers to entry are extremely low. With relatively little initial capital, a firm can purchase third-party software at minimal cost (royalties included) and apply for a license in any of a number of jurisdictions throughout the world. As a result, "given the portability of pure online services,"[8] jurisdictions now compete to draw online operators, resulting in attractive tax regimes for firms willing to be flexible in their location. This leads to increasing levels of competition for consumers, driving up marketing spending. The added complexity of the various legal issues regarding the jurisdiction of the consumers themselves further muddles the legal waters.

The following is a short summary of the various issues surrounding Internet gambling. Current legislation in various countries runs the gamut from complete prohibition of Internet gambling to its legalization and regulation. In the United States, the Department of Justice (DOJ) holds that all Internet

gambling is illegal, under the 1961 Federal Wire Act, which prohibits bets made over telephone and other "wires."[9] Although the DOJ has no intention of prosecuting casual gamers, it is adamantly opposed to allowing firms located within U.S. jurisdiction to run online gambling sites. Harrah's and MGM Mirage, for example, both formed Internet casinos, only to shut them down under pressure from the DOJ.[10] The DOJ's strategy has been to put pressure on financial intermediaries, fining them for processing illegal online gambling transactions. For example, the DOJ fined PayPal $10 million in 2003 for such violations.[11]

Interestingly enough, the legality of certain actions by the Department of Justice in restricting online gambling subsequently came under scrutiny. A case filed by the island of Antigua, a small Caribbean center for offshore Internet gambling, charged that the Department of Justice had restricted the "cross border supply of gambling and betting services" in violation of U.S. obligations under the General Agreement on Trade in Services (GATS) and World Trade Organization (WTO) regulations. The outcome proved ambiguous, with both sides claiming victory. Nevertheless, it appears that the United States ultimately will have to acquiesce to the WTO ruling—either through "total prohibition, including currently legal forms of online gambling . . . or liberalization [*sic*] and permissive regulation of online gambling."[12]

Unlike U.S. legislation, legislation within the United Kingdom was updated following passage of the Gambling Act in 2005. Although online betting had not previously been illegal in the United Kingdom, the Gambling Act explicitly legalized online gambling and clarified a number of issues regarding advertising to the UK audience.[13] But although the United Kingdom's stance on Internet gambling is quite clear (permissive regulation), the greater European landscape is still divided. The 1957 Treaty of Rome established free trade principles regarding services, yet the European Court of Justice "has wrestled with the conflicting claims of member state laws predicated on sovereign power over gambling and free trade principles."[14] The political structure and conflicting interests of greater Europe are immeasurably complex, but research analysts at Deutsche Bank believe that the European market ultimately "will open up to cross-border online gambling . . . through rulings from the EU (European Union) courts rather than through a directive."[15]

Regardless of the short-term developments in global regulation, the expansion of Internet gambling is inevitable in the long term. The blistering growth rates of online revenues, seemingly endless consumer demand, and market capitalization figures usually reserved for blue-chip stocks all point in one direction: Internet gambling will continue to grow as more firms enter the industry and cater to "an audience that is discovering that it actually quite enjoys casual gambling."[16] In regard to the future of Internet gambling, the competition will

eventually be whittled away until, as the industry matures, only the most successful operators remain. Consolidation is also likely as smaller sites are bought up by major firms, several of which, after very successful IPOs, now have plenty of acquisition currency.

What, therefore, is in store for traditional land-based gambling operators, given the unfettered explosion of Internet gambling in the United States—despite legislation aimed at preventing American gamers from utilizing online services? Like many things in life, it all depends on whom you ask, and even then, the answer you get today is likely to change over the course of the next few years. One study that analyzed the relationship between the increasing prevalence of Internet gambling and states' casino revenues came to the conclusion that "Internet gambling has not had a statistically significant negative impact on the gambling revenues of . . . Nevada and New Jersey."[17]

Yet by the close of this first decade of the twenty-first century, the advent of Internet gambling will have had a significant impact on traditional land-based revenues, negatively *or positively*. One scenario foresees Internet gambling reducing revenues through a substitution effect. The UK Treasury currently holds a different view, stating that the "assumption of a substitution effect between traditional based gambling offerings and remote gambling is false. Remote gambling is a unique customer experience."[18] Although the experience is indeed different, this does nothing to imply that casual gamers will not choose one form of entertainment over the other. Internet gambling cannot replicate the experience of a weekend in Vegas, but online gambling sites offer betting services from the home. For consumers looking only to place a bet—with no interest in luxurious hotels, restaurants commanded by world-renowned chefs, or bustling nightlife—the online gambling experience may be preferable to what traditional casinos have to offer. In order to succeed in a post-Internet environment, casinos and other traditionally based gambling operations will have to evolve from being merchants of gambling services to merchants of *entertainment* services. This process has already begun, with certain casinos on the Las Vegas Strip earning less than half their revenues from gambling activities. If online gambling does negatively impact traditional operators, those operators will have to adapt their product offerings in order to replace lost revenues.

A second possible outcome scenario would find Internet gambling actually benefiting traditionally based games operators. Internet gambling—online poker, specifically—has introduced a record number of Americans to casual gambling. The stigma attached to gambling by moral authorities is diminished every time a consumer visits an online gambling site. The trend was started when Las Vegas shed its image as a seedy gambling hotspot and began to be perceived as a more family-friendly destination. What the Internet has done is

accelerate a shift in public opinion about gambling as a form of leisure, from a vice to an acceptable, and oftentimes enjoyable, pastime. Should this scenario hold true, casinos would see increases in their gambling revenues *as well as* in their other operating revenues. According to this "rising tide lifts all boats" view, casinos would see not only higher room occupancy rates *but also a greater percentage of guests actually sitting down to play at gambling tables*. Not only is there the opportunity to draw more people to casinos and other traditional gambling sites, but the opportunity also exists to encourage them to gamble more. Whereas Americans spend an average of 1 percent of after-tax earnings on gambling, Australians, for example, spend approximately 3.5 percent on gambling.[19] Clearly, the market for American gambling services is far from saturated.

For horseracing in particular, with declining purses caused by years of declining race attendance, Internet gambling may be the last hope for survival. Simulcast races and off-track betting offer two ways to stimulate the sluggish industry by increasing purse sizes. Not everyone is embracing Internet gambling, though. Betting exchanges, which operate by matching a gambler who sets odds with another willing to take the bet (the operator of a betting exchange site takes no risk), can offer better prices than traditional bookmakers. Traditional bookmakers must manage their risk exposure and are also subject to higher taxes. Therefore, traditional bookmakers are unambiguously harmed by the existence of betting exchanges, because betting exchanges reduce the margins bookmakers can hope to earn.

To say that the Internet has revolutionized the gambling industry is far from an overstatement. If anything, the word "revolution" has been so overused that it fails to capture the tremendous impact Internet gambling will have on the industry for years to come.

NATIVE AMERICAN GAMBLING

Although online gambling has taken the globe by storm, it is not the only segment of the gambling industry to experience double-digit growth in recent years. Within the United States, gambling ventures operated under the Indian Gambling Regulatory Act have proven highly lucrative for Native American tribes as well as for investors lucky enough to have gotten in on the action. Collectively, the tribes now have the largest gambling industry segment in the United States, having surpassed the gross gambling revenues of the state of Nevada in 2001.[20] The climate of tribal gambling in the United States has become increasingly political, a fact that will shape the future of tribal gambling to come.

The most heated political debates revolve around tribal recognition and off-reservation casinos. Formal recognition of a tribe's legitimacy can mean the difference between financial success and poverty, and the political maneuverings employed by tribes seeking recognition, the investors backing them, casino operators, and other tribes who fear competition demonstrate the varied interests at play in tribal gambling. In 2005, the U.S. Senate Committee on Indian Affairs met to discuss the recognition process for Indian tribes. As one commentator noted, "Connecticut's governor, both its senators, and three of its congressmen showed up to testify on a matter none would have cared much about a decade or so ago."[21] Connecticut already has two well-established tribal casinos, and those in office are not interested in a third.

Senator John McCain, who then chaired the Senate committee, noted that wealthy investors have a vested interest in helping tribes win recognition because the tribes would, in turn, provide the investors with profitable investment opportunities.[22] Indeed, lobbying expenditures by tribes—whose poverty-stricken members are purportedly among the poorest in America—can total in the millions of dollars. As a matter of illustration, the Schaghticoke tribal nation spent approximately $12 million in its efforts for recognition, financed in part by the founder of the Subway restaurant chain, and the Eastern Pequots of Connecticut were supported financially by none other than Donald Trump.[23] Although it would be nice to consider these acts as particularly altruistic, partnering with Native American casino operators can prove highly profitable. Witness a recent partnership between the Creek Indians and Harrah's Entertainment Inc. in trying to expand gambling in Rhode Island.[24]

Off-reservation casinos are also a hot-button issue within the scope of tribal gambling. Whereas landless tribes maintain that restricting off-reservation gambling deprives them of opportunities to raise their socioeconomic status by opening a casino, opponents of off-reservation casinos, led by Senator McCain, argue that Congress (in passing the Indian Gaming Regulatory Act in 1988) never intended Native Americans to build off-reservation, Vegas-style casinos.[25] Opposition can even come from a seemingly unlikely source—other Indian tribes. Many tribes oppose off-reservation casinos strictly for fear of competition with their own tribal casinos.

Future prospects for Indian gambling are similar to those for traditional land-based casinos and other gambling sites. Although rapid expansion continues, the market segment is already showing signs of slowing growth rates as tribal casinos face increased competition from private casinos, state-run gambling operations, online gambling sites, and other tribal casinos themselves. As tribal casinos become established in their marketplace, fewer tribes will seek to enter a saturated market, especially if casinos are restricted to reservations not

frequented by the majority of the public. Though tribal gambling's fantastic rate of growth has been second only to that of Internet gambling, according to one economist, the "double-digit growth is over."[26]

THE EXPANSION OF GAMBLING BY STATES: SLOTS, SPORTS GAMBLING, AND PRIVATIZATION

The final segment of the gambling industry to be discussed is the state of gambling in traditional gambling operations within the United States, including casinos, lotteries, and slot machines. As mentioned at the opening of this chapter, 2005 was a banner year for brick-and-mortar casinos throughout the country. Despite the threat posed by Internet gambling and tribal gambling, casinos in Las Vegas and Atlantic City are positioned to continue their financial success of recent years into the near future. The gambling industry as a whole will benefit from a favorable demographic shift in the U.S. population. "The average gambling patron is 49 years old, placing the average U.S. gamer in an age category that is growing three times faster than the overall U.S. population."[27] It is also noteworthy that the Las Vegas Strip tends to be a supply-driven economy, in that increasing the number of rooms available in effect increases the demand for said rooms. With the addition in 2005 of the $2.7 billion Wynn Las Vegas, revenues from the hotel side of Vegas casinos could reach an all-time high.

Moving away from Las Vegas, casinos become less about profits and more about tax revenues. Permitting slot machines has become a favorite tactic of legislators seeking to raise revenue for state treasuries without raising taxes. Oftentimes, states vie for each other's residents' gambling dollars, building casinos just across their borders in hopes of luring revenues from out of state. As an example, Maryland is currently considering the legalization of slot machines in the state. James Browning, former executive director of Common Cause Maryland, which oversees campaign spending by the gambling industry, makes a deft comparison:

> If you look at the other states, Pennsylvania got slots. West Virginia is talking about table gambling. It's like an arms race between the states, and campaign contributions and lobbying expenditures are the weapons to win.[28]

Although the analogy may seem a bit overblown, it does bring to light an underlying theme of state-run gambling. The initial success of many of the first movers was a result of an inflow of out-of-state money into state-run casinos. Indiana, for example, has taken advantage of differences in legislation by allowing riverboat gambling near the border of two states that do not, those states

being Ohio and Kentucky.[29] In effect, Ohio and Kentucky residents are subsidizing lower taxes for residents in Indiana—a politician's dream come true.

In a similar vein, states such as New Jersey are considering legalizing sports gambling in order to protect their current flow of gambling revenue. In 1976, New Jersey became the first state besides Nevada to legalize casino gambling. Casino gambling was confined to Atlantic City in the hope that Atlantic City would recover some its cachet as a resort community. It has been a success in that Atlantic City is the second-largest casino gambling market in the United States, yet Atlantic City casino gambling has experienced slow growth from 2001 to 2006. With bordering states such as Delaware, Pennsylvania, and New York permitting slot machines at racetracks as well as other venues, Atlantic City's prospects appear bleak. Hence, legalized sports gambling is seen as the newest weapon in New Jersey's arsenal to protect its gambling revenues by reinvigorating Atlantic City as a casino destination. This is a highly controversial move on New Jersey's part. Many professional sports leagues, such as the National Football League and the National Basketball Association, as well as the National Collegiate Athletic Association, are adamantly opposed to legalizing sports betting.

Finally, the state of Illinois, among others, has proposed selling its lottery to private operators. Currently, the state lotteries are owned and operated as government agencies. This proposal does gives rise to two series of questions: (1) Why should government have a monopoly over lotteries? Isn't our entire economy built on the merits of competition? Wouldn't the bettor be better off with competitors offering a variety of games and odds? and (2) Why is gambling regulated by government at all? If so, what is the appropriate amount of regulation, and can government really regulate an industry from which it draws so much revenue?

Of course, all of the these questions have one common denominator: revenue! As the reader proceeds to examine all of the various facets of gaming, Tables 15.1, 15.2, and 15.3 should provide an idea as to why gambling has been become such an important topic for public policy officials.

Although the threads of competition and regulation run through any discussion regarding any segment of the gambling industry, it is a daunting task to make any statement summarizing the present climate or future outlook of "the" gambling industry, for the industry is manifested in many distinct forms. What *can* be said about the gambling industry in its entirety is that each part is connected to the whole more deeply than ever before. Consumers enjoy gambling, and firms are just scratching the surface in terms of developing innovative service offerings to cater to the gambling public. Yet although the public enjoys additional forms of gambling, opposition to the expansion of gambling remains quite strong.

Table 15.1
State Prohibitions on Gaming

State	Lottery	Casino/ Resort	Native Casino	Video Lottery Terminals	Betting Tracks
Alabama	No	No	Yes(3)	Yes	Yes
Alaska	Yes (non-profits)	No	No	No	No
Arizona	Yes	Yes	No	Yes	Yes
Arkansas	No	No	No	No	No
California	Yes	Yes	Yes	Yes	Yes
Colorado	Yes	Yes	Yes	Yes	Yes
Connecticut	Yes	No	Yes(2)	Yes	Yes
Delaware	Yes	No	No	Yes	Yes
Florida	Yes	Yes	Yes(4)	No	Yes
Georgia	Yes	No	No	No	No
Hawaii	No	No	No	No	No
Idaho	Yes	No	Yes(1)	Yes	No
Illinois	Yes	Yes	No	Yes	Yes(?)
Indiana	Yes	Yes	No	Yes	Yes
Iowa	Yes	Yes	Yes	Yes	Yes
Kansas	Yes	Yes	Yes(4)	Yes	Yes
Kentucky	Yes	No	No	No	Yes
Louisiana	Yes	Yes	Yes(3)	Yes	Yes
Maine	Yes	No	No	No	Yes
Maryland	Yes	No	No	No	Yes
Massachusetts	Yes	No	No	No	Yes
Michigan	Yes	Yes	Yes	Yes	Yes
Minnesota	Yes	No	Yes(18)	Yes	Yes
Mississippi	No	Yes	Yes	Yes	Yes(?)
Missouri	Yes	Yes	No	Yes	No
Montana	Yes	No	No	Yes	No
Nebraska	Yes	Yes	Yes(1)	Yes	No
Nevada	No	Yes	No	Yes	Yes
New Hampshire	Yes	No	No	No	Yes(4)
New Jersey	Yes	Yes	Yes	Yes	Yes

Table 15.1 (continued)

State	Lottery	Casino/ Resort	Native Casino	Video Lottery Terminals	Betting Tracks
New Mexico	Yes	Yes	Yes(7)	Yes	Yes(4)
New York	Yes	Yes	Yes(2)	Yes	Yes
North Carolina	No	Yes	Yes(1)	Yes	No
North Dakota	No	No	Yes(4)	Yes	No
Ohio	Yes	No	No	No	Yes(7)
Oklahoma	No	No	Yes(4)	No	Yes
Oregon	Yes	No	Yes(6)	Yes	No(?)
Pennsylvania	Yes	No	No	No	Yes(4)
Rhode Island	Yes	No	No	Yes	Yes
South Carolina	Yes	No	No	No	No
South Dakota	Yes	Yes	Yes(10)	Yes	No(?)
Tennessee	Yes	No	No	No	Yes
Texas	Yes	No	Yes(2)	No	Yes
Utah	No	No	No	No	No
Vermont	Yes	No	No	No	Yes
Virginia	No	No	No	No	No
Washington	Yes	Yes	Yes(20)	Yes	Yes
West Virginia	Yes	No	No	Yes	Yes(4)
Wisconsin	Yes	No	Yes(17)	Yes	No(?)
Wyoming	No	No	No	No	Home Only

Source: Retrieved from http://www.gamblingandthelaw.com, and http://www.naspl.org.

Table 15.2
Gambling Taxes (Millions $) Collected by States (2005)

State	Lottery	Casino/ Resort	Rac- inggo	Chari- table	Pari- Mutual	Total
Alabama	0	0	0	0	0.5	0.5
Alaska	0	0	0	0	0	0
Arizona	104.57	0	0	5.61	0.64	110.82
Arkansas	0	0	0	0	2.59	2.59
California	945.16	0	0	20.72	37.43	1,003.31
Colorado	93	95.6	0	4.68	0.52	193.80
Connecticut	270.37	0	0	1.36	4.52	275.93
Delaware	32.87	0	175.7	0.09	0.24	208.89
Florida	1,178.36	0	0	4.95	8.91	1,192.23
Georgia	768.16	0	0	1.1	0	769.25
Hawaii	0	0	0	0	0	0
Idaho	20.7	0	0	0	0.47	21.17
Illinois	578.08	719.9	0	9.21	12.01	1,319.20
Indiana	188.47	702.7	0	0	4.12	895.28
Iowa	48.85	141.3	68.4	1.18	0.19	259.93
Kansas	63.43	0	0	1.33	1.65	66.41
Kentucky	187.66	0	0	14.12	5.6	207.58
Louisiana	119.25	517.66	76.7	4.57	4.83	723.01
Maine	44.02	0	0	0	1.74	45.77
Maryland	464.59	0	0	5.75	1.89	472.23
Massachusetts	971.78	0	0	4.15	0.98	976.90
Michigan	609.75	250.2	0	12.59	11.87	884.41
Minnesota	79.17	0	0	25.26	0.17	104.60
Mississippi	0	325	0	2.7	0	327.70
Missouri	214.74	377.2	0	2.89	0	594.82
Montana	7.01	46.13	0	0.09	0.1	53.33
Nebraska	62.13	0	0	2.32	0.76	65.21
Nevada	0	776.5	0	0.34	5.01	781.85
New Hampshire	71.28	0	0	3.52	2.14	76.94
New Jersey	824.20	414.5	0	4.81	0	1,243.50
New Mexico	32.86	0	37.7	0.72	0.64	71.92

Table 15.2 (continued)

State	Lottery	Casino/ Resort	Rac- inggo	Chari- table	Pari- Mutual	Total
New York	2,144.51	0	n/a		27.85	2,172.35
North Carolina	0	0	0	0.89	0	0.89
North Dakota	0	0	0	5.39	4.01	9.40
Ohio	580.66	0	0	22.49	10.08	613.23
Oklahoma	0	0	0	2.15	3.33	5.48
Oregon	68.97	0	256.66	0	0.76	326.40
Pennsylvania	519.19	0	0	6.14	19.71	545.04
Rhode Island	69.75	0	188.8	0.41	3.26	262.22
South Carolina	0	0	0	3.87	0	3.87
South Dakota	6.25	11.6	138.93	0.64	0.18	157.61
Tennessee	0	0	0	0	0	0.00
Texas	1,004.74	0	0	16.85	3.38	1,024.96
Utah	0	0	0	0	0	0.00
Vermont	20.52	0	0	0.28	0	28.80
Virginia	377.58	0	0	0	2.29	379.88
Washington	63.82	93.42	0	6.1	1.84	165.18
West Virginia	54.35	0	324.6	0	1.02	379.97
Wisconsin	120.21	0	0	2.93	0	123.15
Wyoming	0	0	0	0.59	0.18	0.76
Totals	$13,088.70	$4,471.80	$1,267.50	$202.80	$187.10	19,217.70

Source: Christiansen Capital Advisors, *Insight,* August 2005, retrieved from http://www.cca-1.com; http://www.naspl.org.

Table 15.3
Gambling's Contributions to State Finances

State	Gambling Revenue	Total Revenue	Percentage
Alabama	$0.50	$5,585	0.01
Alaska	0	2,471	0
Arizona	110.8	6,031	1.84
Arkansas	2.6	3,251	0.08
California	1003.3	79,412	1.26
Colorado	193.8	6,137	3.16
Connecticut	275.9	12,016	2.3
Delaware	208.9	2,918	7.16
Florida	1192.2	21,197	5.62
Georgia	769.3	16,383	4.7
Hawaii	0	3,923	0
Idaho	21.2	1,941	1.09
Illinois	1319.2	25,161	5.24
Indiana	895.3	10,446	8.57
Iowa	259.9	4,484	5.8
Kansas	66.4	4,260	1.56
Kentucky	207.6	7,444	2.79
Louisiana	723	6,662	10.85
Maine	45.8	2,564	1.78
Maryland	472.2	10,469	4.51
Massachusetts	976.9	23,363	4.18
Michigan	884.4	8,895	9.94
Minnesota	104.6	14,180	0.74
Mississippi	327.7	3,494	9.38
Missouri	594.8	7,669	7.76
Montana	53.3	1,322	4.03
Nebraska	65.2	2,622	2.49
Nevada	781.9	2,139	36.55
New Hampshire	76.9	1,336	5.76
New Jersey	1243.5	23,223	5.35
New Mexico	71.9	4,339	1.66

Table 15.3 (continued)

State	Gambling Revenue	Total Revenue	Percentage
New York	2172.4	40,328	5.39
North Carolina	0.9	14,271	0.01
North Dakota	9.4	870	1.08
Ohio	613.2	22,558	2.72
Oklahoma	5.5	4,687	0.12
Oregon	326.4	3,969	8.22
Pennsylvania	545	20,679	2.64
Rhode Island	262.2	2,735	9.59
South Carolina	3.9	5,040	0.08
South Dakota	157.6	891	17.69
Tennessee	0	8,126	0
Texas	1025	31,064	3.3
Utah	0	3,560	0
Vermont	20.8	884	2.35
Virginia	379.9	12,204	3.11
Washington	165.2	11,666	1.42
West Virginia	380	3,139	12.1
Wisconsin	123.1	10,772	1.14
Wyoming	0.8	768	0.1
Totals	19,217.70	523,548	3.67

Source: Statistical Abstract of the United States for 2004–05, State Regulatory Agencies.

This chapter has focused on the current state of the various segments of the gambling industry, on why states compete for expanded gambling revenues, and on the forces expected to shape how that competition will take place. There are various ethical concerns that public policy officials need to take into account before they develop a coherent gaming strategy for their states. How states formulate and implement these strategies will, of course, determine whether they are successful in achieving the goal of a delicate balance between the revenue needs of the state and the social costs that invariably accompany the expansion of gambling activity.

NOTES

1. From *The Gambling Debate,* Richard A. McGowan; Chapter 1, pp. 3–18, "The Current Climate of Gambling in the United States." Copyright 2007 by Richard A. McGowan. Reproduced with permission of Greenwood Publishing Group, Inc. Westport, CT.
2. Goldman, Adam, "Nevada Casinos Win $11 Billion from Gamblers in Fiscal 2005," *AP Newswire,* August 12, 2005.
3. Christiansen, Eugene Martin, "The 2004 Gross Annual Wager of the United States," *Insight* 3 (2005): 1.
4. Ibid.
5. Christiansen, Eugene Martin, "The 2004 Gross Annual Wager of the United States," *Insight* 3 (2005): 14.
6. Sinclair, Sebastian, "Pocket Aces: Poker and Other Table Games," *Insight* (2003).
7. Christiansen, Eugene Martin, "Free Trade vs. Gambling Monopolies," *Insight* 11 (2005): 2.
8. World Online Gambling Law Report, *www.e-comlaw.com/woglr* (2005). Subscription required.
9. Ng, Serena, "For Audit Firms, All Bets Are Off," *Wall Street Journal Online,* July 21, 2005.
10. Christiansen, Eugene Martin, "The 2004 Gross Annual Wager of the United States," *Insight* 3 (2005): 6.
11. Cabot, Anthony, "The Internet and Horseracing's Future," *Insight* (2003).
12. Champion, Simon, and Collyer Tarasewicz, "Online Gambling Industry: Surreal or Real Returns," Deutsche Bank AG London, 2005.
13. Christiansen, Eugene Martin, "Out of the Past," *Insight* 2 (2005): 12.
14. Christiansen, Eugene Martin, "The 2004 Gross Annual Wager of the United States," *Insight* 3 (2005): 12.
15. Champion, Simon, and Collyer Tarasewicz, "Online Gambling Industry: Surreal or Real Returns," Deutsche Bank AG London, 2005.
16. Ibid.
17. McGowan, Richard, "Internet Gambling and Its Effect on Traditional Gambling Revenues." *Insight* (2003).
18. World Online Gambling Law Report (2005).
19. Gregg, Deirdre, "Gambling Limits Loom," *www.msnbc.com,* June 19, 2005.
20. Werner, Erica, "McCain Takes Aim at Indian Gambling," *Ithaca Journal,* June 29, 2005.
21. Lucas, Fred, "State Slams Tribal Agency," *The New Times*. May 12, 2005.
22. Werner, 2005.
23. Nitkin, David, "Gambling Industry Puts Their Money on State Lobbyists," *The Baltimore Sun,* June 1, 2005.
24. Wilemon, Tom, "Choctaw's Leader Welcomes Competition," *The Sun Herald,* May 5, 2005.

25. Eaton, Adam "Shawnee Chief, Voinovich Arguments on Ohio Casinos," *The Plain Dealer,* July 28, 2005.
26. Gregg, "Gambling Limits Loom."
27. Ibid.
28. Nitkin, "Gambling Industry Puts Their Money on State Lobbyists."
29. Pawlina, Jason, and Eugene Martin Christiansen, "Balance of Trade: Indiana Funds Its Budget in Ohio and Kentucky," *Insight* (2003).

This page intentionally left blank

Chapter **16**

Online Gaming Addiction: Symptoms, Risk Factors, and Treatment

Kimberly S. Young, PhD

My son is a sophomore in high school and he appears to be addicted to online video games. I think his ideal life would be sitting in front of a computer monitor with an IV in his arm to deliver enough nutrients and caffeine that he wouldn't have to eat or sleep. He also has started to lie to his father and me—and to his teachers—about his schoolwork to maximize his access to the video games, particularly those on the web. He has some friends, but they are limited to other "gamers" and he went from being a straight "A" student to failing out of school. In one sense, I'm glad he isn't out on the streets getting into drugs or other forms of trouble. But I fear his life has become so one-dimensional that he will be damaged as a result of this obsession.

—Linda, a concerned mother in California

Adolescents are encouraged to utilize the Internet to enhance school performance and competitiveness; however, heavy use has several negative consequences. Internet addiction is one of the most serious problems (Young, 1998a; Griffiths et al., 2003; Kelly, 2004). Adolescents who are addicted to the Internet also usually suffer from problems in their daily routine, school performance, family relationships, and mood (Leung, 2004; Ng & Wiemer-Hastings, 2005). It is important, therefore, for mental health professionals to develop effective intervention strategies for preventing Internet addiction among adolescents. It is also important to examine the warning signs, risk factors, and treatment strategies associated with gaming addiction, to aid in detection and prevention.

WHAT IS ONLINE GAMING ADDICTION?

Online gaming addiction is an addiction to online video games, role-playing games, or any interactive gaming environment available through the Internet (Young, 1998b). Online games such as EverQuest, the Dark Age of Camelot, or Diablo II—dubbed "heroinware" by some players—can pose much more complex problems. Extensive chat features give such games a social aspect missing from offline activities, and the collaborative/competitive nature of working with or against other players can make these games a hard habit to break (Ducheneaut & Moore, 2004).

"I really want my life back," explained one gaming addict. "Three years ago I was one of the most popular kids at school. I got invited to all the parties, got lots of girls, had too many friends. Then I discovered an online game called Counter-Strike. It's very hard for me to stop. I wake up in the morning, no shower, get on the computer, stay on till the wee hours of the mornings, go to sleep, repeat. I don't know how to get off, I've tried. . . . It's just too hard. I heard this is a very common problem but I really want to get my life back and I'd give anything."

In the early days of the Internet, interactive online games were a takeoff on the old Dungeons and Dragons games, often known as Multi-User Dungeons, or MUDs, that drew upon power, dominance, and recognition within a role-playing, make-believe virtual world. Young men traditionally gravitated toward these role-playing games to assume a character role associated with specific skills, attributes, and rankings that fellow players would acknowledge and treat accordingly (Turkle, 1998). MUDs differed from traditional video arcade games in that instead of a player's hand-eye coordination improving, the actual strength, skills, and rankings of the character improved. MUD players earned respect and recognition from fellow players (Turkle, 1998), and younger men, especially those with low self-esteem and weak interpersonal skills, were at greatest risk of becoming addicted if they developed a powerful persona within the game (Young, 1998b).

Interactive gaming has taken on new themes beyond Dungeons and Dragons that are easier to learn and appealing to those who are more mainstream. In several documented cases, interactive gaming has led to divorce, job loss, and health problems among those suffering from an addiction. While online gaming addiction is not as prevalent as addictions to cyberporn or online chatting, online gaming has grown substantially over the last few decades, reaching millions of users. Gaming also encompasses traditional board games such as YAHZEE or Bingo, which have taken on an interactive and social nature when

played online, and virtual casinos have also grown rapidly, especially among teenagers and college-aged populations who now easily access black jack, roulette, or poker tables online.

Globally, recent reports have indicated that interactive online gaming has reached addictive proportions in China, Korea, and Taiwan (Lee, 2007). About 10 percent of China's more than 30 million Internet gamers are said to be addicted. To battle what has been called an epidemic in some reports, Chinese authorities regularly shut down Internet cafes, many illegally operated, in crackdowns that also include huge fines for their operators. The Chinese government has also instituted laws to reduce the number of hours adolescents can play online games (BBC News, 2007) and in 2005 opened the first treatment center for Internet addiction in Beijing. Online gaming addiction continues to raise such serious concerns that the first detox center for video game addiction has opened in Amsterdam (CBSNews.com, 2006), and most recently the American Medical Association, at its annual policy meeting, has considered calling video game overuse an addiction (Tanner, 2007).

SIGNS OF ONLINE GAMING ADDICTION

As online gaming becomes more popular, more parents discover how addictive it can be for their children. "My son has completely withdrawn from his family and from all reality," explains one mother. "At first, his father and I were happy that he seemed interested in the Internet. He had no other hobbies and he seemed to make friends online, but soon, the game took over his life. He didn't shower, didn't eat, and didn't leave his room. My husband and I became worried and tried to set time limits, but he just got mad. I mean angry and hateful towards us. It was a side of our son we had never seen. All he wanted was the game. We are so scared and don't know where to turn. Counselors we have talked to just tell us it is a phase and to ignore it, but we can't. His whole life is the game. This is more than a phase—it is an obsession. We are desperate to find him help."

Parents often feel alone and scared as their children become hooked on something that no one seems to understand. Parents search for information and help of any kind as they helplessly watch their children become absorbed in the computer and begin to see the warning signs of a dangerous pattern. Gamers who become hooked show clear signs of addiction. Serious gamers, who play for extended periods of time (over four hours at a time), get restless or irritable if they can't play, and sacrifice other social activities just to game, are

showing signs of addiction (Griffiths, Davies, & Chappell, 2003). Other common warning signs include the following:

- A preoccupation with gaming
- Lying or hiding gaming use
- Disobeying time limits
- Loss of interest in other activities
- Social withdrawal from family and friends
- Psychological withdrawal from the game when forced away from it
- Using gaming as an escape
- Continuing to game despite its consequences

Preoccupation with Gaming

The addiction process begins with a preoccupation with gaming. Gamers will think about the game when offline and often fantasize about playing the game when they should be concentrating on other things. Instead of thinking about the paper that needs to be completed for school, or going to class, or studying at the library, the gamer becomes completely focused on playing the game. Gamers start to miss deadlines and neglect work or social activities, as being online and playing the game becomes their main priority.

Lying or Hiding Gaming Use

Some gamers spend days and nights online. They don't eat, sleep, or take showers because of the game. They lie to family and friends about what they are really doing on the computer. Students tell their parents that they are doing their homework, spouses tell their family that they are using the computer for work, and friends will make up excuses for why they can't go out—all to find more time to play the game.

Loss of Interest in Other Activities

As the addiction grows, gamers become less interested in hobbies or activities that they used to enjoy and become more fascinated by the game. One mother told me about her son who loved baseball and played Varsity on his high school team until he discovered XBox Live. "His grades plummeted after he discovered the game, but it wasn't until he quit the baseball team that I knew that something was seriously wrong. He loved baseball too much. He even won

a baseball scholarship for college and dreamed about playing professionally. Now, nothing else matters to him except the game."

Social Withdrawal

Some gamers experience personality changes the more addicted they become. A once outgoing and social daughter withdraws from friends and family only to spend more time alone in front of the computer. A normally happy son becomes withdrawn, preferring to make friends in the game as the people that were once important in real life become less important. As one mother explained, "If no one else existed, he would play all day." If children do have real-life friends, they are usually fellow gamers. In some cases, gamers are introverts who have problems making social connections in real life and turn to the game for companionship and acceptance.

Disobeying Time Limits

Because of their addiction, gamers become defensive about their need to play the game and angry when forced to go without it. Parents who try to put time limits on the game describe how their sons and daughters become angry, irrational, and even violent. In one case, a mother told me about her son who spent his nights gaming and his days sleeping. "When I took away his computer, he pushed me, slammed the door to his room, and wouldn't come out all night. When I came home from work the next day, he took a sledgehammer to my computer, which was off limits to him. This isn't my son. He was a good kid and never gave me a moment's trouble until I lost him to the game."

Psychological Withdrawal

Gamers who can't access the game experience a loss. They want to be on the game and they miss playing the game. This feeling can become so intense that they become irritable, anxious, or depressed when they are forced to go without the game. They can't concentrate on anything other than going back online to play. Their minds become so fixated on the game that they can experience a psychological withdrawal from the game such as depression and irritability. Their feelings intensify and they stop thinking rationally and begin to act out toward other people in their lives. All that they can think about is getting back to the game, and they become vent their anger and bitterness against anyone who threatens to take it away.

Using Gaming as an Escape

Gaming addicts use the online world as a psychological escape. The game becomes a safe means to cope with life's problems. It is a legal and inexpensive way to soothe troubling feelings and can quickly become a convenient way to instantly forget stresses and pains. Like drug addicts or alcoholics who use drugs or alcohol as a way to escape problems that they aren't able to deal with, gaming addicts use the game to avoid stressful situations and unpleasant feelings. They escape into the gratification of the game and the feelings they associate with playing it. Gamers who feel socially awkward, isolated, and insecure in real life can transform themselves into people who feel socially confident, connected, and self-assured in interacting with others through the game. As gamers progress further into the game, they make friends (or maybe their friends were the ones who first introduced them to the game) and these social relationships with other players become highly significant. While playing, gamers feel more accomplished, more accepted, and better about themselves. Through their characters, gamers live out a fictional life that is more satisfying and interesting than their own.

Continuing to Game despite Its Consequences

Gamers often want to be the best at the game. In order to grow in the game, they need to play. Especially in quest-type games that include a shared activity, they hunt for items together, and can take several hours to complete one quest. Gamers who become hooked become obsessed with the need to be the best at the game. They want to feel powerful and to be recognized by other players; in order to do this they must spend time in the game. They continue to use the game despite its effects on their lives. Adolescents may fail out of school, lose a scholarship, break up with a girlfriend or boyfriend, and ignore basic hygiene, just to be online. Adults may lose a job or a relationship, or their marriage may be on the brink of divorce, but still they remain loyal to the game.

PSYCHOSOCIAL RISK FACTORS

People of all ages are quickly becoming immersed in the virtual fantasy world in which they can easily escape problems in their real lives (Kolo & Baur, 2004). "For me, gaming was a way of coping with my divorce," said Susan, a regular player of the game EverQuest. "A guy I talk to has been through three girlfriends and even more jobs because of the game." Like other players, Susan struggles to find a workable balance between gaming and the responsibilities

in her life. She spends nearly eight hours a day online and often questions her devotion to the game. "I think of quitting all the time," she said. "I'm neglecting my kids and my husband but the game is so powerful I feel helpless to stop." Recent research has explored several potential risk factors associated with addictive online gaming behavior.

Low Self-Esteem

Individuals who suffer from low self-esteem or other emotional problems are at greater risk for developing an addiction to online gaming. Recent studies suggest that hardcore players may have a tendency toward neuroticism or may suffer from emotional problems or low self-worth and esteem (Yee, 2007). The studies suggest that individuals who have other emotional problems may be more at risk of developing an addiction to interactive gaming. In the game, the interactive environments allow individuals to experiment with parts of their personality: they can be more vocal, try out leadership roles, and try out new identities. The problem comes when players rely upon these new online personae and the distinction between reality and a fantasy role-play game becomes blurred. Kevin was a 21-year-old from Rochester, NY, who was dismissed from college because of his gaming habit. When I asked him about the game, he said that his life seemed to be important when he was playing the game. He was important in the game, but in real life he was a person who couldn't make friends and wasn't meeting his parent's expectations. He had failed in school, not so much because he couldn't pass the tests but because he couldn't make it to class. He didn't have a direction in his life, and because of this, he didn't feel good about his life. But in the game, all that changed. He was good at the game, had a network of fellow gamers who, he felt, were his closest (and only) friends, and he felt validated and confident when playing the game.

Poor Social Relationships

A large part of gaming is about making social relationships. Gamers often make friends with other gamers (Kolo & Baur, 2004). Ultimately, online gaming is a social activity. Most online games include copious amounts of chatting, allowing players to interact with each other in the guise of the characters they represent. The social aspect is a primary factor in many game addictions (Leung, 2007). Many adolescents have trouble with social relationships and feel lonely, as if they have never truly belonged. Adolescents can develop a sense of belonging in the game. In some cases, the game provides the only friends they interact with. Gamers can become hooked on the social aspect of the game.

They may join guilds that provide a strong sense of community and accomplishment when they take out monsters or strategize about their next online session. Through quests or nightly turns playing the game, gamers can form close bonds and friendships with fellow players that provide the social contact that has been missing from their lives.

Highly Intelligent and Imaginative Individuals

Gaming provides players with an outlet for their imaginations (Turkle, 1998). Adolescents who are academically bright and who feel understimulated in school turn to the game as a place for adventure and intellectual stimulation (Kelly, 2004). Games also lure players with complex systems of goals and achievements. The players are drawn into the virtual fantasy world of the game; they internalize the game as a real place, and other characters are seen as real people, not fictional characters. Especially in goal-oriented games such as EverQuest**,** players engage in activities designed to develop their characters and compete to find valuable in-game elements such as armor and weapons. Players can find themselves wrapped up in the game for hours as they struggle to gain one more skill or weapon.

Need for Recognition and Power

Turkle (1998) describes how gamers often achieve recognition and power through online gaming. "I'd say the most addictive part for me was definitely the gain of power and status," explained Mark, a gamer hooked on Diablo II. "The way you progressively gain power you become more of an object of awe to the other players. . . . each new skill isn't enough." Among most multi-user gamers, each goal leads to another goal, and gamers make critical choices along the way. They invest significant time and thought in developing a character. They feel they have wasted their time unless they reach the next goal. For example, Mark, a college sophomore, explained, "By day I am a mild-mannered student, but at night, I become the most aggressive warrior online." Mark had always been a loner. He described how, growing up as a middle child, he felt ignored by his parents, who gloated over his older sister, a medical doctor, and his younger sister, a freshman at Brandeis, while he attended a state university. He had built up a great deal of resentment toward his siblings, and deep down he resented his parents for their neglect. "On the outside, I looked like the perfect child and no one knew of the anger and resentment that I felt inside," Mark explained. "I was afraid to give into my anger, yet within the game, I confronted my fears and liked dominating other players. I became known as the most powerful and the

most respected player in the game, and I needed that. The game was the only place in my life where I felt important."

The Younger They Start

Some research has suggested that the younger children start to use the Internet, the more they are at risk of developing an addiction to online gaming (Yee, 2007). Dan started gaming by age 12. He was drawn to Gameboy, Sony Play Station, and Nintendo with his friends, and gradually progressed to XBox. He was able to manage the time he spent gaming until he went on XBox Live. "It was like a whole other world opened up to me," he explained. Suddenly, he was able to interact with fellow players instead of sitting beside friends while playing the game. Gaming had already become a large part of his personal identity, and despite suffering from attention deficit disorder (ADD) he was able to sit in front of the computer for hours. His parents became concerned when his gaming habit turned into an obsession. "He went into a trance-like state every time he went online but unlike other hobbies, he never lost interest in this," his mother explained. "When he quit the track team, which he loved, we knew he had a serious problem and the game took over his life." Interestingly, children with ADD have been prone to gaming addiction and are more likely to form an addiction to gaming due to the stimulation that the interaction with other online players and the challenge of the game can provide.

Family History of Addiction

Recent studies in Taiwan suggest that family factors also increase risk for developing online gaming addiction (Yen, Yen, Chen, Chen, and Ko, 2007). Adolescents from households of lower economic levels, whose parents are separated or divorced, whose families have high parent-adolescent conflict, or whose families have a history of addiction are at greater risk of developing gaming addiction. Many addictions stem from a history of addiction in the family as a way of coping with painful feelings and difficult situations. Seeing that an aunt, uncle, or other relative copes with problems through drinking, drug use, gaming, or smoking might indicate to an adolescent that this is the way to cope with all problems. Gamers who become addicted, especially adolescents, often use the game as a way of escaping conflict or turbulence in their lives. Adolescents experiencing a traumatic transition such as divorce, or family relocation, or the acquisition of a new step-parent face a personal crisis and learn to cope through the Internet. Adolescents addicted to gaming explain that they feel alone, feel emotionally removed from others, or feel that their parents are somehow

disappointed with them (Young, 1998b). Like those addicted to drugs or alcohol, they use the game to escape these painful feelings and momentarily feel a sense of acceptance and accomplishment in their lives.

TREATING GAMING ADDICTS

As in any treatment program, the primary step to take in the path to recovery is to accept and not refute "denial," a defense mechanism that addicts frequently employ and that effectively stops them from accepting treatment. Once this obstacle is overcome, treatment can be more effective. It is important to understand that compulsive online gaming is treatable. It affects the gamers, their families, their schoolwork or employer, and their community. For players who do admit they have a problem, the most common response is a guilt-and-purge cycle, which is common to many addictions (Kelly, 2004). Many players who realize that they are addicted will kill their characters and delete the game software with no regrets; however, many other game addicts aren't as successful in doing this. For most players, true recovery involves looking at the issues underlying the game habit. Addicted players need to examine the emotional motives that prompt them to play a game excessively and look for alternate ways to satisfy those needs.

Knowing the Clinical Signs

It is important to be able to recognize the symptoms of online gaming addiction and possible warning signs. The sooner one seeks help for an adolescent experiencing online gaming addiction, the more beneficial it will be. Consistent patterns of addiction in adolescence are in most cases a sign that there are prevalent issues in the adolescent's immediate environment that need to be addressed. Some warning signs that an adolescent may be abusing games include change of friends, change in physical health, behavioral problems, academic problems, change in attitude, indifference, increased irritability and hostility, frequent changes in mood, eating, and sleeping patterns, and depression and isolation. Young (2007) developed an eight-item screening test based upon the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) to help clinicians identify the most common signs of compulsive behavior related to online games:

1. Do you need to play for increasing amounts of time?
2. Are you preoccupied with gaming (thinking about it when offline, anticipating your next online session)?

3. Have you lied to friends and family members to conceal the extent of your gaming?
4. Do you feel restless or irritable when attempting to cut down or stop online gaming?
5. Have you made repeated unsuccessful efforts to control, cut back, or stop online gaming?
6. Do you use gaming as a way of escaping from problems or relieving feelings of helplessness, guilt, anxiety, or depression?
7. Have you jeopardized or lost a significant relationship because of your online gaming habit?
8. Have you jeopardized a job, educational, or career opportunity because of your online gaming habit?
9. Do you suffer from eye strain, back strain, or lack of sleep because of the amount of time you spend at the computer?
10. Do you suffer from carpal tunnel syndrome or other repetitive stress injuries because of the amount of time you spend at the computer?

Answering "yes" to give or more of questions without the response being better accounted for by a manic episode suggests that a client may suffer from online gaming addiction. These signs suggest that a client has lost control, lied, risked a relationship or job, or possibly suffered from physical problems because of online gaming.

Symptom Management

Understanding how and when a gamer uses the computer is an initial step in the recovery process. Keep a daily log to track how the gamer actually uses the Internet, then take a few minutes to consider his or her current online habits. On what days of the week does he typically log online? At what time of day does she usually begin? How long does he stay online during a typical session? Where does he usually use the computer? Does a pattern emerge? Now, using the daily log, construct a new schedule, or what Young (1998b, 56) refers to as "practicing the opposite." The goal of this exercise is to disrupt the client's normal routine and construct new patterns of time use in an effort to break the online habit. Let's say the client's Internet habit involves playing the game from 5 a.m. until after midnight. Instead of going online, the client should take a shower or start breakfast first instead of logging on. Or, perhaps, a client uses the Internet only in the evening, and has an established pattern of coming home and sitting in front of the computer for the remainder of the evening. Instead, he should wait until he has had dinner and watched the news before logging on. Practicing the opposite will disrupt clients' normal Internet patterns and increase their ability to effectively manage their online time.

Online gaming is an emotionally draining and time-consuming activity, and to create more time for the computer, addicts neglect sleep, diet, exercise, hobbies, and socializing. The initial loss of online gaming means an increase in idle time or boredom, which only increases the temptation to surf, making it vital for clients to create positive lifestyle changes to fill the void created by time no longer spent at the computer.

Symptom management also involves creating positive lifestyle changes that take clients away from the computer and improve their emotional and physical well-being (Young, 2007). This varies, depending upon the client's specific situation. Some strategies may involve finding spiritual fellowship in the form of personal prayer or pastoral counseling as part of spiritual wellness and daily recovery (Young & Klausing, 2007). Clients may practice mediation to focus their energy during recovery and use prayer and scripture to improve their ability to fight the temptation to return to the computer. Other activities include getting the proper rest, going to bed at a reasonable hour, joining a gym, and improving diet to manage overall physical health.

As in food addiction, symptom management is an essential part of recovery. In food addiction, certain foods trigger binge behavior. Chocolate or potato chips will trigger binges but celery sticks will not, so avoidance of the trigger foods is a necessary part of recovery. This means that recovery from binge eating is about relearning how to eat in order to make more informed and healthier food selections, with success being measured through objective goals such as changes in caloric intake and weight loss. The same logic is applied to addictive online gaming. In part, recovery involves relearning how to use the computer, making better choices about its use. Reducing the number of hours a gaming addict spends online is an important first step, but to move toward full recovery, the addict must also address the underlying issues that led to gaming (Young, 1998b).

Addressing Underlying Issues

The ability to mentally absorb oneself in a virtual environment that seems more exciting and more interesting than one's real life reinforces the addictive behavior and can be used as a coping mechanism to deal with missing or unfulfilled needs. That is, gaming momentarily allows the gamer to forget his or her problems. In the short term, gaming may be a useful way to cope with the stress of a hard situation; however, addictive behaviors used to enable the individual to escape or run away from unpleasant situations in the long run only make the problem worse. For the gaming addict, situations such as a death of a loved one, a divorce, or a job loss may trigger using the game as a mental distraction that

temporarily makes such problems fade into the background. Since the escape is only temporary, addicts return to gaming as a means of making themselves feel better without dealing with and resolving the underlying feelings of depression or anxiety in their lives.

The game produces a "high" that provides an emotional escape, altered state of reality, or mental rush (Ng & Wiemer-Hastings, 2005). That is, online gaming, through the excitement of becoming someone new in a role-playing game, the challenge of winning the quest, and the pleasure of making new friends through the game, provides an immediate mental escape from problems and serves to reinforce future behavior.

Therefore, treatment requires addressing those needs that the game fulfills. For James, a 21-year-old college freshman addicted to XBox Live, the hardest part of getting better was finding something else in life that mattered as much as the game. "At 21, I was going nowhere fast," James explained. "I didn't like school, I didn't have any friends except those I knew in the game, but yet I saw others around me moving on with their lives. The friends that I had during freshman year were going to graduate while I got myself kicked out of school because I couldn't stop gaming. My older brother was graduating from graduate school, had a girlfriend and was getting married in the fall, while I did nothing and felt completely stuck in the game. I tried quitting the game so many times, but I felt there was nothing important enough in my life to really quit it for."

James repeatedly relapsed into the game because he still wasn't dealing with his feelings about his career goals. Each time he thought of registering for classes, taking a course, or studying for a test, he felt the pressure and internal judgment of feeling like a failure. He constantly compared himself to his friends and his brother, and relapsed into the game as a way to relieve his underlying feelings of depression and self-doubt.

As part of his recovery, James started taking night classes at a community college, taking one or two classes to see what type of work or vocation he wanted to pursue. He liked finance, a far cry from his major in engineering at the University of Buffalo, and with the financial and emotional support of his parents, he re-enrolled full-time at the university and graduated with a degree in business and financial management. His interest in online gaming diminished as he spent his free time studying, going to classes, and making new friends—and he was finally doing something he loved.

Whatever the situation, confronting the issues that initially drove a client toward the addiction will not be easy, but it is the only way to achieve the personal growth necessary to maintain long-term recovery. In our work together, James gained a deeper sense of why he gamed. Important questions to ask clients, in order to help them understand their gaming behavior are as follows:

What types of needs does gaming fulfill? Does gaming give you a sense of power, but offline, do you still feel out of control? Does gaming make you feel better about yourself, but offline, do you still feel unsure about the choices you have made in life? Instead of using gaming to avoid difficult feelings, therapists should work with their clients to learn how and why gaming has become a way to deal with what hasn't been working in people's lives.

Family Therapy

Adolescent gaming addiction continues to be a major issue in our society as teen gaming abuse is growing at an alarmingly high rate. Adolescence alone, regardless of involvement in the Internet, is an extremely challenging and complex transition for young individuals. Exploring and attempting to discover one's identity as an adolescent can be an overwhelming stage in one's life. In the event that an adolescent is using online games, it is more than likely that many more obstacles will be encountered and as a result a teen will struggle with unmanageable physical and emotional consequences (Kelly, 2004).

Peer pressure and environmental stresses are the main influences on an adolescent who becomes involved with gaming. Friends are often gamers, and, as discussed above, family dynamics can play a role in the development of online gaming addiction. Furthermore, children of substance-abusing parents have been shown to have an increased risk of using gaming as a means to cope with problems such as developmental issues, school problems, health problems, delinquency, sexual problems, mental issues, and family problems.

It is very hard for a teen to recover from gaming addiction, especially when the computer is often a necessary component of the teen's home and school environments. Effective treatment requires that the dynamics of the family should be assessed and that family members must also be helped to achieve health, or relapse is much more likely.

For most adolescents, referral to treatment is involuntary and is usually mandated by parents, teachers, or the judicial system. When asked at the intake stage what the problem is, adolescents' most common answers are "Don't know" or "Somebody [family, teacher, policeman] just overreacted." When pressed, most adolescents say they are doing nothing different from their peers or explain that they aren't online as much as other friends who game.

Successful treatment must not only address the gaming behavior but also help an adolescent navigate the normal developmental tasks of identity formation that are often neglected while gaming is being used as a means of coping with life's problems. Treatment should focus on effective problem solving and the social skills necessary to build self-esteem. Many gamers lack a strong sense

of self, using gaming as means to form their identities. However, their self-esteem in real life is fragile or nonexistent. Family therapy must focus on ways to build or rebuild their identities within a nongaming environment.

Gamers often minimize the extent to which they game and avoid dealing with the family issues that may be driving their desire to game. It is important to consider an adolescent's specific family situation when treating the addiction. Comprehending the teen's immediate environment in most cases enhances the understanding of the addiction's causes. It is necessary to look at family dynamics, such as family history of addiction, background, communication dynamics, or conflict and to look at the ways in which these factors may be impacting a teen's developmental stages, emotional well-being, and self-esteem.

Finally, family therapy needs to include educating the family on ways in which they can help the addict, whether or not he or she is in individual counseling or treatment. This may include counseling for family members, education on problem/compulsive gaming for the family, strategies on how to cope with the addict's anger and loss of trust, and education on the emotional costs of online gaming. Often, gaming addiction is addressed as a part of a weekly family program. Each week, topics related to addiction are addressed, to help family members understand the process of recovery, the possible relapse triggers, and the importance of maintaining healthy boundaries. This is especially important for parents as they struggle to understand a son or daughter's compulsive need to game and the underlying dynamics associated with the addiction.

Communication Skills

Communication skills may also need to be learned. Many gamers cannot communicate well in face-to-face situations (Leo Sang-Min, 2003). This one reason why children game in the first place. Communicating online seems safer and easier for them. However, a lack of communication skills can cause poor self esteem and feelings of isolation and create additional problems in life among adolescents, so therapy needs to address the way they communicate with others offline. Some basic guidelines for therapists include the following:

- Enlisting the aid of an older child who may help to engage your client in short conversations to help develop skills.
- Engaging as many of your client's senses as you can during a conversation and teaching the client to do the same. The client's interest in the discussion will remain higher.
- Using books, magazines, and television to talk to your client about facial expressions and what they mean, and to enable the client to watch for body

language so as to improve the understanding of what the other person is feeling.

- Using role-playing conversations to build the client's confidence. You should start one on one and introduce other children one at a time as the client begins to build confidence in his abilities. Older siblings, cousins, or neighbors might be more than willing to help.
- Taking the time to tell to your clients what you think they meant. If needed, you should help them find ways to better explain themselves.
- Asking your client to tell you what she thinks you said. This will help you see how well your client listens during a conversation.
- Using regular eye contact and having your client practice using eye contact when speaking to other people.

Residential Care

Residential care may be required to provide intensive therapy when the effects of the game have become severe. Often gamers refuse treatment until they become deeply depressed, are dismissed from school, are terminated from a job, are threatened with divorce and separation, or are thinking about suicide. Once the problems have become this severe, it is important to seek professional help in evaluating the situation. Residential treatment programs often last for four to six weeks of intensive treatment. Some gamers may require more or less time, so recommendations will be made following an initial assessment.

In most cases, the treatment program of a residential care facility is specifically designed to fit the needs of the client, and most sessions focus on individual treatment, educational groups, and family therapy where appropriate to best manage and address the intense feelings surrounding the addiction. Often, parents will initiate residential care for a child addicted to online gaming. It may be difficult to find a facility that understands the special requirements in treating compulsive online gaming, but gradually more inpatient addiction rehabilitation centers are learning about this new form of addictive behavior.

REFERENCES

BBC News. (2005). *China imposes online gaming curbs*. Retrieved August 7, 2007, from http://news.bbc.co.uk/1/hi/technology/4183340.stm

CBSNews.com. (2006). *Detox for video game addiction? Experts say gaming can be a compulsion as strong as gambling.* Retrieved August 7, 2007, from http://www.cbsnews.com/stories/2006/07/03/health/webmd/main1773956.shtml

Ducheneaut, N. & Moore, R. J. (2005). More than just "XP": Learning social skills in massively multiplayer online games. I*nteractive Technology and Smart Education, 2*(2), pp. 89–100.

Griffiths, M. D., Davies, M. N. & Chappell, D. (2003). Breaking the stereotype: The case of online gambling. *CyberPsychology and Behavior, 6*(1), 81–91.

Kelly, R. V. (2004). *Massively multiplayer online role-playing games: The people, the addiction and the playing.* Jefferson, NC: McFarland & Company.

Kolo, C., & Baur, T. (2004). Living a virtual life: Social dynamics of online gaming. *International Journal of Computer Game Research, 4*(1), 1–10.

Lee, M. (2007). *China to limit teens' online gaming for exercise.* Retrieved August 7, 2007, from http://www.msnbc.msn.com/id/19812989/

Leo Sang-Min, Whang, L., Lee, S., & Chang, G. (2003). Internet over-users' psychological profiles: A behavior sampling analysis on Internet addiction. *CyberPsychology and Behavior, 6*(2), 143–150.

Leung, L. (2004). Net-generation attributes and seductive properties of the internet as predictors of online activities and internet addiction. *CyberPsychology and Behavior, 7*(3), 333–348.

Leung, L. (2007). Stressful life events, motives for Internet use, and social support among digital kids. *CyberPsychology and Behavior, 10*(2), 204–214.

Ng, B. D.. & Wiemer-Hastings, P. (2005). Addiction to the Internet and online gaming. *CyberPsychology and Behavior, 8*(2), 110–113.

Tanner, L. (2007). *AMA considers video game overuse an addiction.* Associated Press. Retrieved August 7, 2007, from http://www.washingtonpost.com/wp-dyn/content/article/2007/06/27/AR2007062700995.html

Turkle, S. (1998). *Life on the screen: Identity in the age of the Internet.* Cambridge, MA: MIT Press.

Yen, J., Yen, C., Chen, C., Chen., & Ko, C. (2007). Family factors of Internet addiction and substance use experience in Taiwanese adolescents. *CyberPsychology and Behavior, 10,* 323–329.

Yee, N. (2007). Motivations of play in online games. *CyberPsychology and Behavior, 9,* 772–775.

Young, K. S. (1998a). Internet addiction: The emergence of a new clinical disorder. *CyberPsychology and Behavior, 1,* 237–244.

Young, K. S. (1998b). *Caught in the Net: How to recognize the signs of Internet addiction and a winning strategy for recovery.* New York: Wiley.

Young, K. S. (2007). Cognitive-Behavioral therapy with Internet addicts: Treatment outcomes and implications, *CyberPsychology & Behavior, 10*(5), pp. 671–679.

Young K. S., & Klausing, P. (2007). *Breaking free of the Web: Catholics and Internet addiction.* Cincinnati, OH: St. Anthony's Messenger Press.

This page intentionally left blank

Chapter **17**

Youth Gambling Prevention and Resilience Education: A Harm Reduction Approach

Isabelle D. Lussier, MA,
Jeffrey L. Derevensky, PhD, and Rina Gupta, PhD

Gambling in its many forms, has permeated every society and culture all the way back to ancient times (Caltabiano, 2003). While gambling has been a source of entertainment for countless people, a small but identifiable minority of people exhibit compulsive gambling behaviors that lead to personal harm and suffering (Abbott, Volberg, Bellringer, & Reith, 2004). Over the last two decades, an unprecedented growth has occurred in the legalization, availability, and accessibility of gambling activities and venues. Internationally, the legitimacy and popularity of gambling continue to rise, and technological advances continue to evolve, attracting both young and old to participate (Abbott et al., 2004; Derevensky, 2007).

Of particular concern is the fact that young people are consistently reported to be at higher risk of demonstrating compulsive gambling behavior (Gupta & Derevensky, 1998; National Research Council [NRC], 1999). It is estimated that 3–8 percent of adolescents meet the criteria for pathological gambling, while another 10–15 percent of adolescents are at risk for the development of severe gambling behavior (Derevensky & Gupta, 2000, 2004; Jacobs, 2000, 2004; NRC, 1999; Shaffer & Hall, 1996). Probable pathological gambling is more prevalent among males than females (NRC, 1999), and the onset of gambling problems occurs early, between the ages of 11 and 13 (Jacobs, 2000), indicating a need for targeted public education and awareness campaigns (Abbott et al., 2004).

As well, there appears to be a large degree of overlap between risk factors that predispose youth to severe gambling problems and those that predispose youth

to other risky behaviors such as delinquency and substance abuse (Derevensky & Gupta, 2004; Ladouceur, Dubé, & Bujold, 1994). These bio-psycho-social risk factors, (e.g., familial attitudes and history of gambling problems, depression, abuse, school failure, delinquency, availability of and access to gambling opportunities, and early onset) span individual, peer, familial, neighborhood, and societal domains (Dickson, Derevensky, & Gupta, 2002; see chapter 12 in this volume for a more comprehensive review of international youth gambling prevalence rates and risk and protective correlates).

The harm reduction/minimization approach, prevalent among alcohol and substance prevention initiatives, has recently attracted considerable attention from youth gambling researchers (Derevensky, 2007, and Derevensky, in press; Dickson, Derevensky, & Gupta, 2004; Gupta & Derevensky, 2008). These researchers advocate the adoption of harm reduction prevention programs (HRPPs) in targeting youth gambling behaviors, but they recommend that these strategies also emphasize the promotion of resiliency traits, by reducing the negative effects of risk factors while enhancing the beneficial moderating effects of protective factors and resiliency traits (Derevensky, 2007, and Derevensky, in press; Dickson et al., 2004; Dickson, Derevensky, & Gupta, 2008). The significant overlap in risk and protective factors for youth with respect to problem gambling and other problem behaviors (Dickson et al., 2002, 2004) has led to the creation of prevention initiatives that target multiple risk behaviors (Jessor, 1998), including problem gambling (Dickson et al., 2002, 2004; Jacobs, 2004). To date, very little resilience research regarding youth gambling behaviors has been conducted (Lussier, Derevensky, Gupta, Bergevin, & Ellenbogen, 2007). However, resiliency skills have long been incorporated into prevention programs for a wide variety of risky behaviors (Jessor, 1998). It is therefore important to extend resilience research, especially in an area such as youth gambling, which has received such little attention (Dickson et al., 2002).

RESILIENCY

Traditionally, the major focus of prevention research has been to identify risk factors and high-risk individuals (Leshner, 1999). However, it is evident that many youth exposed to high levels of risk never develop the anticipated negative problem behavior(s), and many thrive in spite of them; a concept referred to as *resilience*. Although there is substantial variation in the definition of resilience, two central constructs exist in most definitions, that is, *risk* or *adversity*, and *positive adaptation* or *competence* (Luthar, 1997). A widely accepted, simplified definition of resilience therefore relates to the presence of manifest competence despite exposure to significant adversity (Rolf & Glantz, 1999). Manifest

competence generally refers to internal states of well-being and/or effective functioning in the environment (Masten, Best, & Garmezy, 1990). Individuals may be resilient in one domain or several, but rarely in all (Luthar, 1997). For example, resilient adolescents who demonstrate high social competence despite much adversity in their lives may also report depressive symptoms. In light of these findings, resilience researchers have become increasingly cautious in using the term resilience, opting instead for more specific terms such as *educational resilience, emotional resilience,* and *behavioral resilience* (Luthar, Cicchetti, & Becker, 2000).

Resilience research has to date experienced three waves (O'Dougherty Wright & Masten, 2005). In the first wave, early studies on resilience focused mostly on the identification of protective factors. Influenced by Urie Bronfenbrenner's ecological (1979) and bioecological (2005) models, a second wave of resilience research emphasized relationships and systems, and integrated biological, social, and cultural processes across time. Ecological theory is conceptualized by Urie Bronfenbrenner as

> the scientific study of the progressive, mutual accommodation, throughout the life course, between an active, growing human being and the changing properties of the immediate settings in which the developing person lives, as this process is affected by the relations between these settings, and by the larger contexts in which the settings are embedded. (2005, p. 107)

According to this model, there are four environmental levels including the *microsystem* (the actions and interactions within the environment that a child is behaving in, at any given moment in time, e.g., home and school); the *mesosystem* (the interrelations among the child's microsystems, e.g., the relationship between a child's parents and school); the *exosystem* (the environmental factors that indirectly influence the child's behavior and development, e.g., parental workplace), and the *macrosystem* (broad social factors and cultural values that influence other settings, e.g., public social policies) (Bronfenbrenner, 2005; Kaminski & Stormshak, 2007).

Risk and Vulnerability Factors

The term *risk* commonly refers to early predictors of probable negative outcomes and to descriptions of negative life conditions such as family conflict and poverty (Kaplan, 1999). Probable negative outcomes may include psychopathology, excessive gambling, drug or alcohol abuse, dropping out of school, and so on. Two terms commonly used in reference to risk include *vulnerability factors* and *risk factors*. Risk and vulnerability factors may include individual attributes,

characteristics, situations, or contexts within the environment that increase the likelihood of acquiring and maintaining maladaptive behaviors (Kaplan, 1999). However, vulnerability factors suggest variables that increase the chances of negative outcomes within the context of adversity, while risk factors refer to variables that increase the chances of negative outcomes regardless of the occurrence of adversity (Rose, Holmbeck, Millstein Coakley, & Franks, 2004).

Researchers and clinicians have long recognized that the relationship between risk and maladaptive behaviors is such that as the co-occurrence and accumulation of risk factors over time increases, so too do maladaptive behaviors (Jessor, 1998; Jessor, Van Den Bos, Vanderryn, Costa, & Turbin, 1995; Rutter, 1990). In other words, the more risk factors an individual is exposed to, the less likely the individual will be to build internal or external assets (Benson, Galbraith, & Espeland, 1995). Risk is often described on a continuum, with a positive end associated with positive outcomes and a negative end associated with negative outcomes (e.g., socioeconomic status) (Masten, 2001). However, not all risk factors may be conceptualized in this manner. For example, teen pregnancy and cigarette use have been shown to be associated with negative outcomes, but the lack of pregnancy or smoking is not necessarily associated with positive outcomes.

Protective and Resource Factors

The term *protection* commonly involves conditions that improve an individual's resistance to negative outcomes. Two terms commonly used in reference to protection include *protective factors* and *resource factors*. Protective factors include variables that decrease the chances of negative outcomes in the context of adversity, whereas resource factors refer to variables that positively influence outcome independent of the occurrence of adversity (Rose et al., 2004). Protective factors may include personal attributes (e.g., temperament, intelligence, social bonding, personal competence, social competence), familial factors (e.g., encouragement of trust, autonomy, and initiative), and community characteristics (e.g., external support systems including church, youth groups, and school) that *moderate* a person's reaction to adversity in a positive manner (Dickson, Derevensky, & Gupta, 2008; Werner, 1995; Werner & Smith, 1992). Fostering the growth and presence of protective factors thus moderates the undesired effects of risk in such a way that development is more positive than if the protective factors had not existed (Masten et al., 1990).

To summarize, when a variable promotes or impedes adaptive outcomes within the context of adversity, it may be conceptualized in terms of protective or vulnerability factors, serving a moderating role. However, when a variable

promotes or impedes adaptive outcomes regardless of the context of adversity, it may be conceptualized in terms of resource or risk factors (Rose et al., 2004).

Most researchers now agree that a child may be identified as resilient at one point in his development but not in another. Similarly, a child may be competent in one context or aspect of life but not in another (O'Dougherty Wright & Masten, 2005). A second wave of research has sought to search for mediating and moderating processes that ultimately lead to resilience. Although this wave of research is far from complete, a third wave of research has already begun, with a focus on intervention strategies designed to promote resilience.

PREVENTION EFFORTS

The importance of resilience research rests in its applicability to the field of prevention. Researchers have begun to incorporate resilience research into prevention and intervention programs for high-risk youth (Coie et al., 1993; Leshner, 1999). The focus in prevention research was initially to identify risk and vulnerability factors and at-risk populations (Garmezy, 1971; Pasamanick & Lilienfeld, 1956). However, the identification of risk and vulnerability factors by themselves has not been of great use to prevention efforts since many of these factors are difficult to minimize (e.g., poverty) or identify (e.g., sexual abuse) (Leshner, 1999) and since many high-risk youth never actually develop the anticipated negative behaviors. As a result, an attempt to identify variables and interactions between variables that might act as buffers or protective factors to counteract the risks associated with aberrant behavior has begun.

Dickson and her colleagues (2004) have integrated adolescent gambling behavior into Jessor's (1998) adolescent risk behavior model. Jessor's model follows current trends in resilience research, where risk and protection are seen as interacting across various domains (biological, social environment, perceived environment, personality, and behavior) and high-risk behaviors. Further, the protection variables listed in each of the domains of the model correspond to broad resiliency traits including *social bonding* (pro-social ties to one's school, family, and community), *personal competence* (one's individual identity and sense of personal development), and *social competence* (one's ability to adjust in social situations) (Springer & Phillips, 1992).

HARM REDUCTION

There are certain risky behaviors, such as alcohol consumption, that, though potentially harmful, have nonetheless become part of the fabric of our society.

This has led certain prevention specialists to reevaluate abstinence models as being unrealistic and impractical (Beck, 1998; Dickson et al., 2004; Poulin & Elliott, 1997). Rather, a movement toward responsible, controlled involvement has led to a harm reduction approach versus an abstinence approach. Harm reduction strategies are designed to limit the harmful effects that may result from involvement in risky behaviors without demanding abstinence per se. Such strategies were first developed to curb alcohol and illicit substance abuse (Erickson, 1997). More recently however, a youth gambling risk prevention model based on a public health perspective has been proposed (Messerlian, Derevensky, & Gupta, 2005). Within this model, primary prevention strategies are designed to prevent the onset of risky gambling behavior by educating youth, parents, professionals, and the public regarding the risks and consequences of problem gambling. As well, based on the assumption that it is not realistic to expect youth to abstain from gambling altogether (especially unregulated forms of gambling), secondary prevention strategies are designed to prevent juveniles at risk of developing serious gambling problems from escalating toward problem gambling. These harm reduction strategies include early identification of gambling problems by educating primary health care workers to identify some of the risk and warning signs of excessive gambling. Finally, tertiary prevention strategies within this model, designed for youth exhibiting excessive gambling behaviors, seek to augment the access to and availability of treatment, services, and support (Messerlian et al., 2005).

HARM REDUCTION AND RESILIENCE EDUCATION INITIATIVES

Given the increasing availability, accessibility, and popularity of gambling, the utility of a harm reduction approach to prevent probable pathological gambling among youth is promising. Based on a harm reduction approach, the International Centre for Youth Gambling Problems and High-Risk Behaviors at McGill University in Montreal has developed a number of prevention initiatives including *The Amazing Chateau* and *Hooked City* (interactive CD ROM games for children and adolescents aged 11–18), prevention workshops for youth, and *Clean Break* (a DVD/VHS docudrama designed for adolescents 13–18) (Derevensky, in press). Although there are very few evaluated outcomes of harm reduction prevention initiatives for youth gambling (Petry, 2005), parents (Ladouceur, Vitaro, & Côté, 2001; Côté, Vitaro, & Ladouceur, 2003) and educators (Ladouceur, Ferland, Côté, & Vitaro, 2004) in Quebec, Canada, are reportedly becoming increasingly aware of the potential risks involved in youth gambling behavior, indicating that primary prevention efforts may be having a

beneficial effect. As well, estimates from a large-scale national prevalence study in the United Kingdom (UK) indicate that lifetime participation in gambling activities and rates of problem gambling among youth have been on a steady decline since 1997 (Wood, Griffiths, Stevens, Bartlett, & Pye, 2006). The authors suggest that this decline may be an indication of the beneficial effects of prevention measures put in place in the country.

Resilience education programs have received some evaluative attention, supporting the plausibility of translating resilience research into effective practice-based prevention and intervention programs (Battistich, Schaps, & Wilson, 2004; Lynch, Geller, & Schmidt, 2004). For example, Battistich and colleagues (2004) examined the effects of an elementary-school intervention program aimed at reducing risk and promoting resilience among youth. Students exposed to the program experienced greater levels of pro-social behavior and engaged in fewer problem behaviors than did the control group. Similarly, social-emotional competence, positive coping skills, and suppression of antisocial and aggressive behavior were strengthened by a carefully designed, research-based resilience program for children (Lynch et al., 2004).

Nation and colleagues (2003) have identified nine qualities that are consistently displayed in effective prevention programs. These principles include (a) comprehensive programming, (b) varied methods of teaching, (c) adequate exposure to the intervention in terms of duration, (d) theory-driven programming, (e) promotion of strong relationships between adults and participants, (f) developmentally sensitive programming such that exposure occurs early enough to have an impact on the problem behavior, (g) sociocultural relevance, (h) clear goals and objectives and documentation of results relative to these goals, and (i) well-trained personnel. Resilience programs that are grounded in research and theory tend to be multifaceted in nature and include multiple strategies designed to strengthen protective factors while concurrently reducing or minimizing risk.

Concluding Remarks

Gambling activities have entertained people across cultures for thousands of years (Caltabiano, 2003). However, over the last 20 years there has been a proliferation in the legalization and expansion of various forms of gambling. Consequently, a reevaluation of how best to prevent youth from developing and maintaining serious gambling problems has fostered the adoption of a harm reduction approach (Dickson et al., 2002; Gupta & Derevensky, 2008; Messerlian et al., 2005). Although such efforts are currently in their infancy, environmental micro-, meso-, exo-, and macrosystems should be taken

into consideration in the design, implementation, and evaluation stages of such initiatives, as these four systems are increasingly recognized as important transactional variables that significantly influence human development (Bronfenbrenner, 2005; Kaminski & Stormshak, 2007).

Despite the promising potential of resilience education programs, various concerns have been raised regarding the hazards of applying such programs in schools. In particular, Pianta and Walsh (1998) delineate numerous cautions to bear in mind regarding the immaturity of the study of resilience, and they draw a parallel between resilience education programs and historical fads such as the e*ffective schools movement*that was popular in the 1970s and early 1980s. During the period of popularity of this movement, schools were identified that had students doing better academically than could be expected, given their high-risk backgrounds. These schools were then used to derive lists of factors that could improve the performance of students in schools with an overrepresentation of high-risk youth. Pianta and Walsh (1998) point out that despite the promise of early findings, methodological flaws in early evaluative studies, eventual lack of evidence, and lack of theory led to the downfall of the movement. They suggest a parallel between the rise and fall of the effective schools movement and the proliferation of recent success stories arising from the resilience movement. Although Pianta and Walsh's article was originally published in 1998, their concerns remain poignant today, as new articles reporting on resilience theory and education programs are published in greater numbers than ever before. Pianta and Walsh caution that a heavy focus on stories of success may deter individuals from appreciating that success is a process that develops over time, and that such a focus may deflect attention away from the harsh realities that high-risk youth are exposed to (Pianta & Walsh, 1998). Similarly, Cantinotti and Ladouceur (2008) caution that harm reduction initiatives for gambling behavior require adherence to the integrity of the original term, harm reduction (i.e., the reduction of negative effects related to gambling, without reducing gambling participation per se), lest the term become so broad as to lose its utility.

Ultimately, the successful prevention of gambling and other problems among youth and the treatment of youth with gambling problems and other addictions is the desired outcome of youth gambling research. Today, it is generally acknowledged that gambling problem prevention efforts, as well as public and industry policies, must be empirically science based (Abbott et al., 2004; Dickson et al., 2002, 2004). The efficacy of programs is largely dependent upon commitment from stakeholders to work together and on conceptually driven research on risk and resilience theory (Abbott et al., 2004; Gupta & Derevensky, 1997; Luthar et al., 2000).

The lack of recognition by youth, parents, educators, and primary health care workers of the prevalence and negative effects of problem gambling is disconcerting, considering that estimates for problem gambling among youth are predicted to rise as gambling activities increase in accessibility, availability, and popularity (Abbott et al., 2004; Derevensky, 2007). Though more complex and controversial in terms of its goals than an abstinence model, the notion of a harm reduction approach that promulgates self-control and responsible involvement may thus become increasingly realistic and palatable as a way of protecting our adolescents.

REFERENCES

Abbott, M. W., Volberg, R. A., Bellringer, M., & Reith, G. (2004). *A review of research on aspects of problem gambling. Final report.* Prepared for the Responsibility in Gambling Trust, Auckland, New Zealand: Auckland University of Technology.

Battistich, V., Schaps, E., & Wilson, N. (2004). Effects of an elementary school intervention on students' "connectedness" to school and social adjustment during middle school. *Journal of Primary Prevention, 24,* 243–262.

Beck, J. (1998). 100 years of "Just say no" versus "Just say know": Reevaluating drug education goals for the coming century. *Evaluation Review, 22,* 15–45.

Benson, P., Galbraith, J., & Espeland, P. (1995). *What kids need to succeed: Proven practical ways to raise good kids*. Minneapolis, MN: Free Spirit Publishing.

Bronfenbrenner, U. (1979). *The ecology of human development: Experiments by nature and design.* Cambridge, MA: Harvard University Press.

Bronfenbrenner, U. (2005). Ecological systems theory. In U. Bronfenbrenner (Ed.), *Making human beings human: Bioecological perspectives on human development.* Thousand Oaks, CA: Sage.

Caltabiano, N. J. (2003). From antiquity to Australia: A brief account of gambling. *International Journal of Mental Health and Addiction, 1,* 16–19.

Cantinotti, M., & Ladouceur, R. (2008). Harm reduction and electronic gambling machines: Does this pair make a happy couple or is divorce foreseen? *Journal of Gambling Studies, 24,* 39–54.

Coie, J., Watt, N., West, S., Hawkins, J., Asarnow, J., Markman, H., et al. (1993). The science of prevention. *American Psychologist, 48,* 1013–1022.

Côté, M.-A., Vitaro, F., & Ladouceur, R. (2003). Attitudes, connaissances et comportements des parents québécois en regard de la pratique des jeux de hasard et d'argent chez les jeunes. *Canadian Psychology, 44,* 152–161.

Derevensky, J., (2007). Youth gambling and problem gambling: Another high risk behavior. (Impulsividade: Jogo entre Jovens e Jogo Problematico). In A. Gigliotti & A. Guimarães (Eds.), *Dependencia, compulusão e impulsividade* (Compulsivity, impulsivity and addiction) (pp. 81–90). Rio de Janeiro, Brazil: Editoro Rubio.

Derevensky, J., (in press). Youth gambling: A Canadian perspective. In J. F. Cosgrave & T. R. Klassen (Eds.), *Gambling in 21st century Canada: Citizens, consumers and the state*. Toronto, Ontario, Canada: McGill-Queen's Press.

Derevensky, J. L., & Gupta, R. (2000). Prevalence estimates of adolescent gambling: A comparison of the SOGS-RA, DSM-IV-J, and the GA 20 Questions. *Journal of Gambling Studies, 16*, 227–251.

Derevensky, J. L., & Gupta, R. (2004). Adolescents with gambling problems: A synopsis of our current knowledge. *e-Gambling: The Electronic Journal of Gambling Issues, 10*, 119–140.

Dickson, L. M., Derevensky, J., & Gupta, R. (2002). The prevention of gambling problems in youth: A conceptual framework. *Journal of Gambling Studies, 18*, 97–159.

Dickson, L. M., Derevensky, J. L., & Gupta, R. (2004). Harm reduction for the prevention of youth gambling problems: Lessons learned from adolescent high-risk prevention programs. *Journal of Adolescent Research, 19*, 233–263.

Dickson, L., Derevensky, J., & Gupta, R. (2008). Youth gambling problems: An examination of risk and protective factors. *International Gambling Studies 8*, 25–47.

Erickson, P. G. (1997). Reducing the harm of adolescent substance use. *Canadian Medical Association Journal, 156*, 1386–1393.

Garmezy, N. (1971). Vulnerability research and the issue of primary prevention. *American Journal of Orthopsychiatry, 41*, 101–116.

Gupta, R., & Derevensky, J. L. (1997). Familial and social influences on juvenile gambling behaviour. *Journal of Gambling Studies, 13*, 179–192.

Gupta, R., & Derevensky, J. (1998). Adolescent gambling behavior: A prevalence study and examination of the correlates associated with problem gambling. *Journal of Gambling Studies, 14*, 319–345.

Gupta, R., & Derevensky, J. (2008). Gambling practices among youth: Etiology, prevention and treatment. In C. A. Essau (Ed.), *Adolescent addiction: Epidemiology, assessment and treatment* (pp. 207–230). London: Elsevier.

Jacobs, D. F. (2000). Juvenile gambling in North America: An analysis of long term trends and future prospects. *Journal of Gambling Studies, 16*, 119–152.

Jacobs, D. F. (2004). Youth gambling in North America: Long term trends and future prospects. In J. Derevensky & R. Gupta (Eds.), *Gambling problems in youth: Theoretical and applied perspectives* (pp. 1–26). New York: Kluwer Academic/Plenum Publishers.

Jessor, R. (1998). *New perspectives on adolescent risk behavior.* Cambridge, UK: Cambridge University Press.

Jessor, R., Van Den Bos, J., Vanderryn, J., Costa, F. M., & Turbin, M. S. (1995). Protective factors in adolescent problem behavior: Moderator effects and developmental change. *Developmental Psychology, 31*, 923–933.

Kaminski, R. A., & Stormshak, E. A. (2007). Project STAR: Early intervention with preschool children and families for the prevention of substance abuse. In P. Tolan, J. Szapocznik, & S. Soledad (Eds.), *Preventing youth substance abuse: Science-based*

programs for children and adolescents (pp. 89–109). Washington, DC: American Psychological Association.

Kaplan, H. B. (1999). Toward an understanding of resilience: A critical review of definitions and models. In M. D. Glantz & J. L. Johnson (Eds.), *Resilience and development: Positive life adaptations* (pp. 17–84). New York: Kluwer Academic/Plenum Publishers.

Ladouceur, R., Dubé, D., & Bujold, A. (1994). Prevalence of pathological gambling and related problems among college students in the Quebec metropolitan area. *Canadian Journal of Psychiatry, 39,* 289–293.

Ladouceur, R., Bouchard, C., Rhéaume, N., Jacques, C., Ferland, F., Leblond, J., & Walker, M. (2000). Is the SOGS an accurate measure of pathological gambling among children, adolescents and adults? *Journal of Gambling Studies, 16,* 1–21.

Ladouceur, R., Ferland, F., Côté, M.-A., & Vitaro, F. (2004). Teachers' knowledge and training needs regarding youth gambling. *School Psychology International, 25,* 472–479.

Ladouceur, R., Vitaro, F., & Côté, M.-A. (2001). Parents' attitudes, knowledge, and behavior toward youth gambling: A five-year follow-up. *Journal of Gambling Studies, 17,* 101–116.

Leshner, A. I. (1999). Introduction. In M. D. Glantz & J. L. Johnson (Eds.), *Resilience and development: Positive life adaptations* (pp. 1–4). New York: Kluwer Academic/Plenum Publishers.

Lussier, I., Derevensky, J., Gupta, R., Bergevin, T., & Ellenbogen, S. (2007). Youth gambling behaviours: An examination of the role of resilience. *Psychology of Addictive Behaviors, 21,* 165–173.

Luthar, S. S. (1997). Sociodemographic disadvantage and psychosocial adjustment: Perspectives from developmental psychopathology. In S. S. Luthar, J. A. Burack, D. Cicchetti, & J. R. Weisz (Eds.), *Developmental psychopathology: Perspectives on adjustment risk and disorder* (pp. 459–485). New York: Cambridge University Press.

Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development, 71,* 543–562.

Lynch, K. B., Geller, S. R., & Schmidt, M. G. (2004). Multi-year evaluation of the effectiveness of a resilience-based prevention program for young children. *Journal of Primary Prevention, 24,* 335–353.

Masten, A. S. (2001). Ordinary magic: Resilience processes in development. *American Psychologist, 56,* 227–238.

Masten, A. S., Best, K. M., & Garmezy, N. (1990). Resilience and development: Contributions from the study of children who overcome adversity. *Development and Psychopathology, 2,* 425–444.

Messerlian, C., Derevensky, J., & Gupta, R. (2005). Youth gambling problems: A public health perspective. *Health Promotion International, 20,* 69–79.

Nation, M., Crusto, C., Wandersman, A., Kumpfer, K. L., Seybolt, D., Morrissey-Kane, E., et al. (2003). What works in prevention: Principles of effective prevention programs. *American Psychologist, 58,* 449–456.

National Research Council. (1999). *Pathological gambling: A critical review.* Washington, DC: National Academy Press.

O'Dougherty Wright, M., & Masten, A. S. (2005). Resilience processes in development: Fostering positive adaptation in the context of adversity. In S. Goldstein & R. B. Brooks (Eds.), *Handbook of resilience in children* (pp. 17–37). New York: Kluwer Academic/Plenum Publishers.

Pasamanick, B., & Lilienfeld, A. M. (1956). Association of maternal and fetal factors with development of mental deficiency: Abnormalities in the prenatal and paranatal periods. *Journal of the American Medical Association, 159,* 155–160.

Petry, N. M. (2005). Prevention: Focus on gambling in youth and young adults. In N. M. Petry (Ed.), *Pathological gambling: Etiology, comorbidity, and treatment* (pp. 269–278). Washington, DC: American Psychological Association.

Pianta, R. C., & Walsh, D. J. (1998). Applying the construct of resilience in schools: Cautions from a developmental systems perspective. *School Psychology Review, 27,* 407–417.

Poulin C., & Elliott, D. (1997). Alcohol, tobacco and cannabis use among Nova Scotia adolescents: Implications for prevention and harm reduction. *Canadian Medical Association Journal, 156,* 1387–1393.

Rolf, J. E., & Glantz, M. D. (1999). Resilience: An interview with Norman Garmezy. In M. D. Glantz, & J. L., Johnson (Eds.), *Resilience and development: Positive life adaptations* (pp. 5–14). Dordrecht, Netherlands: Kluwer Academic Publishers.

Rose, B. M., Holmbeck, G. N., Millstein Coakley, R., & Franks, E. A. (2004). Mediator and moderator effects in developmental and behavioral pediatric research. *Developmental and behavioral pediatrics, 25,* 58–67.

Rutter, M. (1990). Psychosocial resilience and protective mechanisms. In J. Rolf, A. S. Masten, D. Cicchetti, K. H. Nuechterlein, & S. Weintraub (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 181–214). New York: Cambridge University Press.

Shaffer, H. J., & Hall, M. N. (1996). Estimating the prevalence of adolescent gambling disorders: A quantitative synthesis and guide toward standard gambling nomenclature. *Journal of Gambling Studies, 12,* 193–214.

Springer, J. F., & Phillips, J. L. (1992). Extended national youth sports program 1991–1992 evaluation: Part two: Individual protective factors index (IPFI) and risk assessment study. Folsom, CA: EMT Associates.

Werner, E. (1995). Resilience in development. *Current Directions in Psychological Science, 4,* 81–85.

Werner, E. E., & Smith, R. S. (1992). *Overcoming the odds: High risk children from birth to adulthood.* Ithaca, NY: Cornell University Press.

Wood, R.T.A., Griffiths, M., Stevens, J., Bartlett, H., & Pye, J. (2006). *Under 16s and the national lottery. Final report.* Prepared for the National Lottery Commission. London: National Lottery Commission.

Index

About the Editor and Contributors

EDITOR

Angela Browne-Miller, PhD, DSW, MPH, is the Set Editor of the *Praeger International Collection on Addictions* (2009), Director of Metaxis Institute for Personal, Social, and Systems Change, based in northern California, United States; Director of Browne and Associates Violence, Substance Abuse, and Trauma Treatment and Prevention Program, also in northern California; has been a keynote speaker at conferences around the world on addiction, violence, and behavior change; and is the author of numerous books, including *To Have and to Hurt: Seeing, Changing or Escaping Patterns of Abuse in Relationships* (2007) and *Rewiring Your Brain to Change Your Behavior* (2009). Dr. Browne-Miller earned two doctorates and two master's degrees at the University of California, Berkeley, where she lectured in three departments for 14 years, and has served as a National Institute of Mental Health Postdoctoral Fellow, a U.S. Department of Public Health Fellow, the public relations director for Californians for Drug Free Youth, the Research Education and Treatment Director for the Cokenders Alcohol and Drug Program, a member of the board of directors of the Employee Assistance Society of North America, an advisor to addiction treatment programs in the United States and several other countries, and project director on three California Department of Health violence prevention projects.

CONTRIBUTORS

Hugo Barrera, MD, completed his internship and general surgery residency training at the University of California, San Diego from 1992 until 1997. Since 1997, he has been with Coast Surgical Group in Chula Vista, California, United States, where he became a partner in 2000. Dr. Barrera's special interests in surgery include surgery for the correction of heartburn using minimally invasive techniques (laparoscopic Nissen fundoplication), advanced laparoscopic surgery including removal of the colon and spleen, and cancer surgery. Dr. Barrera is the Medical Director of the Bloodless Medicine and Surgery Program at Sharp Chula Vista Medical Program. This is a program focusing on the implementation of specialized techniques to minimize operative blood loss and decrease the use of blood transfusions.

Ronald J. Burke, PhD, University of Michigan, is currently professor of organizational behavior, Schulich School of Business, York University, Toronto, Ontario, Canada. His research interests include gender in organizations, work and health, work hours and work addiction, and the use of human resource management research to build more humane and more effective organizations.

Jeffrey L. Derevensky, PhD, is professor in the School of Applied Child Psychology, Department of Educational and Counseling Psychology, also professor in the Department of Psychiatry, McGill University, Montreal, Quebec, Canada. He is codirector of the McGill University Youth Gambling Research and Treatment Clinic and the International Centre for Youth Gambling Problems and High-Risk Behaviors. He is a child psychologist who has published widely in the field of youth gambling. Dr. Derevensky remains on the editorial board of several journals and is an international consultant on responsible gambling practices.

Mark Griffiths, PhD, is a chartered psychologist and professor of gambling studies at Nottingham Trent University, Nottingham, England, and director of the International Gaming Research Unit. He has spent over two decades in the field, is internationally known for his work on gambling and gaming, and has won seven national and international awards for his research. He has published over 200 refereed research papers, two books, 50 book chapters, and over 550 other articles. He has served on numerous national and international committees and gambling charities (e.g., [the] Society for the Study of Gambling, Gamblers Anonymous General Services Board, National Council on Gambling, and as national chair of GamCare). He has won eight national and international awards for his work, including the John Rosecrance Prize (1994), the CELEJ Prize (1998), and the Joseph Lister Prize (2004). He also

does a great deal of freelance journalism and has appeared on over 1,800 radio and television programs.

Rina Gupta, PhD, is a clinical child psychologist and assistant professor of school/applied child psychology, Department of Educational and Counseling Psychology at McGill University, Montreal, Quebec, Canada. She has published widely and has focused her research and social policy work in the area of youth high-risk behaviors. Her early work testing a general theory of addictions was recognized with the doctoral dissertation award of the National Council on Problem Gambling. Most recently, Dr. Gupta was named the National Centre for Responsible Gambling's young researcher of 2006. Dr. Gupta is the founder and codirector of the International Centre for Youth Gambling Problems and High-Risk Behaviors, where her research focus is on prevention. She has worked hard to effect social change and has been invited to provide expert testimony before a number of governmental committees internationally.

Norman Jackson, MS, is a senior learning skills counselor with University of San Diego (UCSD) Center for Academic Research and Training in Anthropogeny (CARTA) in San Diego, California, United States. Prior to working at UCSD, he worked for the Information and Education Section in the California Department of Health Services Immunization Branch as an educational specialist. He was awarded an academic fellowship from the Andrus Foundation and completed a master's of science degree from San Diego State University's College of Health and Human Services. He has over 14 years of health promotion and education work experience in the nonprofit and government sectors. He is committed to working to decrease the health disparities in populations of color, historically disadvantaged populations, and the aged.

Cynthia R. Kalodner, PhD, is professor of psychology at Towson University in Baltimore, Maryland, United States. She teaches graduate and undergraduate courses in abnormal psychology, group counseling, and counseling techniques. Aside from her work in eating disorders, Dr. Kalodner is well known for her work in group counseling and psychotherapy. She coedited a book entitled *The Handbook of Group Counseling and Psychotherapy* (2004). She also writes fiction for young adults.

Robert W. Kubey, PhD, is director of the Center for Media Studies and professor of journalism and media studies at Rutgers University in New Brunswick, New Jersey, United States. Professor Kubey received his doctorate from the Committee on Comparative Human Development at the University of Chicago. Dr. Kubey has been an Annenberg Scholar in Media Literacy at the University of Pennsylvania, and a National Institute of Mental Health research fellow in

the Program in Social Ecology at the University of California at Irvine. He has been a visiting professor at Stanford University. Professor Kubey's books include *Creating Television* (2004), *Television and the Quality of Life* (1991), coauthored with Mihaly Csikszentmihalyi, and *Media Literacy in the Information Age* (1997/2001). Dr. Kubey has written articles for the *New York Times, Scientific American, Newsweek*, and the *Christian Science Monitor* as well as for numerous other major newspapers and magazines. News reports on his research have been carried by all of the world's major wire services and news outlets.

Isabelle D. Lussier, MA, PhD candidate, is a doctoral student in the School/Applied Child Psychology Program, Department of Educational and Counseling Psychology, McGill University, Montreal, Quebec, Canada. Her master's thesis examined youth gambling problems and resilience, and was recognized by the National Council on Problem Gambling with its master's thesis award. Her current research explores personal resiliency traits as moderators of the relationship between environmental risk and gambling problems among low socioeconomic status youth.

Richard A. McGowan, MA, MDiv, ThM, DBA**,** is associate professor in the Carroll School of Management at Boston College in Boston, Massachusetts, United States, and research associate at Harvard Medical, Division on Addictions also in Boston. The focus of his research is on the interaction of the business and public policy processes, especially as they relate to the gambling, tobacco, and alcohol industries. Father McGowan has published five books, entitled *State Lotteries and Legalized Gambling: Painless Revenue or Painful Mirage* (1994); *Business, Politics and Cigarettes: Multiple Levels, Multiple Agendas* (1995); *Industry as a Player in the Social and Political Arenas* (1996); and *The Search for Revenue and the Common Good: An Analysis of Government Regulation of the Alcohol Industry* (1997). He has also published 48 referred articles in various academic journals and made over 80 academic and professional presentations.

Basant K. Puri, MA, PhD, MB, MRCPsych, MMath, of the Medical Research Council (MRC) Clinical Sciences Centre, Hammersmith Hospital, and the Imperial College in London, England, received his primary and postgraduate degrees in medicine from the University of Cambridge and carried out postdoctoral work in molecular genetics at the MRC and the University of Cambridge, and in imaging at the Royal Postgraduate Medical School at Hammersmith Hospital, London. He also has a first-class honors degree in mathematics and postgraduate degrees in mathematics and is a member of the Royal College of Psychiatrists.

Paul Rose, PhD, is an assistant professor of psychology at Southern Illinois University, Edwardsville, Illinois, United States. His current research is focused on consumer behavior. He has also published on the self and romantic relationships.

Amanda Ruiz, MD, trained in psychiatry at the University of California San Diego, in San Diego, California, United States. She also completed a fellowship in forensic psychiatry at the University of California, San Francisco. Dr. Ruiz currently provides consultation services to physicians, attorneys, and companies on mental illness and risk management.

Dan J. Segrist, PhD, is an assistant professor of psychology at Southern Illinois University, Edwardsville, Illinois, United States. His research interests include college student alcohol use, clinical practice issues, and the teaching of psychology.

Mamta Sood, MD, earned her MD degree from Ranchi University in India, followed by a diploma in psychiatry. She has been elected as a fellow of the Indian Association for Social Psychiatry in India. She worked as a psychiatric specialist in the Army Medical Corps of India and subsequently joined the All India Institute of Medical Sciences, New Delhi, Delhi, India as an assistant professor. She has several publications and has received the award for the best paper in military psychiatry and industrial psychiatry.

Ian H. Treasaden, MB, LRCP, MRCS, FRCPsych, LLM, is head of forensic neurosciences, Lipid Neuroscience Group, Imperial College, London, England, and, since l984, he has been consultant forensic psychiatrist at the Three Bridges Medium Secure Unit for mentally disordered offenders, West London Mental Health NHS Trust, where he has also been clinical director. He qualified in medicine in 1975 from the London Hospital Medical College, University of London, where he was awarded the James Anderson Prize in Clinical Medicine. He undertook training in forensic psychiatry at the Maudsley and Bethlem Royal Hospitals in London and Broadmoor Special Hospital, Berkshire, England, between 1982 and 1984. He is the author of papers on forensic psychiatry, and he is also coauthor of the *Textbook of Psychiatry and Mental Health Law: A Practical Guide*. His current research interests include the relationship between mental disorder and offending, including violence among those with schizophrenia and associated lipid neuro-imaging abnormalities.

Meera Vaswani, PhD, earned her PhD degree from Delhi University, a leading university in India. She is a member of the All India Institute of Medical Sciences in New Delhi, Delhi, a prestigious and internationally recognized

institute in India, where she was first a postdoctoral fellow, subsequently became a lecturer, and rose to be a professor. During this time, she was elected as a fellow of the Royal Society, London, and member of the National Academy of Medical Sciences, India. She was selected for a United Nations fellowship, during which she worked in the University of Glasgow, in Scotland, UK. She was one of three scientists in the world selected for the Distinguished International Scientist Collaborative Award (DISCA) from the U.S. National Institute on Drug Addiction (NIDA), the U.S. National Institutes of Health (NIH), 2007. She has completed several projects on addiction (alcohol and heroin) and published many papers in both national and international journals. She has been a guest speaker at several national and international forums. She was the only delegate invited by Japan to represent India in the Asia Pacific Society of Biological Research in Alcoholism. Subsequently, she was elected as a member of the board of directors of the Asia Pacific Society for Alcohol and Addiction Research (APSAAR).

Kimberly S. Young, PhD, is an internationally known expert on Internet addiction and online behavior. She serves as the director of the Center for Internet Addiction Recovery, founded in 1995, and travels nationally conducting seminars on the impact of the Internet. She is the author of *Caught in the Net* (1998), the first book to address Internet addiction, which has been translated into six languages; *Tangled in the Web* (1998); and her most recent, *Breaking Free of the Web: Catholics and Internet Addiction* (2009). She is a professor at St. Bonaventure University in St. Bonaventure, New York, United States, and has published over 40 articles on the impact of online abuse. Her work has been featured in media outlets such as the *New York Times*, *USA Today*, *Newsweek*, *Time*, CBS News, Fox News, *Good Morning America*, and ABC's *World News Tonight*. Dr. Young received the Psychology in the Media Award from the Pennsylvania Psychological Association, and in 2000 she received the Alumni Ambassador of the Year Award for Outstanding Achievement from Indiana University at Pennsylvania in Pennsylvania, United States.