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APA
Addiction Syndrome Handbook
VOLUME 1
Foundations, Influences, and Expressions of Addiction

Howard J. Shaffer, Editor-in-Chief
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To my parents, Milton and Ruth, for providing me with the freedom to be curious; to my brother and sister, Rick and Jayne, who let me be rebellious; and to my children, David and Paige, who necessitated that I be vigilant—collectively furnishing me with the cornerstones of a life in science.

—Howard J. Shaffer

In memory of my father, John, for teaching me to live life on my own terms.

—Debi A. LaPlante

To my parents, Ann and Marshall, for giving me the confidence to pursue my goals, and to my husband, Travis, for helping me choose goals worth pursuing.

—Sarah E. Nelson
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Howard J. Shaffer, PhD, is an associate professor of psychology at Harvard Medical School and director of the Division on Addiction at the Cambridge Health Alliance, a Harvard Medical School teaching affiliate. Dr. Shaffer is also licensed as a clinical psychologist in the Commonwealth of Massachusetts and is certified by the National Register of Health Care Providers in psychology. He has more than 35 years of treatment experience; during his career, he has studied and treated the full range of addiction expressions (e.g., gambling, opioid, cocaine, nicotine, shopping, computer, and sexual dependence).

Dr. Shaffer's major interests include the social perception of addiction and disease, the philosophy of science, impulse control regulation and compulsive behaviors, addiction treatment outcome, and the natural history of addictive behaviors. He writes extensively on the treatment of addictive behaviors and the nature of addiction. His most recent book, Change Your Gambling, Change Your Life: Strategies for Managing Your Gambling and Improving Your Finances, Relationships, and Health, was published in February 2012. Additionally, he is a fellow of the American Psychological Association and the former editor of the Journal of Gambling Studies and Psychology of Addictive Behaviors. His clinical and research work has been an influential force in shaping how the field views and treats addiction. During 2010, Shaffer received the American Psychological Association Division 50 (Society of Addiction Psychology) Award for Outstanding Contributions to Advancing the Understanding of Addictions.

In addition to maintaining an active private practice and being a member of many scientific editorial boards and review panels, Dr. Shaffer consults internationally to a variety of organizations in business, education, human services, and government. He has also served on the National Academy of Sciences, National Research Council, Committee on the Social and Economic Impacts of Pathological Gambling.
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One of the responsibilities of my first postdoctorate job in 1964 was to help staff at the Bos­
ton City Hospital Alcoholism Clinic, which met once a week for 2 hours. As a graduate stu­
dent, I had had virtually no exposure to addiction, so much of what I needed to learn about alcoholism I had to acquire on the job. Among the first lessons patients and staff alike
wanted to instill was that Alcoholics Anonymous was the only treatment anyone had any
confidence in, even though its success rate was admittedly modest. I also learned of a variety
of beliefs and opinions central to then-current understandings of addiction: Alcoholism
invariably took one of two forms unique to alcohol addiction, as outlined in Jellinek’s iconic
Disease Concept of Alcoholism⁴; alcoholism ran in families, most often in troubled families, for
whom alcoholism was a consequence of a chaotic growing up; the personality, social and
emotional history, symptoms of addiction, and response to treatment of people with alcohol­
ism differed greatly from those of people with drug addiction as well as from those of people
with mental disorders; and the prognosis for treatment-seeking patients who attended the
Boston City Hospital clinic, most of them on welfare, was extremely guarded. When I talked
to colleagues from other parts of the country, I got the impression that most of these beliefs
were consensual.

My first academic job began a few years later, when I moved from Boston in 1969 to join
the Rutgers University clinical psychology program. As I learned shortly after my arrival, the
New Jersey Department of Public Health maintained separate divisions on alcoholism, drug
addiction, and mental health. Each had its own staff, budget, and specific obligation to fund
treatment and prevention efforts for the group of disorders for which it was responsible.
New to the institutional traditions of the substance abuse field, I recall my surprise that three
separate agencies were required to oversee disorders that coexisted in so many patients.

The director of the alcoholism division, himself a recovering alcoholic, shared his belief
during our first meeting that in etiology, behavior, and response to treatment people with
drug addiction and people with alcoholism were almost totally dissimilar. I shortly came to
realize that the three sets of administrators and treatment providers in the New Jersey system
held similar views; these convictions helped justify the continued separation of the divisions.
It was also the case that providers of services for patients with alcoholism, patients with drug
addiction, and patients with mental disorders tended to come to their jobs with different
backgrounds and experiences. Providers of alcoholism services, for example, were most
often recovering alcoholics who were convinced they owed their sobriety to Alcoholics

Anonymous. This experience was thought to be one of the most important means by which a person became an effective alcoholism treatment provider. As a result, because I was not a recovering alcoholic, my research on and treatment efforts with people with alcoholism were viewed with considerable skepticism during the first years I spent in New Jersey.

Although the decade from 1965 to 1975 marked the brief flowering of the community mental health center movement throughout the United States, the New Jersey community mental health centers preferred to leave the diagnosis and treatment of substance abusers, both those with alcoholism and those who were drug addicted, to their own separately funded treatment facilities. These two patient groups were thought to require special handling that mental health clinicians were not trained to provide, in part because they were considered disruptive and inaccessible to successful treatment. This treatment arrangement in New Jersey seemed to be replicated in many other parts of the United States.

During the same decade, New Jersey's decision to maintain separate agencies overseeing treatment and prevention efforts for patients with alcoholism, patients with drug addiction, and patients with mental illness was replicated at the federal level. The National Center for the Prevention and Control of Alcoholism, which emerged from the National Institute of Mental Health during the late 1960s, was renamed the National Institute on Alcohol Abuse and Alcoholism (NIAAA) in 1971. The National Institute on Drug Abuse came into being shortly thereafter. Although all three agencies were tasked to support basic and applied research, including treatment research, they did so with separate staffs, generally undertook their own grant reviews, and vigorously competed with each other in Congress for funding. As an NIAAA grantee, I shared the continuing lament of NIAAA-funded researchers over the National Institute on Drug Abuse's more generous research budget. This budget discrepancy existed even though virtually everyone in the substance abuse field knew there were many more people in the United States with alcoholism than with drug addiction and that this majority cost the country much more than did the minority who struggled with drug abuse. However, domestic politics required the government to pay special attention to the problem of drug addiction because of the violent crime it was said to foster.

This institutional separation of state and federal funders of mental health, alcoholism, and drug abuse research, treatment, and prevention was repeated locally, including on university campuses. Since its founding, the Rutgers Center of Alcohol Studies, for example, had focused its faculty and staff research energies on the sociological and, later, psychological and biological parameters of the disorder. Despite a growing recognition during that early period that the three sets of disorders were commonly comorbid and might share some etiologic and treatment response features as well, the mission of the Rutgers Center remained focused on alcohol. Reflecting that commitment, the center's Journal of Alcohol Studies rarely published articles that did not largely or wholly report research on alcoholism and people with alcoholism. Today, things are very different—the journal has been renamed the Journal of Studies on Alcohol and Drugs, and center faculty and staff study a wide range of both substance-related and non-substance-related phenomena.

Much of what I had learned about the causes, addictive mechanisms, distinguishing diagnostic features, and treatment responses of people with alcoholism, people with drug addictions, and people with mental illness during those early years has since been replaced by findings from the extraordinary research advances during the past 40 years, most of it supported by the three federal institutes. A number of these advances, chronicled in this handbook, revealed the substantial similarities in the causes, properties, expressions, and responses to treatment of people with the addictive disorders, notably including
nonsubstance addictions such as pathological gambling. These findings are what led Howard J. Shaffer and his colleagues to develop the addiction syndrome model (e.g., as described in the Introduction to this handbook) so thoroughly examined in these volumes.

An important early impetus to research focusing on the variety of expressions of addiction, detailed by Widiger and Smith (Chapter 3 of this volume), was the publication of the third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM–III).* The instrument's operational criteria and empirical bases, both of them first-time features of the DSM, enabled substantially more reliable diagnoses. Reliable diagnosis, of course, is of crucial importance for studies comparing addiction-related diagnoses along a range of dimensions. DSM–III subsumed alcohol and drugs under a single diagnostic heading, another first for the instrument, reflecting commonalities that the substance disorders shared in behaviors associated with intoxication, withdrawal, tolerance, and dependence. DSM–III's text, as well as voluminous research before and after publication of the instrument, emphasized the substantial comorbidity of alcoholism and drug addiction (see Chapters 4 and 7 of this volume and Volume 2, Chapter 14, this handbook).

The successive development of more and more sensitive imaging procedures from the 1970s to the present initially enabled structural, then functional, studies of the living brain that revealed surprising similarities between alcohol and drug abusers in the effects of these substances on the brain as well as their impact on the brain's reward system (see Volume 2, Chapter 8, this handbook); findings that nonsubstance expressions of addiction demonstrate brain effects very similar to those associated with addictive substances have more recently been reported (see the Introduction and Chapter 15 of this volume). In related explorations, targeted neurobiological studies have revealed the essential role that neurotransmission anomalies play in substance-related and non–substance-related addictive disorders, as well as in many mental disorders (see Chapter 15 of this volume). Large-scale genetic studies of substance abusers, many apparently mediating the expression of neurobiological dysfunctions, confirm the central function of genes in the etiology and expression of addictive disorders, including nonsubstance addictions (see Chapter 15 of this volume).

Psychological research focusing on personality and temperament has also contributed to efforts to understand the role of these uniquely human traits in substance and nonsubstance addiction (Chapter 9 of this volume). In like fashion, research investigating how people with different expressions of addiction respond to treatment has revealed common pathways of change that transcend diagnostic categories (Chapter 5 of this volume). Trials of behavioral treatments developed for addictive disorders, both substance related and non–substance related, have convincingly indicated that some of these treatments are clearly of value for more patients than those with solely the anxiety and depression for which they were originally developed (Volume 2, Chapter 4, this handbook). Some of these treatments, developed or adapted for those who abuse alcohol—including motivational interviewing, cognitive–behavioral therapy, and relapse prevention—have turned out to be useful with people addicted to drugs and pathological gamblers as well (see the Introduction to this handbook).

Despite these very significant gains in understanding, which confirmed the broad reach of commonalities linking the substance-related disorders with each other as well as with the non–substance-related addiction disorders, the three federal categorical institutes remained entirely separate until very recently; at present, efforts to combine the institutes have begun.

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to accelerate. At the state level, the single state agency model has been the rule for many years, reflecting both state officials' recognition of similarities among the various expressions of addiction, especially in their response to treatment, as well as the need to effect cost savings by reducing duplicative administrative costs. At the local level, many providers of treatment for substance abuse, with limited access to or interest in research findings, continue to believe in the unique nature of the substance use disorder they treat, including its etiology and care.

For more than a decade, Shaffer has written and spoken of the need for his colleagues in addiction studies to recognize the similarities in etiology, expression, and treatment response that link the apparently disparate addictive disorders. Doing so, he has made clear, leads to new understandings of these matters. In the process of undertaking this effort, Shaffer has also developed and promulgated a new, more heuristic view of addiction. Although he has not been alone in his advocacy for these changes, Shaffer has been especially persistent—and quite eloquent—in making the case for the addiction syndrome model.

One of the cardinal traits of Shaffer's approach to science is epitomized in the following words from the Introduction to this handbook:

We asked each contributor to provide evidence to support or refute the addiction syndrome model. We hope that this approach stimulates an enthusiastic dialogue that can advance the field by revising and improving the etiological models that have in the past guided and currently guide the conventional wisdom about addiction and its causes and consequences. (p. li)

These two brief sentences epitomize Shaffer's characteristic openness to data regardless of whether they confirm his own beliefs.

My reading of the material in these volumes reinforces my belief in the worth of Shaffer's characterization of the addiction syndrome model. Even when aspects of the model do not fully accord with research findings, as a few of this handbook's chapters suggest, light is cast on important etiological and treatment issues. Such, of course, is the nature of the scientific enterprise, and such, I believe, is a principal reason why Shaffer, along with associate editors Debi A. LaPlante and Sarah E. Nelson, decided to undertake the arduous task of seeking, organizing, and editing this voluminous material.

I am grateful to Dr. Shaffer for proposing a theory and a model that have now generated a plethora of productive research. There is little question that research will lead to further advances in our long-continued effort to more fully understand the causes and characteristics of and effective treatments for addiction-related disorders.

Peter E. Nathan, PhD

The APA Addiction Syndrome Handbook is the fifth publication to be released in the American Psychological Association's latest reference line, the APA Handbooks in Psychology™ series. The series comprises multiple two- and three-volume sets focused on core subfields, and sets will be issued individually over the next several years. Some 20 are currently envisioned, with more than half already commissioned and in various stages of completion. Additionally, several handbooks on highly focused content areas within core subfields will be released in conjunction with the series, of which this two-volume set is the first.

Thus, the APA Handbooks in Psychology series now joins APA's three critically acclaimed, award-winning, and best-selling dictionaries—the APA Dictionary of Psychology (2006), the APA Concise Dictionary of Psychology (2008), and the APA College Dictionary of Psychology (2009)—as part of a growing suite of distinguished reference literature.

Each handbook set is formulated primarily to address the reference interests and needs of researchers, clinicians, and practitioners in psychology and allied behavioral fields. A secondary purpose is to meet the needs of professionals in pertinent complementary fields (i.e., by content area), be they corporate executives and human resources personnel; physicians, psychiatrists, and other health personnel; teachers and school administrators; cultural diversity and pastoral counselors; legal professionals; and so forth. Finally, the entire series is geared to graduate students in psychology who require well-organized, detailed supplementary texts, not only for “filling in” their own specialty areas but also for gaining sound familiarity with other established specialties and emerging trends across the breadth of psychology.

Under the direction of small and select editorial boards consisting of top scholars in the field, with chapters authored by both senior and rising researchers and practitioners, each reference set is committed to a steady focus on best science and best practice. Coverage focuses on what is currently known in the particular subject area (including basic historical reviews) and the identification of the most pertinent sources of information in both core and evolving literature. Volumes and chapters alike pinpoint practical issues; probe unresolved and controversial topics; and present future theoretical, research, and practice trends. The editors provide clear guidance to the “dialogue” among chapters, with internal cross-referencing, demonstrating a robust integration of topics that leads the user to a clearer understanding of the complex interrelationships within each field.

With the imprimatur of the largest scientific and professional organization representing psychology in the United States and the largest association of psychologists in the world, with content edited and authored by some of its most respected members, the APA
Handbooks in Psychology series will be the indispensable and authoritative reference resource to turn to for researchers, instructors, practitioners, and field leaders alike.

Gary R. VandenBos, PhD
APA Publisher
Handbook Preface

It is possible to trace the origins of the APA Addiction Syndrome Handbook to the middle 1970s, when I was working in the city of Boston's East Boston methadone maintenance treatment program. Methadone is a longer acting synthetic opioid that provides users with a pharmaceutically pure substitute for shorter acting opioids; methadone, like its shorter acting counterparts, can produce physical dependence. At the time, our treatment team was struggling with some of the common psychological complications associated with methadone detoxification. Patients would frequently begin detoxification and then, somewhere around a daily methadone dose of 20 mg to 30 mg (diluted in a drink of Tang), many would experience a renewed and overwhelming craving for opioids; they would stop the detoxification. As a result, the treatment team developed a yo-yo detoxification procedure that would confuse patients about their dose status; that is, on any given day their dose of methadone could be higher or lower than it was the day before. Over time, the protocol was to lower the dose. Unknown to the patients, even after they achieved withdrawal we kept them on daily doses of Tang for a period. Then, when the treatment plan indicated, I had the privilege of telling patients that they were drug free and no longer opioid dependent. For some patients, this straightforward event stimulated a sense of accomplishment, ambivalence, and trepidation about their future. Others, however, on hearing that they were no longer receiving methadone, immediately experienced opioid withdrawal, despite not having ingested any opioids for weeks and no longer being tolerant to opioids. It occurred to me that “drug effects” were not simply the result of drugs. This clinical experience suggested that popular assumptions about drugs of addiction (i.e., that the drug caused drug effects) might be inaccurate. Consequently, I began to search for opportunities to study and treat people who appeared to engage in addictive behavior patterns (e.g., intemperate gambling, shopping, running) in the absence of ingesting psychoactive drugs. This transition was the beginning of what would become a new conceptual approach to addiction. Drugs were not addictive; instead, addiction was the result of a meaningful relationship between the person with addiction and the object or objects of his or her addiction.

During the 1980s, the idea that drugs were not addictive was met with disinterest and considerable criticism. After all, everyone knew that drugs were addictive and activities were not. Over time, however, the evidence that inanimate objects are not inherently addictive grew. For instance, the epidemiology of substance use showed that most psychoactive drug users never developed addiction. As the scientific evidence was accumulating, my clinical experience with patients was teaching me that addiction emerged from a relationship with objects, whether chemical or activity. Over time, the field began conceptually and clinically
to distinguish among drug use, abuse, dependence, addiction, and impairment; this was an important development because these distinctions allowed the field to consider that one of these states does not necessarily involve the other. For example, use does not necessarily require abuse, and dependence does not necessarily equal addiction. Gradually, neurobiological and neuroimaging research began to reveal that the reward pathways of the brain react similarly to different pleasurable activities, whether or not these activities involve the use of psychoactive drugs. The meaningfulness, reliability, and robustness of the object of addiction to influence these pleasure-related neurocircuits interact to determine a person’s subjective state. Not all psychoactive states are desirable for all people. Consequently, it became increasingly apparent that addiction is not the passive and inevitable result of drug influences.

This Galileian (i.e., interactive) mode of thinking provided a much more complex view of addiction than the orthodox Aristotelian (i.e., main effects) perspective that dominated the conventional wisdom. For example, an Aristotelian view explains that the dependence-producing characteristics of heroin cause heroin addiction. More generally, the Aristotelian perspective considers that many different types of addiction exist and that each addiction results from the “addictive” properties of the object of addiction. Kurt Lewin, in his 1931 journal article “The Conflict Between Aristotelian and Galileian Modes of Thought in Contemporary Psychology,” first noted the limitations of this mode of thinking in psychology and encouraged a more interactive view. From Lewin’s topological and Galileian perspective, human behavior is always a function of the person in an environment (i.e., $B = f(P, E)$). For addiction, this Galileian perspective considers the relationship between the object (e.g., heroin) and the situation in which it is used.

Norman Zinberg, a mentor and colleague, was among the first Galileian thinkers to study addiction. He considered addiction from a macro rather than a molecular perspective. Norman recognized that the effects of drugs were not as simple as they first appeared. Using his interactive framework of drug, set, and setting, he taught me that drug effects are not simply the result of chemistry. He taught me to pay close attention to the social setting and how it modifies the typical drug purpose. For example, consider the use of beverage alcohol. Alcohol is a central nervous system depressant. However, in low doses and in certain social settings (e.g., cocktail parties), instead of suppressing the nervous system it suppresses only inhibitory processes; the result is that alcohol can act as a disinhibitory influence, a social lubricant. In other social settings (e.g., at home on the couch), the same low dose of beverage alcohol can serve as a sedative, producing a relaxed and sleepy result. The same person using the same dose of beverage alcohol can experience different effects in different settings. This phenomenon is true for other objects of addiction that do not involve the ingestion of psychoactive drugs. Consequently, as Norman taught me, the effects of drugs (e.g., marijuana) and activities (e.g., gambling) can vary widely as a function of the social setting and the meaningfulness of the setting to the user. The drug or activity, the set (i.e., expectations and psychological attributes of the user), and the social setting synergistically interact to produce the subjectively desirable effects that influence the development and maintenance of addiction. Undoubtedly, Norman’s teaching and perspectives influenced my early clinical observations—and these influences have endured to affect and guide my thinking.

Historically, there has been considerable confusion about the concept of addiction. There has been so much perplexity that past and current versions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) have excluded any mention of addiction and omitted *addiction* from the diagnostic nomenclature and taxa. Currently, an American Psychiatric Association working group for the fifth edition of the DSM has proposed to include it for the first time; however, there is also some discussion about continuing to exclude addiction from the DSM. Sometimes, resolving conceptual confusion requires stepping back from the problem at hand and taking a wider view of the issues. As discussed in the Introduction to this handbook, the discovery that many and apparently disparate opportunistic illnesses shared a common etiological pathway through an immune system compromised by HIV led to the discovery of AIDS. This syndromal view that many diverse signs and symptoms, not always present at the same time, can emerge from a common etiology seems to fit the variety of co-occurring problems that are associated with addiction.

As the Harvard Medical School’s Division on Addiction expanded from its early beginnings in the neurobiology department, it extended its scope during the early 2000s from medical school teaching and curriculum development to conducting research with a very comprehensive mission. This development allowed my colleagues and me to take a step back and consider the available addiction science in new ways; in turn, new perspectives enabled and encouraged us to organize, extend, and advance my early clinical observations into an evidence-based, testable model of addiction. Specifically, this collaborative work yielded a review paper, “*Toward a Syndrome Model of Addiction: Multiple Expressions, Common Etiology,*” that introduced the syndrome model of addiction. However, that work did not stop with the *Harvard Review of Psychiatry* article. Since then, my colleagues and I have been systematically examining the links and syntax that connect the fundamental constructs that make up the syndrome model of addiction. For example, we have focused on the psychiatric comorbidity that often precedes pathological gambling, studied the psychiatric comorbidity that repeat driving-under-the-influence offenders overwhelmingly evidence, and investigated the role of exposure and adaptation in the development of gambling addiction.

A project of this magnitude does not reach its goals easily or without widespread support. On behalf of my coeditors, I want to acknowledge the many diverse funding sources (federal, state, foundation) that supported the research that germinated the ideas that in turn led to the syndrome model of addiction and ultimately this handbook. For this support, we extend our thanks to the National Institutes of Health, the Iowa Department of Public Health, the Massachusetts Department of Public Health, and the National Center for Responsible Gambling. We also thank American Psychological Association staff Susan Reynolds, Ted Baroody, and Gary VandenBos for their encouragement and support. In particular, we thank Trish Knowles for her vigilant and diligent work administrating the many chapter revisions and communications between editors and chapter contributors. She completed these tasks with precision, style, and grace. We also extend thanks to our colleagues at the Division on Addiction and Harvard Medical School for their continuing support. The division was born at Harvard Medical School and the Department of Neurobiology. Here, under the guidance of Deans Daniel Tosteson and James Adelstein, the division began to develop into a research setting that provided the context for advancing new ideas and conducting research to test these ideas. We thank Steve Hyman, Joseph Coyle, and Gerald Fischbach for their support.

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during these growing years. We also extend our special thanks to our colleagues who provided us with the space, support, and freedom to work on these ideas and this volume. Thanks to Chrissy Thurmond, Tasha Chandler, Michael Stanton, Richard LaBrie, Rachel Kidman, Anthony Donato, John Renner Jr., Edward Khantzian, and Mark Albanese for stimulating our thinking, encouraging us to be better, and reining us in when we went too far.

Preparing books requires time—lots of time. For giving us the space to complete this project, we extend a very special thank you to our families. They have been patient and understanding.

Last but certainly not least, we extend our thanks and appreciation to each of the contributors for their careful, diligent, creative, fascinating, and insightful work. The end product is very special to us and, we hope, to the readers.

Howard J. Shaffer
Editor-in-Chief
Addiction is an extremely complex and common public health problem. During 2007, roughly 22.2 million people age 12 years or older (9% of the total population) qualified as having a substance use disorder (i.e., either dependence or abuse; Substance Abuse and Mental Health Services Administration, 2009). This number included people abusing alcohol, illicit drugs, or prescription drugs for nonprescription purposes. Among the commonly used psychoactive substances (i.e., those substances that change one's mood, thinking, or behavior), nicotine and alcohol account for most addiction problems. During 2006, 35.5 million Americans age 12 years or older (14.4% of the population) met the criteria for past 30 day nicotine dependence, and during 2008, 18.3 million (7.3% of the population) met the criteria for either alcohol abuse or dependence (Substance Abuse and Mental Health Services Administration, 2008b, 2009). In addition, about 2.3% of Americans have problems with excessive gambling (Kessler et al., 2008). These estimates only begin to reveal the extent of addiction; they ignore the countless people struggling with excessive sex, shopping, eating, and other intemperate behaviors.

Whatever the prevalence of addictive behaviors, one thing is clear: The problem is very costly. Among brain disorders, addiction incurs more economic costs than Alzheimer’s disease, stroke, Parkinson’s disease, or head and neck injury (Uhl & Grow, 2004). According to the National Institute on Drug Abuse (2008), the overall cost of substance abuse in the United States exceeds half a trillion dollars, including health-related expenses as well as losses in productivity. The costs associated with addiction are, in many cases, very difficult to define, and the science associated with such estimates is immature (Walker, 2003). Nevertheless, costs undoubtedly extend beyond financial factors and result from excessive behaviors as well as substance use disorders. Specifically, addiction harms individuals as well as their families, friends, and communities. Substance abuse (i.e., drug and alcohol abuse) can lead to the development of health problems, family disintegration, domestic violence, criminal behavior, and child abuse (Dunn et al., 2002; Khalsa, Treisman, McCance-Katz, & Tedaldi, 2008; Lundqvist, 2005; Mathers, Toumbourou, Catalano, Williams, & Patton, 2006; Perkins, 2002). Substance abuse is also among the most important influences contributing to risky sexual practices that can spread HIV and other diseases, lead to unplanned pregnancy, and raise the risk of violence (Ayoola, Brewer, & Nettleman, 2006; Santibanez et al., 2006; see Volume 2, Chapter 17, this handbook). Finally, gambling problems often similarly result in significant financial, social, and health distress for individuals and their families (Petry, 2005; Shaffer & Korn, 2002).
Long-term use of psychoactive substances can lead to serious health consequences (see Chapter 16 of this volume). For example, nearly 13,000 people die each year from alcohol-related liver disease alone (Heron et al., 2009). Stimulant drugs, such as amphetamines and cocaine, attack the heart instead of the liver, sometimes bringing on stroke or heart attacks (National Institute on Drug Abuse, 2009). Substance abuse can land people in the hospital for reasons other than long-term health consequences as well. Of the roughly 113 million recorded emergency room visits in the United States in 2006, more than 1.7 million were associated with substance use or misuse (Substance Abuse and Mental Health Services Administration, 2008a). That number includes people who attempted to commit suicide and people who sought help for their substance abuse; yet, in most cases, the substance use was not the primary reason for the visit. Many of the drug- and alcohol-related visits to the emergency room involve car accidents. According to the National Highway Traffic Safety Administration (2010), an alcohol-related car crash kills someone every 48 minutes. Just less than 18% of drivers killed in motor vehicle accidents test positive for drugs other than alcohol (Jones, Shinar, & Walsh, 2003), but it is worth noting that people generally use these illicit drugs in combination with alcohol. These numbers are, perhaps, not surprising, considering that more than 16% of drivers age 21 years and older report having driven under the influence of alcohol or drugs during the past year (Substance Abuse and Mental Health Services Administration, 2005).

This handbook addresses many of these wide-ranging consequences. For example, Chapters 15 through 17 of this volume cover the shared and unique consequences of addiction. In Chapter 15, Bühringer, Kräpelin, and Behrendt provide an overview of the universal consequences of addiction. In Chapter 16, Madras focuses on unique consequences of addiction. In Chapter 17, Black, Kuzma, and Shaw discuss the various consequences associated with behavioral addiction. In Volume 2 of this handbook, two chapters also address the consequences of addiction. In Chapter 14, Bickel et al. discuss the behavioral economics associated with addiction. In Chapter 15, Nelson and Tao provide an overview of driving under the influence, one of the most publicly harmful consequences of addiction. In Volume 2, Chapter 17, this handbook, Sorenson, Larios, and Manuel consider the relationship among HIV, AIDS, and addiction, including HIV as a potential consequence of some expressions of addiction.

Despite the prevalence and harmful consequences of addiction to substances and other—as they are referred to in this book—objects or expressions of addiction (e.g., gambling, shopping, running), the scientific understanding of addiction and its development remains imprecise and scattered. Shaffer, Hall, and Vander Bilt (2000) pointed out that although there are simple working definitions of addiction, the essence of addiction remains elusive to researchers, clinicians, and others.

Without a clear understanding of addiction, researchers will find it very difficult to reach consensus regarding addiction prevalence rates, etiology, and the necessary and sufficient causes that stimulate recovery (Shaffer, 1997a, 1999a). Also, without a precise understanding of addiction, clinicians will encounter diagnostic and treatment matching difficulties, and satisfactory treatment outcome measures will remain lacking. Without such an understanding, public policymakers will find it difficult to establish regulatory legislation, determine treatment need, establish health care systems, and promulgate new guidelines for health care reimbursement. To begin to remedy this situation, this handbook brings together some of the top minds in the field of addiction studies to discuss and debate the nature of addiction.
In this introduction, I first examine the idea of addiction and its features. Second, I consider past and contemporary models for identifying, defining, understanding, and treating addiction. I then conclude with a presentation of the syndrome model of addiction as an integrative conceptual model for the field of addiction studies and treatment and for this handbook.

**IDEAS ABOUT ADDICTION AND ITS FEATURES**

As Shaffer (1997a) noted,

> Addiction is a lay term, often used by scientists. Dependence is a more scientific construct, used occasionally by lay people. While there are many working definitions of addiction, the essence of the construct has remained elusive to nosologists. Consequently, addiction remains an imprecise lay concept and has not yet been welcome in diagnostic manuals such as DSM-IV or ICD-10. (p. 1445)

Although addiction is conceptually complex, it can be thought of as one thinks of mountains and seasons: You know them when you see them (Vaillant, 1982). Nevertheless, there are identifiable characteristics associated with addictive behavior, which has encouraged the development of professional best-practice guidelines to help clinicians and others identify and treat people struggling with different expressions of addiction. In this section, we review three key aspects of addiction. First, we review the spectrum of disorders often associated with addiction. Second, we discuss some primary defining characteristics of addiction. Third, we reflect on the identification and diagnosis of addiction.

**Distinguishing Use, Abuse, Dependence, and Addiction**

Many people fail to distinguish among the terms use, abuse, dependence, and addiction. By using these terms interchangeably, treatment providers and programs might confuse people who use psychoactive drugs or behaviors (e.g., gambling) with those who abuse these potential objects of addiction. In addition, clinicians and other stakeholders can mistake both of these groups with people who are drug or gambling dependent (i.e., people who experience neuroadaptation; see the next section). As we discuss later in this introduction, unnecessary hospitalizations, increased medical costs, and patients who learn to distrust health care providers too often result from this failure to distinguish these very different conceptual classes of object-user relationship. Consequently, without a precise definition of addiction, treatment providers risk failing to provide patients with the care they require.

**Dependence and abuse.** Although there are many professional systems for categorizing addiction-related behavior, clinicians rely on the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision; DSM–IV–TR; American Psychiatric Association, 2000). This manual is in the process of revision and some elements will be changing, but at present, the *DSM–IV–TR* considers substance use disorders within two diagnostic classes: abuse and dependence (e.g., alcohol or drug abuse and dependence; see Volume 2, Chapter 1, this handbook). The physiological symptoms associated with dependence include the emergence of neuroadaptation. *Neuroadaptation* is the technical term for the observation that (a) an increased dose of an object is needed to experience the same subjective effects as with a previously lower dose and (b) there is a stereotypical pattern of discomfort on stopping use. These elements of neuroadaptation are called *tolerance* and *withdrawal*, respectively.
Tolerance among heroin users, for example, refers to the observation that regular users require more heroin to get the same level of intoxication experienced previously at a lower dose; withdrawal means that these users get sick when they stop using the drug and that using the drug again can make this stereotypical pattern of illness stop. More important, although people have typically discussed neuroadaptation in terms of the brain's response to the ingestion of a psychoactive substance, in subsequent sections of this introduction we explain how neuroadaptation extends beyond such objects of addiction.

The DSM-IV-TR defines substance abuse—less serious than substance dependence—as "a maladaptive pattern of substance use manifested by recurrent and significant adverse consequences related to the repeated use of substances" (American Psychiatric Association, 2000, p. 198). In addition to these substance use disorders, the American Psychiatric Association (2000) has classified a variety of other substance-related and substance-induced disorders in the DSM-IV-TR as well as pathological gambling, which shares some features with substance dependence but is classified as an impulse disorder.

Dependence versus addiction. Not all drug-dependent patients evidence addictive behavior. Postoperative surgical patients who receive opioids as part of a pain management treatment plan gradually will show neuroadaptation as a consequence but will rarely seek these or other psychoactive drugs after their pain subsides. The absence of drug seeking among those who have evidenced neuroadaptation suggests that dependence and addiction are not synonymous. To illustrate, the social setting has some limited influence on the features of dependence. However, the social context of psychoactive drug use or experience-shifting behaviors can have large effects on the presence and nature of addiction. For example, Shaffer and Albanese (2004) described how a political struggle in Italy significantly limited the supply of cigarettes, consequently increasing their value. The social results of this value shift were both stunning and conceptually instructive: Sex workers traded their services for cigarettes, people were robbed of their cigarettes, people went to extraordinary lengths to acquire cigarettes, and a black market for cigarettes emerged (Reuters News Service, 1992, p. 50). This natural experiment revealed that the social context exerts considerable influence on the emergence of addictive behavior and its adverse consequences but does not influence the nature and extent of dependence, which is more a unique consequence of the stereotypical characteristics associated with drug use patterns (e.g., sedative dependence, opioid dependence; see Chapter 16 of this volume). This situation also revealed that the object of addiction is not sufficient to explain the experience of addiction and many of its adverse consequences.

Essential Characteristics of Addiction
Ego syntonic versus ego dystonic. Clinicians can distinguish addiction from obsessive-compulsive patterns of behavior by the quality of associated subjective states. People experience ego-syntonic behaviors as consistent with their sense of self and, particularly early in the development of addiction, do not experience their pattern of behavior as troublesome while engaging in it (Shaffer, 1997b). People experiencing ego-dystonic or ego-alien states feel as though something else is in control (e.g., obsessive compulsive or anxiety disorders). Despite not wanting to act on their urges, people with compulsive disorders act to relieve their mounting tension. The extent of ego-dystonic thoughts and distress that accompany these disorders distinguish them from addiction, for which subjective anguish is usually revealed only after the fact (Shaffer, 1994).
Addiction is a dynamic process. It is important to note that contrary to its implicit status as a static state, addiction is a dynamic process that waxes and wanes. People with addiction often go through phases of exacerbation and abstention; many also have episodes of controlled activity whereby they use the object of their addiction intermittently or more often but within manageable limits (see Chapters 4 and 5 of this volume). There is considerable evidence that people with addiction can recover with and without treatment (e.g., Klingemann & Sobell, 2007; Volume 2, Chapter 6, this handbook). Despite the connotation of an addict as someone who cannot change and is resistant to the influence of treatment, people with addiction do change and often respond positively to treatment experiences. The term addict is sufficiently pejorative that it is no longer acceptable in many journals (e.g., the Journal of Substance Abuse Treatment was one of the first publications to establish an editorial policy that avoids the use of the term addict). In this volume, we use the term addict only in its historical, not in its pejorative, sense.

Three chapters in this handbook address the dynamic nature of addiction. In Chapter 4 of this volume, Anthony provides a discussion of dynamic epidemiology and its relevance for addiction. In Chapter 5 of this volume, Prochaska focuses on the intrapersonal dynamics of addiction and the process of change. Finally, in Volume 2, Chapter 6, this handbook, Bischof, Rumpf, and John review what is known about the dynamic nature of addiction as it relates to natural recovery.

Objects of addiction. According to the Compact Edition of the Oxford English Dictionary (1971), the first use of the term addiction emerged during the 17th century (i.e., Roman law, c. 1625) in reference to a formal giving over or surrender of one individual to a master by sentence of the court. Shortly thereafter (i.e., 1641), the idea of being self-addicted to a habit or pursuit emerged; devotion was the essence of addiction. For example, these habits or pursuits included reading and involvement with agricultural activities, which suggests that contrary to conventional wisdom, the idea of addiction was originally derived from the excessive habits and pursuits of everyday life—not the ingestion of psychoactive substances. Not until the 18th century did the idea of addiction begin to refer to the state of being addicted to a drug and the compulsion or need to continue taking a drug as a result of taking it in the past.

The concept of addiction is currently returning to its origins. Although scientists historically have used addiction to refer to a physical dependence on alcohol and other drugs, brain scan technology has suggested that the brain is similarly rewarded by both substances and experiences. When reward is present, a risk of addiction exists. Scientists are increasingly recognizing the commonality between substance addiction and other compulsive behavior (Holden, 2001).

One way to define objects of addiction is by their ability to shift subjective experience reliably and robustly. Reliable shifters of subjective states are those activities that consistently change experience in a desired direction (e.g., activities that consistently make people feel good); robust shifters of subjective states are activities that are strong enough to ensure that experience will change, despite circumstances to the contrary. The most reliable and robust shifters hold the greatest potential to stimulate the development of addictive disorders. However, because the strength and consistency of these activities to shift subjective states vary across individuals, social settings, and dose, scientists have been unable to predict with accuracy which people will become addicted to what after experiencing a desirable shift in subjective state. Nevertheless, psychoactive drugs and certain other activities correlate highly with shifting subjective states because these activities reliably influence and change emotional experiences (e.g., Hyman,
Consequently, psychoactive drug use and certain other behaviors (e.g., gambling) tend to rank highly among the activities that can become expressions of addiction. As Albanese and Shaffer note in Chapter 1 of this volume, from substance abuse to shopping (Baker, 2000; Catalano & Sonenberg, 1993; Christenson et al., 1994), to eating carrots (Cerny & Cerny, 1992), to drinking water to intoxication (Pickering & Hogan, 1971; Rowntree, 1923), to committing antisocial or criminal acts (Hodge, McMurran, & Hollin, 1997), to using computers (Epript, Maureen, Stern, & Theiss, 1999; Mitchell, 2000; O'Reilly, 1996; Shaffer, 1996; Shaffer, Hall, & Vander Bilt, 2000; Starker, 1983), social observers apply the notion of addiction to many and varied human activities (Orford, 1985). For example, in Volume 2, Chapter 12, this handbook, LaPlante explores the relationship between technology and addiction; she considers the potential for certain forms of technology to serve both as objects of addiction and as devices that can help to prevent or reduce addiction. Scientists and clinicians have referred to nondrug addiction activities such as gambling, computer use, or shopping—activities that do not involve the ingestion of psychoactive substances—as process or behavioral addictions (e.g., Shaffer, 1996, 1997a, 1999a).

Neurobiological evidence about objects of addiction. Historically, researchers and others have attempted to consider the commonalities across substance and behavioral addictive behaviors (Levison, Gerstein, & Maloff, 1983). However, limited neurobiological evidence restricted the theoretical advance of these early efforts. The early evidence for commonalities derived primarily from the shared manifestations and experiences across apparently disparate expressions of addiction. The early views suggested that common features and shared experiences were associated with disparate expressions of addiction but did not identify a common cause. Some pioneers recognized the likelihood of common neurobiological pathways (Sunderwirth & Milkman, 1991). As great strides in neurobiology and the imaging of neurobiological processes emerged, a new evidence base, which we describe later, became available to support a consideration of the common etiology for the phenotypically similar characteristics typically associated with addiction—across behavioral and substance-involved expressions. Nevertheless, not all scientists accept the idea of behavioral expressions of addiction (e.g., Marks, 1990), partly because of the absence of an ingested substance (Holden, 2001). We review this issue and the evidence in more detail in the Syndrome Model of Addiction section later in this introduction. In Chapter 6 of this volume, Grant, Schreiber, and Harvanko review the neurogenetic evidence relevant to the commonalities and differences between behavioral and chemical expressions of addiction.

Relationship between addiction and objects of addiction. The most common conceptual error committed by clinicians, researchers, and public policymakers is to think that addiction resides as a latent property of an object (e.g., a drug or game of chance). Conventional wisdom, for example, refers to “addictive drugs” or “addictive gambling.” However, addiction is not the product of a substance, game, or technology, although each of these things has the capacity to influence human experience. To illustrate, if psychoactive drug using was a necessary and sufficient cause for addiction, then addiction would occur every time this pattern of drug using was present, and addictive behaviors would be absent every time drug using was absent. However, tolerance, withdrawal, and adverse consequences of excessive behaviors are often present when drug using is absent. For example, on stopping, pathological gamblers who do not use alcohol or other psychoactive drugs can experience physical symptoms that appear to be very similar to opioid, stimulant, or polysubstance withdrawal (Potenza, 2001; Wray & Dickerson, 1981). In addition, people often exceed their drug-using
limits and lose a modicum of self-control without experiencing addiction. Therefore, drug using is neither a necessary nor a sufficient cause of addiction. This observation provides insight into the necessity of considering a more complex relationship between a person who might develop addiction and the object of that person's desire.

When a particular pattern of behavior can reliably and robustly change emotional experience in a desirable way, the potential for addiction emerges. The relationship of the addicted person with the object of that person's excessive behavior—not just the attributes of the object—is what defines addiction. Consequently, the causes of addiction are multifactorial (Zinberg, 1984). The confluence of psychological, social, and biological forces is what determines addiction. No single set of factors can define addiction precisely. Unfortunately, the concept of relationship is also difficult to define. Therefore, until experience provides more insight into the synergistic nature of these factors, clinicians, scientists, and policymakers alike are forced to operationally define addiction in relatively narrow ways so that they can share a common perspective (Shaffer, 1992, 1999a; Shaffer & Robbins, 1991, 1995). Nevertheless, these definitions often compete and can lead to conceptual confusion.

Identifying Addiction and Establishing a Gold Standard

Attempts to define addiction. Contemporary addiction workers have come to think of addictive behavior as having three primary components: (a) some element of craving or compulsion, (b) some level of losing control over intended behavior, and (c) continuing the behavior in question despite experiencing associated adverse consequences. Although these dimensions provide a useful map for understanding the elements of addiction, one must remember that the map is not the territory (Shaffer & Robbins, 1991), and a diagnosis is not the disease (Szasz, 1991). This means that diagnostic guides do not necessarily provide a valid representation of the underlying construct that the nosology targets. To illustrate, DSM-IV-TR provides a multidimensional map for diagnosing mental disorders; it improves the reliability of making a diagnosis at the considerable expense of validity (Barron, 1998; Vaillant, 1984). That is, contemporary diagnostic schemas help ensure that different clinicians, or the same clinician at different times, will make consistent diagnostic decisions. However, despite this intra- and interclinician reliability, what disorder they are diagnosing remains uncertain. As Alan Leshner (1999), former director of the National Institute on Drug Abuse, once asked, “When is addiction addiction?” This question has not been resolved and cuts to the heart of the matter: When clinicians and scientists identify a behavior pattern as an expression of addiction, even if they can identify it reliably, how do they know that it is indeed an expression of addiction? To accomplish such a diagnosis, clinicians must be able to distinguish overwhelming impulses to act from an unwillingness to resist these impulses (e.g., Davies, 1996); similarly, they must distinguish the effects of co-occurring disorders (e.g., obsessive–compulsive disorder, depression, anxiety) that can mimic addiction (see Volume 2, Chapter 1, this handbook). Two handbook chapters are devoted to the issues associated with identifying and diagnosing addiction. In Chapter 3 of this volume, Widiger and Smith review the nosology of addiction. Later, in Volume 2, Chapter 1, this handbook, Langenbucher discusses the assessment and diagnosis of addiction.

Behavioral excess as evidence for addiction: Avoiding tautologies. As diagnostic manuals evolved and improved the reliability of efforts to classify mental disorders, the refined criteria associated with the signs and symptoms of drug use and gambling disorders increasingly evidenced tautology. Available manuals and other diagnostic systems assess the presence
of different expressions of addiction using a circular and tautological process—effectively describing the adversities that accumulate from repetitive behaviors by labeling the pattern as a specific expression of addiction, that is, describing the same thing in different terms. For example, as we mentioned before, addiction is commonly understood as a pattern of behavior over which people have little or no control; however, the available assessment methods do not allow clinicians and others to determine the extent of control that is available to people. Instead of using a measure of control—or other influential moderators and mediators—clinicians identify addiction by its consequences. To rectify this problem, they need etiological models of addiction that permit them to predict the development of the disorder absent its adverse consequences. For example, the risk factors for cardiovascular disease are often identifiable before an adverse cardiovascular event. In Chapter 14 of this volume, Tarter, Horner, and Ridenour review the developmental timing of addiction and offer an epigenetic model of substance use disorder liability that does not rely on addiction-related consequences to determine risk. Standards, independent of addiction consequences, are necessary to avoid the current tautology associated with addiction. These independent standards are known as gold standards.

Toward a gold standard. To avoid tautological explanations, diagnosticians use an independent gold standard as a definitive index against which they can judge a patient’s condition. For example, in medicine, a test that evidences 100% sensitivity and 100% specificity is a gold standard: This test would identify everyone with the disorder and never falsely identify someone as having the problem. Absent these “perfect” psychometric characteristics, diagnosticians use proxy measures that approximate these standards. A physician might examine a patient with gastrointestinal discomfort and an abdominal bulge, diagnosing this as a hernia; magnetic resonance imaging (MRI) serves as the independent gold standard to confirm the diagnosis. Similarly, the independent gold standard for the diagnosis of AIDS is a blood test that determines the extent of T-cells and the presence of HIV; for diabetes, a blood test determines the blood glucose level. These tests do not require the presence of symptoms (e.g., thrush, carcinomas, coma) that emerge as a consequence of AIDS or diabetes.

For addiction to become a meaningful scientific construct, absent tautological features, to explain and guide the treatment of psychoactive drug use or intemperate patterns of behavior (e.g., pathological gambling, excessive shopping, overindulgent sex), investigators need to establish a gold standard against which clinicians can judge the presence or absence of disorder. This gold standard must be independent of the disorder being judged. At present, there is no independent gold standard for judging the presence or absence of addiction. Even the existence of neuroadaptation—tolerance and withdrawal—does not represent a gold standard against which clinicians can judge the presence or absence of addiction. As mentioned previously, neuroadaptation can occur in the absence of addiction. Absent a gold standard, addiction will continue to suffer from the “myth of mental illness” stigma (e.g., Szasz, 1987, 1991). “The psychiatric community seems determined to ground its medical legitimacy on principles that confuse diagnoses with diseases” (Szasz, 1991, p. 1574). Without a clear standard and because of tautological explanations, many observers are critical of the construct of addiction, often blaming people with addiction for their difficulties and viewing the problem as a matter of willful misconduct.

If addiction represents a distinct disorder with unique features and course, then clinicians must be able to independently validate the disorder and its impaired regulatory mechanisms.
Clinicians must avoid defining addiction tautologically by its adverse consequences. As Albanese and Shaffer (Chapter 1 of this volume) note, if addiction is a primary disorder, independent of its consequences, then clinicians and scientists should be able to identify the disorder without knowing its consequences. Despite this scientific necessity, clinical significance and human suffering require that clinicians attend to addictive behavior patterns whether or not they can be distinguished with precision.

Most likely, an independent gold standard will derive from neurogenetic (e.g., biomarkers), biobehavioral (e.g., neurotransmitter challenge), implicit and explicit behavioral attributes, or some combination of these sources (see Chapters 3 and 14 of this volume). Early neuroscience research is encouraging. For example, research has suggested that pathological gamblers have altered dopaminergic and serotonergic functions (e.g., Bergh, Sodersten, & Nordin, 1997; DeCaria, Begaz, & Hollander, 1998). As Bühringer, Kräplin, and Behrendt show in Chapter 15 of this volume, focusing on the universal characteristics of addiction, researchers have also identified biogenetic vulnerabilities among pathological gamblers (e.g., Comings, 1998), and evidence has suggested that there might be genetic markers for novelty-seeking behavior that can predispose people to take chances (Benjamin et al., 1996; Ebstein et al., 1996). In addition, as we and other contributors (see Chapters 1, 6, 15, and 16 of this volume) describe in more detail elsewhere, new evidence has suggested that common reward circuits in the central nervous system are responsible for the experiences associated with the anticipation of substance use effects, the acquisition of money, and the appreciation of beauty (Aharon et al., 2001; Breiter, 1999; Breiter, Aharon, Kahneman, Dale, & Shizgal, 2001). Finally, with respect to implicit behavioral attributes, research is mounting that suggests that addictive behavior is associated with nonverbal communication deficits (e.g., Bari-beau, Braun, & Dube, 1986; Borrill, Rosen, & Summerfield, 1987; Kornreich et al., 2001; Marinkovic et al., 2009). The benefit of such independent standards is that they can avoid the issues associated with tautological diagnostic processes.

Evolution of Models of Addiction

Stakeholders approach the idea of addiction with a set of assumptions that often remains implicit. These ideologies provide a framework for how they understand and approach addiction-related problems. It is essential that clinicians, public policymakers, and others be aware of these ideologies. Ideologies often represent tacit paradigms. As with scientific paradigms in general, these perspectives focus attention on various addiction features; simultaneously, these views serve as blinders that might prevent one from acknowledging other characteristics of addiction that are essential to attempts to understand, prevent, or treat addiction (e.g., Gambino & Shaffer, 1979; Kuhn, 1970). Two chapters in this handbook trace the development of conceptual approaches to addiction through history. In Chapter 1 of this volume, Albanese and Shaffer describe the origins of addiction and its evolution into the contemporary concept commonly held today. In Chapter 2 of this volume, Freed traces the history of addiction from a sociological perspective.

To our knowledge, nobody has articulated a unifying principle that explains all of addiction. Instead, there are a variety of different clinical, scientific, and lay viewpoints on addiction and addictive behavior patterns, readily grouped into biological, psychological, and sociological perspectives. These perspectives can take the form of informal but influential formulations about the nature of temerity; alternatively, they can represent a well-developed and reasoned theory of excessive behaviors. Most models that clinicians use to guide their treatment efforts reside somewhere between these extremes. Despite the fact that
the majority of models lie in between these ends of a continuum of views, the perspectives are quite varied; a brief review of better known models reveals considerable breadth and diversity of ideas and implications. For example, Shaffer and Burglass (1981) summarized the evolution of classic models of addiction. These theoretical approaches represent common perspectives that have been offered to explain many expressions of addiction; historically, these perspectives can be loosely organized into moral, biological, psychological, and social models. For example, the earliest models of addiction explained excessive behavior patterns as a moral turpitude that required values conversion and piety (e.g., Quinn, 1891; Weems, 1812). Gradually, disease or illness models of addiction emerged (Alcoholics Anonymous, 2009; Gamblers Anonymous, 2006; Narcotics Anonymous, 2009). These perspectives considered excessive behavior as a chronic, fundamental disorder for which there is no cure other than abstinence. The basic biological models of addiction evolved to include more complex components. For example, addiction was characterized as an impulse control problem (e.g., American Psychiatric Association, 1994; Bergh et al., 1997; Christenson et al., 1994; Comings et al., 1999; France, 1913; Hollander, Buchalter, & DeCaria, 2000; Marks, 1990). This perspective hypothesized that deficiencies or changes in neurobiological or genetic structures or processes influence regulatory problems that result in impulsive behavior patterns. Gradually, a more sophisticated biological view of addiction postulated that genetic vulnerability moderated impulsive and intemperate patterns of behavior (Comings, 1998; Comings et al., 1999; Crabbe, 2002). With the advent of neuroimaging technology, even more reductionistic and complex biological models emerged. These models recognized the neurocircuitry of reward and pleasure as deficient, thereby making some people more vulnerable to the psychoactive effects of drugs and experience of shifting activities (e.g., Aharon et al., 2001; Bergh et al., 1997; Breiter et al., 2001; Ebstein et al., 1996; Wise, 1996). This perspective postulates that addiction results from shifts in the reward system and reflects dynamic changes in the pattern of activity among neurotransmitters or a deficit in the capacity of this system to yield pleasurable experiences.

In addition to the development of neurobiological models, there were many advancing theoretical ideas about the psychological factors that influence the development and maintenance of addiction. For example, social learning and reinforcement contingencies exert influence over people to promote excessive patterns of drinking, drugging, or gambling (e.g., Seager, 1970; Skinner, 1969). These reinforcement contingencies vary by culture and social setting. Bad judgment and erroneous thought patterns also play a role in the development and maintenance of addiction in general and gambling in particular (e.g., Ladouceur, Paquet, & Dube, 1996; Ladouceur & Walker, 1998; Rosecrance, 1983, 1988). This psychological view considered that various expressions of addiction, gambling in particular, represent poor decision-making strategies, usually displayed by naive gamblers who do not fully understand the games they play. That is, players view gambling outcomes and their capacity to influence the outcomes as a product of illogical cognition concerning laws of probability. Psychodynamic models of addiction proposed that psychological deficiency led to patterns of self-medication (e.g., Jacobs, 1989; Khantzian, 1975, 1997; Radó, 1933) that served as an anodyne for these circumstances. From this perspective, personality and emotional vulnerabilities invite addiction as an adaptive response. Under some conditions, these excessive behavior patterns serve to keep people from regressing to a more primitive state. In short, addiction represents an attempt to manage uncomfortable psychological states. This view is similar to psychological models that postulate an underlying psychodynamic neuroticism. That is, addiction results from intrapsychic conflicts that take root during earlier
developmental stages; these problems and the adaptations that follow sustain the excessive pattern of behavior (e.g., Dodes, 2002, 2011; Lindner, 1950).

Important sociological models offer explanations for the development of addiction. These perspectives hypothesize that moral and sociocultural factors influence the incidence and growth of excessive patterns of behavior; Bennett and Golub (Chapter 10 of this volume) provide a comprehensive review of the sociological influences that affect addiction. Eventually, interactive models of addiction began to emerge and dominate the field. Psychosocial (Orford, 2001; Zinberg, 1984) and biopsychosocial models became commonplace. Finally, public health models (Korn & Shaffer, 1999; Marlatt, 1996; Marlatt, Baer, Donovan, & Kivlahan, 1988) provided stakeholders with the opportunity to step back and consider the population issues associated with addiction. A public health viewpoint acknowledges that addiction is a multidimensional health concern for which potential biological, psychological, economic, and social costs must be considered.

Addiction develops in a social context and likely has benefits as well as costs for the sufferer and the social setting.

As our previous examples have shown, in the absence of a gold standard, the panoply of theoretical perspectives can influence clinicians to apply various and perhaps substantially different addiction treatment strategies. In the discussion that follows, we offer a syndrome model of addiction. This model provides a broad umbrella under which many different micromodels and perspectives can be integrated and evaluated.

**SYNDROME MODEL OF ADDICTION: MULTIPLE EXPRESSIONS, COMMON ETIOLOGY**

The important thing in science is not so much to obtain new facts as to discover new ways of thinking about them.

—William Bragg

New lines of scientific research are encouraging a reconsideration of addiction. Specifically, this research has suggested that addictive disorders are not a collection of unique addictions at all but rather are indicators of a more complex pattern of activity than initially thought. Reassessing the concept of addiction has profound implications for science, health care, and public policy. Reconsidering addiction as a syndrome holds the potential to inform existing understandings of specific expressions of addiction. For example, research on behavioral disorders, such as excessive gambling, can advance the understanding of substance use disorders because such problems can be studied without the confounding influence of intoxicants.

In this section of the introduction, we integrate evidence from a variety of sources to provide a macro model of addiction: This model views addiction as a syndrome with multiple opportunistic expressions. The various expressions of an addiction syndrome rest on multiple and interacting biological, psychological, and experiential elements (i.e., basic factors) and emerge as behavioral and substance-related patterns of abuse and dependence. Before we present the addiction syndrome in more detail, it is important that we describe syndromes in general and the diagnostic challenges that syndromes can present.

**On Syndromes**

At minimum, a syndrome is a cluster of signs and symptoms related to an abnormal condition. Kendell (1989) advanced this definition by proposing that two essential elements
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comprise a clinical syndrome: (a) a cluster of related symptoms and (b) a distinctive temporal progression. In addition, syndromes have unique and shared components: Unique components identify the presence of a syndrome and the specific expression of that syndrome; shared components overlap between expressions of the syndrome and with other disorders. Shared components often account for observations of comorbidity between apparently different disorders. Finally, not all symptoms, signs, or disorders that make up a syndrome need be present in every expression of the syndrome. This circumstance can make diagnosis very difficult.

The history of the identification of AIDS provides a useful illustration. During the early 1980s, health researchers noticed a surge in a variety of what had previously been rare or low-prevalence diseases. Gradually, they were able to link these cases to a shared factor: immunosuppression. This revelation brought about the formal recognition of AIDS but not identification of the root cause. Ultimately, researchers identified the cause as HIV. When HIV infection sets the stage, an assortment of diseases gain opportunity and can become the proximate cause of mortality. The importance of understanding the role of opportunistic diseases in the consequences of syndromes cannot be overestimated. Scientists recognize that although each opportunistic disease is important to consider and even treat independently, without considering the underlying syndrome researchers might never have developed vital and effective treatments for AIDS.

If scientists and doctors had failed to connect the presence of rare opportunistic diseases associated with AIDS with the presence of HIV, the underlying disorder would likely have remained untreated. For example, imagine that scientists and doctors continued to work on each of these opportunistic diseases diligently and carefully but never actually understood the common root that made it possible for this array of diseases to emerge and thrive. The prevention programs and medication cocktails that treat HIV would not be available to extend the lives of many people who have HIV infection.

Addiction researchers and treatment providers seem to be working in a disconnected investigative and clinical environment that is similar to the early days of AIDS research and treatment. The conventional wisdom among addiction researchers and treatment providers has been that people develop various and distinct addictions independently and that commonalities among addictions are likely spurious or a consequence of the addiction experience. These researchers and practitioners might be so focused on the individual trees that they fail to see the larger forest. Returning to AIDS to illustrate this point, opportunistic infections can present dramatically differently yet share the same etiology (i.e., HIV infection). Similarly, the expressions of an addiction syndrome could outwardly vary considerably (e.g., disordered gambling, alcohol dependence) yet derive from common etiology. In the absence of an explicit gold standard, such as the presence of a virus or the number of T-cells, investigators must identify the existence of syndromes indirectly. Identifying an addiction syndrome requires recognizing the presence of premorbid, comorbid, and postmorbid commonalities, or phenomena shared by seemingly disparate disorders, across both chemical and behavioral expressions of addiction.

In the discussion that follows, we review the empirical evidence for an addiction syndrome and organize this material into three primary areas: (a) shared neurobiological antecedents, (b) shared psychosocial antecedents, and (c) shared experiences (e.g., manifestations and sequelae). This handbook offers the syndrome model of addiction and the various contributors’ considerations of this model based on research from these interactive domains. Instead of viewing addiction as a collection of discrete disorders, the addiction syndrome
model views addiction as a syndrome with multiple opportunistic expressions (e.g., substance use disorders, pathological gambling) resulting from multidimensional susceptibility. Moreover, addiction to any particular object is due, in large part, to exposure, access, and the capacity to produce a predictable and desirable subjective shift in mental state.

Evidence for an Addiction Syndrome: Shared Neurobiological Antecedents

Neurobiological system nonspecificity: Common pathways of addiction. To stimulate and sustain repeated involvement with various objects of addiction, these objects or activities must energize the brain’s reward system. Originally, the reward system likely existed to ensure the propagation of the species by implicitly teaching people that sexual intercourse was pleasurable and they should repeat it. As the context of human behavior shifted and society matured, the same reward system eventually became associated with other pleasurable experiences (e.g., using psychoactive drugs, gambling, shopping).

The reward system of the brain includes the ventral tegmental area, located in the anterior ventral midbrain, and the nucleus accumbens, located in the ventral forebrain. The dopaminergic neurons that have their cell bodies in the ventral tegmental area and terminals in the nucleus accumbens are part of the brain’s mesolimbic dopamine system. Dopamine is one class of neurotransmitters that scientists have implicated in the development and maintenance of addictive behaviors. Substances such as alcohol, cocaine, and opiates activate the brain’s reward system. As Wise (1996) stated, “The drugs known to synergize with brain stimulation reward . . . are, for the most part, drugs of abuse” (p. 327). Furthermore, both psychoactive drugs (e.g., alcohol, cocaine, heroin) and behaviors (e.g., gambling, shopping, eating) have the capacity to stimulate neurobiological systems in general and the brain’s dopamine reward system in particular (Betz, Mihalic, Pinto, & Raffa, 2000; Burger & Stice, 2011; Daigle, Clark, & Landry, 1988; Frascella, Potenza, Brown, & Childress, 2010; Gearhardt et al., 2011; Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009; Wise, 1996). Functional neuroimaging studies, which display images of a living, functioning brain responding to a task, have revealed that anticipation of cocaine, money, and beauty energize the reward system (e.g., Aharon et al., 2001; Breiter et al., 2001). New research has revealed that food, like psychoactive drugs, can also energize the neurocircuitry of reward (Burger & Stice, 2011; Gearhardt et al., 2011; Stice, Yokum, Burger, Epstein, & Small, 2011). The observation of disparate objects stimulating similar neurobiological pathways indicates that, regardless of the object of addiction, the brain is the final common pathway for addictive behaviors. When people do not experience the subjective pleasure that is associated with the stimulation of the brain’s reward system, they typically stop engaging in these behaviors. It is interesting that although opiates enhance the reward system, opiate antagonists, such as naloxone and naltrexone, diminish the system’s operation.

Blum et al. (2000) implicated a reward deficiency syndrome as a vulnerability to addiction, whether the expression of addiction is substance or behavior related. This model posits that addiction emerges, in part, as a result of hypofunction of the dopamine system described in the last paragraph. People with such dysfunction fail to experience sufficient pleasure; they adapt to this circumstance by engaging in behaviors that stimulate the faulty reward circuitry. Moreover, epidemiological, genetic, and neurobiological evidence support the notion that vulnerability to addiction (as well as impulsive and compulsive behaviors) is genetically transmitted. It is not necessary to establish that all addiction is caused by genetic vulnerability. Heavy exposure to alcohol and other drugs may set in
motion perturbators of neurochemistry and receptors which may have similar end results. (Blum et al., 2000, p. 2)

It is vital to note that the dopamine system does not function in isolation. A complex neurobiological context influences the effects of dopamine action. There are a variety of brain receptor systems, and the assortment of abusable substances can also affect the other transmitter systems (e.g., norepinephrine, GABA, glutamate, serotonin) that in turn interact with the dopamine reward system. Ingested psychoactive substances—and human activities in general—exert their influence on subjective experience through the brain's many endogenous neurotransmitter systems. For example, opiates function through the endogenous opioid system and alcohol through the GABA system. These neurotransmitter systems, as well as others, impinge on and modulate the dopamine reward system. In addition, although neurobiological reward activity represents the most well-known evidence that supports an addiction syndrome, other systems deserve consideration. As Breiter and Gasic (2004) reminded us, the observations of the dopamine reward system should not minimize the potential contribution of learning and memory in the hippocampus and emotional regulation in the amygdala in the development and maintenance of addiction. Overall, the observation that disparate objects and behaviors can stimulate similar neurobiological pathways (e.g., Potenza, 2001; Potenza, Sofuoglu, Carroll, & Rounsaville, 2011) suggests that, regardless of the object of addiction, the neurobiological circuitry of the central nervous system is the ultimate common pathway for addictive behaviors.

Genetic overlap. There is evidence suggesting substantial genetic and environmental nonspecificity across addictive behaviors (Betz et al., 2000; Johnson, 2003; Nestler, Barrot, & Self, 2001). For example, genetic studies have revealed common molecular mechanisms for drug addiction and compulsive running behavior (Werme, Lindholm, Thorén, Franck, & Brené, 2002; Werme, Thorén, Olson, & Brené, 2000). Pathological gambling similarly shares a common genetic vulnerability with alcohol dependence (Slutske et al., 2000). A study of male twins showed that shared genetic and environmental risk factors for psychoactive substance abuse are largely substance nonspecific (Kendler, Jacobson, Prescott, & Neale, 2003). Kendler, Jacobson, et al. (2003) noted, "We could not find evidence for genetic factors that increase risk for individuals to abuse substance A and not also to abuse substances B, C, and D" (p. 692).

Other evidence has also supported nonspecific genetic vulnerability to addictive behavior. For example, Merikangas, Stolar, et al. (1998) found that similar direct (e.g., exposure to drugs) and indirect (e.g., resultant family discord) factors augment genetic risk for both drug and alcohol abuse. In their study of female twins, Karkowski, Prescott, and Kendler (2000) found (a) genetic and environmental factors significantly influenced substance use in general and (b) no evidence of a heritability or familial environmental effect for specific substances. Similar results were found in a study of Vietnam-era drug users: With the exception of heroin—which exhibited unique substance-specific genetic risk—investigators observed a common vulnerability to multiclass drug use among study participants (Tsuang et al., 1998). Finally, Bierut et al. (1998) observed that "although studies support the familial transmission of alcohol and substance dependence, individuals are frequently dependent on multiple substances, raising the possibility of a general addictive tendency" (p. 987). These findings provide evidence that the genetic link to addiction does not account for vulnerability to specific objects of addiction; rather, genetics account for a general and increased risk for addiction.
Evidence for an Addiction Syndrome: Shared Psychosocial Antecedents

Among the participants in the National Comorbidity Survey Replication study with a past-year diagnosis, 55% carried only a single diagnosis, 22% evidenced comorbid mental disorders, and 23% reported experiences that reflected three or more mental disorders (Kessler, Chiu, Demler, & Walters, 2005). The prevalence of psychopathology is increased among individuals who are dependent on multiple psychoactive substances (e.g., heroin, alcohol, cocaine; Conway, Kane, Ball, Poling, & Rounsaville, 2003), which is perhaps another indication of a shared vulnerability. Many substance abuse treatment seekers (e.g., those in treatment for opioid dependence or for driving under the influence of alcohol) have increased rates of anxiety and depressive disorders (Lapham, Baca, McMillan, & Lapidus, 2006; Shaffer et al., 2007; Silk & Shaffer, 1996). Likewise, populations with psychopathology (e.g., major depression, generalized anxiety disorder, posttraumatic stress disorder) often exhibit increased prevalence of drug use disorders (Merikangas, Mehta, et al., 1998; Regier et al., 1990). New multinational research has suggested that earlier life disorders are associated with the onset of similar kinds of disorders at some point later in life; two higher order variables (i.e., internalizing and externalizing dimensions) are common to both of these associated antecedent and consequent disorders (Kessler et al., 2011). Consequently, antecedent problems tend to endure and relate to later or consequent difficulties. Similarly, childhood adversities (e.g., parental death, divorce, substance abuse, mental illness, experience of sexual abuse) are associated with substance use and other mental disorders during adulthood, although this association is general rather than disorder specific (Kessler et al., 2010). Finally, several studies have shown that comorbid psychiatric conditions typically precede alcohol abuse, cocaine use, and gambling problems (Kessler et al., 1996, 2008; Nelson, Heath, & Kessler, 1998; Shaffer & Eber, 2002). To determine whether other behaviorally expressed addictions follow these patterns observed for substance disorders and gambling, more research is needed. In Chapter 7 of this volume, Tsuang, Genderson, Zink, and Lyons provide a comprehensive review of psychiatric comorbidity among people with addiction.

Subclinical and social risk factors (e.g., impulsivity, poor parental supervision, delinquency) are also common across chemical and behavioral expressions of addiction (Brener & Collins, 1998; Welte, Barnes, & Hoffman, 2004; Whalen, Jamner, Henker, & Delfino, 2001). Furthermore, research has shown that individuals who engage in one problem behavior are also likely to engage in others (Caetano, Schafer, & Cunradi, 2001; Shaffer & Hall, 2002; Vitaro, Brendgen, Ladouceur, & Tremblay, 2001). For example, in Volume 2, Chapter 16, this handbook, Derringer and Krueger discuss the relationship between addiction and a larger problem behavior syndrome. Various sociodemographic risk factors (e.g., poverty, geography, family and peer group) can influence the onset and course of drug use and other behavioral activities (e.g., gambling) that increase the risk of addiction (Christiansen, Vik, & Jarchow, 2002; Evans & Kantrowitz, 2002; Gambino, Fitzgerald, Shaffer, Remer, & Court- nage, 1993; Lopes, 1987; Robins, 1993; Shaffer, Vander Bilt, & Hall, 1999; Wechsler, Davenport, Dowdall, Grossman, & Zanakos, 1997). Volume 1, Part II, of this handbook includes multiple chapters devoted to these psychosocial risks that can influence the development of addiction. In Chapter 8 of this volume, Alquist and Baumeister review the research focusing on self-regulation as a key factor in the development and maintenance of addiction. In Chapter 9 of this volume, Zuckerman provides a thorough review of the relationship between personality factors and addiction, and in Chapter 12 of this volume, Winters et al. discuss social
Influences that can affect the development of addiction. Two chapters in Volume 2, Part III, this handbook, also address the influence of sociodemographic factors on addiction. In Volume 2, Chapter 13, this handbook, Alegria, Valentine, Li, and Min discuss the development of addiction among vulnerable populations, and in Volume 2, Chapter 18, this handbook, Stein, Grella, Conner, and Gelberg review the evidence for addiction among a particularly vulnerable population, those who are homeless.

Evidence for an Addiction Syndrome: Shared Experiences

Shared manifestations and sequelae. Different expressions of addiction share various manifestations and sequelae. Accordingly, Zinberg (1984) suggested that "the experience of addiction diminishes personality differences and makes all compulsive users seem very much alike" (p. 111). In addition to reducing preexisting personality differences, various and distinct expressions of addiction also stimulate similar biopsychosocial sequelae. Several studies have supported this notion. Psychosocially, people who engage in substance abuse, pathological gambling, or excessive shopping commonly have recognizable sequelae (Christenson et al., 1994; Shaffer & Hall, 2002; Zinberg, 1984). Chemical and behavioral expressions of addiction also have similar neurobiological consequences, as discussed earlier, including the emergence of neuroadaptation (e.g., tolerance and withdrawal; Frascella et al., 2010; Goodman, 2008; Wray & Dickerson, 1981). Neurocognitive manifestations between behavioral and chemical expressions of addiction are also similar. For example, individuals with alcohol dependence and disordered gambling shared increased impulsivity and risky decision making compared with healthy age- and education-matched control participants; both of these deficits are associated with ventral prefrontal cortical dysfunction (Lawrence et al., 2009). In addition to the shared features of gambling and alcohol disorders, there were unique features: The participants with alcohol dependence evidenced impaired working memory and decision-making deliberation times compared with both those with disordered gambling and the healthy control participants (Lawrence et al., 2009).

Parallel natural histories. There is a natural history to the course of addiction that begins with risk factors and always includes exposure to potential objects of addiction (Slutske, Jackson, & Sher, 2003; Vaillant, 1983). Once addictive behavior patterns emerge, there is a similar natural history across various substances. For example, Hunt, Barnett, and Branch (1971) presented seminal research, based on 84 studies, demonstrating remarkably similar relapse patterns for heroin, smoking, and alcohol use. The observation that drugs with important biochemical differences follow the same course suggests that the object of addiction is less relevant to the course of addiction than previously thought. These patterns likely reflect the dynamics of a common underlying addiction process and therefore challenge the conventional wisdom that there are various and distinct addictive disorders (Marlatt et al., 1988; Marlatt & Gordon, 1985; Prochaska, DiClemente, & Norcross, 1992; Shaffer, 1997a, 1999a, 1999b, 2003). In the absence of sufficient longitudinal evidence, we cautiously suggest that the natural histories of behavioral expressions of addiction are similar to the histories of many chemical expressions of addictions. Despite this dearth of information, emerging evidence suggests that, as with substance use disorders, gambling disorders are dynamic: Over time, individuals with these disorders might experience worsening symptoms, but they also might recover. People who recover do so through self-directed change and treatment influences alike. As with alcohol and other drugs, it is likely that the majority of cases who remit from various other expressions of addiction accomplish this on their own and without formal treatment (e.g., Klingemann &
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Sobell, 2007; Nathan, 2003; Schachter, 1982; Shaffer & Jones, 1989; Slutske, 2006; Sobell, Cunningham, & Sobell, 1996; see Volume 2, Chapter 6, this handbook). However, although more people stop addiction on their own, it is likely that the success of this change strategy is similar to that of treatment seekers (e.g., Cohen et al., 1989), and the rate of success varies with the severity of the disorder. For a review of the longitudinal studies and trajectories associated with gambling disorders, interested readers should consult LaPlante, Nelson, LaBrie, and Shaffer (2008) and Shaffer and Martin (2011).

Object nonspecificity. Research has suggested that addiction is not necessarily inextricably linked to a particular substance or behavior. For example, circumstantial opportunity plays a more influential role in the development of addictive behavior than do individuals' preferences for certain drugs (Harford, 1978). Moreover, with or without treatment, it is very common for people recovering to hop from one expression of addiction (e.g., opioids) to another (e.g., cocaine, alcohol, gambling, exercise) before successfully recovering from addiction. Hser, Anglin, and Powers (1990) examined longitudinal patterns of alcohol and narcotic use and observed a decrease in alcohol consumption at the time that narcotic addiction began; likewise, during periods of decreased narcotics use, alcohol consumption rose. This hopping between addiction objects has been demonstrated for illicit drugs and nicotine (Conner, Stein, Longshore, & Stacy, 1999), alcohol abuse and bulimia (Çepik, Arikam, Boratav, & Isik, 1995), and substance abuse and pathological gambling (Blume, 1994). Finally, clinical research has shown that during early treatment for opioid dependence, as both opioid and cocaine use decreased, sedative use increased (Shaffer & LaSalvia, 1992).

Concurrent manifestations of addiction. The prevalence of polysubstance abuse and dependence is well documented (Grant et al., 2004; Kessler et al., 1997, 2005), but the co-occurrence of chemical and behavioral expressions of addiction is also common (e.g., Kessler et al., 2008). For example, intemperate shoppers and gamblers both evidence higher rates of substance use disorders than groups without these patterns of economic excess (Baker, 2000; Christenson et al., 1994; Feigelman, Wallisch, & Lesieur, 1998; Lejoyeux, Ades, Tassain, & Solomon, 1996). Conversely, compared with those without substance use disorders, individuals who are dependent on psychoactive substances are more likely to be pathological gamblers (Black & Moyer, 1998; Feigelman et al., 1998; Lesieur & Heineman, 1988). Research demonstrating the frequent co-occurrence of different expressions of addiction signals the potential for the presence of an underlying force responsible for addiction.

In addition to the co-occurring manifestations of addiction and shared psychosocial antecedents (e.g., mental disorders), evidence has suggested that various expressions of addiction are associated with identifiable subclinical shadow syndromes that influence the experience, manifestation, and treatment outcomes associated with addiction (e.g., Boudreau, LaBrie, & Shaffer, 2009; Kessler, Merikangas, Berglund, & Eaton, 2003). Shadow syndromes are subclinical clusters of signs and symptoms. Everyone has characteristic shadows; these are the features of human personality and character (Ratey & Johnson, 1997). However, shadows typically go unnoticed or undetected by observers because they think of these shadows as a person's personality style (Shapiro, 1965). In the case of shadow syndromes related to gambling disorders, research has indicated that individuals with a diagnosis of pathological gambling experience characteristic reliable and identifiable subclinical patterns of symptom clusters associated with psychiatric disorders other than those associated with the diagnostic criteria for a gambling disorder (e.g., dysthymia, anxiety, specific phobias; Boudreau et al., 2009). Although it remains to be determined, it is likely that every
expression of addiction has a characteristic shadow that influences how the expression becomes manifest and how it is sustained. These shadows might be unique to specific expressions of addiction or shared across expressions.

**Treatment nonspecificity.** Pharmacological treatment nonspecificity (i.e., a drug-specific treatment reducing the immoderate use of another drug or activity) also provides support for a syndrome model of addiction (e.g., Barson et al., 2009; McKee et al., 2009; Potenza et al., 2011). Recently, scientists have identified interesting pharmacological treatment spillover effects (see Volume 2, Chapter 3, this handbook). For example, naltrexone, an opioid antagonist used for the treatment of opioid abuse and dependence disorders, has shown efficacy for the treatment of pathological gambling (Kim, Grant, Adson, & Shin, 2001). In a double-blind, placebo-controlled study, varenicline, a partial nicotine agonist used for the treatment of nicotine dependence, has shown efficacy in reducing consumption among heavy alcohol users (McKee et al., 2009). Treatment programs featuring methadone, an opioid agonist, have shown efficacy in reducing cocaine abuse among opioid-dependent patients (Shaffer & LaSalvia, 1992). Other research has shown that topiramate, an adjunctive treatment for seizure disorders that acts on the brain's dopamine pathways, has efficacy in treating alcohol dependence disorders (Johnson et al., 2003). Similarly, researchers have speculated that bupropion, an antidepressant used in smoking cessation protocols to treat nicotine dependence, might be efficacious because of its dopaminergic and noradrenergic activities, “with the dopaminergic activity affecting areas of the brain having to do with the reinforcement properties of addictive drugs and the noradrenergic activity affecting nicotine withdrawal” (Hurt et al., 1997, p. 1201), rather than its antidepressant properties. Additional spillover treatment effects to other dopamine-mediated expressions of addiction would provide support for a syndromal theory of addiction. Finally, several nonpharmacological treatments (e.g., cognitive-behavior therapy, psychodynamic therapy, behavior therapy) are commonly used interchangeably and effectively to treat both chemical and behavioral expressions of addiction (e.g., Potenza et al., 2011).

**Modeling an Addiction Syndrome**

The extant evidence has suggested that (a) many commonalities occur across different expressions of addiction and (b) these commonalities might reflect shared etiology: a syndrome. As we mentioned before, at a minimum, a syndrome is a cluster of symptoms and signs related to an abnormal condition; not all symptoms or signs must be present in every instance of the syndrome, and some expressions of a syndrome have unique signs and symptoms. Kendell (1989) advanced this definition by proposing that two essential elements make up a clinical syndrome: (a) a cluster of related symptoms and (b) a distinctive temporal progression. Figure 1 illustrates the development of an addiction syndrome and its consequences.

As Figure 1 shows, antecedents of the addiction syndrome include personal vulnerability levels, object exposure, and object interaction. More specifically, throughout the course of development people encounter and accumulate specific combinations of neurobiological and psychosocial elements that can influence their behavior. Some elements increase the likelihood of addiction; others are protective and reduce the chance of addiction (e.g., social support networks or dimensions of religiosity; Kendler, Liu, et al., 2003; Vander Bilt, Dodge, Pandav, Shaffer, & Ganguli, 2004). Similarly, during their lifetimes, individuals are exposed to and have access to different objects of addiction. Exposure and access to an object of
addiction increases an individual's likelihood of interacting with that object. Interacting with an object of addiction can expose at-risk individuals to neurobiological consequences that are both common to all objects of addiction (e.g., activation of reward circuitry) and unique to specific objects of addiction (e.g., psychoactivity). In Chapter 11 of this volume, Shadel and Scharf examine the relationship between exposure and addiction, addressing the important interactions that occur between individual and environment to influence addiction.

When (a) individuals engage in repeated interactions with a specific object or objects of addiction and (b) the neurobiological or social consequences of these interactions produce a desirable subjective shift that is reliable and robust, the premorbid stage of the addiction syndrome emerges. During this stage of the syndrome, people teeter on a delicate balance that can shift them toward either more or less healthy behavior. Although distal antecedents of addiction (see Figure 1) are well documented, the proximal antecedents that influence the likelihood of further syndrome development remain poorly identified. In Chapter 13 of this volume, Najavits discusses some of the potential proximal influences that can affect the development of addiction in a public health framework. Despite the dearth of research in this area, we suggest that these proximal influences are likely to be biopsychosocial factors similar to those associated with distal influences. For example, despite being distal, genetic dispositions directly and indirectly influence current patterns of behavior; long-standing dispositions affect psychological characteristics that have an impact on how people manage current social situations. Furthermore, as we mentioned before, distal antecedents have

contemporary consequences that serve as more proximal risk factors for developing or maintaining addiction.

The addiction syndrome can manifest itself in many different ways; its premorbid characteristics and some sequelae are dependent on the object with which people interact. To illustrate, if one interacts with cigarettes (e.g., by repeatedly smoking) or if one interacts with a slot machine (e.g., by repeatedly gambling) and the addiction syndrome emerges, then the manifestation of this syndrome and its sequelae will have some characteristics that uniquely reflect each of these objects. In addition, assorted expressions of the addiction syndrome (e.g., substance use disorders, pathological gambling) will share common manifestations (e.g., shifts in schedule, changing personal priorities) and sequelae (e.g., depression, neuroadaptation, deception, personal debt). Researchers and clinicians can identify the presence of the addiction syndrome when premorbid characteristics are accompanied by at least one of the shared manifestations and sequelae summarized in Figure 1. As we noted before, the addiction syndrome can be recursive and its sequelae can generate an entirely new vulnerability profile (e.g., provoke a reward system malfunction in a previously normal system). The development of the addiction syndrome therefore places people with the syndrome at increased risk for continuing addictive behavior and for developing new addictive behaviors. This chain of events is evident in many ways but most specifically in the parallel natural histories of different manifestations of addiction, including relapse patterns, addiction hopping, treatment nonspecificity, and addiction comorbidity.

Clinical Implications of the Addiction Syndrome Model

Although distinct expressions of addiction have unique elements, they also share many neurobiological and psychosocial antecedents and consequents. Coupled with repeated premorbid shifts toward a desirable subjective state, neurobiological and psychosocial characteristics both define and result from the addiction syndrome. This situation creates important treatment challenges. The recursive nature of sequelae can exacerbate the difficulties associated with addiction treatment. For example, when clinicians pay insufficient attention to the etiological causes of addiction, patients can experience a cycle of remitting and exacerbating expressions of addiction. Moreover, the emerging behavior often serves as a risk factor for other expressions of addiction and comorbidity, increasing the likelihood of developing new or different manifestations of the addiction syndrome. Moreover, without an independent diagnostic gold standard—free from the problems of impression management that can bias self-report—the manifestations and sequelae of addiction serve tautologically as the primary evidence that clinicians use to make diagnostic decisions and inferences about the presence of addiction.

Unlike other approaches that focus almost exclusively on the objects of addiction—in addition to understanding distal influences—the syndrome model requires researchers and clinicians to consider developing and using methods that can identify the shared and unique proximal antecedent elements (e.g., trauma, depression, anxiety, financial stress) of addiction at the premorbid stage. This strategy encourages scientists to advance and clarify an etiological model. Doing so will lead to a more precise understanding of the precursors of addiction expressions, eventually yielding a diagnostic gold standard. Armed with an improved understanding of both proximal and distal influences and a diagnostic gold standard that is not dependent on the assessment of sequelae, clinicians and public health workers will be able to advance primary and secondary prevention programs. Volume 2, Part II of this handbook focuses on the prevention of addiction. In Chapter 9, Scheier provides an overview of primary prevention models for addiction. In Chapter 10, Fosco, Dishion, and
Stormshak offer a practical evidence-based guide for secondary and high school addiction prevention. Finally, in Chapter 11, using the addiction syndrome as context, Borsari focuses on prevention in the college setting.

Rethinking addiction as a syndrome also has many direct implications for treatment. At the population level, the syndrome model provides a map to the etiology of addiction. Therefore, it provides a public health guide to the primary, secondary, and tertiary prevention of addiction. As summarized in Figure 1, primary prevention efforts should target the left side of the model; secondary prevention should target the middle of the model, which depicts the premorbid stage of addiction; and, finally, tertiary prevention should focus on the right side of the model (i.e., expressions of addiction).

At the individual level of analysis, 80% to 90% of people entering recovery from addiction will relapse during the 1st year after treatment (Marlatt & Gordon, 1985). This circumstance might result in part from the prevalent use of focused object-specific treatment approaches despite research suggesting that objects of addiction cannot sufficiently account for the dominant underpinnings of addiction. From the syndromal perspective, the most effective addiction treatments are multimodal cocktail approaches (Marlatt, 1988) that include both object-specific and addiction-general treatments. Addiction can exacerbate vulnerabilities and neurobiological changes. This circumstance might, in part, explain high relapse rates and new manifestations of the syndrome that often appear during the course of the addiction. The syndrome model of addiction encourages clinicians to recognize that patients develop new risk factors during treatment that can interfere with recovery efforts. This model requires clinicians to develop multidimensional treatment plans that account for the many relationships among the multiple influences and consequences of addiction. Viewing addiction as a syndrome also obligates providers to assess repeatedly the impact of these relationships on relapse, addiction hopping, the course of the illness, and many other treatment-related outcomes.

Understanding distinct addictive behaviors as opportunistic rather than separate encourages the development of new diagnostic tools to identify the shared elements of the addiction syndrome. Other than measuring the intensity and duration of withdrawal signs, current diagnostic protocols typically rely on self-reports of past behavior patterns to identify addiction; this strategy essentially requires patients to diagnose themselves. Adopting a syndromal perspective on addiction should stimulate further investigation of more objective diagnostic measures (e.g., functional MRI, event-related brain potentials, implicit behavioral tools) that limit demand characteristics and reduce socially desirable responses; this strategic shift will move the field toward increasingly reliable, valid, and clinically meaningful diagnoses. Developing more objective diagnostic tests will help limit the use of unnecessary clinical resources and reduce the application of treatments inappropriate for individuals at certain stages of the addiction syndrome.

Despite the evidence supporting a syndromal view of addiction, the dominant clinical philosophy continues to focus on the addictive nature of chemicals, yielding unnecessarily narrow treatment protocols. Because evidence for the efficacy of many pharmacological and psychological non-object specific treatments for addiction already exists (McLellan, Arndt, Metzger, Woody, & O'Brien, 1993), perhaps the existing treatments are more advanced than the addiction philosophy. This discrepancy between theory and practice might inadvertently contribute to less than optimal treatment outcomes given that conventional wisdom discourages clinicians from paying sufficient attention to the underlying core of addictive behaviors. Moreover, because the therapeutic boundaries of various addiction treatments are unknown, clinicians might overlook effective chemical addiction treatments for behavioral addictions and useful behavioral treatments for chemical addiction. Ultimately, addiction treatment
outcomes might be improved by revising and reordering existing clinical activities and developing new unobtrusive diagnostic tools. This observation leads to the interesting and promising conclusion that the necessary tools for improving addiction treatment might already be available—all that is required to enhance the use of these devices is a rethinking of addiction. Many of the chapters in Volume 2, Part I, this handbook, which is devoted to the treatment of addiction, provide support for the need to rethink addiction to advance clinical outcomes. For example, there is evidence for the effectiveness of different screens and treatments across multiple expressions of addiction. The chapters in Volume 2, Part I address brief screening (Chapter 8), psychotherapies (Chapter 4), pharmacotherapies (Chapter 3), supervision of addiction treatment (Chapter 2), relapse prevention (Chapter 5), and self-help aids (Chapter 7).

**EMPIRICAL CHALLENGES FOR THE ADDICTION SYNDROME**

At this time, the neurobiological and psychosocial antecedent evidence for the syndrome model is strong; however, many important aspects of the model remain under tested. For example, research focusing on chemical and behavioral addiction hopping, temporal patterns of psychiatric comorbidity (e.g., sign, symptom, and disorder patterns), and treatment nonspecificity is limited. Similarly, there is a paucity of research involving secondary behaviors and object-specific natural history; both of these areas of research are essential to advancing an addiction syndrome model. To assess the validity of the syndrome model, it is important to develop and pursue empirical research that tests the model against competing frameworks and theories. In this final section, we propose several research challenges that will help evaluate, revise, and potentially transform the syndrome model of addiction.

**Testing the Influence of Antecedents: Underlying Vulnerability**

If addiction is a syndrome, then different expressions of addiction should share common antecedents. We also expect that the shared variance will be greater for the distal underlying vulnerabilities than for the more proximal antecedents, in part because the enduring distal effects are larger and more complex compared with the more limited and focused impact of proximal antecedents. In other words, a person's underlying vulnerability predisposes him or her to addiction but not to any specific expression of addiction. Despite having less general impact than distal factors (e.g., genetics), proximal events (e.g., precipitating events) have more focused influence in determining the emergence of a particular expression of addiction. Unlike the interactive, Galileian approach of the syndrome model, competing Aristotelian models link specific antecedents to specific expressions of addiction. To test these rival models, researchers need to conduct prospective studies that measure a wide array of potential antecedents and examine their relationship to the emergence of different forms of addiction. Given the low base rates of certain disorders (e.g., pathological gambling), a catchment area design would provide an ideal setting for these studies. This design (e.g., sampling in Las Vegas) will maximize the potential incidence of the low base-rate disorders. The sampling plan for this kind of research also needs to begin during adolescence or earlier; this will permit researchers to capture participants who have not yet manifested expressions of addiction. Given such a design, Figure 2 shows the two alternative models that can be tested. Both models postulate that clusters of antecedent variables will make up precursor constructs. These clusters could be as simple as different measures of depression or anxiety defining depression and posttraumatic stress disorder constructs, or more
complex models in which multiple disorders or behaviors define higher order constructs (e.g., psychiatric symptoms clustering into internalizing and externalizing disorders; Krueger, Caspi, Moffitt, & Silva, 1998). For the syndrome model, these antecedents or antecedent profiles predict the development of addiction but do not differ across its unique expressions. In competing models, these antecedents relate directly to specific expressions of addiction.

Testing the Influence of Exposure on Addiction
According to the syndrome model, given similar vulnerabilities, the development of addiction depends on an individual's exposure to potential objects of addiction. This exposure is necessary for addiction to develop; it also helps determine what expression of addiction will emerge. To test this assumption, researchers need to use an investigative design similar to the design we described previously, with the added requirement that the methods include (a) sampling from areas or time periods with different levels of exposure to different objects of addiction and (b) matching the sample according to individuals' level of vulnerability to addiction. Again, to test for exposure effects, the research design needs to be longitudinal and needs to sample young people who have not yet developed addiction. Many resources are available to examine the effects of exposure. Regional databases now exist that include information about substance-related crimes, the presence of casinos, liquor licenses, and so forth.
Testing Premorbid Addiction Syndrome States: Desirable Subjective Shifts and Repeated Interactions

One criticism sometimes leveled at the addiction syndrome model is that according to this model almost any object could be a potential object of addiction. Although this is strictly true, it ignores the critical part of the model that connects distal and proximal influences to desirable shifts in subjective states. The potential for any object to become an object of addiction depends, in large part, on its ability to create a reliable and desirable subjective shift for the individual interacting with the object. Although this is a complex psychological process, distinct for each individual, different objects have different overall potentials to create such a shift.

To test this postulate of the syndrome model, unlike many of the questions posed here that require costly long-term prospective designs to answer, research needs to develop a measure of desirable subjective shift. Ideally, scientists could administer this measure via ecological momentary assessment (e.g., monitor craving, withdrawal symptoms, moods, and activities over time; using smartphones as electronic diaries; Shiffman, Stone, & Hufford, 2008). In addition to self-report, which is required to reveal desirable subjective shifts, independent assessments (e.g., functional MRI, reaction time, nonverbal measures) can provide collateral data. Although an involved longitudinal study is not necessary to test these kinds of measures, a large representative sample is required. With such a sample, one could then place different objects of addiction along a gradient of addictive potential (e.g., Hyman, 1994).

Testing Expressions of Addiction: Unique and Shared Consequences

If addiction is a syndrome, then we also expect that different expressions of addiction will evidence unique consequences and share some consequences. The same design proposed to test antecedents to the emergence of addiction could be used to test shared consequences of addiction.

Once these components of the syndrome model have been established, refined, and tested, additional studies can test the syndrome model as a whole. These studies would need to be based on a longitudinal sample followed over many years, similar to the sample established for the Framingham Heart Study (Levy & Brink, 2005; National Heart Institute, 1966) or in the book The Natural History of Alcoholism (Vaillant, 1983). With such a study, researchers can measure antecedents and exposure, reported subjective shifts, and the timing and expression of addiction among those in whom it develops. With the subgroup that develops addiction, the study will have the opportunity to study the consequences of the various expressions of addiction and, for those in treatment, the treatment effects. This study can chronicle the natural history of addiction, with the added facets of including multiple potential expressions of addiction and measures of the actual experiences with the objects of addiction. Just as the Framingham Heart Study did for cardiovascular disease, a study of this magnitude holds the potential to unlock many secrets of the addiction syndrome.

THE APA ADDICTION SYNDROME HANDBOOK

This two-volume handbook includes a comprehensive review of addiction. Volume 1 includes this introduction and three sections focusing on the background and history of addiction, the distal and proximal influences on addiction, and expressions of addiction. Volume 2 also has three major sections. These sections examine the many aspects associated with recovery from addiction, the prevention of addiction, and other essential issues
commonly associated with addiction, for example, technology, driving under the influence, and homelessness. Readers will also find a foreword in Volume 1 and an epilogue in Volume 2 that provide insight into and perspective on the addiction syndrome and the place of this handbook within the development of a science of addiction.

As we assembled this handbook, we asked the contributors to consider their areas of interest with respect to the addiction syndrome model. In addition, to advance the conceptual framework that guides addiction research and treatment, we asked each contributor to provide evidence to support or refute the addiction syndrome model. We hope that this approach stimulates an enthusiastic dialogue that can advance the field by revising and improving the etiological models that have in the past guided and currently guide the conventional wisdom about addiction and its causes and consequences.

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Editor-in-Chief

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Introduction


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PART I

BACKGROUND AND HISTORY OF ADDICTION
Addiction is... an attachment to, or dependence upon, any substance, thing, person or idea so single-minded and intense that virtually all other realities are ignored or given second place—and consequences, even lethal ones, are disregarded. (Mack, 2002)¹

It is best to think of any affliction—a disease, a disability... as a text and of “society” as its author. (Blum, 1985, p. 221)

DECONSTRUCTING THE CONCEPT OF ADDICTION

Addiction can take many and seemingly disparate forms. From substance abuse to shopping (Baker, 2000; Catalano & Sonenberg, 1993; Christenson et al., 1994), eating carrots (Cerný & Cerný, 1992), drinking water to intoxication (Pickering & Hogan, 1971; Rowntree, 1923), committing antisocial or criminal acts (Hodge, McMurran, & Hollin, 1997), and using computers (Eppright, Allwood, Stern, & Theiss, 1999; Mitchell, 2000; O'Reilly, 1996; Shaffer, 1996; Shaffer, Hall, & Vander Bilt, 2000; Starker, 1983), social observers have applied the notion of addiction to many and varied human activities (Orford, 1985). These many and varied expressions of addiction contribute to its ideological complexity. The conceptual complexity of addiction also derives partly from the fact that the adverse consequences of addiction typically include biological, psychological, and social harms. The negative biological consequences include the emergence of neuroadaptation,
which manifests as tolerance (i.e., the observation that an increased dose is needed to experience the same subjective effects experienced with a lower dose before) and withdrawal (i.e., a stereotypical pattern of discomfort on stopping use that resumed use can alleviate). Neuroadaptation reflects changes that have occurred at the neurotransmitter, presynaptic, and postsynaptic levels of the brain. Of note, tolerance and withdrawal can be evident whether the expression of addiction involves a substance or a behavior. For example, disordered gamblers often evidence a pattern of increasing bets to achieve the same level of excitement that they experienced previously at a lower level of wagering; gamblers have also reported symptoms of withdrawal when they cut back or stop gambling (Wray & Dickerson, 1981).

However, not all scientists accept the idea of behavioral addiction (Holden, 2001), and the consequence of this debate is conceptual chaos about the construct of addiction (Shaffer, 1997a). For example,

People toss around the term “addiction” to describe someone’s relationship to a job, a boyfriend, or a computer. But scientists have traditionally confined their use of the term to substances—namely alcohol and other drugs—that clearly foster physical dependence in the user. That’s changing, however. New knowledge about the brain’s reward system, much gained by super refined brain scan technology, suggests that as far as the brain is concerned, a reward’s a reward, regardless of whether it comes from a chemical or an experience. And where there’s a reward, there’s the risk of the vulnerable brain getting trapped in a compulsion. “Over the past 6 months, more and more people have been thinking that, contrary to earlier views, there is commonality between substance addictions and other compulsions,” says Alan Leshner, head of the National Institute on Drug Abuse (NIDA) and incoming executive officer of the American Association for the Advancement of Science, publisher of Science. (Holden, 2001, p. 980)

Addiction has primarily become a lay term, although scientists often use it as well. Even though future diagnostic manuals are likely to include addiction, it is not included in the current diagnostic manuals (e.g., the Diagnostic and Statistical Manual of Mental Disorders, 4th ed., text revision [DSM-IV-TR; American Psychiatric Association, 2000] or International Classification of Diseases—10 [World Health Organization, 1992]). As we discuss later, the American Psychiatric Association is preparing the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5); in this new version, nosologists have proposed that substance use and gambling disorders be classified under addiction and related disorders. Recognizing the problems associated with the meaning of addiction, Vaillant (1982) suggested that instead of seeking a strict operational definition, one should think of alcoholism as one does mountains and seasons: You know them when you see them. Clinicians working with the full range of addictive disorders (e.g., substance, gambling, and other excessive patterns of activity) often apply similar subjective strategies as they try to determine whether it is present and whether it requires treatment.

Reducing Conceptual Complexity
Although addiction is a complex construct, there are useful guidelines to help identify people struggling with this disorder. For example, DSM-IV-TR takes a categorical approach to defining addiction and, consequently, discusses chronic substance use disorders in terms of abuse and dependence. DSM-IV-TR defines the main features of substance dependence as “a cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues use of the substance despite significant substance-related problems” (American Psychiatric Association, 2000, p. 192). DSM-IV-TR defines substance abuse—less serious than substance dependence—as “a maladaptive pattern of substance use manifested by recurrent and significant adverse consequences related to the repeated use of substances” (American Psychiatric Association, 2000,
Before we consider the practical and applied aspects of the *DSM-IV-TR*’s categorical approach to identifying addiction, it is important to consider five conceptual consequences associated with the idea of addiction that can influence clinical judgments about the assessment and treatment of addiction. These include (a) distinguishing among use, abuse, physiological dependence, and addiction; (b) clarifying that the object of addiction does not cause addiction; (c) describing the difficulties distinguishing addiction from other mental states; (d) avoiding tautological explanation; and (e) dealing with the absence of a diagnostic gold standard.

**Distinguishing Among Use, Abuse, Physiological Dependence, and Addiction**

To avoid unintended consequences, such as unnecessary hospitalizations, increased medical costs, and patients who learn to distrust health care providers, addiction specialists need to distinguish among use, abuse, dependence, and addiction. Use results in no measurable biopsychosocial sequelae. For example, a glass of wine at dinner or an informal bet between two friends about the outcome of a sports event typically has little adverse consequence. In fact, some patterns of use might be beneficial, as the recent literature focusing on the apparent health benefits of moderate amounts of red wine suggests (Hansel et al., 2010; McCaul et al., 2010). Abuse, as noted earlier, does result in negative consequences, but less intensely than addiction. For some people, abuse represents a less serious illness; for others, it represents a stop along the way to addiction.

The distinction between addiction and physiological dependence is also very important. For example, after surgery many people receive opioid analgesics (e.g., oxycodone) for their pain. Using a sufficient dose of opioids for an adequate period, many of these patients experience neuroadaptation; that is, they exhibit symptoms of opioid withdrawal if the dosage is significantly reduced or eliminated. However, most postoperative patients never seek these medications after their pain subsides. After being treated sufficiently with narcotic analgesics for an extended time, they do not behave as though they are addicted, despite being physiologically dependent.

Conversely, physiological dependence is not a necessary condition for the presence of addiction. To illustrate, consider a man who does not drink all week, then drinks excessively every weekend; he never exhibits tolerance or withdrawal. His drinking, however, does result in an arrest for driving under the influence and considerable family discord. Furthermore, he puts his job in jeopardy because of an excessive number of Monday absences. In this instance, addiction is present, but physiological dependence is not.

Finally, as we noted previously, a person does not have to ingest a substance to exhibit physiological dependence. For example, on stopping, pathological gamblers who do not use alcohol or other psychoactive drugs can evidence physical symptoms that appear to be similar to narcotic, stimulant, or poly-substance withdrawal (Wray & Dickerson, 1981).

The reality that addiction can exist with or without physical dependence encourages and supports a broader concept of addiction; this new perspective includes both substances and activities (e.g., Shaffer, 1996, 1997a, 1999). For example, neuroadaptation reflects changes in the brain neurotransmitter receptors. Just as ingested substances cause release of neurotransmitters that vie for receptor sites within the brain, human activities also stimulate release of neurotransmitters (e.g., R. Hyman, 1995; S. E. Hyman, 1994; S. E. Hyman & Nestler, 1993). The activity of these naturally occurring psychoactive substances are likely a mediating cause of addictions. As we will show, the biological substrate is similar for both substance and behavioral addictions.

As we mentioned, the American Psychiatric Association has made available its draft of *DSM-5*. From our perspective, it is an improvement on *DSM-IV-TR*, as that version was an improvement on its predecessors. One change being proposed is to rename the substance-related disorders category; it would become *addiction and related disorders*. The word *dependence* would be reserved once again for neuroadaptation, not out-of-control compulsive

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1. Although a full discussion of this matter is beyond the scope of this chapter, it is also important to note that not all people with addiction are impaired in every aspect of their daily lives.
Albanese and Shaffer

substance use. As noted earlier, physicians and others have mistakenly thought that people who develop physiological dependence on medications they use appropriately—despite having helped them to become more functional—also have addiction. Another change would move pathological gambling, currently included in the category impulse-control disorders not elsewhere classified, to the category of addiction and related disorders as gambling disorder. This move acknowledges the evidence that has been accumulating of a heritage shared by both substance and nonsubstance addictions. Similarly, nosologists could include other behavioral disorders such as Internet addiction in this category, if sufficient data accumulate to support this diagnosis. We believe these developments represent a step closer to a spectrum or syndrome approach to addiction and a step further away from a categorical understanding.

The Object of Addiction Does Not Cause Addiction

A contemporary view of addiction suggests that the objects of addiction are those things that reliably and robustly shift subjective experience: that is, those activities that consistently change experience in a desired direction (e.g., activities that always make people feel better). The most reliable and robust “shifters” hold the greatest potential to stimulate the development of addictive disorders. However, because the strength and consistency of these activities to shift subjective states vary across individuals and settings, scientists have been unable to predict with accuracy precisely who will become addicted. That is, one cannot eliminate any substance or activity as a potential object of addiction if the object can shift someone’s subjective state at some place and time. Nevertheless, it is important to note that psychoactive drugs and certain other activities correlate highly with shifting subjective states because these activities reliably influence and change emotional experiences. Consequently, psychoactive drug use (e.g., S. E. Hyman, 1994) and certain other behaviors (e.g., gambling) tend to be ranked highly among the activities that are often associated with addictive behaviors.

Observers make a conceptual error when they think that addiction resides as a latent property of an object (e.g., a drug or game of chance). Conventional wisdom, for example, refers to “addictive drugs” or “addictive gambling.” However, addiction is not the product of a substance, game, or technology, although each of these has the capacity to influence human experience. Experience is the currency of addiction. When a particular pattern of behavior reliably and robustly changes emotional experience, the potential for addiction emerges. In other words, addiction is the description of a relationship between organisms and objects within their environment, not simply the result of an object’s attributes. As such, the causes of addiction are multifactorial (Shaffer et al., 2004; Zinberg, 1984).

The syndrome model avoids the conceptual trap of considering addiction to be a property of an object or activity. Some observers might argue that this opens the door to a variety of meaningless claims of addiction. However, human suffering comes in many varieties, and the objects of this suffering do not reflect a theoretical debate. Addiction theory needs to make room for and explain the seemingly rare instances of addiction to some objects as well as the more common prevalence of addiction to others. Nevertheless, the addiction syndrome model does acknowledge that some substances or behaviors are more likely to become an object of addiction for some people at some times. That is, some substances or behaviors are better at reliably and robustly shifting subjective experiences in a user’s desirable direction. Taken to a logical extreme, the syndrome model must be open to the possibility that any substance or activity can become an object of addiction. To rule out a priori anything as such an object is not scientifically based because many objects hold the potential to shift subjective states; this potential derives from psychoactive drug effects, cultural influences, family values, and many other influences. Despite this wide range of possibilities, those objects that most robustly and reliably shift subjective experiences (e.g., nicotine, alcohol, sex) will continue to be among the most prevalent objects of addiction compared with other potential objects (e.g., grapefruit juice, spinach, folding laundry).

Objects of addiction—objects that can shift subjective experience—produce neurobiologically similar events in the central nervous system. To
stimulate and sustain involvement with any of the various objects of addiction, the brain's reward system must be activated. Originally, the reward system existed to ensure the propagation of the species by implicitly teaching people that sex was pleasurable and should be repeated. As the context of human behavior shifted and society matured, the same reward system eventually became associated, for some people, with other pleasurable experiences (e.g., using psychoactive drugs, gambling, eating, shopping).

Other chapters in this handbook address the neurobiology of addiction. At this point, however, it is important to note that psychoactive substances, such as alcohol, cocaine, and opiates, stimulate the brain's reward system. Furthermore, functional neuroimaging studies have revealed that money, beauty, and the anticipation of cocaine energize the reward system (e.g., Aharon et al., 2001; Breiter, Aharon, Kahneman, Dale, & Shizgal, 2001). The observation that disparate objects stimulate similar neurobiological pathways, which is consistent with the syndrome model of addiction, indicates that, regardless of the object of addiction, the brain is the final common pathway for addictive behaviors. A recent imaging study of intemperate Internet game users, who appear to exhibit similar behavior to people with alcohol dependence or pathological gambling, revealed activation of the same neural pathways as those people with alcohol or gambling disorders (Park et al., 2010). When people do not experience the positive subjective effects associated with the stimulation of the brain's reward system, they typically stop engaging in these behaviors.

To this point, we have implied that simply using drugs or engaging in certain activities does not cause addiction. Now let us be more explicit: From a logical perspective, the objects of addiction cannot cause addictive behavior patterns. To illustrate, if psychoactive drug using was a necessary and sufficient cause for addiction, then addiction would occur every time drug using was present. Similarly, if drug using was a necessary cause for addiction, addictive behaviors would be absent every time drug using was missing. However, as we described before, tolerance, withdrawal, and adverse consequences of excessive behaviors are often present when drug using is absent. In addition, people often exceed their drug-using limits and lose a modicum of self-control without experiencing addiction. Therefore, drug using is neither a necessary nor a sufficient cause to produce addiction. Furthermore, as we have suggested, it might not even be the primary cause of addiction. Even though drug using is highly correlated with addiction because psychoactive substances reliably shift subjective experience and alter neurochemistry, drug taking does not cause addiction. As in the case of pathological gambling and excessive sexual behaviors—that do not fall within the domain of obsessive-compulsive disorders—addiction can exist in the absence of drug taking. This observation provides insight into the necessity of considering a more complex relationship between a person who might develop addiction and his or her object of addiction. The syndrome model of addiction provides the architecture for this more complex understanding.

**Difficulties Distinguishing Addiction From Other Mental States**

As the syndrome model illustrates, the relationship of a person with an object of excessive behavior is what defines addiction. The confluence of psychological, social, and biological forces determines addiction. No single set of factors can define addiction precisely. Unfortunately, as with addiction, the concept of relationship is also difficult to define. Therefore, until experience and scientific evidence provide more insight into the synergistic nature of these factors and help researchers determine the interactive threshold or thresholds that might apply, they are forced to operationalize addiction so that researchers, clinicians, and policymakers can share a common perspective (Shaffer, 1992, 1999; Shaffer et al., 2004; Shaffer & Robbins, 1991, 1995). This need to develop an operational definition has encouraged contemporary Western addiction workers to think of addictive behavior as having three primary components: (a) some element of craving or compulsion; (b) loss of control; and (c) continuing the behavior pattern despite adverse consequences.

Addiction is different from compulsive patterns of behavior because of the quality of associated subjective states. Addictive behaviors tend to be ego
syntonic, particularly early in the development of the addiction (Shaffer, 1997b). People experience ego-syntonic behaviors as consistent with their sense of self and do not anticipate that these activities will cause personal problems. In fact, most addiction emerges from positive experiences (e.g., winning a bet, relieving psychological or physical discomfort by ingesting drugs). People tend to experience other psychiatric illnesses as ego-dystonic, or ego alien. Ego-dystonic states feel as though something—an "otherness"—is taking control against one's will and directing behaviors toward undesirable ends. Despite not wanting to act on these renegade urges, people with compulsive disorders act to discharge the mounting pressure; they then feel some measure of tension relief. Clinicians can often distinguish obsessive-compulsive disorders from addiction by the extent of ego-dystonic thoughts and distress that accompany these disorders compared with addiction, for which subjective anguish is usually revealed only after the fact (Shaffer, 1994a). It is worth noting that there is little evidence suggesting that addiction represents a unique taxon among the various mental disorders (cf. Widiger & Samuel, 2005). Instead, the current evidence has suggested that it reflects activity on a dimension that ranges from little or no involvement to intense and frequent involvement with a particular object or activity (e.g., Braverman, LaBrie, & Shaffer, 2011).

Avoiding Tautological Explanation: Considering Behavioral Excess as Evidence

For scientists, the current concept of addiction represents a troublesome tautology. This situation has contributed to keeping addiction a very popular lay concept because it can be invoked to explain almost any pattern of excessive behavior. This tautology operates when observers notice adverse consequences, stimulated by repetitive behavior patterns, apparently occurring against the actor's better judgment; under these circumstances, they often infer the presence of addiction.

The problem is that there is no independent way to confirm that the "addict"
cannot help himself and therefore the label is often used as a tautological explanation of the addiction. The habit is called an addiction because it is not under control but there is no way to distinguish a habit that is uncontrollable from one that is simply not controlled. (Akers, 1991, as cited in Davies, 1996, p. S41)

As organized currently, diagnostic manuals such as the DSM-IV-TR increase the likelihood that clinicians can repeatedly classify disorders, such as pathological gambling, correctly. However, these systems fail to address the construct validity of what is being classified because the "addictive" disorder (e.g., pathological gambling and substance use disorders) is assumed to exist by inference from the consequences associated with the behaviors in question (e.g., Barron, 1998).

Social consensus among scientists is insufficient to establish a definitive diagnosis, although the consensus-building process can promulgate criteria that scientists can evaluate for concurrent validity. To achieve definitive status, the diagnostic benchmark must be independent of the disorder being judged. If addiction represents an uncontrollable impulse and not an uncontrolled habit, then there must be independent validation of the irrepresible impulse or the impaired regulatory mechanisms (e.g., Kipnis, 1997). Addiction cannot be limited to intemperate behaviors that only result in adverse consequences. If addiction is a primary disorder, independent of its consequences, then clinicians and scientists should be able to identify the disorder without knowing its consequences. Of note, clinicians do not serve as a proxy for definitive diagnosis, because clinicians who perform diagnostic evaluations are not as reliable as many people assume (e.g., Meehl, 1954, 1973; Rosenhan, 1973; Ziskin, 1970). Clinicians are extremely vulnerable to biases in clinical judgment. Consequently, diagnostic systems that rest on a mix of self-report and corroborating perspectives do not resolve the problem. Such a system can result in two biased perspectives: (a) the perception that an external object alone stimulated the excessive behavior pattern or (b) the cause of intemperance is a relatively stable underlying trait (e.g., weak character or addictive personality).
Dealing With the Absence of a Diagnostic Gold Standard

For addiction to emerge as a viable scientific construct, whether intemperate psychoactive drug use or behavior patterns in the absence of drug ingestion are the concern, investigators need to establish a gold standard against which clinicians can judge the presence or absence of the disorder. An independent gold standard—a definitive index against which diagnosticians and researchers can judge the presence or absence of a clinical state—will likely come from a combination of neurogenetic or biobehavioral attributes. Early neuroscience research has been encouraging. For example, altered dopaminergic and serotonergic functions have been found among pathological gamblers (e.g., Bergh, Sodersten, & Nordin, 1997; DeCaria, Begaz, & Hollander, 1998). Researchers have also identified biogenetic vulnerabilities among pathological gamblers (e.g., Comings, 1998; Slutske, Zhu, Meier, & Martin, 2010), and there is evidence to suggest that genetic markers might exist for novelty-seeking behavior among normal individuals that can predispose people to take chances (Benjamin et al., 1996; Ebstein et al., 1996).

Also, neuropsychological testing suggests that people with gambling disorders and alcohol dependence share deficits in impulsive decision-making (Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009). Finally, as noted earlier, new evidence has suggested that common central nervous system reward circuits are responsible for the desirable subjective experiences associated with the anticipation of substance use effects, the acquisition of money, and the appreciation of beauty (Aharon et al., 2001; Breiter, 1999; Breiter et al., 2001). The particular mix of neurogenetic and biobehavioral attributes, combined with the psychosocial context within which these factors exist, will determine whether and how addiction expresses itself for each individual. Any future gold standard for diagnosing addiction will require an algorithm and criteria that reflect this complex, multidimensional confluence of factors.

SEEING THE SYNDROME FOREST AMONG THE CATEGORICAL TREES: CONSIDERING THE DSM

Despite the scientific necessity for a diagnostic gold standard, clinical significance and human suffering require that treatment providers attend to addictive behavior whether or not they can distinguish it with precision, and the DSM-IV-TR (American Psychiatric Association, 2000) provides an operational guide to making such diagnoses. An especially appealing aspect of the DSM (i.e., American Psychiatric Association, 1987, 1994, 2000) is its multidimensional formulation of disorders in terms of (a) biological, psychological, and social aspects (i.e., a biopsychosocial approach) and (b) its appreciation for the spectrum of, and heterogeneity among, substance use problems. This heterogeneity of substances and the suggestion that different substance-using patterns represent different kinds of addiction reflect an Aristotelian approach to classification; this categorical strategy inadvertently implies that various substances cause different kinds of addiction. However, the current DSM-IV-TR is also Galileian in the sense that it recognizes the interaction of causal factors that stimulate and sustain excessive patterns of behavior. The recognition of these interactive factors helps to make the transition from a strictly categorical to a syndromal understanding of addiction.

During our earlier review of the DSM-IV-TR substance abuse and dependence definitions, we described two of the criteria (i.e., tolerance and withdrawal) that refer to the biological neuroadaptive features of addiction. The other DSM-IV-TR criteria (American Psychiatric Association, 2000, p. 197) include psychosocial standards: (a) Important social, occupational, or recreational activities are given up or reduced because of the substance, and (b) the substance use is continued despite knowledge of having a physical or psychological problem that is likely to have been caused or exacerbated by the substance. One of the most important social consequences of addictive behavior, driving while intoxicated, is the main topic of Volume 2, Chapter 15, this handbook. We would be remiss if we did not mention that addiction also has social precursors, which are described in Chapter 10 of this volume.

For example, Khantzian and Albanese (2008) wrote, Social and economic contexts or environments and the availability of addictive substances are factors that cannot be ignored in the development of substance
use disorders. It is not surprising that the occurrence of [substance use disorders] is significantly greater among poor, oppressed minorities, and victims of social upheaval and unrest. Yet the advantages of wealth and privilege do not protect against addictive vulnerability. It is said that time and money are risk factors for addiction. (pp. 32–33)

As many chapters in this volume show, the determinants of addiction are not limited to social influences. For example, Khantzian's (1997) self-medication hypothesis (SMH) emphasizes the psychological antecedents of addiction and serves as a primary influence on the development of the syndrome model of addiction. The SMH is a psychodynamic model that understands substance abuse as an attempt—by people with self-regulation deficits in affect, self-care, self-esteem, and interpersonal relationships—to provide some internal regulation (Khantzian & Albanese, 2008). The SMH acknowledges the psychological pain and suffering that often precede substance use and misuse. Sometimes this suffering stems from psychiatric disorders that are more prevalent among people with addiction, but sometimes only painful emotions associated with everyday life make a substance or behavior especially desirable as a vehicle to shift and attenuate these painful subjective states. The psychological correlates of addiction are not limited to precursors; there are also many psychological consequences of addiction, for example, exacerbation of mood and mood lability, anxiety disorders, and the emergence of guilt and shame. These emotional consequences contribute to sustaining addiction and stimulating new expressions of addiction.

The distribution and biopsychosocial determinants of substance use vary across population segments. Some people never use alcohol or drugs, some use occasionally, and some exhibit the DSM-IV-TR substance use disorders of abuse or dependence. In addition to these substance use disorders, the DSM-IV-TR classifies a variety of other substance-related disorders. For example, alcohol and drugs can cause intoxication and withdrawal, each of which suggests that there is an abuse or dependence problem, but neither of which is adequate to make such a diagnosis. Substance intoxication (American Psychiatric Association, 2000, p. 201) consists of (a) the development of a reversible substance-specific syndrome caused by recent ingestion of a substance and (b) clinically significant maladaptive behavioral or psychological changes that are due to the effect of the substance on the central nervous system and develop during or shortly after use of the substance. Similarly, withdrawal consists of (a) the development of a substance-specific syndrome caused by the cessation of, or reduction in, substance use that has been heavy and prolonged and (b) clinically significant distress or impairment in social, occupational, or other important areas of functioning caused by the substance-specific syndrome (American Psychiatric Association, 2000, p. 202). In addition, there are numerous other substance-induced disorders, such as substance-induced delirium, substance-induced psychotic disorder, and substance-induced mood disorder. The DSM-IV-TR therefore provides a spectrum of different addiction-related diagnoses that imply an underlying syndrome. In the remainder of this chapter, we relate a syndrome perspective to the assessment and treatment of addiction.

TRANSLATING THEORY INTO PRACTICE: FORMALIZING IDEAS INTO MODELS THAT INFLUENCE CLINICAL UNDERSTANDING

To approach the complex construct of addiction and make it pertinent to assessment and treatment in the clinical context, theoretical models provide useful guideposts, typically highlighting one aspect of the DSM-IV-TR's biopsychosocial approach. As Table 1.1 illustrates, stakeholders have used a wide variety of models to help explain addiction. However, just as models can clarify clinicians' understanding of complex behavior patterns and focus their perspective, these models can also blind them to alternative views. Observers of addiction—researchers, clinicians, public policy-makers, and lay people—typically evaluate excessive behavior with a particular model in mind. It is not unusual for a particular ideology, which might not be articulated explicitly, to inform the
**TABLE 1.1**

Common Perspectives on Addictive Disorders

<table>
<thead>
<tr>
<th>Perspective</th>
<th>Illustrative publications</th>
<th>Conceptualization</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Moral model</strong></td>
<td></td>
<td></td>
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<tr>
<td>Moral turpitude</td>
<td>Johnson (1986), Quinn (1891), Weems (1812)</td>
<td>Addictive behavior is a moral problem; the resolution of addiction requires piety and values conversion.</td>
</tr>
<tr>
<td><strong>Biological models</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disease or illness</td>
<td>Gamblers Anonymous, Alcoholics Anonymous, Narcotics Anonymous</td>
<td>Excessive behavior is a chronic disease for which there is no cure, except abstinence as a way to manage the disease.</td>
</tr>
<tr>
<td>Impulse control disorder</td>
<td><em>DSM-III, DSM-III-R</em> (American Psychiatric Association, 1980, 1994); Bergh, Sodersten, &amp; Nordin (1997); Cardoso (2002); Christenson et al. (1994); Comings et al. (1999); France (1913); Hollander, Buchalter, &amp; DeCaria (2000); Marks (1990)</td>
<td>Deficiencies or changes in neurobiological and genetic structures or activities influence regulatory problems that result in impulsive behavior patterns. Impulses are discharged too readily, and either short or more prolonged bursts of behavior result.</td>
</tr>
<tr>
<td>Reward deficiency and neurophysiological adaptation</td>
<td>Aharon et al. (2001); Bergh et al. (1997); Breiter, Aharon, Kahman, Dale, &amp; Shizgal (2001); Ebstein et al. (1996); Wise (1996)</td>
<td>Addiction results from shifts in the reward system and reflects changes in the pattern of activity among neurotransmitters or a deficit in the capacity of this system to yield pleasurable experiences.</td>
</tr>
<tr>
<td><strong>Psychological models</strong></td>
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<td></td>
</tr>
<tr>
<td>Bad judgment</td>
<td>Rosecrance (1985, 1988)</td>
<td>Addictive disorders (e.g., gambling) represent poor strategies, usually displayed by naive gamblers who do not fully understand the games they play.</td>
</tr>
<tr>
<td>Psychological deficiency and self-medication</td>
<td>Jacobs (1989), Khantzian (1975, 1997), Radó (1933)</td>
<td>Personality and emotional vulnerabilities invite addiction as an adaptive response that serves as an anodyne for these problems; under some conditions, these excessive behavior patterns can serve to keep people from regressing to a more primitive state. Addictive behavior patterns are viewed as an attempt to manage uncomfortable psychological states.</td>
</tr>
<tr>
<td>Psychodynamic neuroticism</td>
<td>Dodes (2002), Lindner (1950)</td>
<td>Addictive behavior patterns result from intrapsychic conflicts that can have roots in earlier developmental stages and the adaptations that follow.</td>
</tr>
<tr>
<td><strong>Social models</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychosocial</td>
<td>Orford (1985, 2001), Zinberg (1984)</td>
<td>Cognition and behavior pertaining to excessive behaviors are influenced by numerous moral and social factors that are responsible for their development and maintenance.</td>
</tr>
<tr>
<td>Public health issue</td>
<td>Korn &amp; Shaffer (1999); Marlatt (1996); Marlatt, Baer, Donovan, &amp; Kivlahan (1988)</td>
<td>Addiction is a multidimensional health risk for which potential biological, psychological, economic, and social costs must be considered. Addiction develops within a social context, and it likely has benefits as well as costs for the sufferer and the social setting.</td>
</tr>
</tbody>
</table>

(Continued)
Albanese and Shaffer

<table>
<thead>
<tr>
<th>Syndrome model</th>
<th>Shaffer et al. (2004)</th>
<th>Etiological multidimensional model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Addiction is an expression of multidimensional influences that affect vulnerable people. Not all of the symptoms and signs of addiction are present at all times. Behavioral and chemical expressions of addiction have similar etiology and consequences.</td>
</tr>
</tbody>
</table>


evaluation and the approach that might follow. Ideologies provide a framework for understanding and approaching problems. It is important to be aware of these ideologies. For example, a biologically minded clinician can view recidivistic alcoholic behavior as a mainly neurochemical problem and treat it with naltrexone. Although this is certainly a valid approach to treating alcohol dependence for some, it is important that clinicians not disregard other factors (e.g., family stress predisposing to relapse) that might also warrant additional treatment approaches (e.g., psychosocial interventions).

Addiction Models in Practice

Clinicians apply their models of addiction to make sense of those who seek their care. Shaffer and Robbins (1991) illustrated this point by presenting three descriptions of “Ms. S.” Each formulation of Ms. S. tells a different story about her problems and suggests a treatment plan on the basis of the belief system or systems used by the clinician-author. Consider the following biological, psychological, and behavioral assessment formulations of Ms. S.

Biological formulation. Ms. S is a 21-year-old woman with a 4-year history of depression and cocaine addiction. Before her use of cocaine, the patient experienced a major depression with severely impaired functioning. The patient has a positive family history for affective illness. Ms. S's mother has been hospitalized twice after depressive episodes, her maternal aunt is diagnosed with bipolar illness, and her father's alcohol dependence might mask a depression.

Psychological formulation. Ms. S is a 21-year-old woman with a 4-year history of drug abuse and self-destructive behavior. Her background of physical and sexual abuse and emotional deprivation has left her with low self-esteem, poor object relations, and an inability to tolerate intense affect. The patient's use of cocaine began at age 17 and continues to be her method of managing the demands of adult life for which she feels ill equipped.

Behavioral formulation. Ms. S is a 21-year-old woman who has been using cocaine excessively for the past 4 years. Her exclusive route of cocaine administration is intranasal. She uses between 0.50 g and 0.75 g of cocaine two to three times daily. Ms. S reports that cocaine acts as a negative reinforcer by relieving her feelings of malaise and discomfort. Because she has few alternative activities that produce positive reinforcement, her repertoire of behaviors has become narrow. Consequently, she has withdrawn from her primary social support systems.

Integrating and Applying Models of Addiction: A Syndrome Approach to Assessment

Although these biopsychosocial perspectives focus attention on specific aspects of addiction, they should not prevent clinicians and others from acknowledging additional perspectives that might enhance understanding and, therefore, advance prevention and treatment efforts. There is then the risk
that clinicians will view addiction only from their individual perspectives, failing to recognize that their view serves as a lens through which they screen the clinical evidence.

In reality, each perspective is not necessarily mutually exclusive. The SMH, for example, neither denies nor is at odds with biological, social, or other psychological understandings of addiction. In fact, the SMH makes clear that it is the combination of genetic predisposition and self-regulation deficits that, when mixed with substances of abuse, can lead to a substance use disorder. To illustrate,

In reviewing the literature on genetic factors in the susceptibility for alcoholism and addiction, it remains impressive how clear and strong a case is made for a genetic model of inheritance when even its strongest proponents concede genetic studies "are still in their infancy." (Khantzian, 1999, p. 371)

Khantzian and Albanese (2008) illustrated this further, when they wrote,

For example, one study has reported on a group of people who had neither an addiction nor a psychiatric disorder. The investigators found that nervousness during the day was associated with an increase in alcohol consumption in the evening. And the alcohol consumption, in turn, was associated with lower levels of nervousness. For those with a genetic predisposition to addiction, this kind of use could evolve into addiction. (p. 9)

The SMH reflects the importance of desirable shifts in subjective states that is central to the syndrome model perspective of the development of addiction. For addiction to emerge, the object of addiction must, at some time, provide a desirable subjective change. This is a unifying principle—biological or psychological—central to the development of addiction, whether activity or substance related. In his collected works, Khantzian (1999) stated that the SMH approach

should be considered in parallel with other approaches and not in competition with them. . . . I do not assume to have all the answers . . . only some special ones, answers that are often not enough considered in an era of biological psychiatry and empiricism. (p. 5)

He continued,

As in any approach there are potentials and limitations. . . . I believe it is the obligation of all of us, however, to try and appreciate the potentials and limitations of the various approaches we adopt, to find complementarity when we can, and, when we cannot, to explore the advantages of other perspectives. (pp. 5–6)

Similarly, Zinberg (1975)—whose approach emphasized the interplay of (a) the drug effect, (b) the user’s psychological set (i.e., expectations), and (c) the social setting within which drug use occurs in stimulating and sustaining the evolution of substance dependence—acknowledged that some people use illicit drugs to deal with painful affects. Of note, most people who experiment with substances do not develop a substance use disorder, probably because they have neither the biological predisposition nor the necessary combination of biology and self-regulation deficits or vulnerabilities.

The syndrome model of addiction provides an integrative approach by including interactive biological, psychological, and social influences within a developmental process; in addition, the syndrome model identifies the unique and shared consequences of addiction that require a cocktail of synergistic treatment components. We believe that it is the integration of clinical approaches that will be most helpful to the people seeking relief from their addictions. Most people come to treatment providers with a variety of problems in addition to addiction. At the very least, as the syndrome model suggests, they frequently have multiple expressions of addiction. They also not uncommonly have at least one comorbid psychiatric illness. These are the so-called “dual-diagnosis” patients. In addition, there are usually social issues, such as unemployment or homelessness, that require attention. To a greater or lesser degree, biology, psychology, and
The influence of these factors has treatment implications. For example, in the following section, we offer two brief illustrations of a conceptually integrated understanding of and clinical approach to our typically complicated patients.

Toward a Better Understanding of Addiction and Co-Occurring Disorders

Psychological distress often derives from human suffering. Frequently, subjective anguish is related to a psychiatric disorder. More often than not, a psychiatric disorder predated the onset of addiction, suggesting a causal relationship (e.g., Kessler et al., 2008). Similarly, Shaffer and Eber (2002), analyzing a large representative community sample from the United States, found that for most people with cocaine dependence and a comorbid psychiatric disorder, the latter preceded the onset of any cocaine use. One disorder associated with a notably high prevalence of addiction is posttraumatic stress disorder (PTSD). A recent and authoritative book about PTSD and addiction makes the point that the majority of patients follow a pattern in which the development of PTSD precedes the development of addiction (Ouimette & Brown, 2003). From a psychological perspective, Khantzian and Albanese (2008) have noted,

We have observed that PTSD is a disorder which disrupts essential aspects of human life—feelings, self-esteem, relationships, and self-care...these facets of life are fundamental to the capacity for self-regulation. Understanding how and why PTSD victims with [substance use disorders] suffer so much, and why substances of abuse become so compelling, is probably best understood by examining how disrupted these capacities become when vulnerable individuals experience major trauma. The suffering becomes pervasive and persistent, and does not readily yield or recede with time. Fortunately, or unfortunately, the suffering and symptoms of PTSD temporarily yield to the effects of substances of abuse and thus they run the risk of becoming addictive. (p. 62)

The syndrome model extends the psychological understanding of people with PTSD and addiction offered by the SMH by integrating recent neurobiological research. For example, new evidence has revealed that childhood trauma can cause persistent dysregulation of the body's stress response system, which includes the amygdala, hypothalamus, pituitary, and adrenals (De Bellis, 2002). Negative affective symptoms result. This circumstance might explain the increased use of psychoactive substances as an effort to self-medicate the distressful feelings. Of note, the stress system also activates in an attempt to balance the addiction-activated dopamine system. Increased substance use—as an attempt to cope with distressful feelings related to the stress system—further dysregulates the stress system in a troublesome cycle. The increased cortisol from this system can contribute to developmental problems in the brain's frontal and prefrontal cortex, with resultant failure in self-regulation and increases in impulsivity.

Depression offers another example in which apparently disparate components are better understood through an integrated, syndromal approach. From a psychological perspective, depression is a disorder of extreme emotional dysregulation. Accumulating biological evidence points to decreased dopamine transmission. As we know, amphetamine boosts dopamine transmission in the reward pathway. It turns out that nonaddicted people with severe major depression, under experimental conditions, describe an experience of greater reward after ingesting amphetamine than do nondepressed or mildly depressed people (Dunlop & Nemeroff, 2007). It is not much of a stretch to speculate that in everyday life some depressed people with certain blends of genes, biology, distress, and social circumstances would end up discovering amphetamine, liking it, and then liking it too much, with detrimental but understandable results.

An Integrated Understanding: Thinking Syndrome

The variety of symptoms associated with addiction reflect a complex syndrome instead of a series of
individual disorders. Overlapping symptoms might represent a common underlying factor. However, when a variety of symptoms are associated with a disorder, but not all the symptoms are always present, a syndrome is in evidence. The idea of addiction as a syndrome suggests that it has both common and unique components. A syndrome's common component (e.g., craving) is shared across various expressions of the disorder (e.g., alcohol abuse, opioid dependence, pathological gambling), whereas its unique components (e.g., betting increasing amounts of money) are specific to a single expression (i.e., pathological gambling). The shared component, which accounts for the comorbidity evidence, reflects broad individual differences that can vary along multiple dimensions (e.g., intensity and duration); the unique component distinguishes pathological gambling from other disorders and is specific only to it (e.g., Widiger & Clark, 2000). The complexity of biological and psychological attributes of the person, in combination with his or her social milieu, determines why a person manifests one expression rather than another.

In addition, although each expression of addiction has unique elements, addiction also has many signs and symptoms that it shares with other disorders (e.g., anxiety, depression, impulsivity). Sometimes these shared elements represent subsyndromal symptoms, and other times they indicate the presence of a comorbid disorder that commonly co-occurs with addiction (e.g., bipolar disorder, PTSD). Consequently, addiction is best thought of as a syndrome. From this perspective, as we describe in the next section and as others describe in later chapters, the most effective treatments for the variety of addictive behaviors will reflect a multimodal cocktail approach that attends to both the specific and the nonspecific elements of addiction.

TREATMENT AND RECOVERY: ADDICTION IS REVERSIBLE

Ideas have consequences, and addiction is no exception. For example, how clinicians think about addiction has an impact on public health services and public policy. In particular, the idea of addiction stimulates the idea of addiction treatment; in turn, the idea of treatment implies that addiction is reversible. The extent to which clinicians agree with this idea affects treatment planning and delivery. Many factors can influence the change from addiction to recovery. Some of these factors are nonspecific, and others are more direct. By moving beyond the view that the object of addiction is its cause, the syndrome model encourages a broader perspective about not only the etiology and maintenance of addiction but also the forces that influence recovery. In this section, we consider nonspecific factors that influence treatment outcomes; matching treatment to stage of change; unassisted recovery; and the settings, strategies, and components of assisted recovery; we end this discussion with a brief consideration of treatment efficacy and outcomes.

Nonspecific Factors That Influence Treatment Outcomes

As we noted earlier, a syndrome model suggests that there are aspects of any illness that are common to a variety of illnesses. Given this circumstance, it is no surprise that nonspecific or common factors account for a considerable amount of treatment outcome (e.g., Frank, 1961; Hubble, Duncan, & Miller, 1999). Hubble et al. (1999) suggested that nonspecific treatment factors include (a) the extratherapeutic attributes that clients bring with them to treatment (e.g., education, family support), (b) relationship factors displayed by the treatment provider (e.g., empathy, caring, warmth), and (c) the hope, expectancies, and placebo effects that are often associated with the start of treatment. Thus, the unique effects of particular treatment programs might easily be mistaken for nonspecific effects that accompany all treatment programs. Estimates of specific effects for treatment programs are best identified via empirical research. A full discussion of the nonspecific factors that influence treatment outcome is beyond the scope and intent of this chapter. However, there are many useful resources for readers interested in the factors common to successful treatment (e.g., Frank, 1961; Havens, 1989; Hubble et al., 1999; Imhof, Hirsch, & Terenzi, 1984; Maltsberger & Buie, 1974; Miller, 2000; Miller et al., 1995; Polanyi, 1967; Schon, 1983; Shaffer, 1994b; Shaffer & Robbins, 1991, 1995). Recognizing nonspecific treatment
effects within treatment episodes holds the potential to maximize treatment benefits.

Just as nonspecific treatment factors can increase the likelihood of positive treatment outcomes, they can also make things worse. When the relationship between clinician and treatment seeker is less than optimal, there is an increased risk of poor treatment outcomes. Arguably the most important adverse influence on the relationship is the presence of countertransference hate (Maltsberger & Buie, 1974; Shaffer, 1994b). In addition to relationship issues, therapist training and experience as well as opportunities for relapse prevention can influence treatment outcomes.

**Countertransference hate.**

When a therapist feels or acts toward a patient in ways that are neither part of the real relationship, rationally justified by the circumstances, nor part of the working alliance, appropriate to the terms of the treatment contract, he is manifesting countertransference. (Weiner, 1975, p. 244)

Think about your instinctive response to someone revealing he or she is getting married or divorced, expecting a child, detoxifying from a dependence-producing drug, or abstaining from gambling. In each case, there is a tendency to feel either congratulatory or sympathetic. When a therapist experiences one of these responses, this is countertransference. Rather than expressing either congratulations or sympathy, a more effective clinical posture would be to ask, “When did you decide?” or “How did you decide?” or “What’s that going to be like for you?” Congratulations might leave relapsing patients in a difficult position: If they share their difficulties with their therapists in the future, there is a risk that they might disappoint the treatment providers. This can limit what patients are willing to say to their therapists (Shaffer, 1994b).

Not only can countertransference influence patient behavior, but it can also influence therapist behavior. When patients experience ambivalence about changing their addictive behavior patterns, treatment providers often get frustrated, angry, and perhaps even malevolent (Shaffer, 1994b). Maltsberger and Buie (1974) suggested that clinical hate and rage consist of three important elements: (a) malice, (b) aversion, and (c) a mixture of these two emotions. Malicious impulses stimulate a disgust that can make patients seem loathsome (e.g., disgust for patients who are self-indulgent). Under these circumstances, patients can become the object of punishing, torturing impulses. However, Maltsberger and Buie were quick to note that malicious impulses are less dangerous than aversive tendencies because malice allows clinicians to maintain a clinical relationship with a patient whether he or she is abominated or loathed. Aversive impulses, in contrast, tempt the therapist to abandon the patient. Finally, unbearable malicious impulses often stimulate aversive actions. Malicious impulses are more painful to clinicians than the tendency to avoid (Maltsberger & Buie, 1974). Therefore, when patients stimulate malevolent impulses, clinicians tend to avoid having to confront or continue working with them.

**Matching treatment to stage of change.** Another important nonspecific clinical consideration is that matching treatment interventions to a particular patient on the basis of, among other things, where the patient is in relationship to changing behavior. Others have written extensively about stage-change concepts that have emerged as an important force in the treatment of addictive behaviors (e.g., Crowley, 1999; Prochaska, DiClemente, & Norcross, 1992; Prochaska, Norcross, & DiClemente, 1994; Quinn, 1891; Rollnick & Morgan, 1995; Shaffer, 1992, 1994b, 1997b; Shaffer & Robbins, 1995; see also Chapter 5 of this volume). In short, stage-change theory suggests that an evaluation of a person’s readiness to change and determination of his or her stage of change is one important step to formulating effective treatment strategies (Shaffer, 1997b; Shaffer & Robbins, 1995).

**Unassisted Recovery**

Conventional wisdom has assumed that there are only two ways out of any addiction: treatment or
death. The idea of addiction and its inherent influence on personal control has stimulated an erroneous belief that people with addiction cannot regain control and recover from addiction without the assistance of others. Given that addiction represents a syndrome of symptoms and signs, it is very important to understand that treatment is just one of several pathways to recovery. Seeking treatment is not the only means of allaying addictive behaviors. For example, almost every adult knows someone who has stopped smoking cigarettes without having participated in treatment (e.g., Schachter, 1982). The evidence that many addictive behavior patterns (e.g., drinking, gambling, smoking, drugging) resolve without formal treatment is considerable (e.g., Cunningham, Sobell, Sobell, & Kapur, 1995; Hodgin, Wynne, & Makarchuk, 1999; Schachter, 1982; Shaffer & Jones, 1989; Sobell, Cunningham, & Sobell, 1996; Sobell, Ellingstad, & Sobell, 2000; Waldorf, 1983; Waldorf & Biernacki, 1979, 1981; Waldorf, Reinerman, & Murphy, 1991; Winick, 1962).

Since Winick (1962) first described “maturing out” of narcotics use, the idea of recovery from addiction without treatment has caught the imagination of many clinical investigators. Recent research has suggested that recovery from addiction without formal or informal treatment is more common than previously expected (Cunningham et al., 1995; Hodgins et al., 1999; Sobell et al., 1996). Those who recover from addictive disorders without treatment tend to have milder forms of the disorder and fewer coexisting problems that complicate the recovery process. Nevertheless, people who recover from addiction without treatment are an interesting and important group to understand because they might provide important insights into treatment seekers and important elements of treatment that are not currently part of formal addiction treatment protocols. Similarly, assisted recovery need not always come from experience with clinicians, because self-help, including Alcoholics Anonymous, Narcotics Anonymous, or self-directed recovery, is also an option.

Assisted Recovery: Treatment

Addiction-related ideas indeed have treatment-related consequences. Not everyone can—or believes that they can—effectively evoke natural recovery processes. Consequently, although many of those who change might revise their behavior without treatment (e.g., Cunningham et al., 1995; Schachter, 1982; Shaffer & Jones, 1989; Sobell et al., 1996), a lot of people seek treatment and recovery via clinical pathways. In the remainder of this section, we briefly consider key treatment-related issues, including treatment settings, strategies, and components. These and other treatment-related matters are explored in much greater depth in Volume 2, Part I, this handbook.

Treatment settings. It is not surprising, given that addiction is syndromal, that people with addiction present for treatment along a spectrum of addiction involvement. Addiction treatment can take place in a variety of settings, with various levels of containment. At one end of the setting spectrum is treatment that occurs in a correctional context. At the other end is treatment that occurs in a client's home (e.g., outpatient detoxification) or workplace (e.g., employee assistance program) or in a church basement (e.g., Alcoholics Anonymous meeting). In between, the range of treatment settings includes hospital inpatient units, residential settings such as group homes and therapeutic communities, hospital partial care, day treatment programs, community mental health centers, and alcohol and drug treatment centers.

Treatment strategies. Addictive disorders evidence a variety of characteristics that invite treatment attention. As noted earlier, as a syndrome, addiction has common and unique elements. As with other syndromes (e.g., AIDS), addiction responds best to a comprehensive biopsychosocial approach (e.g., McLellan, Arndt, Metzger, Woody, & O'Brien, 1993). This treatment strategy considers and combines many different resources for helping patients. For example, pharmacotherapy and various forms of psychotherapy and counseling (e.g., individual, group, family, and financial) are combined to address biological, psychological, and behavioral problems. Each of these treatments is available in short- and longer term configurations. Medications are available to people with a range of addiction problems. These various treatment elements are both additive and interactive, a
circumstance necessary to deal with the syndromal nature of addictive disorders.

In the case of patients with comorbidities, comprehensive biopsychosocial treatment integrated with biopsychosocial psychiatric treatment produces better outcomes than less thorough care. Because no single modality is best, a cocktail approach to addiction treatment offers the most favorable outcomes. For example, McLellan et al. (1993) controlled the dose of methadone for 92 men who abused intravenous opiates to 60 mg to 90 mg per day. These study participants were then randomized to one of three levels of psychosocial treatment for a 6-month trial: (a) methadone alone (i.e., minimum methadone services; MMS); (b) methadone plus counseling (i.e., standard methadone services; SMS); and (c) methadone plus counseling and onsite medical–psychiatric, employment, and family therapy (i.e., enhanced methadone services; EMS). Although MMS patients exhibited reductions in opiate use, 69% of them met criteria for protective transfer to SMS (i.e., eight consecutive weekly urines positive for heroin or cocaine or three or more medical–psychiatric emergencies). This was significantly different from the 41% of SMS patients and 19% of EMS patients who met these criteria. End-of-treatment outcomes revealed that the SMS group showed significantly more and larger improvements than the MMS group, and the EMS group showed significantly better outcomes than the SMS group. MMS patients who had been protectively transferred to SMS showed significant reductions in opiate and cocaine use within 4 weeks. In summary, this study underscores the notion that combining adequate biological and psychosocial interventions results in better treatment outcomes for people struggling with addiction.

Treatment components: Pharmacotherapy and psychosocial modalities. In the remainder of this section, we examine some of the components of a syndromal approach to addictions treatment. Given the multidimensional nature of addiction, we examine biological (i.e., pharmacotherapy), psychological, and social aspects of treatment (e.g., psychotherapy, self-help). Each of these treatment components is not necessary in every case. Some treatment plans include only some of these interventions or use interventions for only a limited time.

As we noted earlier (and as is discussed at greater length in Volume 2, Chapter 3, this handbook), pharmacological agents can be one very important component of a comprehensive approach to treating substance use disorders. Several classes of agents could be useful. The first treatment agent type includes agonists. These medications are used as substitutes for substances for detoxification or maintenance treatment. Examples include methadone for heroin and nicotine patches for cigarettes. The second pharmacotherapeutic category includes antagonists or partial agonists, which compete with substances of abuse, preventing the substances from interacting at the nerve cell receptors where they exert their effects. For example, naltrexone is an antagonist, and buprenorphine a partial agonist, of heroin at the brain's opioid receptors. A third category is aversive agents, which cause an unpleasant effect if a substance is ingested while the person is taking the medication. An example is disulfiram, which prevents the complete metabolism of alcohol, resulting in the accumulation of an unpleasant metabolite. A fourth category is antitarget agents, which act to decrease craving for a substance. Naltrexone seems to work at least partially via this mechanism to decrease alcohol ingestion. A fifth class of pharmacological agents is antidrug-seeking agents other than those that are antitarget. Because of comorbidities, several other categories of agents are also frequently used. Among these are agents for comorbid medical problems, for example, HIV medications. Also, medications for comorbid psychiatric disorders (e.g., antidepressants) are often prescribed. Finally, some pharmacological agents are used to address both the addiction and the psychiatric disorder. For example, some antidepressants seem to improve both the depression and alcohol problems, and some of the newer antipsychotics seem to alleviate both the psychotic symptoms and the substance use.

We mentioned earlier that matching treatment to client is crucial. Suffice to say, this holds true when matching medication to patient subtype. For example, some studies (e.g., Pettinati, 2001) have suggested that different subgroups of alcoholic patients exhibit distinct responses to selective serotonin reuptake inhibitors (e.g., citalopram, fluoxetine),
and other data have suggested that men and women respond differently to these medications (Naranjo, Knoke, & Bremner, 2000).

Given the multidimensional and syndromal nature of addiction, the range of psychosocial treatment modalities is considerable. These modalities include self-help programs (e.g., Alcoholics Anonymous, Gamblers Anonymous), individual therapy, couples therapy, family therapy, network therapy, and group therapy. Furthermore, within these modalities, there are a variety of treatment approaches. For example, the various modalities can be psychoeducational, cognitive, behavioral, or psychodynamic. Cognitive–behavioral approaches, which focus on the thoughts and behaviors of substance abusers, include relapse prevention and dialectical behavioral therapy. These approaches teach clients cognitive and behavioral skills, such as relaxation and stress management techniques, to avoid substance use. Psychodynamic approaches focus on the intrapsychic forces predisposing to, and a consequence of, substance abuse. As noted before, among people who suffer with regulatory deficits, the SMH of addictions maintains that substance use is an attempt to control affect, self-care, self-esteem, and relationships by providing some or enhancing fragile internal regulation (Khantzian, 1985). The group therapy approach that derives from the SMH, modified dynamic group therapy, focuses on helping group members increase self-awareness of their self-regulatory deficits and improve the way in which they deal with these deficits (Khantzian, Halliday, & McAuliffe, 1990).

As noted earlier, when pharmacological interventions are combined with psychosocial treatments, clinical outcomes improve. In general, however, studies have not demonstrated the superiority of one psychosocial approach over others. For example, in the Harvard Cocaine Recovery Project, cocaine-dependent individuals received either (a) one of two group treatments or (b) no group modality. Both recovery training and self-help (i.e., a cognitive–behavioral approach) and modified dynamic group therapy were similarly more effective than treatment without a group modality (Khantzian et al., 1990). Similarly, Project MATCH found, for the most part, that people with alcohol disorders showed improvement, whether they received coping skills, motivational enhancement therapy, or 12-step facilitation (TSF) therapy (Project MATCH Research Group, 1997). Despite the observation that participants in Project MATCH improved regardless of treatment type, as with pharmacological treatments, there is some support for matching psychosocial interventions:

In the outpatient arm of the trial there was a matching effect for one specified contrast of the psychiatric severity hypothesis. Although the original conceptualization of this hypothesis was that individuals high in psychopathology would have better drinking outcomes with CBT [cognitive–behavioral therapy] rather than TSF, results indicated that there was no reliable difference in the outcome of high psychopathology subjects. On the other hand, subjects without psychopathology had significantly more abstinence in 7 of the 12 follow-up months when treated with TSF rather than CBT. The TSF advantage over CBT was on average approximately 4 more abstinent days per month. (Project MATCH Research Group, 1997, p. 12)

Just as one theory about addictions does not preclude the utility of other theories, one psychosocial treatment approach does not preclude others. For example, we have developed an inpatient state hospital dual-diagnosis treatment program and an outpatient substance abuse treatment program that include a variety of groups (e.g., relapse prevention, Alcoholics and Narcotics Anonymous, and modified dynamic group therapy) that are available for clinicians to mix and match according to patient needs identified during a comprehensive clinical evaluation.

**Considering Treatment Efficacy and Outcomes**

Currently, the information about long-term effectiveness with every existing treatment approach is limited. It is important to keep in mind that addiction treatment outcomes should be evaluated against
standards similar to the criteria clinicians use to assess the effectiveness of cancer treatments. That is, treatment for addictive behaviors should be judged against 5-year follow-up rates. Similarly, although recovery is best understood by treatment seekers as one day at a time, the scientific evaluation of recovery should determine how they have progressed over a 5-year follow-up period. Anything less than this time frame can be misleading. Many treatments provide short-term gains that do not last. Similarly, short-term gains should not provide a false sense of security that all risk of relapse is over. Because syndromes are multidimensional, these disorders do not typically respond favorably to a single treatment modality. Whether one views addiction as primary or secondary, unique or syndromal, intemperance inflicts human suffering (e.g., driving while intoxicated). If addiction is a primary disorder, it will often require professional assistance; if it is a disorder secondary to another problem, it still requires specialized modalities focusing on the object of addiction in addition to the problems related to the primary disorder. Future research will help clarify these theoretical, research, and clinical issues.

CONCLUSIONS

Addiction-related ideas have evolved dramatically from the earliest application of addiction to activities that reflected fervor and passion to later usage that chiefly described behaviors that involved the ingestion of psychoactive drugs. Currently, the concept of addiction is being transformed back to its roots by new theory and technology (e.g., neuroimaging) that describes a wide range of behaviors, including both patterns of excessive activity without psychoactive drug use (e.g., pathological gambling) and the substance use disorders (Holden, 2001; Shaffer, 1997a, 1999). The current state of the science suggests that addiction represents a syndrome, which encompasses a spectrum of disorders. This syndrome is characterized by variations in the manifestation and severity of signs and symptoms as well as an almost unlimited range of potential objects of addiction.

People subjectively experience the many apparently disparate objects of addiction in a similarly sought-after and positive way. Objects of addiction take advantage of the brain’s neurochemistry—and a psychosocial context—to achieve this shift in subjective experience. Because this subjective change is experienced as desirable (i.e., positive), the shift is inherently reinforced and therefore repetitively pursued. This sequence of activities begins a self-sustaining cycle of socially adverse events that often have some psychologically redeeming value. Of note, not everyone with a biogenetic vulnerability will develop addiction; similarly, not everyone without such predisposing risk factors will avoid addiction.

The idea of addiction and its evolution is a relatively new event within the history of language. The concept of addiction emerged only 400 years ago (Shaffer & Albanese, 2004). As we have argued, the concept of addiction has important consequences for understanding human behavior. Addiction implies that people have urges or cravings that make it difficult to control or manage their behavior. One important consequence of this seemingly simple idea is that people with addiction might not be able to conform their behavior to the requirements of community life because of impulses beyond their control. Ultimately, the idea of addiction stimulates its development and sustains its conceptual alter ego—control—which has its roots in 15th- and 16th-century language. Control originally referred both to relationships with one’s passions and to power relationships between people (Compact Edition of the Oxford English Dictionary, 1971). In the U.S. criminal justice system, these ideas about control have influenced the notion of mens rea, or “evil mind,” and serve as the architectural superstructure of insanity. However, addictive behavior does not share the full exculpatory power of insanity because most states assume some modicum of personal responsibility for taking the drink or drug that led to a criminal act (Shaffer & Albanese, 2004; Sullivan et al., 2008). The ideological struggle between intemperance and control has been waged throughout history within and across many domains, including intrapersonal psychology, personal behavior, public policy, public health, and religion. These conceptual alter egos need each other to survive, and clinicians must attend to both sides of this
complex and fascinating equation to provide optimal care.

Clinicians attempting to diagnose addiction face a complex set of assessment tasks. The challenges are both endemic to the nature of addiction and inherent in their subjective view of addiction. Clinicians must determine the multitude of biological, psychological, and social factors that contribute to an expression of addiction. In addition, clinicians frequently encounter issues that are not intrinsic to addiction but nevertheless closely related (e.g., anxiety, depression, deprivation, trauma). Although addiction is a dynamic process that waxes and wanes more than most observers and people with addiction typically go through phases of exacerbation and abstinence—there is considerable evidence that people with addiction recover both without treatment and with an array of treatments (e.g., Mclellan, 1994; Mclellan et al., 1992; Shaffer & Jones, 1989; Sobell et al., 1996, 2000).

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HISTORICAL PERSPECTIVES ON ADDICTION

Christopher R. Freed

On March 29, 2007, Joseph R. Biden Jr., then U.S. senator (D-DE) and chair of the Senate Judiciary Subcommittee on Crime and Drugs, issued a press release to announce Senate Bill S. 1011, Recognizing Addiction as a Disease Act of 2007. Biden's bill proposed renaming the National Institute on Drug Abuse as the National Institute on Diseases of Addiction, and the National Institute on Alcohol Abuse and Alcoholism as the National Institute on Alcohol Disorders and Health. In the press release, Biden (2007) explained,

Addiction is a neurobiological disease—not a lifestyle choice—and it's about time we start treating it as such. We must lead by example and change the names of our Federal research institutes to accurately reflect this reality. By changing the way we talk about addiction, we change the way people think about addiction, both of which are critical steps in getting past the social stigma too often associated with this disease.

Biden's (2007) appeal to promote addiction as a neurobiological disease by renaming the United States' most influential centers for substance abuse research underscores the latest medical and scientific thinking about chronic alcohol and drug use. However, the neurobiological model is only one of several perspectives on addiction. Indeed, since the early 19th century, no single model of addiction has received universal or lasting medical, scientific, or public support (e.g., see Campbell, 2007), perhaps best illustrated by the conceptual uncertainty about addiction that has historically pervaded the addiction field itself. According to Shaffer (1997), the editor-in-chief of this handbook,

If the field of addictions is to mature as have other domains of science, we must diligently work toward conceptual clarity. To develop theoretical precision, the field of addictions must escape from the cloak of partisan ideas. Conceptual clarity does not require that clinicians, researchers, and social policymakers agree. However, it does require that as addiction specialists we define our concepts and work precisely and operationally. (p. 1578)

The syndrome model of addiction (see the Introduction to this handbook) is a major advance toward the conceptual clarity and theoretical precision that Shaffer (1997) advocates. The syndrome model is etiologic: It proposes that addiction is a disorder with "multiple and interacting biopsychosocial
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antecedents, manifestations, and consequents—within and among behavioral and substance-related patterns of excess" (Shaffer et al., 2004, p. 367). In other words, addiction is caused by a combination of biological, psychological, and social vulnerabilities alongside exposure to and experience with substances or activities. The syndrome model of addiction acknowledges the myriad circumstances and conditions, both distal and proximal, under which individuals become susceptible to addiction.

In this chapter, I review a sample of historical perspectives on addiction to provide a framework to contextualize the syndrome model. In the first section, I review 19th-century perspectives on addiction, beginning in the early 1800s with the discovery of addiction and turning to Washingtonian "moral suasion" and the late 19th-century disease concept of inebriety. In the second section, I review 20th-century perspectives on addiction, starting in 1901 with Charles B. Towns of the Charles B. Towns Hospital, to explore the idea promoted by Alcoholics Anonymous that an allergy to alcohol caused alcohol addiction. After briefly examining the psychiatric model of addiction that dominated the first third of the 20th century, I trace the development and popularization of the disease concept of alcoholism. Next, I present social and behavioral science perspectives on addiction from the second half of the 20th century that expose social and cultural factors that cause and contribute to chronic alcohol and drug use. Finally, in the third section of the chapter, I consider how, today, the fields of addiction medicine and addiction psychiatry conceptualize addiction and reveal how addiction medicine physicians and addiction psychiatrists are still learning about addiction, before contextualizing the syndrome model with respect to the addiction paradigms that it follows.

19TH-CENTURY PERSPECTIVES ON ADDICTION

Early 19th-century Americans considered alcohol “the good creature of God” (Levine, 1978; Rorabaugh, 1979). They drank at home; at work; on holidays; and during family, social, and civic events such as weddings, funerals, house raisings, barbeques, dances, land clearings, elections, and court sessions. Americans drank alcohol to celebrate business deals and farm harvests, and the tavern was central to community life (Rorabaugh, 1979). Americans drank all day, every day, and when they drank they got drunk (Levine, 1978): “Early nineteenth-century America may not have been ‘a nation of drunkards,’ but Americans were certainly enjoying a spectacular binge” (Rorabaugh, 1979, p. 21). During 1830, annual per capita consumption of distilled liquor such as rum, whiskey, gin, and brandy surpassed 5 gallons (Rorabaugh, 1979). Today, annual per capita consumption of distilled alcohol is 0.73 gallon (National Institute on Alcohol Abuse and Alcoholism, 2009).

Most early 19th-century Americans were not obviously bothered by widespread drinking and drunkenness. Those who were troubled likely regarded such behavior as an inevitable outcome of colonial American drinking practices (see Levine, 1978). The nation’s political leaders, however, expressed a more acute concern about pervasive alcohol consumption. As early as 1736, Benjamin Franklin wrote about the “mischief” that accompanied drunkenness, including theft and violence. John Adams remarked that business competition forced community taverns to serve the destitute, depraved, and disorderly of society, which put the public at risk (Rorabaugh, 1979). Over time, then, Americans recast alcohol as the “demon rum” in light of the crime, poverty, family discord, and general disorder that powerful, educated, and upper class citizens attributed to common drunkenness. A growing capitalist elite blamed drinking for the poor health and general suffering of workers and consumers and maintained that alcohol threatened U.S. productivity, commerce, and progress (Levine, 1978; Rorabaugh, 1979).

Early Responses to “The Great Destroyer”

Benjamin Rush, a prominent colonial physician who advised the likes of George Washington and Thomas Jefferson and a cosigner of the Declaration of Independence (Kobler, 1973), articulated much of this concern. In an essay titled An Inquiry Into the Effects of Ardent Spirits on the Human Body and Mind, first published in 1784, Rush warned Americans about the dangerous effects of chronic drinking. “We see poverty and misery, crimes and infamy, diseases
and death, are all the natural and usual consequences of the intemperate use of ardent spirits" (Rush, 1784/1811, p. 9). On the basis of this concern, Rush developed the first medical model of addiction. Specifically, Rush argued that distilled alcohol was inherently addicting, similar to today's conventional wisdom about illicit drugs such as crack cocaine. In addition, Rush described alcohol addiction as a disease, or a loss of control over drinking that only permanent abstinence could cure (see Levine, 1978).¹ "My observations authorize me to say," Rush cautioned, that persons who have been addicted to [ardent spirits], should abstain from them suddenly, and entirely. "Taste not, handle not, touch not," should be inscribed upon every vessel that contains spirits in the house of a man, who wishes to be cured of habits of intemperance. (p. 32)

Mindful of the nation's spiritual zeal, Rush (1784/1811) asked religious leaders to help disseminate his message:

Ministers of the gospel of every denomination, in the United States!—aid me with all the weight you possess in society, from the dignity and usefulness of your sacred office, to save our fellow-men from being destroyed by the great destroyer [italics added] of their lives and souls. In order more successfully to effect this purpose, permit me to suggest to you, to employ the same wise modes of instruction, which you use in your attempts to prevent their destruction by other vices... Denounce by your preaching, conversation and examples, the seducing influence of toddy and grog when you aim to prevent all the crimes and miseries, which are the offspring of strong drink. (p. 23)

The 19th-century temperance movement endorsed Rush's model of alcohol addiction. Temperance initially promoted moderate alcohol consumption, or "the sobriety of the previously sober" (Baumohl & Room, 1987, p. 138), but as the movement matured to include more than a half million people, one of the largest social movements of the 19th century, the idea that chronic drunkards had lost control over their drinking dominated temperance ideology (Levine, 1978, 1984). Temperance advocates believed that if they could eliminate drunkenness from society, they could also solve the alcohol-related social problems that Rush identified. Indeed, "the elimination of drunkenness would prove crucial to avoiding internal civil disruption—thus literally preserving the republican experiment itself" (Lender & Martin, 1987, p. 66). Yet the temperance movement never effectively explained how to reform those addicted to alcohol. Instead, the Washingtonian movement provided the nation's first organized method of addiction treatment (Baumohl & Room, 1987). Founded in 1840, the Washingtonian movement consisted of working-class, recovering drunkards whose motto, "Let every man be present, and every man bring a man," led Washingtonians to taverns, their former drinking circles, or wherever else chronic drunkards gathered to recruit new members (see White, 1998, p. 9). Washingtonians attended "experience lectures" during which sober members of the movement described their drinking history as others listened, shared their own personal stories, and pledged permanent abstinence from alcohol (Baumohl & Room, 1987). Washingtonian "homes" in Boston and later in San Francisco and Chicago attempted to rebuild the character of chronic drinkers through a combination of self-determination and moral and spiritual support from other recovering alcohol addicts. In sum, the Washingtonians tried to cure chronic drunkards using "moral suasion" (White, 1998).

By the late 1840s, however, the Washingtonian movement had ended because of its lack of leadership, organization, and treatment focus. "The Washingtonian movement was like a cry of 'Fire!' in a crowded theater. It had aroused great emotion to get everyone outside the theater, but then no one was

¹Although Rush (1784/1811) promoted permanent abstinence from distilled alcohol, he encouraged Americans to consume cider, malt liquor, wine, and coffee for enjoyment and nourishment. "If we reject spirits from being a part of our drinks," Rush asked, "what liquors shall we substitute in their room?" (p. 11).
ensure what to do” (White, 1998, p. 12). Although the exact reasons for the Washingtonians' failure remain unknown, perhaps one explanation relates not only to the model of moral suasion but also to the physicians who became influenced by the Washingtonian movement, so-called “neo-Washingtonians” (Baumohl & Room, 1987). Neither the model of moral suasion nor the neo-Washingtonians supported the disease concept of alcohol addiction. Neo-Washingtonians, in particular, conceptualized chronic drinking as a complex social, psychological, and spiritual issue but not as a medical problem (Baumohl & Room, 1987). For instance, Albert Day of the Boston Washingtonian home emphasized the moral regeneration of drunkards and not a loss of control over drinking. “I have no desire to apologize for gross intemperance,” Day said, “or to attribute it to an uncontrollable force or involuntary action. . . . None are less willing to so consider it than the victims themselves, who know that self-control, though weakened, is not wholly lost” (Day, 1867, p. 49). Most Americans, however, accepted the Rush and temperance doctrine on alcohol addiction.

The Disease Concept of Inebriety

During the second half of the 19th century, civil war in the United States weakened the relevancy and influence of the temperance movement. With the demise of the Washingtonians as well as failed legislative and punitive efforts to solve alcohol addiction, inebriety specialists intervened (E. M. Brown, 1985). Late 19th-century inebriety specialists typically defined inebriety as the disease of chronic drunkenness, but they also used the term inebriety to describe opium and cocaine addiction as well as excessive coffee and tea consumption (e.g., see American Association for the Study and Cure of Inebriety, 1893). Inebriety specialists proposed that degeneration and neurasthenia caused inebriety (Jaffe, 1978).

In 1857, Benedict-Augustin Morel, a French psychiatrist, developed the concept of degeneration. Broadly defined as “a morbid deviation from an original type” (Chamberlin, 1985, p. 265), degeneration was the theory that individuals with a physical, psychological, or immoral deficiency transmit an even worse variant of this trait to members of the next generation, and so on throughout future generations, until an entire family line becomes extinct. Among the environmental factors that caused degeneration was exposure to, and abuse of, alcohol and other drugs (Wilkerson, 1966). As inebriety specialists interpreted degeneration, inebriates experienced a physiological or psychological decline from normalcy and morality to “abnormal, instinctive, and animalistic levels” (Jaffe, 1978, p. 140).

In 1869, the neurologist George Miller Beard discovered neurasthenia, a nervous condition that Beard believed was caused by modern advancements such as clocks and watches, the telegraph, excessive noise, railroad travel, and the proliferation of business and politics. Progress created new demands on Americans, Beard (1881) argued, especially among the educated and upper class who experienced these changes before other groups. The ensuing psychological strain and nervous exhaustion increased susceptibility to the addictiveness of alcohol and thus to the disease of inebriety. “Notably in our country,” Beard warned, “where nervous sensitiveness is seen in its extreme manifestations, the majority of brain-workers are not entirely safe so long as they are in the habit of even moderate drinking” (Beard, 1881, p. 38). Indeed, the heightened sensitiveness of Americans forces them to abstain entirely, or to use in incredible and amusing moderation, not only the stronger alcoholic liquors, whether pure or impure, but also the milder wines, ales, and beers, and even tea and coffee. (Beard, 1881, p. 31)

T. D. Crothers, arguably the most influential inebriety specialist of the late 19th century, incorporated aspects of both degeneration and neurasthenia into his view of inebriety. Crothers was the superintendent of Walnut Hill Home, a treatment facility for alcohol and opium inebriates in Hartford, Connecticut, as well as the longtime secretary of the American Association for the Study and Cure of Inebriety, an organization of medical professionals that promoted inebriety as a disease (Blumberg, 1978). Crothers (1881) identified three categories of inebriates. For the U.S middle-class “brain labor” (Crothers, 1881, p. 180), burdened by overwork and mental anxiety, “the use of alcohol commences as a
temporary relief, and culminates in a toxic condition or intoxication” (Crothers, 1881, p. 181). Wealthy inebriates, who justified their alcohol consumption as part of a healthy lifestyle, inherited a nerve or brain condition that caused them to periodically lose control over their drinking. The most common category of inebriates, Crothers (1881) stated, were “strongly marked cases of defective brain and nerve force, alternately criminals, insane and inebriates, from accident and circumstance” (p. 179). According to Crothers (1877), “Nothing can be more appalling than this vast army of moving drunkards, drifting from place to place, a perpetual burden and tax on society, scattering vice and crime everywhere” (p. 117). Crothers (1881) recommended institutionalizing these inebriates, permanently if necessary, in special inebriate asylums to protect the public from their “feeble will and impulse” (p. 173).

Following Crothers’s lead, late 19th-century inebriety specialists committed inebriates to asylum treatment to not only eliminate chronic alcohol and drug use from society but also to increase the medical and scientific legitimacy of inebriety as a disease (E. M. Brown, 1985; White, 1998). To be sure, the disease concept of inebriety had several detractors. The Association of Medical Superintendents of American Institutions for the Insane insisted that inebriety was a temporary condition, not a permanent disease, that diverted resources from more important psychological disorders (see Jaffe, 1976). Other medical professionals believed that long-term drug consumption or opiate treatment for physical or emotional pain caused inebriety, not psychological problems that stemmed from inherited traits or social change (see Courtwright, 2001). Religious leaders regarded the disease concept of inebriety as the “medicalization of sin” (see White, 1998, p. 26) or, as one physician concluded, an excuse for chronic drunkards to continue drinking (Bucknill, 1877). Therefore, the remedies for drink craving, as it has been called, are all moral. Even the inebriate asylum is a moral remedy directed to change the character, not to cure the disease; for if cure only were aimed at, the drunkard would be dismissed in a few days, as soon as he could digest his

food and sleep o’ nights like sober folk; but the cry is that he must be detained for not less than two years, in order that his character may be changed by absence of temptation. This surely is directed to the moral side of his nature. (Bucknill, 1877, p. 439)

By the early 1900s, most Americans recognized that inebriety specialists had failed to reduce inebriety in society as well as to solve social problems such as unemployment and homelessness that inebriety specialists claimed chronic alcohol and drug use caused. Increasingly, the public described people with addiction as pests and criminals who should be punished, not as victims of a disease who deserved medical care (Blumberg, 1978). Nevertheless, perspectives on addiction continued to evolve.

20TH-CENTURY PERSPECTIVES ON ADDICTION

In 1901, Charles B. Towns, a businessman with no medical training in addiction, opened the Charles B. Towns Hospital, a private alcohol and drug treatment facility in New York City. Towns (1912) argued that “the ‘American type,’ highly nervous, living under pressure, always going to the full limit, or beyond” (p. 2) was particularly susceptible to substance abuse. However, Towns (1917a) rejected the notion that chronic drunkenness was a disease:

Medical men have been largely responsible for making the alcoholic believe that alcoholism is a disease. Stop and think for a moment and you will see how absolutely absurd this is! The only extent to which a man can be alcoholically diseased is the extent to which he has been taking alcohol, in such quantities and in such regularity over a certain period that he has established a certain definite tolerance; and if he has been taking it in sufficient quantities, this tolerance would mean, in the end, that if he were suddenly deprived of his stimulant, delirium tremens and all of the unfavorable consequences that come out of that condition
would result. Now, delirium tremens is due to nothing more or less than alcoholic poisoning. If this man is medically unpoisoned, he cannot experience any such condition. Nor can the unpoisoned alcoholic have any physical or mental craving for alcohol. (pp. 26–27)

Towns (1917b) described himself as a “pioneer” (p. 7) with the “one as yet entirely successful known method” (p. 9) for treating addiction. This method consisted of the “persistent administration of the belladonna mixture in small doses and the thorough elimination by means of some form of mercury as a cathartic” (Lambert, 1913, p. 1933). In other words, patients at Towns Hospital were administered extract from the hallucinogenic belladonna and hyoscyamus plants along with elements of the cure-all xanthoxylum shrub (Markel, 2010; Pittman, 1988). Alexander Lambert, professor of clinical medicine at Cornell University Medical College and a national expert on addiction, popularized this treatment method in the medical literature. Lambert (1909) acknowledged,

If some years ago any one had told me that it was possible to take away the desire for morphine, cocaïn [sic], or alcohol in less than five days with a minimum of discomfort and suffering to the patient, I should have felt justified in treating the statement with a polite skepticism. Such, however, is the fact, if the treatment ... is carefully carried out. (p. 985)

The Allergy Model of Alcoholism and Alcoholics Anonymous
William Griffith Wilson, a failed New York City stockbroker and chronic drinker, became the most renowned patient treated at Towns Hospital. In 1934, Wilson, today known as Bill W., the cofounder of Alcoholics Anonymous, was admitted to Towns Hospital for the third time. During his treatment he experienced a “hot flash” (see Pittman, 1988, p. 169) and exclaimed,

“If there is a God, let Him show Himself! I am ready to do anything, anything!”

Suddenly the room lit up with a great white light. I was caught up into an ecstasy which there are no words to describe. It seemed to me, in the mind’s eye, that I was on a mountain and that a wind not of air but of spirit was blowing. And then it burst upon me that I was a free man. (Alcoholics Anonymous Comes of Age, 1957/1971, p. 63)

Wilson first thought that his hot flash was a hallucination. He turned to William D. Silkworth, the medical director of Towns Hospital, for an explanation. According to Silkworth, Wilson had a spiritual conversion that was powerful enough to cure his addiction to alcohol (Alcoholics Anonymous Comes of Age, 1957/1971). Wilson, a lifelong agnostic, doubted Silkworth’s assessment until a friend named Ebby, a recovering alcoholic, urged him to read The Varieties of Religious Experience by the philosopher William James (1902/1999), a mix of “religious impulses with other principles of common sense” (p. xiv). Wilson read James “from cover to cover” (Alcoholics Anonymous Comes of Age, 1957/1971, p. 64) and interpreted his hot flash as a deflation of character, caused by suffering, that only a “higher power” could cure. Equally as important, Ebby convinced Wilson of the positive impact that one alcoholic could have on another. As Wilson stated,

On Dr. Silkworth’s say-so alone maybe I would never have completely accepted the verdict, but when Ebby came along and one alcoholic began to talk to another, that clinched it. My thoughts began to race as I envisioned a chain reaction among alcoholics, one carrying this message and these principles to the next. More than I could ever want anything else, I now knew that I wanted to work with other alcoholics. (Alcoholics Anonymous Comes of Age, 1957/1971, p. 64)

“The whole of what became Alcoholics Anonymous appeared in these words—almost” (Kurtz, 1979, p. 21). Wilson also learned from Silkworth that alcoholics are physically allergic to alcohol. “The inevitable conclusion is that true alcoholism
is an allergic state," Silkworth (1937, p. 251) said, "the result of gradually increasing sensitization by alcohol over a more or less extended period of time. The constancy of the symptoms and progress is too fixed to permit any other explanation." Wilson left Towns Hospital with a new perspective on alcoholism: He was physically sick, not morally weak as most Americans labeled alcoholics (Johnson, 1973).

During the months that followed, Wilson was surprised at how difficult it was to explain the concept of alcoholism as an illness to other chronic drinkers. "You've got to deflate these people first," Silkworth told him.

So give them the medical business, and give it to them hard. Pour it right into them about the obsession that condemns them to drink and the physical sensitivity or allergy of the body that condemns them to go mad or die if they keep on drinking. (Alcoholics Anonymous Comes of Age, 1957/1971, p. 68).

In 1935, Wilson met an alcoholic named Robert Holbrook Smith, a surgeon from Ohio known as "Dr. Bob." "I remembered all that Dr. Silkworth had said," Wilson recalled. "I just talked away about my own case until he [Smith] got a good identification with me, until he began to say, 'Yes, that's me, I'm like that'" (Alcoholics Anonymous Comes of Age, 1957/1971, p. 68).

In 1935, Wilson and Smith founded Alcoholics Anonymous (AA). In 1939, Wilson, Smith, and other early members of AA assembled their key documents into Alcoholics Anonymous: The Story of How More Than One Hundred Men Have Recovered From Alcoholism, dubbed the "Big Book" because its authors had it printed on heavy-duty paper so that their work might appear more substantial (S. Brown & Brown, 2001). The Journal of the American Medical Association criticized Alcoholics Anonymous as "a curious combination of organizing propaganda and religious exhortation ... [with] no scientific merit or interest" ("Book Notices," 1939, p. 1513). The Big Book's centerpiece, outlined in the chapter "How It Works" (see Alcoholics Anonymous, 2001, pp. 58–71) was the 12-step program of recovery (see Exhibit 2.1).

The Big Book included testimonials from recovering alcoholics about the effectiveness of the 12-step program as well as praise from Silkworth himself. "The subject presented in this book seems to me to be of paramount importance to those afflicted with alcoholic addiction," Silkworth wrote. "I say this after many years' experience as Medical Director of one of the oldest hospitals in the country treating alcoholic and drug addiction" (Alcoholics Anonymous, 1957/1971, p. 68).

2A full discussion of how Wilson and Smith founded AA is beyond the scope of this chapter. For two excellent histories, see Kurtz (1979) and Pittman (1988).
Silkworth's account of Wilson's last stay in Towns Hospital appears in every copy of the Big Book:

In late 1934 I attended a patient who, though he had been a competent businessman of good earning capacity, was an alcoholic of a type I had come to regard as hopeless. In the course of his third treatment he acquired certain ideas concerning a possible means of recovery. As part of his rehabilitation he commenced to present his conceptions to other alcoholics, impressing upon them that they must do likewise with still others. This has become the basis of a rapidly growing fellowship of these men and their families. This man and over one hundred others appear to have recovered. I personally know scores of cases who were of the type with whom other methods had failed completely. These facts appear to be of extreme medical importance; because of the extraordinary possibilities of rapid growth inherent in this group they may mark a new epoch in the annals of alcoholism. These men may well have a remedy for thousands of such situations. (Alcoholics Anonymous, 2001, pp. xxv–xxvi)

By 1945, AA had 15,000 members (Johnson, 1973). A positive article about the fellowship in The Saturday Evening Post (see J. Alexander, 1941) generated a great deal of interest in the organization. Wilson, of course, credited AA's early success to Silkworth. "Dr. Silkworth taught us how to till the black soil of hopelessness out of which every single spiritual awakening in our fellowship has since flowered" (Alcoholics Anonymous Comes of Age, 1957/1971, p. 13).

The Psychiatric and Disease Models of Alcoholism and Drug Addiction

During the first third of the 20th century, most people with addiction could not afford treatment in private facilities such as Towns Hospital that had addiction specialists such as Silkworth on staff. In fact, because most medical professionals considered addiction treatment a waste of time and resources, any treatment at all for addiction, affordable or not, was difficult to find (Johnson, 1973). However, a small number of psychiatrists specialized in addiction to reduce what, from their perspective, was a growing medical and social problem (Johnson, 1973). These psychiatrists conceptualized addiction as a symptom of a mental disorder.

In 1908, for example, Karl Abraham (1908/1927) argued that chronic drinkers consumed alcohol to repress their "sexual complexes" (p. 88). "For the same reason that the neurotic protects his symptoms," Abraham suggested, "the drinker fights in defense of his alcoholism. It represents his sexual activity" (p. 88). Lawrence Kolb (1925), whose work for the U.S. Public Health Service Lexington Narcotic Hospital legitimized narcotic addiction as a mental problem (see Courtwright, 2001), said that people with addiction use drugs to deal with "a sense of inadequacy, imagined or real, or with unconscious pathological strivings" (Kolb 1925, p. 304). Furthermore, alcoholics drink for the same reasons that other individuals abuse different psychotropic drugs: "The so-called intoxication and narcotic impulses are identical" (Kolb, 1925, p. 313). Robert P. Knight (1938) identified overprotective mothers and unaffectionate fathers as among the sources of chronic drinking. Successful treatment required a "reconstruction of the personality" (Knight, 1938, p. 1444). Finally, Karl Menninger (1938) believed that priests, social workers, and prohibitionists mistakenly linked alcohol addiction to life difficulties and diseased states. "If it were so," Menninger insisted, "we should all become alcoholics" (p. 147). Chronic drinkers are insecure and childlike. As such, alcohol addiction is not a disease, Menninger concluded, but "a suicidal flight from disease, a disastrous attempt at the self-cure of an unseen inner conflict" (p. 147) that requires "a complete and thoroughgoing reconstruction of the entire personality" (p. 159).

Not all mental health professionals endorsed the psychiatric perspective on addiction. For instance, Harry M. Tiebout, the medical director of Blythewood Sanitarium, a psychiatric and drug treatment center...
in Greenwich, Connecticut, was a steadfast advocate of the AA allergy model of addiction. According to Tiebout (1951/1999a), “to insist on treatment of the original causes is like focusing upon the cause of the life-threatening fever or upon the irritation leading to cancer. The cause and the origins are irrelevant to the immediate danger” (p. 8). From Tiebout’s (1956/1999b) perspective, most medical professionals lacked “expert” knowledge about addiction. In 1937, the Research Council on Problems of Alcohol adopted a similar position. The Research Council (1940) sought to “ascertain the facts” (p. 105) about alcoholism and to disseminate this information to the public. Made up of alcohol and addiction experts such as Lawrence Kolb and Howard W. Haggard of Yale University, the Research Council planned a systematic program of study on alcoholism on the basis of a review of the scientific literature. The Research Council hired E. M. Jellinek, a biometrician with no experience in the alcohol studies field, to conduct its literature review. In this review, titled “The Study of the Effects of Alcohol on the Individual” (see Jellinek & Jolliffe, 1940), Jellinek cataloged and abstracted 3,000 scientific works on alcohol. His efforts and acquired knowledge impressed Haggard, who wanted to integrate a social scientific approach into his alcohol research at the Laboratory of Applied Physiology at Yale. In 1943, Haggard formed the Section of Alcohol Studies at his laboratory and named Jellinek the section’s director (Page, 1988, 1997).

Jellinek was at the Yale Section for 1 year when Ruth Fox, a New York City internist, contacted him on behalf of her close friend and colleague Marty Mann. Mann was a former patient of the psychiatrist Harry M. Tiebout and the first woman to stay sober by following the AA 12-step program (see Alcoholics Anonymous, 2001, pp. 200–207). Mann’s experience in AA convinced her not only that alcoholics were sick but also that medical professionals and the public were largely unaware that alcoholism was an illness (Mann, 1944). “Convinced of the validity of the A.A. ideology, [Mann] envisioned a society in which these ideas would be common knowledge and accepted as scientific facts” (Johnson, 1973, p. 266). Mann devised a three-part plan to achieve these objectives. First, she recommended a series of lectures on alcoholism for doctors, nurses, and community leaders who could then publicize the medical “facts” about alcohol addiction. Second, Mann wanted to open alcoholism information centers nationwide to advertise alcoholism as an illness. Last, Mann thought that by lobbying hospitals to admit alcoholics for treatment, health care professionals and the public could be persuaded that alcoholism was a legitimate medical condition (Johnson, 1973).

Jellinek was so energized by Mann’s plan that he offered Mann financial and scientific support from the Yale Section. As a result, in 1944 Mann founded the National Committee for Education on Alcoholism. “The total effort of the National Committee,” Mann (1944) explained, will be directed toward bringing the facts of alcoholism before the public and spreading knowledge of alcoholism by word of mouth and through the printed page, and fostering the creation of clinics and other facilities for the rehabilitation of tens of thousands of alcoholics. (p. 358)

Mann hoped to expose the “unguarded secret” (p. 354) that alcoholism was a disease, and the National Committee quickly received nationwide attention. Stories about the medical model of alcoholism appeared in publications such as Time magazine as Mann delivered hundreds of speeches about alcoholism and established local affiliates of the National Committee across the United States (Johnson, 1973).

Mann used Jellinek’s research, of course, to support her efforts. In 1946, Jellinek analyzed data that the AA Grapevine, the official newsletter of AA, collected about its readership’s drinking history. Foreshadowing contemporary stage-change models, Jellinek (1946) identified basic, intermediate, and compulsive phases of alcoholism that ultimately led

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1Fox was married to an alcoholic named McAllister Coleman, a prominent New York socialist and candidate for Congress (Johnson, 1973). In 1941, after reading about AA in The Saturday Evening Post (see J. Alexander, 1941), Fox attended an AA meeting to see whether the fellowship could help her husband. Mann, at the time a new member of AA, spoke at this meeting. Fox and Mann tried unsuccessfully to keep Coleman sober (S. Brown & Brown, 2001).
to a terminal phase, or a loss of control over drinking. Jellinek (1946) described these results as “highly suggestive and interesting” (p. 79) but the questionnaire from which they derived was “superficial” (p. 78). “Statistical thinking should not begin after a survey or an experiment has been completed but should enter into the first plans for obtaining the data. In the questionnaire under consideration this requirement was neglected” (p. 5). Jellinek drew his results from 98 male AA members. He excluded questionnaires that women completed, 15 in total, claiming that the data were inadequate and contradicted information from the male portion of the sample. Jellinek discarded 17 additional questionnaires that AA Grapevine readers improperly completed and one questionnaire on which 28 members of one AA group combined and averaged their answers. Overall, the questionnaire response rate was 10% out of the AA Grapevine’s circulation of 1,600 copies (Jellinek, 1946).

In 1945, the sociologist Selden D. Bacon, Jellinek’s successor as the director of the Yale Section of Alcohol Studies, argued that modern society neglected the medical model of alcoholism (e.g., see Bacon, 1943, 1945/1972) might have been the deciding factor (S. Brown & Brown, 2001; Johnson, 1973).

The Christopher D. Smithers Foundation provided the funding for the Disease Concept of Alcoholism (Jellinek, 1960). Named after one of the founders of IBM, the Smithers Foundation spent at least $12 million to promote alcoholism as an illness. Its founder, R. Brinkley Smithers, was a recovering alcoholic whom Jellinek treated and who personally spent more than $23 million to support the idea that alcoholism was a disease (White, 1998).
offers numerous advantages but that the specialization, stratification, and individualism of modern society increases emotional insecurity. "Since alcohol can reduce the impact, can allow escape from the tensions, fears, sensitivities, feelings of frustration, which constitute this insecurity, its role will be more highly valued" (Bacon, 1945/1972, p. 192). In short, the "functional foundation of alcohol" (Bacon, 1945/1972, p. 190) in modern society can cause chronic drinking. Horton (1945/1972), an anthropologist, suggested that drinking reduces anxiety from danger. In primitive societies, sickness and survival are at issue. In modern society, poverty, job dissatisfaction, powerlessness, and rapid social change cause alcohol addiction. In fact, in 1946 the sociologist Bales stated that modern culture promotes heavy drinking not only to alleviate the worry and tension that the culture itself generates but also because the culture lacks alternative resources to help individuals handle these emotions. In the early 1960s, as Consultant of Alcoholism at the World Health Organization, even Jellinek (1960, 1962) noticed the impact of culture on alcoholism. "An analysis of this international experience," Jellinek (1960) acknowledged, "suggests that an understanding could be reached much more readily if the term alcoholism were extended beyond the conception which is current in America and some Anglo-Saxon countries" (p. 15).

Although Jellinek recognized that the medical model of alcoholism needed revision, some social scientists have altogether dismissed the notion that alcoholism is a disease. They have called the disease concept "a moral judgment" (Seeley, 1962, p. 587) that conceals that "a step in public policy is being recommended, not a scientific discovery announced" (Seeley, 1962, p. 593); a "social accomplishment" (Schneider, 1978) based on the assumption that science can solve chronic drinking; and a "folk disease" (Rodin, 1981), given the widespread recognition of alcoholism in the United States' "medical culture" (Rodin, 1981, p. 823) yet uncertainty about its nature and doubt over the efficacy of medical treatment.

In 1969, MacAndrew and Edgerton, a psychologist and an anthropologist, respectively, questioned the disease concept's central premise of a loss of control over drinking. People learn what their society "knows" about drunkenness; and, accepting and acting upon the understandings thus imparted to them, they become the living confirmation of their society's teachings" (p. 88). If cultural norms and values socialize people in how to behave while drinking, then even chronic drinkers can at times, under certain circumstances, control their alcohol consumption. In 1988, the philosopher Fingarette contended that Americans have been so inundated with the idea that alcoholism is a disease that they overlook personal and scientific observations that indicate heavy drinkers can control their alcohol consumption. Heavy drinkers have in common only that drinking is a "central activity" in their lives: "Life is pervaded by a preoccupation with drinking, shaped and driven by the quest for drink, drinking situations, and drinking friends" (Fingarette, 1988, p. 100). Moreover, no one story reflects the onset and persistence of heavy drinking except to say that "specific elements of a heavy drinker's way of life are not symptoms of a disease but rather clues to the character of that life" (Fingarette, 1988, p. 107).

Regarding drugs other than alcohol, in 1947 the sociologist Lindesmith (1968) discovered that opiate users experience addiction only if they learn from "social heritage" (p. 78), or by observation or experience, how to recognize opiate withdrawal, link withdrawal to their opiate use, and then use opiates again to relieve their withdrawal distress.

Persons who interpret withdrawal distress as evidence of the onset of an unknown disease act accordingly, and, if they are not enlightened, do not become addicted. Persons who interpret the symptoms of opiate withdrawal as evidence of a need for the drug also act accordingly and, from using the drug after they have understood, become addicted. (Lindesmith, 1968, pp. 95–96)

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3It deserves note that Bacon spent most of his scientific career defending the modern disease concept of alcoholism that AA, Marty Mann, and the Yale Section constructed (Freed, 2010b; Levine, 1991). In fact, Bacon was a founding director of Mann's National Committee for Education on Alcoholism (Straus, 1991).
In 1953, the sociologist Becker observed that novice marijuana users will continue to use marijuana only if they learn from experienced users how to properly smoke marijuana, how to recognize its effects, and how to enjoy those effects. Presumably, then, marijuana addiction is not possible without first experiencing this learning process.

In 1972, the psychiatrist Zinberg determined that the “setting” of drug use contributed to whether U.S. soldiers in the Vietnam War could control their drug use. Soldiers who abused heroin during the war stopped using the drug once they left “the noxious” (Zinberg, 1984, p. xi) war environment and returned home (e.g., see Robins, Davis, & Goodwin, 1974). In 1974, Szasz, another psychiatrist, said that the medical profession and the government overstated and exploited drug problems to maintain social control. The word addiction, then, reveals a “moral attitude and political strategy” (Szasz, 1974, pp. 50–51). Relatedly, the medical historian Musto (1999) documented how, during the early 20th century, Southern Whites falsely claimed that cocaine caused Blacks to be violent to preserve their racial power. Simultaneously, psychiatrists and pharmacologists labeled opiate users as junkies to legitimize psychiatry in the medical community and to help establish the U.S. pharmaceutical industry (Acker, 2002). Courtwright (1982, 2001), another historian, traced how after World War II, young, poor, urban, Black men who were addicted to heroin became stigmatized and stratified. In contrast, during the late 19th century, morphine addiction among White middle- and upper class women drew little public attention. “What we think about addiction,” Courtwright (2001) concluded, “very much depends on who is addicted” (p. 4).

Scholars have also long recognized that individuals who are powerless to achieve mainstream goals through conventional means might “retreat” into addiction (Merton, 1938). These individuals “see in the substance the ability to accomplish what they need or want but can’t do on their own” (Peelle, 1989, p. 158). For example, in the early 1990s the sociologist Williams argued that addiction to crack cocaine is caused by the hopelessness that accompanies racism, poverty, unemployment, family instability, and community decay. “Those who can command resources, who have the power to effect change in their lives, are very hard to find in the crackhouse” (Williams, 1992, p. 3). In 1991, the sociologists Waldorf, Reinarman, and Murphy studied cocaine addiction from the opposite perspective. A “stake in conventional life”—family, finances, a job, and social status—helps heavy cocaine users control their use or even quit using cocaine (Waldorf et al., 1991). Health problems and diminishing pleasure from cocaine also control use. Whatever heavy cocaine users value and prioritize in their lives is more powerful than the drug itself (Waldorf et al., 1991).

**TODAY’S MEDICAL SPECIALTY PERSPECTIVES ON ADDICTION**

Critiques of the medical model of alcoholism and drug addiction from social and behavioral scientists might not surprise some readers. Perhaps less anticipated is that, even today, the fields of addiction medicine and addiction psychiatry, the only two medical disciplines in the United States that exclusively focus on chronic alcohol and drug use, struggle with the concept of addiction.8

Addiction medicine was born in 1954 when Marty Mann and Ruth Fox, the New York City internist who approached E. M. Jellinek about Mann’s plan to popularize alcoholism as an illness, founded an organization of physicians interested in alcohol addiction called the New York City Medical Society on Alcoholism. The New York Society promoted the concept of alcoholism as a disease that AA, Mann, and Jellinek constructed. In fact, a number of physicians in the society were themselves recovering alcoholics who turned to AA for care (Freed, 2007), a trend that continued in the 1970s.

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8 There are several reasons why only two medical disciplines in the United States exclusively focus on addiction. Briefly, since at least the late 19th century, physicians who study and treat addiction have not been highly regarded (e.g., see Blumberg, 1978). As noted in this chapter, colleagues in other medical fields believed that addiction wasted time and resources, and the public saw substance abuse as a moral problem that the church should handle (Block, 1959; Johnson, 1973). In the 1970s, some physicians called the medical treatment of addiction “dirty medicine” (see Macy Foundation, 1973, p. 2), and today drug treatment providers from all medical backgrounds are “not immune from the same stigma that has tracked addiction problems in patients” (Pichot, Starck, Harris, & Benzick, 1997, para. 4).
and 1980s as doctors from organized medicine's impaired physician movement (e.g., see Steindler, 1984; Talbott, 1988) and self-described “addictionologists” (doctors in recovery who dedicated their medical careers to alcoholism and drug treatment) joined physicians with a strictly professional interest in substance abuse to help the field of addiction medicine grow nationally. Today, the leading organization in addiction medicine, the American Society of Addiction Medicine (ASAM), has about 3,000 members.

Addiction psychiatry originated in 1985 when a small group of academic psychiatrists from the American Psychiatric Association Committees on Alcoholism and Drug Abuse founded their own organization of addiction specialists that today, with approximately 1,000 members, is called the American Academy of Addiction Psychiatry. The psychiatrists insisted that substance dependence was a mental illness that they could treat more effectively than ASAM physicians. In 1991, concluding that ASAM physicians had supplanted them on the front lines of treatment (Group for the Advancement of Psychiatry Committee on Alcoholism and the Addictions, 1991, p. 1292), the addiction psychiatrists won subspecialty recognition for addiction psychiatry from the American Board of Medical Specialties (see Freed, 2007).

The scientific comorbidity literature from the 1980s that confirmed that addiction and mental illness co-occur at “higher than chance levels” (Kessler, 2004, p. 730) helped to ensure board subspecialty recognition for the psychiatrists (Freed, 2007, 2010a). One study, for instance, found that 93% of people with narcotic addiction also had a mental disorder, such as depression (Khantzian & Treece, 1985). Other research (Lewis, Robins, & Rice, 1985) uncovered a strong correlation between alcoholism and antisocial personality disorder (e.g., see also Hasin, Grant, & Endicott, 1988; Lewis, Helzer, Cloninger, Croughan, & Whitman, 1982; Powell, Penick, Othmer, Bingham, & Rice, 1982; Rounsaville, Weissman, Kleber, & Wilber, 1982; Weissman, Myers, & Harding, 1980).

In 1990, the Epidemiologic Catchment Area study broke new ground in comorbidity research (see Regier et al., 1984, 1990). The Epidemiologic Catchment Area study found that 37% of individuals with an alcohol disorder and 53% of individuals with drug disorders also had a mental problem such as schizophrenia, antisocial personality disorder, anxiety disorder, or mood disorder. Drug abusers were four times more likely to have a mental problem than those who did not abuse drugs. Additionally, more than 22% of individuals with a lifetime mental disorder abused alcohol or were alcohol dependent, and almost 15% of people with mental illness were drug abusers or drug dependent. Among individuals with no lifetime history of mental illness, 11% and 3.7% abused alcohol or drugs, respectively. The Epidemiologic Catchment Area study concluded that mental disorders must be addressed as a central part of substance abuse prevention efforts. . . . For mental health professionals, it is also important to recognize the high rate of substance abuse disorders among those with severe mental disorders. (Regier et al., 1990, p. 2517)

The 1990s also became “the decade of the brain” (Wise, 2000, p. 27) as addiction research merged with modern neuroscience. Alan I. Leshner, the director of the National Institute on Drug Abuse from 1994 to 2001, noted that science knows more about drugs and the brain than about most other ways the brain works (Leshner, 1999). Addiction, Leshner stated, is a chronic disease triggered by the effect of compulsive drug abuse on the brain. More specifically,

a metaphorical switch in the brain seems to be thrown as a result of prolonged drug use. Initially, drug use is a voluntary behavior, but when that switch is

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9The field of addiction medicine is not recognized as a medical specialty or subspecialty by the American Board of Medical Specialties. A full explanation of why is beyond the scope of this chapter. Suffice it to say that ASAM physicians believe that their medical knowledge and experience provide the basis for board recognition for addiction medicine. However, recently the American Board of Medical Specialties has resisted recognizing new medical disciplines, although in 2006 ASAM once more set out to acquire medical specialty status for the field of addiction medicine (see American Society of Addiction Medicine, 2006).
thrown, the individual moves into the state of addiction, characterized by compulsive drug seeking and use. (Leshner, 1997, p. 46)

In 2001, ASAM crafted a definition of addiction that borrowed from Leshner’s brain metaphor. Appearing in Principles of Addiction Medicine (Ries, Fiellin, Miller, & Saitz, 2009), ASAM’s main clinical text, the definition reads that

[addiction is] a primary, chronic, neurobiologic disease with genetic, psychosocial, and environmental factors influencing its development and manifestations. It is characterized by behaviors that include one or more of the following: impaired control over drug use, compulsive use, continued use despite harm, and craving. (Ries, Fiellin, Miller, & Saitz, 2009, Appendix)

The word addiction is not included in the revised fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000), psychiatry’s official analytic text. Instead, the phrase substance dependence refers to

a cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues use of the substance despite significant substance-related problems. There is a pattern of repeated self-administration that can result in tolerance, withdrawal, and compulsive drug-taking behavior. (American Psychiatric Association, 2000, p. 192)

The diagnosis of substance dependence is based on experiencing at least three of seven clinical criteria within a 1-year period: (a) tolerance; (b) withdrawal; (c) heavy drug use over an extended time period; (d) uncontrolled use; (e) effort and time finding drugs, using them, and recuperating after use; (f) reluctance to participate in normal life events; and (g) continued use of the substance regardless of adverse physical or psychological consequences (American Psychiatric Association, 2000). The DSM–IV–TR also lists substance abuse, which, unlike dependence, does not involve physical tolerance, withdrawal, and compulsive use (American Psychiatric Association, 2000).

One medical specialist in addiction declared that terminology leaves addiction medicine and addiction psychiatry in “a hell of a fix” (AM/AP–16). Doctors from both fields use terms such as addiction, substance dependence, substance abuse, and chemical dependency interchangeably. Conceptual uncertainty, said another addiction specialist, comes with the job:

The likelihood that anyone has . . . the psychiatrically defined disease of “substance abuse” is almost zero. Of my patients, probably 95% . . . have dependence or addiction, and the other 5% have something else but none have “[substance] abuse.” . . . Other doctors may say, “most of my patients have [substance] abuse. I must be seeing a very different group of people than Dr. [X] is.” Probably not. If we swapped places my percentages would be the same. It’s just a matter of definition. (AP–21)

Indeed, a close look at ASAM’s definition of addiction and the DSM–IV–TR description of substance dependence reveals that both terms link the brain, behavior, and biology to chronic drug use. For the brain, ASAM has chosen the term neurobiologic, and psychiatry uses cognitive. To describe the development of behavior, ASAM refers to psychosocial and psychiatrists to behavioral. For addiction’s biological component, ASAM uses the word genetic, but psychiatry prefers physiological. ASAM has stated that these factors lead to craving and impaired control despite harm. In the DSM–IV–TR, the above symptoms lead to withdrawal and compulsive use despite substance-related problems. Last, although a link between chronic drug use and

10For the remainder of this chapter, I use the following identifiers for information that derives from addiction medicine physicians and addiction psychiatrists: AM = addiction medicine physician, AP = addiction psychiatrist, and AM/AP = physician certified in addiction medicine and addiction psychiatry. The number appearing with these identifiers indicates the order in which these doctors were interviewed for two earlier studies about addiction medicine and addiction psychiatry (see Freed, 2007, 2010a).
mental illness is one criterion for substance dependence, that diagnosis is not contingent on the existence of a psychiatric problem but rather a psychiatric problem "that is likely [italics added] to have been caused or exacerbated by the substance" (American Psychiatric Association, 2000, p. 197). In sum, despite differences in terminology, "inside the world of psychiatry and inside the world of addiction medicine there is considerable consensus about [addiction]" (AM/AP–22).

Yet this consensus hides the conceptual uncertainty that has pervaded the fields of addiction medicine and addiction psychiatry for more than a half century. During the mid-1970s, addiction medicine defined alcoholism but not chronic drug use (see Seixas, Blume, Cloud, Lieber, & Simpson, 1976). During the early 1990s, ASAM published a definition of alcoholism (see Morse & Flavin, 1992) that is remarkably similar to its current definition of addiction—a definition that itself significantly changed between the first and third editions of Principles of Addiction Medicine (see Graham & Schultz, 1998; Graham, Schultz, Mayo-Smith, Ries, & Wilford, 2003; Miller, 1994). In the first and second editions of the DSM, printed in 1952 and 1968, respectively, psychiatry distinguished between episodic excessive drinking, habitual excessive drinking, alcohol addiction, other [and unspecified] alcoholism, and drug dependence (American Psychiatric Association, 1968). Since 1980, psychiatrists have used the term substance dependence to characterize addiction (American Psychiatric Association, 1980). Recently, some influential mental health and medical professionals have suggested that addiction should replace substance dependence in the forthcoming fifth edition of the DSM (see O’Brien, Volkow, & Li, 2006).

"Unfortunately, there's a great deal of art in this game," acknowledged an addiction psychiatrist. "Often you're taking a shot at [addiction] based on your intuition, but you don't even know if you're right because the [research] studies are so difficult to do, so expensive, and so lacking." The National Institute on Drug Abuse has promoted addiction as a brain disease, this psychiatrist continued, "and that implies that you need scanning, medicine, and psychiatrists. But the truth is scanning hasn't shown anything in psychiatry except pretty pictures" (AP–9). According to one ASAM physician, "I think we'll keep describing [addiction] and characterizing it, but I doubt there's going to be a single answer to all of this" (AM–11).

Another addiction psychiatrist stated that a lot of medicine is a combination of science and craft, and that's especially true in addiction psychiatry. A lot of things aren't that well researched out and a lot of people do things on the basis of the way they were trained and what their experience was. (AP–7)

This assessment applies to ASAM's definition of addiction as well as the DSM–IV–TR's description of substance dependence. "These are evolving concepts, nothing is written in stone . . . Not everything is absolutely crystal clear or scientifically proven" (AP–7). An ASAM physician agreed:

I give groups . . . to patients and they always ask me what chemical dependency is. I show them three circles: all the people in America who don't use [drugs], all the people who use [drugs] occasionally . . . and other people who use [drugs] and get a disease. People really can get that. I wonder sometimes whether we in medicine get it. (AM–4)

One addiction psychiatrist responded. "I think we’re still at the level of humors, as in [the Greek physician] Galen. The truth is we know a lot about medicines . . . We know a lot about comorbid . . . We've . . . worked with the 12 steps . . . We're learning" (AP–9). That today's addiction medicine psychiatrists and addiction psychiatrists are still learning about addiction likely resembles the experience of their 19th- and 20th-century predecessors.

THE SYNDROME MODEL OF ADDICTION IN HISTORICAL CONTEXT

In this chapter, I have reviewed a sample of historical perspectives on addiction from the early 19th century to the present. Historical reviews of this
nature have limitations. Some readers might have preferred that I cover different perspectives on addiction. Probably no historical review of addiction can present every perspective that deserves attention. For instance, I did not address the numerous drug scares in the United States since the early 19th century (e.g., see Reinarman & Levine, 1997), the Harrison Narcotic Act of 1914, the nation's first federal law against drugs (e.g., see Musto, 1999), or the government's efforts since the 1930s to highlight the addictiveness of marijuana over its medicinal benefits (e.g., see Chapkis & Webb, 2008). In addition, I have all but omitted perspectives on addiction from outside the United States, and the perspectives that I do present, most of which are medical or scientific, receive more detailed attention from the scholars and experts whose work on addiction has informed parts of this chapter. I alone am responsible for any misinterpretations of their work.

Despite these limitations, this review provides a useful framework to contextualize the syndrome model of addiction. To reiterate, the syndrome model is etiologic: It maintains that addiction is caused by numerous and interrelated biological, psychological, and social factors that become manifest in various and excessive behavioral and substance-related activities (see the Introduction to this handbook). Distal antecedents, which include exposure to object, underlying vulnerability, psychological and genetic risk factors, and neurobiological system risk and immediate neurobiological consequences, trigger the addiction syndrome. Since the early 19th century, medical and scientific professionals as well as recovering drug users have also contemplated distal antecedents of addiction, the precedent for the syndrome model's consideration of such and the focus of this conclusion.

When Benjamin Rush (1784/1811) labeled alcohol as an inherently addicting substance to which all U.S. drinkers were vulnerable, he underscored the dangers of exposure to object in an era of pervasive drunkenness. The Washingtonian movement, and neo-Washingtonian physicians such as Albert Day (1867), focused on the moral risk factors for chronic alcohol consumption, or underlying vulnerabilities, that related to social, psychological, and spiritual issues. When late 19th-century inebriety specialists proposed that degeneration and neurasthenia caused inebriety, they isolated genetic and psychological risk factors that affected existing and future generations. In the first third of the 20th century, Karl Abraham (1908/1927), Lawrence Kolb (1925), Robert P. Knight (1938), and Karl Menninger (1938) analyzed psychological risk factors for addiction, and William D. Silkworth's (1937) idea that an allergy to alcohol caused alcoholism, popularized by AA, and later assertions from E. M. Jellinek (1946, 1952, 1960) and Marty Mann (1944) that alcoholism was a disease, signified an underlying vulnerability to alcohol that only certain drinkers possessed.11 Today, when addiction medicine physicians describe addiction as a brain disease, they acknowledge neurobiological system risk and immediate neurobiological consequences, whereas addiction psychiatrists, who focus on the link between substance dependence and mental illness, recognize underlying vulnerability.

The distal antecedents of addiction that these historical perspectives expose mainly identify biological and behavioral markers of addiction, not unlike the syndrome model. Setting aside the various critiques and revisions of these paradigms, a closer look at some of these perspectives also reveals the influence of social markers, or social risk factors, on addiction. Social markers not only predict and cause addiction but also illustrate the social utility of the concept of addiction.

Consider that Benjamin Rush developed the first disease concept of addiction during a time of great social change in the United States. To ensure a prosperous and thriving nation, one that was becoming increasingly disconnected from traditional social bonds and forms of social support, Americans had to maintain self-control, self-reliance, and a sense of collective responsibility. In this social context, alcohol itself did not cause addiction. On the contrary,

11It deserves note that the origin of alcohol addiction changed with the advent of the modern disease concept of alcoholism. Whereas Rush (1784/1811) proposed that all drinkers were susceptible to alcohol addiction because alcohol itself was inherently addicting, according to the modern disease concept, only some drinkers, for reasons still debated today, suffer from a genuine disease called alcoholism (see Levine, 1978). Indeed, after the repeal of national alcohol prohibition in 1933, Americans questioned the 19th-century model of alcohol addiction when it appeared that some individuals could drink, even quite heavily, without experiencing a loss of control over drinking.
addiction, or chronic alcohol consumption, was a response to, and based on the consequences of, the development of market capitalism and an increasingly specialized society. Indeed, “where there is heavy drinking there is significant underlying anxiety” (Rorabaugh, 1979, p. 146). Similarly, George Miller Beard (1881) theorized that modern advancements such as the clock and telegraph, excessive noise, and the growth of business and politics caused neurasthenia and inebriety among the educated and upper classes. Beard’s model “constituted an indictment of many of this society’s failings, of premature industrialism, of materialism, and of the futile anxieties which this materialism fostered” (Rosenberg, 1962, p. 256). T. D. Crothers (1877, 1881) took into account not only the nature of one’s alcohol consumption but also social class when he distinguished between different categories of inebriates. Among the minority of middle- and upper class inebriates was the majority of lower class, criminal, and social and mental degenerates who, according to Crothers, were unproductive, often untreatable, and particularly problematic for society.

In the early 20th century, the businessman Charles B. Towns (1912) of the Charles B. Towns Hospital argued that the pressure of U.S. society caused addiction, and in the mid-1940s the sociologist Selden D. Bacon (1945/1972) explained that “complex society” might lead to chronic drinking. Since then, sociologists, anthropologists, psychologists, philosophers, psychiatrists, and historians have studied the impact of culture, daily life, group interaction, and social setting on addictive behavior as well as how the concept of addiction can be used for social control, to preserve racial power, to establish professional primacy, and to maintain social class hierarchies. In sum, social risk factors for addiction—social change, social class, modern society, and social environment, for example—are as powerful as biological and behavioral risk factors.

Although some historical perspectives on addiction suggest that the syndrome model might underestimate social markers, or social risk factors, of addiction, where the model’s etiologic focus—particularly on distal antecedents—is especially valuable is in the development of a “diagnostic gold standard” (see the Introduction to this handbook) for addictive behavior. For instance, the fields of addiction medicine and addiction psychiatry use different terms to identify addiction yet fundamentally define it the same way. The syndrome model might reduce the “art” (AP-9), to repeat one addiction psychiatrist, that stems from this clinical contradiction. The syndrome model specifies numerous and interrelated causes of addiction rather than, as Shaffer (in the Introduction to this handbook) points out, relying on patient self-report, comparing that self-report against a fixed diagnostic definition or set of criteria, and then attaching a fixed diagnostic label that simply reaffirms the original self-report (see also Odegaard, Peller, & Shaffer, 2005; Shaffer et al., 2004). In other words, if medical specialists and other clinicians in the addictions look for multiple but specific biological, psychological, and social markers for addiction, and identify and isolate these markers as expressed by individual patients, then one comprehensive diagnostic measure for addiction might be developed that corresponds to the central characteristics of addictive behavior. Perhaps such a measure is idealistic, but the alternative is to continue to rely on more narrow concepts of addiction that most clinicians seem to agree are shortsighted and antiquated. This energy would be better spent on constructing a broader and more flexible diagnostic inventory to improve clinical assessment and patient outcomes.

If historical perspectives on addiction reveal anything, it is that no model of chronic alcohol and drug use is infallible. However, the syndrome model advances the latest medical and scientific thinking about addiction. It is broad enough to identify the myriad circumstances and conditions under which individuals become susceptible to addiction yet precise enough to pinpoint these elements to maximize diagnosis, treatment, and prevention. The syndrome model of addiction has enormous potential to capture what addiction paradigms for more than 2 centuries have not: the complexity and nuance of addictive behavior.

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12The psychologist B. K. Alexander (2008) has provided one of the latest iterations of this perspective, proposing that addiction is a substitute for membership and personal meaning in a “globalising free-market society” (p. 60) that prizes productivity, competition, and status. Individuals who cannot conform to these norms experience psychosocial “dislocation” (B. K. Alexander, 2008) that they manage by abusing alcohol and other drugs.
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Shaffer (in the Introduction to this handbook) provides a thoughtful, incisive, and cutting-edge proposal for the concept of an addiction syndrome that would subsume (in part) what are currently understood in the American Psychiatric Association's (APA's) Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision; DSM-IV-TR; APA, 2000) as substance dependence and pathological gambling. We embrace this proposal, although in this chapter we discuss certain cautions and express disagreements with particular aspects. We begin with an historical perspective, discussing how the proposal of a behavioral addiction syndrome has developed through several editions of the APA's diagnostic manual and how it is likely to be represented in its fifth edition (DSM-5). We then provide an illustrative theoretical model for the addiction syndrome, followed by concerns with respect to the proposal, including a neurophysiological reductionism, potentially opening the floodgates for weakly supported diagnostic constructions, and the future research and treatment of pathological gambling.

**HISTORICAL PERSPECTIVE**

Substance dependence has long been equated in the minds of the general public and among many mental health professionals as being equivalent to a physiological dependence. This equivalency was made explicit in the third edition of the DSM (DSM-III; APA, 1980), wherein the diagnostic criteria for substance dependence were confined simply to the presence of tolerance and withdrawal, the two primary, fundamental indicators of a physiological dependence, the neuroadaptation that often occurs in response to a sustained drug usage. In fact, the postoperative patients described by Shaffer (in the Introduction to this handbook) who receive prescribed opioids for pain and experience neuroadaptation as a result of opioid use but never seek these drugs elsewhere or experience any of the common negative consequences of a drug dependence were considered in DSM-III to have substance dependence.

In [some] rare cases the manifestations of the disorder are limited to physiological dependence. . . . An example would be an individual's inadvertently becoming physiologically dependent on an analgesic opioid given to him by a physician for the relief of physical pain. (APA, 1980, p. 165)

For reasons cogently described by Shaffer et al., these people are explicitly excluded from their diagnosis of an addiction syndrome, despite the fact that they clearly do have the neuroadaptation.

The concept of substance dependence shifted substantially in the revised edition of the DSM-III (DSM-III-R; APA, 1987). Substance dependence was no longer equated with physiological dependence. Tolerance and withdrawal were only two of nine optional diagnostic criteria, only three of which were necessary for the diagnosis. It was technically easy to meet the DSM-III-R criteria for substance dependence without having either tolerance or withdrawal, that is, without having any physiological...
Substance use, continued use despite adverse consequences to physical health, but it was no longer a requirement. In fact, in direct contrast to DSM-III, DSM-III-R explicitly stated that the “surgical patients [who] develop a tolerance to prescribed opioids and experience withdrawal symptoms without showing any signs of impaired control of their use of opioids” (APA, 1987, p. 166) would no longer be considered to have a substance dependence.

Substance dependence in the DSM-III-R was closer to a concept of a behavioral dependence (Edwards, 1986) than a physiological dependence. This revision of the conceptualization of substance dependence was in response to a substantial body of research that indicated that the pathology of substance dependence did not appear to be the neuroadaptation of the brain to continued drug usage (Hyman, 2005; Rounsaville & Kranzler, 1989). Physiological dependence was relatively easy to treat, whereas the dependency seen with most patients was not. Withdrawal symptoms are no longer a problem when a patient leaves a drug treatment center, yet people often quickly return to harmful drug usage soon after successful treatment of the physiological dependence. Tolerance could still be present, but tolerance is hardly an adequate explanation for much of the relapsing behaviors commonly seen in purportedly treated patients.

An additional innovation of the DSM-III-R was the explicit alignment of the diagnostic criteria for pathological gambling and substance dependence (APA, 1987). This alignment was done intentionally (Lesieur, 1988), albeit no mention was made within the text of the DSM-III-R of either disorder. Each disorder included nine diagnostic criteria, with an almost perfect isomorphic relationship between them, including even the physiological dependence criteria corresponding to tolerance (i.e., a “need to increase the size or frequency of bets to achieve the desired excitement”; APA, 1987, p. 325) and withdrawal (i.e., “restlessness or irritability if unable to gamble”; APA, 1987, p. 325).

In other words, pathological gambling was seen as a form of dependence, including even perhaps a neuroadaptation despite the absence of any ingestion of a substance. “While pathological gambling does not involve the use of a substance, research conducted by numerous scholars has noted its similarity to addictive behaviors” (Lesieur, 1988, p. 40). Nevertheless, pathological gambling remained in the section for impulse control disorders not elsewhere classified. It could not be shifted to the section for psychoactive substance use disorders in the absence of substance ingestion. In a related vein, DSM-III-R also included an explicit reference to a “nonparaphilic sexual addiction” (APA, 1987, p. 296) as a possible variant of a sexual disorder not otherwise specified, recognizing again that some instances of “repeated sexual conquests” (p. 296) closely resemble the pattern of behavior seen in people with a substance dependence.

The close alignment of the diagnostic criteria for substance dependence and pathological gambling was abandoned in the fourth edition of the DSM (DSM-IV; APA, 1994) for a couple of reasons. First, priority had been given in DSM-III-R to the construction of the diagnostic criteria for substance dependence, compelling the authors of the criterion set for pathological gambling to conform to the number and content of what the substance use disorder researchers decided. As a result, the criterion set for DSM-III-R pathological gambling was considered to be a bit procrustean, leaving out, for instance, valid diagnostic criteria for the sake of consistency with substance dependence (Rosenthal, 1989). “Several clinicians and academicians attributed this to the need to have the criteria conform to those for psychoactive substance dependence” (Bradford, Geller, Lesieur, Rosenthal, & Wise, 1996, p. 1009). “The general feeling was that pathological gambling was in fact an addiction” (Bradford et al., p. 1009) but that its diagnostic criteria should be constructed in a manner that would optimize its clinical and research usage rather than give priority to simply conforming precisely to the criterion set for substance dependence, particularly if no mention of this conformity and correspondence was going to be acknowledged in the diagnostic manual. As a result, the criterion set for pathological gambling deviated somewhat from the criterion set for...
substance dependence in DSM-IV, albeit there were still the two criteria that suggested tolerance ("needs to gamble with increasing amounts of money in order to achieved the desired excitement"; APA, 2000, p. 618) and withdrawal ("is restless or irritable when attempting to cut down or stop gambling"; APA, 2000, p. 618).

Equally as important in DSM-IV was the decision to subtype substance dependence as being either with or without physiological dependence (APA, 1994). The subtype of physiological dependence was based on the presence of tolerance and withdrawal. The inclusion of this subtype was to help clinicians recognize that substance dependence was no longer considered equivalent to a physiological dependence (Frances, First, & Pincus, 1995). In the minds of many clinicians, substance dependence continued to mean a physiological addiction. By having the potential to diagnose a patient with substance dependence in the absence of a physiological dependence, it was evident that substance dependence and physiological dependence were no longer equivalent to one another. This subtyping also conversely allowed clinicians and researchers to focus their interest specifically on the physiological dependence, if they were so inclined (Nathan, 1994).

The authors of DSM-5 propose to take the next natural step in this progression (P. R. Martin, 2005b; Petry, 2006; Potenza, 2006), collapsing pathological gambling and substance use disorders into one common class, addiction and related disorders (see APA, 2010, "Include Pathological [Disordered] Gambling Within Addiction and Related Disorders"). This proposal for DSM-5 is largely consistent with the proposal of Shaffer (in the Introduction to this handbook) for an addiction syndrome. Ironically, perhaps, the term dependence will be confined in DSM-5 to instances of physiological dependence, whereas the term addiction will be used to refer to the broader concept of a behavioral addiction, consistent with Shaffer.

**A RISK MODEL FOR AN ADDICTION SYNDROME**

We do believe there is compelling theoretical and empirical evidence for an addiction process that is not specific to the object of the addiction, as suggested by Shaffer (in the Introduction to this handbook). In this section of the chapter, we present our model and then review additional research findings that help explain why different individuals develop addictions to different objects.

One characteristic shared across various expressions of addiction is that the behaviors engaged in provide some form of immediate reinforcement (a high, relief from distress, distraction from woes), yet repeated engagement in these behaviors can result in harm to one's long-term goals and interests. Our risk model involves an integration across functional brain systems, including neurotransmitter functioning, microgenetics, personality, and psychosocial learning, to describe a process by which some individuals are biased to pursue immediate reinforcement at the expense of their long-term interests and how that bias can lead to addiction.

**Functional Brain Systems**

There are extensive connections between the prefrontal cortex (particularly, the orbitofrontal cortex [OFC] and the ventromedial prefrontal cortex [VMPFC]) and the amygdala (Barbas, 2007; Bechara, 2005). The amygdala is understood to play a role in directing attention to stimuli that are emotionally salient—in particular to stimuli that are disturbing or distressing (Davidson, 2003).

"Bottom-up" connections between the amygdala and the prefrontal cortex carry information about the emotional significance of sensory input, thereby influencing activity in the OFC and VMPFC. These signals are thought to help orient one to what is important and help trigger responses to distress. In turn, "top-down" connections from the OFC and VMPFC back to the amygdala appear to exert a regulating effect on the amygdala and brain stem (Bechara, 2005; Lewis & Todd, 2007). The OFC can interrupt the connection between emotion and response; that is, OFC activity can override amygdala-driven emotional responses by providing information and a bias toward longer term goal-directed behavior (Lewis & Todd, 2007). Thus, in well-functioning individuals, this combination of bottom-up and top-down processing leads to a recognition of emotionally important and distressing events, a
preparedness to respond to those events, and the use of information so that one's response is consistent with one's long-term interests.

There is considerable evidence that low levels of serotonin (5-hydroxytryptamine [5HT]) in this brain system are associated with reduced OFC activity and hence fewer information-based, cautioning signals to the amygdala and an accompanying increase in emotion-driven action without full consideration of the action's long-term consequences (see reviews by Cyders & Smith, 2008b; Davidson, 2003). In part, 5HT modulates dopamine (DA) activity, such that reduced 5HT is associated with increased DA activity in this brain system, which is associated with increases in emotional dysregulation and risk-taking behaviors (Depue & Collins, 1999; Spoont, 1992). Predictable behavioral expressions of reduced 5HT activity have been observed across species (Cyders & Smith, 2008b). For example, low levels of 5HT are associated with impulsive aggression but not planful aggression in monkeys (the latter form of aggression facilitates moves up the group hierarchy; the former has the opposite effect; Frankle et al., 2005). In humans, drugs that increase the level of 5HT in the synaptic cleft are effective in regulating emotion and reducing risky behaviors in a variety of clinical disorders, including borderline personality disorder (Barbe, Rubovsky, Venturini-Andreoli, & Andreoli, 2005). Thus, low levels of 5HT and accompanying high levels of DA in the brain system are associated with emotion-driven rash action that tends to harm one's long-term interests.

It is possible that this pattern of gene polymorphisms contributes to low 5HT and high DA in the OFC/VMPFC–amygdala brain system (Cyders & Smith, 2008b). Indeed, this pattern is associated with alcohol dependence and impulsive, but not planned, aggression (Davidson, 2003; Noble, 2003).

**Personality Traits of Positive and Negative Urgency**

As proposed by Cyders and Smith (2008b), this pattern of gene polymorphisms, and the accompanying profile of low 5HT and high DA activity in the OFC/VMPFC–amygdala brain system, might contribute to the development of a disposition to act in rash, ill-advised ways when experiencing intense emotion. The personality trait terms positive urgency and negative urgency refer to the tendencies to act rashly when experiencing intense positive and intense negative affect, respectively (Cyders & Smith, 2008b; Whiteside & Lynam, 2001). These constructs refer to the disposition to act in response to one's immediate affect, without sufficient regard for the impact of one's actions on one's long-term interests.

The traits positive and negative urgency concurrently and prospectively predict engagement in multiple addictive behaviors. Prospectively, they predict increases in drinking quantity and problem drinking (Cyders, Flory, Rainer, & Smith, 2009; Settles, Cyders, & Smith, 2010), pathological gambling (Cyders & Smith, 2008a), risky sex (Zapolski, Cyders, & Smith, 2009), illegal drug use (Zapolski et al., 2009), and bulimia nervosa (Anestis, Selby, & Joiner, 2007; Fischer, Stojek, & Collins, 2009). Cross-sectionally, the positive and negative urgency traits are predictive of addictive behavior involvement as early as late elementary school: They predict drinker status (Gunn & Smith, 2010) and binge eater status in both girls (Combs, Pearson, & Smith, in press) and boys (Pearson, Combs, & Smith, in press). They are also cross-sectionally predictive in young adults of problem drinking, pathological gambling, binge eating, excessive reassurance seeking, dependence on cellular phone use, compulsive shopping, and tobacco craving (Anestis, Selby, Fink, & Joiner, 2007; Billieux, Rochat, Rebetz, & Van der Linden, 2008; Billieux, Van der Linden, & Ceschi, 2007; Billieux, Van der Linden, D'Acremont, Ceschi, &
Zermatten, 2007; Smith, Fischer, et al., 2007). In each of these studies, the predictive power of the urgency traits was compared with that of other impulsivity-related traits, such as sensation seeking, lack of planning, and lack of perseverance (White­side & Lynam, 2001). In each case, urgency traits predicted above and beyond those other traits, and in most cases, none of these other traits predicted beyond prediction from the urgency traits.

Positive and negative urgency are thought to lead not just to involvement in risky behaviors that can become addictions but also to contribute to the emergence of addiction itself (Cyders & Smith, 2008b). Each time an individual engages in one of these behaviors (drinking, drug use, sex, gambling), the behavior is immediately reinforced and therefore more likely in the future. At the same time, each of these behavioral occurrences represents another occasion on which alternative strategies for responding to one's extreme mood (such as strategies more facilitative of one's long-term interests) are not explored, not engaged in, and hence not developed as part of one's behavioral repertoire (Fischer, Smith, Spillane, & Cyders, 2005). Accordingly, over time, rash actions in response to intense moods become more and more likely, and alternative action possibilities more consistent with long-term goals are not developed and so become less and less likely. Eventually, the rash actions come to feel compelled (Pearson, Guller, Spillane, & Smith, in press), and the compulsive quality of addiction is present.

Taken together, these bodies of literature suggest that there are neurophysiologically based individual differences in the capacity to defer responding to intensely emotional experiences in light of one's long-term interests. Individuals low in this capacity tend to develop the traits of positive and negative urgency, and some of them tend, through an ongoing reinforcement process, to feel increasingly compelled to engage in actions that provide immediate reinforcement but that are harmful to their long-term interests: They develop addictions.

Specific Objects of Addiction: The Importance of Psychosocial Learning

The risk process we have described is general: Nothing about the urgency-based risk process is specific to any particular object of addiction. To understand why some individuals high on the urgency traits develop problems with alcohol and others develop problems with gambling, binge eating, smoking, or other behaviors, we consider people's psychosocial learning histories. To which behaviors have individuals been exposed and from which behaviors have they learned to expect reinforcement? We have addressed this question by measuring individual differences in (a) learned expectancies for reinforcement from target behaviors (Goldman, Brown, Christiansen, & Smith, 1991) and (b) motives for engaging in target behaviors (Cooper, 1994).

Measures of expectancies and measures of motives are highly predictive of numerous addictive behaviors, both concurrently and prospectively. For example, alcohol expectancies predict the onset of drinking and the onset of problem drinking during adolescence (Christiansen, Smith, Roehling, & Goldman, 1989; Smith, Goldman, Greenbaum, & Christiansen, 1995); alcohol expectancies predict increased consumption across the 1st year of college (Settles et al., 2010); expectancies for reinforcement from eating and from dieting or thinness predict the onset of binge eating and purging in middle-school girls (Combs, Smith, Flory, Simmons, & Hill, in press; Smith, Simmons, Flory, Annus, & Hill, 2007); smoking expectancies differentiate lighter and heavier smokers and concurrently predict higher levels of nicotine dependence (Brandon & Baker, 1991; Schleicher, Harris, Catley, Harrar, & Golbeck, 2008); and gambling expectancies differentiate among pathological gamblers, likely pathological gamblers, and nongamblers (Walters & Contri, 1998). Motives to consume alcohol to cope with problems or to enhance positive experiences predict subsequent and current drinking (Cooper, 1994; Settles et al., 2010).

Crucial to the present theory, expectancies for reinforcement from different addictive behaviors differentiate among individuals engaging in those different behaviors. Fischer, Anderson, and Smith (2004) found that alcohol expectancies concurrently predicted problem drinking but not eating disorder–related symptoms, and eating expectancies predicted binge eating but not alcohol problems. Fischer and Smith (2008) found that negative urgency was
associated with binge eating, problem drinking, and pathological gambling, but that expectancies were problem specific: Eating expectancies were associated only with binge eating, alcohol expectancies were associated only with problem drinking, and gambling expectancies were associated only with pathological gambling. Because there is extensive evidence from decades of research in basic psychological science indicating that expectancies represent summaries of individuals' learning experiences (Bolles, 1972; MacCorquodale & Meehl, 1953; Tolman, 1932), we infer that, in part, high-risk individuals engage in the addictive behavior for which they have learned to expect high levels of reinforcement.

It is of course also true that other factors influence individuals' choice of addictive behaviors. As noted by Shaffer (in the Introduction to this handbook), social and cultural factors are important in understanding addictions. Women are much more likely than men to develop bulimia nervosa (Striegel-Moore & Bulik, 2007), and bulimia nervosa appears to be a disorder largely confined to economically advanced, Western cultures (Keel & Klump, 2003). There are also biological factors that alter the likelihood of developing an addiction to any one object. For example, low responsivity to alcohol increases risk (Schuckit & Smith, 2006). It is beyond the scope of this chapter to consider these factors further.

Our risk model calls for a perspective on addictions that is quite consistent with the addiction syndrome described by Shaffer (in the Introduction to this handbook). We believe there is a complex set of genetic, neurobiological, and personality factors that, together, increase the likelihood of developing an addiction to some object. From our perspective, it is perhaps unfortunate that different sets of individuals study each expression of addiction separately from other such expressions. For example, there are different societies, with different members, that emphasize the study of eating disorders, alcohol abuse, smoking, and so on. One reason this Aristotelian state of affairs is unfortunate is because it increases the chance that different individuals studying different addictive disorders will make the same discoveries as each other, without benefiting from each other's work and without realizing the breadth of the impact of what they find.

**CONCERNS AND ISSUES FOR AN ADDICTION SYNDROME**

We believe a central advantage of our model is that it also provides a theoretically sound way to understand and explain how different high-risk individuals develop addictions to different objects. Perhaps the capacity to explain involvement with different objects of addiction by different individuals paradoxically contributes to the viability of a single, overall model of addiction. After all, a single model must account for the variability in the objects of addiction. However, we also feel that there are a few fundamental concerns with respect to the concept of an addiction syndrome (or a behavioral addiction) that warrant careful consideration, including the implications of a neuroadaptation of learning, the floodgates of related conditions, and the future research and treatment of pathological gambling. We discuss each of these concerns in turn.

**Neuroadaptation of Learning**

One of the central arguments being made in favor of conceptually shifting from a physiological dependence to addiction is that the latter will still be fundamentally a neurophysiological disorder. The pathology of dependence will no longer be the direct result of a drug's activity within the brain but will still represent a form of physiological brain disease (Akil et al., 2010; Hyman, 2005; Kandel, 2001, 2009). The neuroadaptation of the brain to sustained drug usage will no longer be the pathology. It will involve a dysregulation in the neuroadaptation of learning (Kalivas & Volkow, 2005). As expressed by P. R. Martin (2005b), “It is now accepted that learning of all forms is accompanied by structural and functional changes in the brain” (pp. 5–6). As expressed by Holden (2001), “Where there’s a reward, there’s the risk of the vulnerable brain getting trapped in a compulsion” (p. 980).

The apparent importance of considering substance use disorders as brain diseases has been present from the very beginnings of these diagnoses within psychiatry. This apparent need is probably related, in part, to the dilemma and risks of diagnosing as a disorder a behavior that carries with it considerable moral condemnation (Nathan & Harris,
1975; Peele, 1984) as well as to the importance within the field of psychiatry of considering itself as a part of real medicine (Guz, Cloninger, Martin, & Clayton, 1986; Kandel, 1998).

We have perhaps left the decade of the brain (Judd, 1998) to enter a decade of the brain disease (Hyman, 1998). Psychiatry originally distinguished itself from neurology by providing psychological models for the etiology and pathology of disorders that were not, at that time, effectively understood or treated by neurologists (Blashfield, 1984; Kandel, 1998; Stone, 1997). Psychiatry is now shifting back toward neurology (Akil et al., 2010; Hyman, 1998; Kandel, 1998). As expressed by a recent director of the National Institute of Mental Health (NIMH), “Mental illnesses are real, diagnosable, treatable brain disorders” (Hyman, 1998, p. 38). As expressed by the current head of NIMH, priority for funding will in the future be given to studies that formally adopt a clinical neuroscience perspective that contributes to an understanding of mental disorders as “developmental brain disorders” (Insel, 2009, p. 132). This is being accomplished in part through the development of research domain criteria (RDoC) that will represent a biological alternative to the DSM, “with a strong focus on biological processes, and emphasis on neural circuits” (Sanislow et al., 2010, p. 633). “The RDoC framework conceptualizes mental illnesses as brain disorders” (Insel et al., 2010, p. 749). As indicated by Miller (2010), “Over the next 2 to 3 years, NIMH will encourage researchers to shift from using DSM criteria in their grant proposals to using the RDoC categories” (p. 1437).

There is much to be gained through a neurophysiological understanding of psychopathology, including pathological gambling. However, as suggested by Shaffer (in the Introduction to this handbook), Kendler (2005), and Miller (2010), there are also risks of too narrowly confining one’s perspective on psychopathology. There is much that will be lost by not studying and understanding psychopathology at the level of psychological and social constructs; that is, much will be lost by reducing psychopathology to neurophysiological mechanisms. “Thoughts, feelings, and impulses matter not only because they are responsible for huge amounts of human suffering but because they do things” (Kendler, 2005, p. 434). Mental disorders can occur within a psychological and social context that needs to be recognized and studied directly if adequate understanding and treatment are to occur (Widiger, 2004). As Kendler (2005) further expressed,

Although humiliation is ultimately expressed in the brain, this does not mean that the basic neurobiological level is necessarily the most efficient level at which to observe humiliation. A full etiological understanding of at least some psychiatric disorders will require consideration of psychological and cultural factors. (p. 436)

It is indeed true “that learning of all forms is accompanied by structural and functional changes in the brain” (P. R. Martin, 2005a, pp. 5–6), but this says little more than the truism that “each and every thought, impulse, affect, perception, and motivation is isomorphic with a commensurate pattern of brain activity” (Ilardi & Feldman, 2001, p. 1072). The fact that all psychological activity must involve some commensurate neurophysiological functioning does not suggest or necessarily imply that the optimal diagnosis, explanation, and understanding of psychological processes will be at the level of that brain activity.

Shaffer (in the Introduction to this handbook) suggests that “adopting a syndromal [behavioral addiction] perspective on addiction should stimulate further investigation of more objective diagnostic measures (e.g., functional MRI, event-related brain potentials . . .)” (p. xlvii). “The RDoC classification assumes that the dysfunction in neural circuits can be identified with the tools of clinical neuroscience, including electrophysiology, functional neuroimaging, and new methods for quantifying connections in vivo” (Insel et al., 2010, p. 749). On the one hand, perhaps someday the diagnosis of pathological gambling could be made simply and more objectively on the basis of how a certain section of the brain lights up in response to a relevant gambling cue. On the other hand, perhaps not. In any case, it is unlikely to occur anytime in the near future.

Substantial attention is being given to structural and functional brain imaging with the expectation
that these instruments could eventually be used to diagnose neurophysiological pathology (Drevets, 2002; Epstein, Isenberg, Stern, & Silbersweig, 2002). Laboratory tests within medical practice go beyond the assessment of symptoms. They provide a more direct and objective assessment of an underlying physical pathology. A hope is that laboratory tests could do the same for psychiatry as they have done for other domains of medicine (Steffens & Krishnan, 2003; Rounsaville et al., 2002). “The increasing use of laboratory tests in psychiatric research raises the question of whether and when these tests should be included within the diagnostic criteria sets” (Frances et al., 1995, p. 22). However, clearly limiting these and other neurophysiological measures’ potential for incorporation within diagnostic criteria sets is the virtual absence of research indicating their ability to provide independent, blind diagnoses. Despite the assumption of the RDoC approach (Insel et al., 2010), currently no studies have assessed the sensitivity and specificity of neuroimaging techniques for the diagnosis or differential diagnosis of any specific mental disorder (Steffens & Krishnan, 2003). There are no direct measures of brain circuits that can independently diagnose the presence of any psychopathology.

This may not be simply a matter of a current lack of adequate understanding of the neurophysiology of a respective mental disorder. It might also reflect the inability to be able to truly or fully reduce at least some mental disorders to a diagnosable brain disease (Kendler, 2005). It may only be in the context of a functional maladaptivity that some mental disorders can be identified. Shaffer (in the Introduction to this handbook) laments the current reliance on impairment to reach a judgment as to the presence of pathological gambling, but impairment is perhaps a fundamental requirement that will always need to be recognized.

Central to the concept of a mental disorder are two key components: dyscontrol and impairment (Widiger & Smith, 1994). The diagnostic criteria for DSM-IV-TR substance dependence and pathological gambling are valid because they provide indicators of both dyscontrol and impairment, neither of which are likely to be able to be assessed through neurochemical or neurological imaging. Mental disorders are dyscontrolled impairments in psychological functioning (Bergner, 1997; Kirmayer & Young, 1999; Klein, 1999; Widiger, 2008; Widiger & Trull, 1991). “Involuntary impairment remains the key inference” (Klein, 1999, p. 424).

Fundamental to the concept of a mental disorder is the presence of impairments in feelings, thoughts, or behaviors over which a normal (healthy) person is believed to have adequate control. To the extent that a person willfully, intentionally, freely, or voluntarily engages in gambling, drug usage, harmful sexual acts, Internet usage, or child abuse, the person would not be considered to have a mental disorder. People seek professional intervention in large part to obtain the insights, techniques, skills, or other tools (e.g., medications) that increase their ability to better control harmful (painful) mood, thoughts, or behavior. Impairment and dyscontrol provide the optimal means by which to identify a meaningful boundary between, or an important parameter for quantifying, normal and abnormal psychological functioning. Impairment and dyscontrol are what determine whether a mental disorder is actually present. Rather than attempt to identify the presence of psychopathology through brain imaging of dysregulated neuroadaptation, it is preferable to improve the behavioral assessment of dyscontrol and impairment.

Dyscontrol as a component of mental disorder does not imply that a normal person has free will, a concept that is, at best, difficult to scientifically or empirically verify (Bargh & Ferguson, 2000; Baumeister, 2008; Howard & Conway, 1986). A person with a mental disorder can be understood analogously to a computer lacking the necessary software to combat particular viruses or to execute effective programs. Pharmacotherapy alters the neural connections of the central nervous system (the hardware), whereas psychotherapy alters the cognitions (the software) in a manner that increases a person’s behavioral repertoire, allowing the person to act and respond more effectively to environmental stress and demands. A computer provided with new software has not been provided with free will, but it has been provided with more options to act and respond more effectively. Of course, changes in the hardware probably facilitate changes in the software: If a drug
leads to increased serotonin and the capacity not to act on immediate impulses, it might create a condition in which individuals learn or develop coping strategies for their impulses that would not previously have been possible. In the same way, changes in software can lead to changes in hardware: If a person learns through dialectical behavior therapy not to act on impulses, that change is surely accompanied by a neurophysiological alteration.

Optimal diagnostic criteria for the Shaffer (see the Introduction to this handbook) addiction syndrome would most likely include scales to assess the extent of inadequate self-control and, separately, the extent of impairment. The inclusion of separate scales is consistent with the current interest within psychiatry to separate the diagnosis of disorder from the assessment of impairment (Sartorius, 2009). However, the separation in this instance is not to imply that a disorder can be present without impairment, because impairment is a fundamental and necessary component of the definition of mental disorder.

Separation of scales for the assessment of dyscontrol and impairment is helpful to provide a more differentiated and distinguished diagnosis of pathological gambling, allowing, for instance, future researchers to determine more precisely whether a particular outcome or correlate of pathological gambling is a reflection of either the dyscontrol or the impairment (Smith & Combs, 2010; Smith, McCarthy, & Zapolski, 2009). This would facilitate, for example, the ability to identify people who are engaging in highly risky gambling (e.g., dyscontrolled behavior without presuming that the behavior is necessarily harmful) as well as recognizing that some behaviors can be extremely harmful yet undertaken with apparent volitional choice. More sophisticated assessment could also establish more precisely that point at which self-control is considered to be sufficiently inadequate, and impairment sufficiently harmful, to warrant professional intervention.

Opening the Floodgates
One of the more common concerns raised in response to a shift to a broader concept of addiction proposed by Shaffer (in the Introduction to this handbook) and by the authors of DSM–5 is the potential opening of a floodgate of weakly supported addictions (Frances, 2010b; Petry, 2005b, 2006). “This ‘slippery slope’ makes it difficult to know where to draw the line demarcating any excessive behavior as an addiction” (Petry, 2005a, p. 7). As Petry (2006) suggested later, “Television, work, exercise, and chocolate addiction have been described” (p. 157). Of course, none of these additional potential expressions of addiction would be provided formal recognition within DSM–5 as distinct diagnostic entities, analogous to the inclusion of separate diagnoses for cocaine, nicotine, opioid, and alcohol dependence within DSM–IV–TR substance dependence. However,

if a clinician felt that the patient were “addicted” to sex, or to shopping, or to the Internet, or to working, or to video games, or to credit card spending, or to surfing, or to suntanning, or (my own personal favorite) to blogging on Blackberries, or to whatever else (the list is long and could easily expand into every area of popular activity)—this could be diagnosed as “Behavioral Addiction Not Otherwise Specified” and thus receive the dignity of an official DSM code. (Frances, 2010b, “DSM 5 Suggests Opening the Door,” para. 1)

It is difficult to believe that this will not occur.

With DSM–IV–TR, it is relatively difficult for clinicians to diagnose what might be considered to be an excessive or harmful interest in sex, pornography, shopping, the Internet, or other activities on which some people spend a considerable amount of time. The best option for such diagnoses could be impulse control disorder not otherwise specified (APA, 2000). With the inclusion of behavioral addiction not otherwise specified in DSM–5, a ready and comfortable home for such diagnoses will be provided. In fact, it is even stated on the DSM–5 Web site that, along with pathological gambling, “other addiction-like behavioral disorders such as ‘Internet addiction’ . . . will be considered as potential additions to this category as research data accumulate” (APA, 2010, “Substance Related Disorders,” para. 1).

One such additional variant is already likely to be included in DSM–5 as an officially recognized
diagnosis: sex addiction. Sex addiction was not provided a formal diagnostic label in DSM-II (APA, 1980), but within the diagnosis of psychosexual disorder not otherwise specified, specific reference was made to “distress about a pattern of repeated sexual conquests with a succession of individuals who exist only as things to be used (Don Juanism and nymphomania)” (APA, 1980, p. 283). This behavior was described within the same diagnosis in DSM-III-R (APA, 1987), which, as noted earlier, even included a specific reference to “nonparaphilic sexual addiction” (p. 296). An explicit reference to sex addiction was deleted in DSM-IV-TR (APA, 2000) precisely out of a concern that it contributed to excessive and inappropriate diagnostic labeling in the absence of an adequate scientific foundation for what was perhaps in many cases simply irresponsible, negligent, or self-centered behavior (Frances, 2010a; Wise & Schmidt, 1997). “The concept of sexual addiction, whether paraphilic or nonparaphilic, has been popularized in a series of books and articles that have no scientific database” (Wise & Schmidt, 1997, p. 1139).

However, proposed for DSM-5 is now full recognition of sex addiction, albeit using the title hypersexual disorder and included in the section of the manual for sex disorders (Frances, 2010a; Kafka, 2010; Ragan & Martin, 2000; Winters, 2010). The proposed diagnostic criteria are clearly modeled after, and closely parallel, the criteria for substance dependence, behavioral addiction, or both (e.g., items concerning failed efforts to control or reduce the behavior and continued engagement in the behavior despite physical or emotional costs). If a person has four of five proposed diagnostic criteria (see http://www.dsm5.org for the complete list), the clinician would further specify whether the hypersexual behavior specifically concerned the use of pornography, cybersex, telephone sex, pornography, strip clubs, or even just masturbation.

A close consideration of the diagnostic criteria for hypersexual disorder might raise concerns as to the potential for an excessive or liberal diagnosis of this behavior as a disorder. “The authors have not thought through how difficult it is to distinguish between ordinary recreational sexual misbehavior (which is very common) and sexual compulsion (which is very rare)” (Frances, 2010a, para. 7). For example, a person who enjoyed Internet pornography would meet diagnostic criteria simply on the basis of enjoying it for longer than 6 months, engaging in the activity for a period of time that a clinician considered to be excessive (i.e., the amount of time considered to be excessive is not objectively specified), doing so at times in response to feeling bored, doing so at times to help relieve stress, attempting to reduce the activity at times because of transient feelings of guilt or doubt but resuming the activity when those feelings were eventually discarded, continuing to enjoy Internet pornography despite a clinician’s belief that it causes emotional harm to the person or even just to someone else, or all of these. These criteria might apply to quite a few people who enjoy Internet pornography.

Some fear that the diagnoses of behavioral addiction and addiction syndrome could also trivialize what are now understood to be serious mental disorders. These diagnoses could “obscure the differences between those that may be better considered bad habits and those that may require professional intervention” (Petry, 2005b, p. 7). The diagnosis of behavioral addiction might also embarrass psychiatry through the expansion into faddish, capricious concepts. An episode of the popular television show South Park nicely satirized the concept of sex addiction in response to the difficulties experienced by Tiger Woods (“South Park Takes on Tiger Woods,” 2010). A further, related concern is that these diagnoses will further weaken the responsibility people should take for their negligent, irresponsible, or selfish behavior. “If we label [these behaviors] as an ‘addiction,’ are we not in essence removing responsibility from the individual, family or society that normalizes, and perhaps even reinforces, such behaviors?” (Petry, 2005b, p. 7).

This tension between taking moral responsibility for one’s actions versus blaming an addiction is, of course, not new (M. W. Martin, 2006). Before the recognition of alcoholism as a disorder (or a disease), people would be blamed for their substance dependence, for their failure to exercise adequate self-control, and for the failure to demonstrate a strong moral character. The development of Alcoholics Anonymous was in part a reaction against the moral condemnation of people who appeared to lack
adequate control over their drinking behavior. Fortunately, the mental health field has embraced the diagnosis of substance dependence rather than leaving it to be understood as simply a moral irresponsibility, albeit the diagnosis of substance dependence in the first two editions of the APA diagnostic manual did still carry some moral connotation in its inclusion in a section of the manual for disorders that involved socially disapproved behaviors (Nathan & Harris, 1975).

The recognition of behavioral addiction and hypersexual disorder in DSM-5 will clearly lead to overdiagnosis. However, of course, their absence leads to underdiagnosis. There seems little reason to believe that pathological gambling and hypersexual disorder would be the only possible forms of a behavioral addiction. It will be a natural and reasonable progression from the recognition of pathological gambling as a behavioral addiction to the inclusion of additional forms, such as excessive shopping, eating, or working. Petry (2005a) suggested satirically that if pathological gambling is an addiction, then why not watching television? She pointed out that some children spend up to 10 hours a day watching television; that it interferes with a healthy social and family life, contributes to poor school performance, and contributes to sedentary behavior and snacking that can have serious consequences for physical health; that efforts to diminish or discontinue the viewing of television can be met with substantial opposition and failure; and that withdrawal from access to television can result in angry temper tantrums or at least irritable mood. Frankly, and ironically, she could be making a very good argument for indeed recognizing some cases of dyscontrolled and harmful television viewing as another reasonable variant of a behavioral addiction.

There is inadequate empirical research to document conclusively that a television addiction would be truly comparable to pathological gambling or sex addiction, but others would suggest that the empirical support to document conclusively that gambling and sex can be expressions of addiction is insufficient. It is difficult to specify with certainty at what point the research is said to be sufficient; this circumstance emerges, in part, because research is unlikely ever to be truly conclusive and without controversy or dispute (Widiger & Clark, 2000; Widiger & Trull, 1993).

Nevertheless, it would seem prudent and wise to at least consider the potential risks and costs of providing official recognition of such diagnoses within the APA manual (Frances, 2010b). It is disappointing, if not seriously troubling, that the official DSM-5 literature review for the inclusion of pathological gambling in a new class of disorders titled addiction and related disorders is confined simply to these two sentences: “Pathological (disordered) gambling has commonalities in clinical expression, etiology, comorbidity, physiology and treatment with Substance Use Disorders. These commonalities are addressed in the following selected papers from a relatively large literature” (APA, 2010, para. 1). These two sentences are followed by a list of articles that pertain indirectly to the proposal by providing data that document various commonalities between substance dependence and pathological gambling (e.g., Cavedini, Riboldi, Keller, D’Annucci, & Bellocchi, 2002; Hodgins, Currie, & El-Guebaly, 2001). None of these articles directly addresses the question of whether pathological gambling is an addiction syndrome, with one exception: an article by Petry (2006), largely in opposition to the proposal.

The APA guidelines for DSM-5 work group members specify that any change to the diagnostic manual should be accompanied by “a discussion of possible unintended negative effects of this proposed change, if it is made, and a consideration of arguments against making this change should also be included” (Kendler, Kupfer, Narrow, Phillips, & Fawcett, 2009, p. 2). Kendler et al. (2009) stated that “the larger and more significant the change, the stronger should be the required level of support” (p. 2) and that the inclusion of a new diagnosis represents the very highest level of change (i.e., a major change). The creation of a whole new class of addiction disorders would appear to be a change of even greater magnitude than a major change. With respect to a major change, it is stated that this proposal must consider the potential harm that could arise by the inclusion (for instance) of a new diagnosis and whether such potential harm might exceed the benefits of its inclusion. “The main concern of DSM-V should be to avoid harm to the affected
individuals, but sometimes broader social or forensic issues that involve potential harm to patient or non-patient groups may warrant consideration” (Kendler et al., 2009, p. 2).

It is clearly evident that the DSM–5 Substance-Related Disorders Work Group has failed to follow the guidelines specified by the APA for making a major change to the diagnostic manual (Frances, 2009). It is not difficult to anticipate a number of possible negative consequences for this major revision, such as the impact on personal sense of responsibility, a potential explosion of unnecessary addiction treatment centers, costs to insurance coverage for these treatments, and efforts to mitigate criminal responsibility in legal defense (Frances, 2010b). Yet, there is no acknowledgment of any of these possible unintended consequences for creating a new class of addiction disorders within the official DSM–5 literature review (APA, 2010), let alone a forthright discussion that would suggest that the benefits would still outweigh the costs.

Shaffer’s Introduction to this handbook could be taken as a literature review to support such a change to the APA diagnostic manual. Shaffer provides a systematic, coherent, and compelling review of the relevant literature in support of recognizing a concept of addiction syndrome, rather than simply referring the reader to articles that would be included in a reference list for such a paper. However, Shaffer as well never acknowledges, let alone discusses, the potential risks and costs of such a broadened concept and in fact appear to embrace the expansion to eventually include shopping and other potential forms of behavioral addiction. This is not necessarily a criticism of their introduction, because they are not members of a DSM–5 work group describing an officially proposed revision to the APA diagnostic manual. Somebody, though, should address the potential negative consequences, not only because it is specifically stated as a requirement within the APA guidelines for making a major change to the diagnostic manual but also because it is the responsible thing to do (Frances, 2009).

**Future Research and Treatment**

An additional concern that has been raised with respect to a shift in the conceptualization of pathological gambling toward a behavioral addiction is the potential effect it may have on future treatment and research. Petry (2006), for example, expressed the concern that “clinics that treat primarily substance abusers may not be as experienced with, or receive a sufficient number of treatment seeking gamblers to have groups dedicated to them” (p. 157). As she indicated elsewhere (Petry, 2005b), treatment programs for pathological gambling have been developed that do not bear a close resemblance to the typical treatments provided for substance dependence. “Completely different interventions focusing on irrational cognitions associated with gambling are also being developed for which no parallel exists in the treatment of substance abuse” (Petry, 2005b, p. 4).

We suspect that treatment will continue as usual with the publication of DSM–5, because there are well-established clinics, clinicians, associations, and conferences devoted specifically to the study and treatment of pathological gambling. Substance use researchers and clinicians are unlikely to immediately assume responsibility for the study and treatment of pathological gambling. However, this concern has some validity.

As Frances (2010b, p. 2, para. 4) suggested in regard to the proposal to broaden substance use disorder to include behavioral addictions, “those working in the addiction field have a natural bias to see the scope of their specialty extended.” Specialists do seem, on average, to find it easier to expand their coverage than to narrow it, and the decisions made during the course of the development of a diagnostic nomenclature will at times be affected by this implicit self-interest. Moreover, when there are differences of opinion as to which section of a diagnostic manual a newly proposed condition belongs, the specialists with the greater power in the decision-making process are more likely to obtain the diagnosis.

For example, during the course of the development of DSM–IV (APA, 1994), Spiegel and colleagues (Cardena, Lewis-Fernandez, Bear, Pakianathan, & Spiegel, 1996; Task Force on DSM–IV, 1991) proposed a new diagnosis, brief reactive dissociative disorder, for inclusion in the dissociative disorders section. The predominant phenomenology consisted of symptoms of dissociation, including derealization, depersonalization, detachment,
stupor, and amnesia. However, brief reactive dissociative disorder closely resembled posttraumatic stress disorder (PTSD), classified as an anxiety disorder but with a shorter duration.

Compelling arguments were made for moving PTSD to the dissociative disorders section (Cardena, Butler, & Spiegel, 2003; Spiegel & Cardena, 1991). For example, the etiology and treatment of people with PTSD more closely resembles the etiology and treatment of dissociative disorders than most anxiety disorders (e.g., panic disorder, social phobia, obsessive–compulsive anxiety disorder, specific phobia). Dissociative identity disorder and dissociative amnesia are almost invariably in response to having experienced, witnessed, or been confronted with a PTSD stressor. The cognitive pathology of PTSD and dissociative disorders concerns difficulties accepting or integrating a severe trauma (expressed dysfunctionally through gross denial, avoidance, recurrent recollections, or all of these). The theories, treatment techniques, and concerns of people who specialize in crisis intervention, trauma, victimization, and abuse may overlap more with those of specialists in dissociative disorders than with those of specialists in anxiety disorders.

Another option was to create a new class of disorders of extreme stress that would include both PTSD and brief reactive dissociative disorder within neutral territory. Nevertheless, the final decision was to change the name to acute stress disorder and to place it in the anxiety disorders section (Frances et al., 1995). Despite having been originally conceptualized as a dissociative disorder, little to no recognition of this perspective has remained, and few dissociative disorders researchers or clinicians are likely still involved in either its research or treatment. A similar outcome befell depressive personality disorder, which morphed into early-onset dysthymia (Widiger, 1999).

A similar outcome is likely to befall pathological gambling, albeit at a slower pace. It may at first be convenient to let pathological gambling specialists continue to predominate the study and treatment of the condition; perhaps they will continue to do so for quite some time. However, it is also possible that the new behavioral addiction (or addiction syndrome) specialty will be predominated by substance abuse and dependence researchers and clinicians because of their greater number, funding, and other sources of power; eventually, they might exercise the predominate control over its diagnosis, research, and treatment. This is not necessarily a bad thing, but it is something that should be addressed openly and forthrightly.

CONCLUSIONS

The field of addiction is shifting in a positive direction away from a purely physiological model of substance dependence toward a broader cognitive–behavioral model of addiction. This evolution reflects a natural progression in the understanding of addictive behavior that has developed over years of systematic and informative research. There are concerns that naturally arise with such a fundamental shift in conceptualization and diagnosis, including the tendency to still hold onto, unnecessarily, a neurophysiological reductionism, opening potential floodgates for faddish and capricious diagnostic constructs and an unknown future for the study and treatment of pathological gambling. Nevertheless, these are all concerns that can be readily managed and addressed through the course of expanding and broadening the scope of addiction diagnosis.

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I started writing this chapter during 2010, on my return from study visits to see former students in three cities of Brazil. In the megacity of São Paulo, cocaine use has reached a relatively high endemic level, akin to the early 1990s experience in the United States. The United States, starting in the early to mid-1960s, experienced 10 to 15 years of epidemic growth of newly incident users, with peak incidence during the mid- to late 1970s. During the 1980s, the estimated annual number of newly incident cocaine users dropped to a plateau of endemic values in the 1990s before more recent declines described later (Anthony, 1992). In Brazil, medical and public health responses to crack smoking and other forms of cocaine use are already activated, as one might expect at this stage of an epidemic. The focus is on treatment and rehabilitation of seriously cocaine-affected individuals, with more modest investments in other forms of primary prevention, including some early outreach to young people at risk of starting to use and to newly incipient users. This pattern of public health investment is typical of what was seen in U.S. communities when the public health response to cocaine use had reached a steady state.

Actually, for many U.S. communities, the public health response to cocaine appears to be declining, consistent with epidemiological dynamics of cocaine use. The most recent results from the U.S. National Surveys on Drug Use and Health (NSDUH) indicate a promising downward shift from the endemic plateau of the 1990s. For example, as of 2002, the estimated number of newly incident users of crack cocaine in the United States was between 300,000 and 350,000. In 2003, that number had become roughly 270,000; by 2004, it was 215,000. By 2008, the estimated number of newly incident users of crack cocaine had fallen to 205,000. The most recent estimates, since 2009, have dropped below 100,000. As for all forms of cocaine, including crack cocaine as well as cocaine hydrochloride powder, the corresponding number of newly incident cocaine users estimated in 2002 was close to 1.2 million; in 2004, it was close to 1 million. By 2008, this incidence value had fallen to a value between 700,000 and 730,000. Recent estimates, since 2009 are below 620,000 (Substance Abuse and Mental Health Services Administration, 2004, 2009). These numbers would seem to represent important downward shifts for the United States.

In contrast to the apparent steady-state high endemic level in São Paulo during 2010, and an apparent decline from a high endemic level in national estimates for the United States during the first decade of this century, there continue to be alarm bells in the more northern Brazilian city of Salvador da Bahia and a continuing dynamic situation in its southern city of Porto Alegre. Of course, in these matters, few other countries have the stable epidemiological surveillance trend estimates of the type found in the United States. In most places, the epidemiological impressions of trends are from the more traditional but still valued key-informant approach of the type used in psychiatric epidemiology generally (Lin & Standley, 1962). In the United States and in other regions, the National Institute on Drug Abuse's Community Epidemiology Work
Groups serve this key-informant function. In a periodic review of epidemiological dynamics of extramedical drug use in the United States and in these regions, Community Epidemiology Work Group meetings elicit and synthesize survey and record-based data with expert judgments of clinicians, public health professionals, and researchers (Kozel, Robertson, & Falkowski, 2002).

Looking to the cocaine situation in other parts of the world, it must be said that 20 to 30 years ago the use of cocaine hydrochloride powder or crack cocaine was not much of a public health concern in Latin America, Australia, New Zealand, Spain, or other middle- to high-income countries of the European Union. Outside North America, the number of cocaine users in general population surveys was often too small for statistical analysis. At that time and in more recent years, global demand for cocaine could largely be traced to the U.S. population. This situation began to change during the 1990s and in the early 2000s.

Epidemiological survey evidence compiled by Maxwell (2008) showed 4% to 5% estimated cumulative incidence proportions for cocaine use in Australia from 1995 to 2004, compared with corresponding U.S. values at the 10% to 13% level. More recent estimates from Australia have indicated increases to the 5% to 6% level (Australian Institute of Health and Welfare, 2008). Corresponding survey-based estimated cumulative incidence proportions from the recent coordinated multisite World Mental Health Surveys have indicated that the United States continues to be in the top tier (14%–16%), with Colombia, Mexico, Spain, and New Zealand in a second tier (4%–6%), and with a third tier that encompasses other participating countries in which cumulative incidence proportions are at less than the 1% to 2% level. The countries in the third tier include Israel, Japan, Ukraine, Nigeria, and China (i.e., not constrained to the low- to middle-income countries, as one might mistakenly infer by considering only the United States, Australia, and New Zealand; Degenhardt, Chiu, et al., 2008). McCrystal, Mayock, and Hannaford (2010) in Northern Ireland recently expressed concern that increasing trends for that country and for other countries of the European Union might follow the trend lines seen for Australia since the late 1980s. There is also recent concern about outbreaks of cocaine use in China, traced back to coca leaf plantations in South America. Apparently, the U.S. Drug Enforcement Administration has started to help the Chinese government with its cocaine supply interdiction efforts (Associated Press, 2006).

What does the present situation concerning the epidemiological dynamics of cocaine use teach us? The public health research community is clearly now able to ask and answer questions with respect to some quite basic, albeit important, topics pertinent to these epidemiological dynamics. Specifically, as illustrated by this introduction to cocaine epidemiology, epidemiologists know how to tap the expertise of key informants for initial clues about outbreaks that rise and fall, to complete transversal (cross-sectional) surveys. In some instances, it has been possible to estimate or approximate cumulative incidence proportions and annual incidence rates as well as other epidemiological parameters of dynamically changing patterns of extramedical drug use. In addition, epidemiologists have a capacity to do so in a fashion that permits initial comparisons from place to place and from time to time. They know how to ask epidemiological questions about what general conditions might account for the observed variation in the cumulative incidence proportion as one parameter of epidemiological dynamics. Nonetheless, much less is known about the underlying mechanisms and which of the various policy instruments or specific public health prevention, early outreach, or treatment and rehabilitation programs might be manipulated to achieve the declines now being observed in the United States.

As such, the present situation represents an exciting time for epidemiologists who have been studying the rise and fall of the U.S. cocaine epidemic of the late 20th century and the later developments in other countries. In this context, what follows is an overview of selected facets of research about epidemiological dynamics, with illustrations that go beyond cocaine epidemiology into the domain of epidemiological research about alcohol and tobacco as well as cannabis and the other internationally regulated psychoactive drug compounds. At the conclusion of each major section, I reflect on the main theme of this
handbook—namely, the syndrome model of addiction, with consideration of the syndromal forms that encompass behaviors other than drug self-administration. It is beyond the scope of this chapter to cover all of the facets of epidemiological research focusing on these compounds and the population studies of their suspected adverse or beneficial effects, prevention, and control. Some readers may be interested in chapters with related content, including a section in the most recent edition of Kaplan and Sadock's Comprehensive Textbook of Psychiatry (Strain & Anthony, 2009) and other source materials (Anthony, 2002; Brook, Kerstin, & Rubenstone, 2008).

SCOPE OF THE CHAPTER

Epidemiological dynamics describes the scope of this chapter, with a focused look at epidemiological dynamics most pertinent to the initiation and persistence or cessation of psychoactive drug self-administration behaviors. I consider some of the associated neuropsychiatric and behavioral syndromes that can emerge under conditions of persistence, along with associated hazard-laden and socially maladaptive consequences of these behaviors and syndromes.

Nevertheless, for the most part, epidemiological researchers working in this area have studied extramedical use of these psychoactive drug compounds—namely, consuming alcohol, tobacco, or a nonprescribed internationally regulated drug without a prescription or exceeding the boundaries of what has actually been prescribed by a clinician (e.g., taking a prescribed drug at doses, in frequencies, or for reasons that go beyond the clinician’s instructions). In some instances, extramedical use of over-the-counter nonprescription psychoactive drug compounds has been studied; here, the drug use exceeds the boundaries of approved indications, as in a young person’s use of a dextromethorphan-containing antitussive cough syrup for kicks or to get high. For the most part, this research has been focused on drug self-administration; in some instances, the developmental consequences of a mother's self-administration of drugs during pregnancy has been studied as a form of secondhand administration or prenatal fetal exposure (e.g., Bandstra et al., 2011).

As noted at the beginning of this chapter, positioned in the second decade of the 21st century, epidemiologists are well equipped to ask and answer questions about the epidemiological dynamics at play when extramedical drug use spreads through a population, first in outbreaks and then in epidemic fashion, and during the endemic steady-state conditions. Whether the same can be said for the epidemiological dynamics at play during outbreaks of other behaviors is not clear, particularly when there is no clearly identifiable and necessary material cause.

For communicable disease research generally, there is an infective agent and the host response to the agent within the context of prevailing environmental conditions, as is expressed in the epidemiologic triangle, sometimes called the agent-host-environment model. The same is true for public health research dealing with extramedical drug use, in which an agent is in the form of the drug compound. The presence of the agent in the community is measurable with appropriate sampling, even without direct assessment of individual hosts (e.g., see Ort, Lawrence, Rieckermann, & Joss, 2010). Agent-host contact can be documented using careful self-report methods designed to enhance reporting and recall accuracy, when a past history of drug-taking is required. In some contexts, researchers can combine toxicological assays with self-report data to improve accuracy of measurement, as has been demonstrated by research on the neurobehavioral and neurocognitive development of newborns when mothers have taken cocaine during pregnancy (e.g., see Bandstra et al., 2011).

For research investigating the epidemiological dynamics of conditions such as sex addiction, for which there is no single necessary agent in the web of causation, a more fruitful source of concepts, principles, and methods might be an interrelated line of epidemiological research about the dancing manias of the Middle Ages, extraordinary popular delusions or rumors that spread through crowds, and other forms of socially shared behaviors and psychiatric disturbances (e.g., see Daley & Gani, 2001; Gruenberg, 1957). In the context of these other addictions, researchers are in a position to ask questions, but their capacity to answer the questions is somewhat less certain than is the case for the
epidemiological dynamics involving extramedical drug use. When researchers measure extramedical drug use in the epidemiological context, they can confirm and document the experience (e.g., via toxicological assays). Experience with one drug compound can be differentiated from experience with another drug compound to the extent that either bioassay or self-report can be relied on to make this differentiation between agents. The degree of certainty is constrained when there is neither a specific agent nor a host response to an underlying necessary causative agent of the type studied in drug dependence epidemiology. Research about the epidemiological dynamics of these syndromes is inherently more difficult when the causative agent is not known.

Keeping in mind the fact that the drug compound is a clearly identifiable and necessary material cause of the neuropsychiatric and behavioral syndromes variously called morphinomania, cocaism, drug habit, drug dependence, and drug addiction, the readers of this chapter may appreciate its orientation to the agent–host–environment interplay of the epidemiologic triangle. Epidemiology and sociology, with psychology or psychiatry, might suffice by themselves when the goal is mastery of the epidemiology of mental disorders in general or the epidemiology of specific mental disorders such as major depression. Nonetheless, it is not possible to master the epidemiology of extramedical drug use or the epidemiological dynamics of drug-caused clinical syndromes without drawing on neuropsychopharmacology as well. To pretend otherwise would be akin to an antiquated claim that it is possible to master the epidemiology of bacterial or viral diseases such as tuberculosis and AIDS without paying attention to bacteriology and virology—notwithstanding an appreciation that tuberculosis and HIV and other infections now are co-occurring in marked association (i.e., are comorbid). Even when one or more underlying shared susceptibility traits can help account for occurrence of these infections, the epidemiological dynamics of the agents and the resulting disease states can and should be differentiated by research steps leading toward models of co-occurrence. When major depression and generalized anxiety disorders co-occur, there is no need to pay attention to specific agents in the overlapping web of causation. When major depression, generalized anxiety disorder, and drug dependence syndrome co-occur, the patterns of co-occurrence are conditioned by whether the major depression and generalized anxiety disorder cases have ever self-administered or been exposed to a psychoactive drug compound. It is regrettable that most psychiatric comorbidity research has neglected this conditioning.

In the rest of this chapter, I cover important issues of this type, showing that epidemiological dynamics are manifest among a broad range of population health studies. I introduce the reader to a limited coverage of that range via a selection of four facets that are most pertinent to epidemiological dynamics when the topic is prevention and control of the drug use disorders, such as drug dependence and other clinically recognizable syndromes that develop concurrent with neuroadaptational changes after effective agent–host contact. The first facet of epidemiological dynamics involves the concept of a reservoir and public health interventions that are intended to disrupt reservoir processes. With respect to psychoactive drug agents in the epidemiologic triangle, the reservoir includes croplands (e.g., for coca leaf or cannabis leaf) as well as factories engaged in legal and illegal manufacture, supply, and distribution lines, as depicted in a graphic illustration of the reservoir for psychoactive prescription drug products (Anthony, 1983). When no specific agent exists in the web of causation of a syndrome or disease, the concept of a reservoir is not applicable.

The second facet of epidemiological dynamics involves the modeling of epidemic spread through populations, typically using deterministic or probabilistic models, in which the parameters of interest are population-level rates as they affect susceptible individuals and as affected individuals become vectors in the person-to-person sharing and spread of drug-taking in a population. I introduce drug-specific concepts of drug exposure opportunity, the first chance to try a drug, and drug use, conditional on exposure, in relation to this facet as well as more general epidemiological concepts such as serial time and induction intervals. I also introduce the topic of peer contagion in this section of the chapter.
The third facet of epidemiological dynamics involves modeling the persistence and rates of drug taking by affected individuals. Typically, these epidemiological parameters are estimated for subgroups of the population soon after the onset of drug taking—perhaps so soon and at such low rates that the just-mentioned clinical syndromes have not yet developed. Here, in theory, the epidemiologist's approach to research focusing on population dynamics can be applied by analogy to syndromes without an agent. That is, the origin of a timeline for estimating persistence and rates of drug taking can be set at the first drug use experience. The epidemiologist can then work his or her way down that timeline to investigate whether, when, and how often drug use occurs again, after that initial drug exposure in the form of actual drug self-administration.

For gambling research, the epidemiologist might set the origin of the timeline on the date of the first gambling experience, irrespective of the type of gambling, or at the first gambling experience of a specific type (e.g., online poker of any type) versus the first gambling experience of a different specific type (e.g., online Texas Hold 'Em poker, Texas Hold 'Em poker at the Elks Club with penny ante, higher stakes Texas Hold 'Em poker at a casino). The epidemiologist can then estimate the persistence of that form of gambling, or of gambling in general, from that time of origin onward.

As Shaffer (in the Introduction to this handbook) makes clear, for some gamblers, the initial occasions and persistence of gambling can start to drive the development of a gambling dependence or gambling addiction syndrome, whereas for other gamblers no such syndrome formation occurs. One presumes that the onset of the syndrome then begins to drive forward the repetitions of gambling behavior. If the process in gambling syndromes is analogous to that seen in drug syndromes, then there are neuroadaptive changes concurrent with initially reinforcing experiences. The reinforcing function of the behavior then begins to drive forward the formation of the syndrome, and a feedback loop occurs such that the syndrome begins to drive forward subsequent behaviors and influence the subsequent persistence and rate of the behavior (Anthony, 2010). This perspective presumes that similar processes are at play across various expressions of the addiction syndrome (e.g., sex addiction, Internet addiction), even in the absence of a necessary causal agent such as a drug, which is the nub of the thesis advanced by Shaffer (in the Introduction to this handbook).

The fourth and final facet of epidemiological dynamics involves estimating the just-described conditional probability of becoming a case of drug dependence once drug use starts as well as other manifestations of transitions across states and processes that cannot be observed until or unless at least one effective agent–host contact has occurred. Nevertheless, a single effective agent–host contact cannot exist except where the susceptible host has a chance of being exposed to the agent. That is, the occurrence of an effective agent–host contact depends in part on what Wade Hampton Frost called exposure opportunity, which can be made operational and measured as the age or time at which a person experiences the first chance to try a drug. The late Lee N. Robins made a pioneering contribution in relation to this facet of epidemiological dynamics in a chapter titled “Estimating Addiction Rates and Locating Target Populations: How Decomposition Into Stages Helps” (Robins, 1977). Later research about this facet of epidemiological dynamics during the earliest stages of drug involvement was derived largely from Frost's idea of exposure opportunity and Robins's associated idea of decomposition into stages.

In contrast to other chapters and monographs, this chapter is more tightly focused on the concept of epidemiological dynamics and the syndrome sometimes termed addiction. Considered from the most frequently expressed viewpoints on the concept of dynamics, a psychological or psychiatric perspective of disease dynamics is oriented toward the study of the individual patient. The Introduction to this handbook expresses this perspective. For example, in the Introduction, the concept of the dynamics of the addiction syndrome is described as follows:

It is important to note that contrary to its implicit status as a static state, addiction is a dynamic process that waxes and wanes. People with addiction often go through
phases of exacerbation and abstention; many also have episodes of controlled activity whereby they use the object of their addiction intermittently or more often but within manageable limits. (Introduction to this handbook, p. xxxi)

A similar perspective can be adopted in epidemiological research. The title of Lee Robins's 1977 monograph chapter mentioned earlier has two advantageous features. First, it reinforces the orientation of epidemiological research toward its source populations. Second, it refers to a concept of stages that, as described, are exchangeable with the just-mentioned concept of phases. Previously published chapters (e.g., Anthony, 2002) provide detailed descriptions of the important staging conceptual model sketched more than 30 years ago in Robins (1977) as well as a critique of that model as offered by Room (1977). In this chapter, I present a more abbreviated version, with citation back to the original source material.

Notwithstanding the individual-level patient-oriented concepts of disease dynamics in this context, readers of this handbook may appreciate that there can be a somewhat different population-level orientation to the concept of these dynamics. Indeed, for most of the history of epidemiology, epidemiological dynamics concepts have been manifest in mathematical models for rates estimated at the population or social group level down to the household level, as in Chapin's concept of the secondary attack rate (Anthony, 2006) and in estimating characteristics and parameters measured at the level of the presumed disease-causing agent (i.e., not just at the level of the patient). To demonstrate the nature of this concept of epidemiological dynamics, as it is understood in epidemiology generally, I turned to the Thomson Reuters Web of Science bibliographic search tool and identified the three most highly cited entries among 64 science articles on epidemiological dynamics. All such articles with epidemiological dynamics in the title, abstract, or keywords had a focus on population-level processes. For example, Ferguson, Galvani, and Bush (2003) found that the modeling of influenza dynamics depended heavily on parameters measured at the supraindividual level, including season of the year, contact rates for mixing of 12 million hosts distributed across 20 geographically disbursed patches, and the basic reproduction number as a manifestation of occurrence of secondary cases once a primary case has occurred. Rambaut et al. (2008) described a source–sink model for the evolution of influenza A virus, according to which viral genetic and antigenic diversity is traced to a dynamic process within the reservoir or source population (e.g., the equatorial tropics of each hemisphere of the world). Thereafter, there is exportation to sink populations of the more temperate zones of the northern and southern hemispheres (Rambaut et al., 2008). Koelle, Rodo, Pascual, Yunus, and Mostafa (2005) modeled variation in the time intervals between outbreaks of diseases such as cholera, taking into account not only population-level characteristics (e.g., the proportion of susceptible individuals in the population vs. those with immunity) but also climate change.

The fourth most highly cited article in the Thomson Reuters epidemiological dynamics search had an individual-level focus. Hu et al. (2001) studied demographic, behavioral, and immune system parameters of 130 HIV-1–infected seroconverters identified at methadone maintenance centers in Thailand as suspected determinants of serial HIV-1 RNA viral load, natural killer cell activity, and CD4–CD8 counts measured after seroconversion, with an eye toward viral load variations traced back to HIV-1 subtypes. After describing the observed patterns of association by studying individual patients as a group, and with a pattern of estimated associations that linked viral load values back to subgroups defined by HIV-1 subtype, Hu et al. acknowledged that the observed pattern of association actually might be due to supraindividual processes—namely, the epidemiological dynamics of HIV transmission in Thailand. That is, my search captured this individually oriented article because its abstract alluded to the more population-level and infective agent facets of the interperson transmission process. Their study was not a study of epidemiological dynamics per se.

I should add that a separate search for the Boolean combination of "epidemiological dynamics" AND "drug" turned up three articles on topics unrelated to drug addiction (e.g., concerning
antibiotic-resistant infective agents). Searches for “epidemiological dynamics” AND “addiction” and for “epidemiological dynamics” AND “drug” disclosed no entries.

Interestingly, the most highly cited article identified in my recent search for the Boolean combination of “dynamics” AND “addiction” is attributed to the team of Robert DuPont and Mark Greene, who with Leon Hunt and Carl Chambers studied and contributed insights into the U.S. mid-century heroin epidemic (e.g., see DuPont & Greene, 1973; L. Hunt & Chambers, 1976). These works deserve the careful study of 21st-century epidemiologists, as do prior contributions about concurrent heroin epidemics in the United States and the United Kingdom during the late 1960s through the 1970s as well as the possibility that epidemics of this type can be disrupted via introduction of new outreach, early intervention, and treatment programs. I discuss these matters later in this chapter in relation to the work of Kosviner et al. (1968); De Alarcon (1969); Hughes and Crawford (1972); and Hughes, Barker, Crawford, and Jaffe (1972).

Before I consider the first facet of epidemiological dynamics, an important note about terminology and word usage should benefit the reader. Lewis Carroll of Through the Looking Glass fame encouraged the mastery of words, lest the words become the masters, and T. S. Eliot in Sweeney Agonistes warned, “I gotta use words when I talk to you.” In public health work, a primary goal is to reach out to the population for the purposes of outreach and interventions as early as possible in the various stages of drug involvement. However, when word usage shifts in the direction of inherently pejorative terms such as illicit (vs. illegal), drug abuse, addict, or addiction, in contrast to word usage based on the less stigma-laden terms recommended by international consensus panels of the World Health Organization since the early 1960s (e.g., drug dependence, harmful drug use), outreach and intervention goals can be disrupted. During early outreach work with drug users at large in the population, the use of stigma-laden terms has not served researchers well because of their unmistakable connotations. This is not to say that these pejorative terms are useless to clinicians, who might wish to take advantage of the stigma-laden content in the context of treatment and rehabilitation (e.g., when states of denial are thwarting successful care and management of a patient who suffers from an addiction).

As was intended by the World Health Organization panels, in public health outreach the term drug dependence has not evoked negative responses of the type seen in response to the term addiction, although in the context of prescribing anti-anxiety or pain medicines some patients express worry about the stigma associated with becoming dependent on these medicines (Rippere, 1978). For this reason, in the work of our research group the terms used are illegal drug use (rather than illicit drug use or drug abuse), except when it is necessary to refer to the Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision; DSM-IV-TR; American Psychiatric Association, 2000) concept of drug abuse. Consequently, in lieu of drug addiction, I use the terms drug dependence or drug dependence syndrome and drug hazards or harms. Nonetheless, I titled this chapter to be consistent with the title of the handbook, and I appreciate that many of the readers of this volume will be clinicians whose work largely is focused on treatment of severely affected patients for whom denial states are profound and for whom the statement “I am an addict” represents a therapeutic milestone of importance.

**Epidemiological Dynamics: The First Facet**

The first facet of epidemiological dynamics to be covered in this section involves the concept of the reservoir. Thereafter, the section covers the distinction between upstream and downstream interventions.

**Concept of a Reservoir**

Once scientists identified specific bacterial and viral agents of disease during the late 19th century, early theories of disease-causing miasma evaporated. The rationale for draining swampland shifted from the logic of miasma clearance to the logic of controlling mosquitoes and other vector-borne agents of infection as well as water sanitation generally. Whenever identifiable infective agents are present that qualify
as necessary causes in disease processes, an especially important facet of epidemiological dynamics research involves the study of the reservoir from which the infective agent emerges to come into contact with susceptible hosts and where the agent breeds or multiplies so as to increase the probability of an effective contact with a host. Some infectious disease epidemiologists' careers have been focused entirely on these agent-generating and dispersion processes. This focus can be traced to a recognition that some of the most effective public health interventions work at the level of the reservoir far upstream from later agent–human contact and the consequences of that contact. Some public health research is also focused on the reservoirs that make psychoactive drug compounds available for extra-medical drug use and the human vectors that convey the drugs from their reservoir sources to susceptible hosts (e.g., Hoffer, Bojshev, & Morris, 2009; Montagne, 1991).

**Reservoirs and Upstream Versus Downstream Interventions**

Control of extramedical drug use and problems associated with drug dependence includes attempts to disrupt reservoir processes, which are often characterized as **upstream interventions** in relation to the position of the individual hosts. An apt analogy for an upstream versus downstream intervention can be drawn in relation to an upstream draining of the swampland to thwart downstream density of mosquito vector-borne infections of humans and subsequent diseases such as yellow fever and malaria. In contrast, a **downstream intervention** would involve window screens and netting to prevent the agent–vector–host contact in the more proximal environment of the susceptible host.

Crop eradication or substitution programs for opium- and coca-growing farmers are analogues to swamp draining and qualify as upstream interventions to disrupt reservoir processes, with intended beneficial effects on population-level occurrence of extramedical drug use and drug dependence. Parenting programs intended to increase levels of parental monitoring and surveillance of children and adolescents are analogues to putting screens on windows and qualify as downstream interventions intended to shield susceptible hosts (e.g., see Chen et al., 2004).

Of course, neither upstream interventions nor downstream interventions are always as effective as theory might lead one to believe. Moreover, it is widely appreciated that a downstream intervention at the level of the individual host, group, or household might have unintended adverse side effects or consequences. For example, later I discuss an illustration of this type in relation to the work by Dishion and Andrews (1995); Dishion, McCord, and Poulin (1999); Duncan, Bojsjoly, Kremer, Levy, and Eccles (2005); and others, who examined peer-to-peer influences on heavy drug involvement and other maladaptive behavior, wherein the grouping of drug-using peers in early intervention groups or university dormitories might have undesirable outcomes. The same has been observed for upstream interventions directed toward the reservoirs for drug use; in some instances, the presumed beneficial effects are questionable, as I illustrate in the next section.

**Questionable Effects of Upstream Interventions: The Controlled Substances Act**

As an example of how an upstream intervention can underachieve with respect to an intended beneficial effect, an appropriate example can be seen in relation to Schedule II of the Controlled Substances Act (CSA) of 1970 and in later revisions of that legislation. As many readers can appreciate, at the federal level, CSA set forth five schedules, or levels of control (Schedules I–V); it assigned to the top four schedules all of the various internationally regulated psychoactive drug compounds. The CSA reserved Schedule V for psychoactive drugs judged to have a safety profile indicating little risk of public health hazard, such as the nonprescription or over-the-counter sedative–hypnotic preparations containing small amounts of phenobarbital or other barbiturate drug compounds.

Schedule IV anchors the lowest level of federal drug control in relation to psychoactive drug products widely available by prescription. At the other end of the control spectrum, Schedule I represents the highest level and is reserved for psychoactive
drug compounds having no federally approved indications for use in clinical practice, even though the drugs might be used quite legitimately with government approval in other countries (e.g., heroin and 3,4-methylenedioxymethamphetamine [MDMA] approved for medical use overseas but not in the United States). Despite medical marijuana laws in many U.S. jurisdictions, cannabis continues to be assigned by the federal government to Schedule I. The list of other psychoactive compounds in Schedule I include heroin, injectable methamphetamine, MDMA, peyote, and other drugs for which the U.S. federal government declines to give approval for use in the legitimate practice of medicine.

Schedule II drugs include cocaine, morphine, and other prescription drugs with federally approved indications for use in the practice of medicine in the United States but with a safety profile that prompts some special drug control restrictions on supply and distribution. For the purposes of this example, one of the restrictions is that a new prescription must be written and signed by an authorized practitioner each time a Schedule II drug is dispensed; the concept of a refill or refillable prescription does not apply to Schedule II drugs.

At the time the CSA was enacted, Schedule IV drug compounds included barbiturate sedative-hypnotic drugs (e.g., pentobarbital, secobarbital) as well as some of the early benzodiazepine anxiolytic drugs (e.g., diazepam) and other psychoactive compounds judged to have a safety profile greater than the Schedule V preparations. For the purposes of this example, prescribers of Schedule III and IV compounds can write prescriptions to allow multiple refills, whereas this is not the case for compounds assigned to Schedule II. The schedules also vary in their degree of surveillance and control of reservoir processes with respect to each drug compound's manufacture, supply, and distribution (Anthony, 1979, 1983).

It is worth noting that the CSA did not involve the scheduling of all psychoactive drug compounds available for legitimate prescribing in the United States. For example, methaqualone (Sopor, Quaalude) was among the sedative-hypnotic drugs on the market at that time, but it was not regulated in the original CSA. Dextropropoxyphene (Darvon) was also a widely marketed unscheduled prescription analgesic compound not scheduled by CSA. Because of increased public concern about its use outside the boundaries of prescribed medical use for an approved indication (i.e., extramedral use), methaqualone was reassigned from unscheduled prescription status to CSA Schedule II in mid-1973. By 1983, it had been withdrawn from the market after a period of declining sales associated with clinician appraisal of its disadvantageous safety profile compared with other sedative-hypnotics then being marketed (e.g., benzodiazepines). In the mid-1970s and again in 2010, the Food and Drug Administration reevaluated dextropropoxyphene's safety profile, first assigning it to the CSA schedules and then directing its removal from the U.S. market. Both methaqualone and dextropropoxyphene continue to be available for approved medical indications in other countries. I mention these two examples because they serve to illustrate how federal-level judgments about safety profiles can change over time and vary from one jurisdiction to another.

After the CSA was enacted, concurrent with expressions of public health concern about methaqualone's disadvantageous safety profile, there were expressions of concern about several other compounds originally assigned to Schedules III and IV. Thereafter, about the same time methaqualone was reassigned from unscheduled status to CSA Schedule II, pentobarbital, secobarbital, and amobarbital were reassigned from Schedule IV to Schedule III. An initial before-and-after analysis of the impact of drug scheduling and rescheduling decisions led to a U.S. government report heralding the beneficial impact of these federal regulatory decisions on the occurrence of drug abuse emergency episodes being detected in Drug Abuse Warning Network surveillance. Regrettably, the simple before-and-after analysis might have produced misleading inferences about the beneficial effects of these regulatory decisions. A less favorable conclusion was drawn after a more detailed reanalysis using the multiple interrupted time series design, with control medicines considered as well (Anthony, 1979).

Nonetheless, an unintended consequence of assigning methaqualone to Schedule II might have been an increase in the size of each new prescription
issued. That is, the evidence from the multiple interrupted time series analysis indicated that reassignment of methaqualone from unscheduled prescription status (with no restrictions on refill prescriptions) to Schedule II was followed by a tangible increase in the number of dosage units dispensed at the time each new prescription was issued. This increase was gauged by comparing time trends in dosage units for the new prescribing of control medicines that had not been assigned to Schedule II when methaqualone was controlled (Anthony, 1983). If the evidence from the multiple interrupted time series “natural experiment” can be believed, then an intended beneficial effect of controlling a drug reservoir process might have produced an undesirable consequence. I suspect that increases in the number of dosage units prescribed with each new prescription was not an intended consequence of CSA and that the increased number of dosage units might not be advantageous for the health of the individual patient or the patient’s family members (e.g., see Garnier et al., 2010).

The U.S. federal government’s upstream interventions intended to control the reservoir of coca leaf production, subsequent processing of cocaine hydrochloride powder, and transshipment of cocaine hydrochloride from countries of origin across the U.S. border might also not have the intended effects. Reservoir-focused interventions in the form of coca leaf crop eradication and crop substitution in the Andes, coupled with increased U.S. border security from the early 1980s onward, do not seem to have had tangible intended effects on cocaine availability, price, or purity in the United States (Manski, Pepper, & Petrie, 2001). In the United States, a general public consensus that these interventions must be having at least some beneficial effect continues to sustain taxpayer support for them. Libertarian sentiments and contemporary efforts to reduce the U.S. federal government budget deficit might disrupt that consensus in the coming decades. However, as I discussed earlier, for other reasons, the U.S. cocaine epidemic of the last 4 decades of the 20th century appears to be ending, with a drop in the number of newly incident cocaine users year by year toward a new low endemic plateau.

**Upstream Interventions With Respect to Tobacco and Alcohol**

Whether large in effect or small, current upstream interventions with respect to the internationally regulated psychoactive drug compounds have not eliminated the availability of psychoactive drugs in response to the demand for extramedical drug use. Supply lawfully rises to meet demand, and within this context, substantial investments in downstream interventions intended to reduce demand and to deal with associated problems have recently been made. These downstream interventions are the topic of other chapters in Volume 2 of this handbook, on matters of treatment, rehabilitation, and prevention and are not covered here, except as they relate to the medical and public health management of epidemics.

Nonetheless, before turning to the second facet of epidemiological dynamics, epidemic spread, readers should note that upstream interventions directed toward alcoholic beverages and tobacco seem to be yielding much more promising results than has been the case for internationally regulated drugs. In relation to these legal drugs, not yet subject to international controls, government-directed regulation of price, taxation, and availability might be the most effective policy instruments that constrain the availability of these compounds and the prevalence of their extramedical use (for recent reviews and new contributed evidence, see Popova et al., 2011; Ribisl, Williams, Gizlice, & Herring, 2011). In addition, whether induced by government policy or action or by other means, disruption of street-level availability of internationally regulated drugs may certainly influence the occurrence of extramedical drug use and drug-related harms (e.g., see Bejerot, 1975; Brill & Hirose, 1969; Lenke & Olsson, 1998). Even among proponents of depenalization for simple possession and use of these drugs, the risks associated with outright legalization are acknowledged (MacCoun & Reuter, 2001).

Finally, the concept of upstream prevention has recently encompassed interventions directed toward community- and school-level ethos about tobacco smoking and other extramedical drug use. Bonell et al. (2010) offered a description of a pilot experiment in which the intervention of primary interest was a manipulation to change the school ethos and
the outcome of primary interest was a reduction in the occurrence of alcohol, tobacco, and other drug use. Reflecting on the thesis of a unitary syndrome model of addiction, which provides an overarching conceptual frame for this volume, there are some challenges from the perspective of epidemiological dynamics, starting with the epidemiological facets just covered. As a reminder to readers, the thesis of a unitary syndrome model being advanced by Shaffer (in the Introduction to this handbook) is based on observations of (a) commonalities in neurobiological substrates, (b) commonalities of suspected causal influences (e.g., inheritance, psychological antecedents, social antecedents), and (c) commonalities of shared experiences (e.g., shared or concurrent manifestations, sequelae, parallel natural histories, object nonspecificity, treatment nonspecificity). Much can be gained by entertaining this thesis, including the virtue of parsimony in an empirically based theory. That is, as William of Occam’s principle is often stated, “Entities are not to be multiplied except as may be necessary.”

Shaffer (in the Introduction to this handbook) has stacked up three separate broad categories and a total of nine subcategories of empirical evidence against multiplication of entities now unified within the overall syndrome model. Nonetheless, counterbalanced with Occam’s Razor is Morgan’s Canon (Morgan, 1891). Scholars of the history of behaviorism know C. Lloyd Morgan mainly for his assertion that animal behavior should be explained by the simplest possible processes before attributing the behavior to higher mental functions. Nonetheless, Morgan also issued a warning: “We do not know enough about the causes of variation [in behavior] to be rigidly bound by the law of parsimony” (Morgan, 1891, p. 5).

An epidemiologist’s reflection on the thesis of a unitary addiction syndrome brings to mind the early 19th-century days of epidemiology, before bacteriology and virology advances during the later years of that century, when it was possible for experts to make the following types of assertions about epidemic diseases:

- Epidemic ... diseases were formerly universally considered to be essentially different in their nature, each being thought to depend upon its own specific contagion. The correctness of this view seemed to be confirmed by the great apparent difference between typhus, scarlet-fever, influenza, and cholera. Subsequently it has been thought that all these diseases may derive their origin from some common agent, modified by peculiarities of climate and other circumstances, and which, under varying conditions, gives to various forms and types of disease. Whichever of these views be adopted, it is agreed by the most eminent investigators that there is a general resemblance between these various forms of disease, and that they have the following characters in common:
  —They are all fevers; they all obey similar laws of diffusion; they all infest the same kind of places; they all attack the same classes [of people], and for the most part persons of the like ages; and their intensity is increased or diminished by the same sanitary and social conditions. (Addison, 1854, p. 6)

A look back to the very logical approach being used by Shaffer (in the Introduction to this handbook) to advance the thesis of a unitary syndrome model illuminates the resemblance to Addison’s (1854) substantiation of his unitary model of epidemic diseases (“They are all fevers ... their intensity is increased or diminished by the same sanitary and social conditions,” etc.; p. 6). Nonetheless, as people now know, Addison was completely wrong in this regard. The idea that cholera, typhus, scarlet fever, and influenza were interchangeable manifestations of a unitary epidemic disease model became preposterous once Robert Koch, in 1884, publicized his isolation of *Vibrio cholerae* as the necessary causal agent in cholera but not in other epidemic diseases. Actually, the Italian Pacini had isolated *Vibrio* in the same year Addison was writing, 1854, but Pacini’s work did not gain recognition until after Koch’s rediscovery of *Vibrio cholerae*. Later, in 1916, Brazilian Henrique da Rocha-Lima isolated *Rickettsia*
Prowazekii, a gram-negative parasitic bacillus, and found it to be the necessary causal agent of epidemic typhus. It was not until 1923 that Gladys and George Dick isolated hemolytic streptococcus as the necessary causal agent in scarlet fever, and in 1931 Richard Shope disclosed evidence that influenza was a virus-caused epidemic disease.

In this historical note about epidemiology of the diseases with specific infective agents as their necessary causes, and with due attention to Morgan’s (1891) warning, cited earlier, one may identify a reason to be somewhat cautious about unitary theories about health outcomes when there is reason for differentiation of the outcomes in relation to underlying necessary causal phenomena in the form of specific agents. For the drug dependence syndromes, as with most diseases caused by infective agents, there is a set of necessary causal agents, which can be illustrated with examples. Eradicate human exposure to the smallpox virus, and smallpox can be eradicated altogether; there has been zero incidence of smallpox infection for decades. If tobacco could be eradicated, there would be no tobacco dependence. The same is true for cocaine and cocaine dependence. The necessary causal agents in sex addiction or in addiction to running and strenuous physical exercise are not so clear, but if gambling and the Internet were eradicated, perhaps gambling and Internet addiction would disappear.

By making this argument, I do not intend to claim that there is no value in thinking about these various syndromes under the same umbrella, and the thesis of a unitary syndrome brings to mind some epidemiological thought experiments that can be followed by empirical research. For example, if the unitary syndrome thesis is correct and there is interchangeability among the various forms, then imagine a place where all but one of the behaviors yoked to the syndrome has effectively been banned—Singapore comes to mind as a possible location. Play out the thought experiment and consider what one might see in an epidemiological addiction syndrome survey of the population in that place. Then, imagine a place where there is no ban on any of the behaviors yoked to the syndrome—Las Vegas comes to mind as a possible location—and consider what might be disclosed via an epidemiological addiction syndrome survey of that place’s population. Is the prediction from the unitary syndrome model that the prevalence of addiction would be the more or less same in both places, such that addiction vulnerability in the first location would be wholly expressed in the single form of addiction to the unbanned behavior, whereas addiction vulnerability in the second location would be expressed in an identical addiction prevalence across multiple forms of possibly comorbid addiction syndromes? For example, one might discover basically the same survey-based addiction prevalence in both locations, with a minority of cases affected by monoaddictions (e.g., sex addiction only) and with a majority of cases affected by comorbidities (e.g., gambling addiction co-occurring with alcohol addiction).

This epidemiological thought experiment in its extreme form cannot be taken to the stage of empirical probing—mainly because there is not apt to be a place where all but only one form of the addiction-yoked behavior can be banned. The true extreme, a place where even sexual behavior is effectively banned, would most likely not be large enough and perhaps not viable long enough to complete an epidemiological survey, and it is likely that the local authorities would also ban the survey taking to which some epidemiologists seem to be addicted. Nonetheless, some partial tests of the theory might possibly be undertaken in new empirical research along the lines of the World Mental Health Surveys Consortium—provided the epidemiological survey assessment were broad enough to encompass all forms of behaviors that give rise to the unitary syndrome.

**Epidemiological Dynamics: The Second Facet**

The second facet of epidemiological dynamics involves the modeling of epidemic spread through populations. The literature on this topic is extensive and largely nonquantitative, enlivened by strongly opposing positions of prominent scientists such as Alfred Lindesmith and Nils Bejerot (e.g., see Agar & Reisinger, 2001, 2002; Bejerot, 1971; Golub &
Johnson, 1999; Lindesmith, 1973) as well as rich material of an ethnographic and sociological character:

The concept of a drug subculture implies that addicts are in contact with each other. In this contact, learning takes place. The learning can be of facts and techniques. For example, the neophyte can learn from more experienced addicts that his withdrawal symptoms are the result of not having had his usual dose of narcotics, and will be relieved by a dose; that the intravenous route enhances the drug effect; how to obtain narcotics, or money for narcotics; new sources of narcotics; how to prepare narcotics for administration, and other knowledge of this kind. (O’Donnell, 1969, p. 84)

A more quantitative approach, closer to the tradition of research on epidemiological dynamics of communicable diseases, has emerged in the direction of what is known as systems science in public health investigations (Mabry, Marcus, Clark, Leischow, & Mendez, 2010).

Modeling Epidemiological Dynamics at the Population Level

In this quantitative facet of epidemiological dynamics, the parameters of central interest are population-level rates as they affect susceptible individuals and as drug users or others become human vectors in the transport of disease-causing agents from the reservoirs of origin, onward toward person-to-person sharing and the eventual spread within a population. Nonetheless, perhaps of greater interest to readers of this chapter, parameter estimates from these models can also help to simulate and forecast effects of public safety and public health interventions, at least in hypothesis-generation mode if not yet hypothesis-testing mode. The promise of this line of research is considerable. As I show, its current utility is a matter of controversy, primarily because of concerns about the quality of available data.

Defined narrowly, epidemiological dynamics is restricted to the estimation of parameters nested within models of epidemic spread of infections and diseases within populations, either in preclinical experiments (e.g., with rodents or rabbits) or clinical (human) investigations. The origins of these studies date back to the early 20th-century mathematical modeling of infectious disease processes in populations. The concept of epidemiological dynamics is implicit in the law of mass action introduced by Sir Ronald Ross under the heading of the proportional happening and in later clarifications of this law, according to which one might assume “that the rate at which an infection passes in a population is proportional jointly to the product of the number of persons ‘I’ who are infectious and the number of persons ‘S’ who are susceptible to the infection” (Wilson & Worcester, 1945, pp. 24–25). As originally formulated, the law of mass action at the population level proved to be inadequate. Some attempts to remediate did not meet with success (e.g., Brownlee’s idea of decaying infectivity). Other refinements have been found to hold more promise, including the concept of the basic reproduction number as well as the differentiation of deterministic and stochastic models of epidemiological dynamics, as discussed by Daley and Gani (2001) in their monograph Epidemic Modelling: An Introduction. This monograph provides details about general epidemiological dynamics research at the population level for interested readers, including a chapter on modeling the spread of rumors.

Modeling Epidemiological Dynamics at the Individual Level

This focus on epidemiological dynamics as estimated for populations does not imply that early epidemiologists ignored the idea of epidemiological dynamics at play within individuals. Stalleybrass, author of the first modern textbook devoted to epidemiology, published in 1931, gave this description of the basis of epidemiology as a science: “the knowledge of the process of infection of the individual and of the responses to infection of the individual [italics added] and of the herd. So clinical medicine, pathology, bacteriology and immunology all bring grist to the epidemiologist's mill” (Stalleybrass, 1931, as quoted in Lilienfeld, 2003, p. 856).

Commenting on these complementary macro-social (i.e., population-level) and microscopic dynamics.
(i.e., individual-level) perspectives on epidemiological dynamics, Matthews and Haydon (2007) characterized the present situation as follows:

At the most fundamental level, the dynamics of infectious diseases are determined by transmission between infected and susceptible individuals: who infects who, and how often. Ironically, this is a level of organization at which studying epidemiology is hard. Data on who infects who are difficult to come by, and inference is often weak (although genetic studies are changing this). Mathematically, models formulated at the individual level can be difficult to analyse (although modern computer power is changing this as well). Fortunately, this level of organization is sandwiched between others at which progress is more easily made. Classically, epidemiological models have often been formulated at higher levels of organization: that of the population, or more recently the metapopulation. With the development of modern genetic and immunological tools, it is now also possible to examine determinants of variation at levels of organization below that of individual hosts or pathogens. As a result, we know a lot more about the underlying basis for host resistance and pathogen virulence. (p. 763)

Parsing each sentence of the passage for readers interested in this chapter's coverage of epidemiological dynamics, I come to the concepts of drug exposure opportunity and the first chance to try a drug. As noted earlier, the idea of drug exposure opportunity can be traced back to the work of early epidemiologist Wade Hampton Frost in the early 20th century (Van Etten, Neumark, & Anthony, 1999; Wagner & Anthony, 2002b). Frost noted that people's understanding of the prevalence of infections and infectious diseases within the population required differentiation of subgroups or individuals who had the opportunity to be exposed to an infective agent; those without an opportunity to be exposed were effectively not at risk of infection or disease. Similarly, those with no chance to try a drug are effectively not at risk of becoming a drug user or developing drug dependence.

Once the susceptible host's opportunity to be exposed to an infective agent has occurred, then a series of questions might be asked: Did an effective contact of agent and host occur (e.g., as manifest in antibody formation after exposure to a bacterial antigen or after first use of a drug)? Is there a latent period after effective contact, during which there is no spread to others (e.g., before the infected hosts becomes infectious)? Do symptoms of the infection become clinically recognizable concurrent with the period of infectiousness, or does the infected host qualify as an apparently healthy carrier with the capacity to spread the infection to others?

These questions and associated concepts of epidemiological dynamics are linked to concepts such as serial interval for infections that are spread from person to person (i.e., the interval from the time one host has become infected until that same host has spread the infection to the next susceptible host). Similarly, one can ask about the serial interval from the time a new drug user first engages in extramural drug use until the time that user offers and shares, barters, or sells the drug to someone else who has never used the drug. An incubation period is also pertinent in epidemiological dynamics here. There is a toxicological analogue in the induction interval concept that expresses itself in the time from exposure to a drug to the appearance of symptoms of an underlying and possibly pathological drug-caused process. For example, the rapid induction interval for tolerance to LSD and some of the other hallucinogens might actually contribute to a reduced risk of developing the hallucinogen dependence syndrome (e.g., see Anthony, Warner, & Kessler, 1994; Halberstadt & Geyer, 2011; Stone, O'Brien, De La Torre, & Anthony, 2007).

Just as Matthews and Haydon (2007) noted the difficulty of knowing who infects whom in the individual-level study of epidemiological dynamics of communicable diseases, it is hard to learn the identity of the human vector source of a drug exposure opportunity. Young people are often unwilling to disclose the names of those who introduced them, although there is a successful line of anonymous
research on illegal sales and purchases (e.g., Garnier et al., 2010; James, Wagner, & Anthony, 2002; O'Donnell, Voss, Clayton, Slatin, & Room, 1976). Nonetheless, even if study respondents are unwilling to identify their sources of drugs or the recipients of drugs they have sold or given away, in the future alternative approaches should help shed light on this facet of epidemiological dynamics. Just as microscopes are used in ballistics laboratories to identify bullets fired by individuals and laboratory molecular genetics techniques can be used to shed light on chains of transmission of viral infections from person to person and from area to area (e.g., via laboratory identification of individual signatures or clades disclosed via molecular analyses), it might become possible to use advanced microscopic or molecular techniques to trace individual samples from batches of illegal drug commodities as they travel from place to place and from person to person. “Pillistics” laboratories have existed in drug law enforcement since the 1960s and 1970s, but they have not yet been tapped for studies of epidemiological dynamics. These developments and applications in future epidemiological dynamics research will evolve concurrently with advances in computational software providing the speed required for individual-level and multilevel modeling of drug exposure opportunities and the transition from first chance to first use, now largely being carried out using population-averaged methods (e.g., see Reboussin & Anthony, 2001, whose population-averaged approaches suggest prevention or delay of drug exposure opportunities as well as drug use conditional on exposure, among subgroups of young people with higher levels of parental supervision and monitoring).

**Systems Science Approach**

The systems science approach in public health, as applied to extramedical drug use and subsequent outcomes such as high levels of cocaine use, has roots that converge in a small set of articles published in the late 1960s and early 1970s. Although not remarkably influential in its citation impact, an early contribution by Simon Rottenberg (1968) is important because of its clarity of vision and organization of principles. Apropos this section’s overview of drug exposure opportunities and drug use conditional on exposure, and its introduction of the concept of serial interval, Rottenberg (1968) wrote,

The optimal allocation of enforcement resources will be affected by the mechanism by which addiction spreads. Two possible hypotheses are: (1) There are constant “returns” to scale, and (2) there are increasing “returns” to scale. If the first of these characterizes the world, one addict will infect x number of non-addicts (causing them to become addicts), and a cluster of ten addicts will infect 10x non-addicts. If the second is true, then if one addict will infect x non-addicts, a cluster of ten addicts will infect some number of non-addicts which is larger than 10x.

If the second hypothesis is correct, it suggests that critical mass of some magnitude is necessary for infection to occur. The process would be analogous to the spread of fire. If a matchstick burns, the probability of neighborhood fires is less than if a house burns. Or if ten houses burn, each of which is separated from any other burning house by ten miles, the probability of disastrous spread of fire is less than if the ten burning houses were in the same neighborhood. (p. 88)

To understand the systems science approach within this context, readers may wish to contemplate systematic variation of the parameters just described by Rottenberg (1968). For example, a simulation-forecasting model can be specified with 10 newly initiated drug users at time \( t = 0 \), and then 100 newly initiated drug users at time \( t = 1 \) (not counting the initial 10 users, who no longer qualify as newly initiated at \( t = 1 \)). Now let us vary the value of the multiplier to be 9 instead of 10, yielding 90 newly initiated drug users between time \( t = 0 \) and time \( t = 1 \). Now let us vary that multiplier across a range that runs from 0 to 20 to generate a basic systems science formulation of what might be achieved across an interval from time \( t = 0 \) to time \( t = 100 \),
depending on whether the value of the multiplier is 0, 1, 2, ..., 100. Then consider what type of intervention might be required to shift the multiplier value in a downward direction—say, from a value of 10 to a value of 5. Understanding this type of simulation is crucial to an understanding of this type of systems science approach in its basic "deterministic" form. The relationship of this approach to the deterministic epidemic modeling described by Daley and Gani (2001) is straightforward. They described the extension of the deterministic approach to a stochastic approach within the context of communicable disease epidemic modeling, with examples of stochastic simulations of value. See Kondakci and Dincer (2011) for an elegant and quite pertinent stochastic model, originally developed for the study of software "worm" infection epidemics and ripe for translation in an extension of the systems sciences as applied to extramedical drug use and its consequences. Examples with this degree of refinement have not yet been found in the published literature focusing on extramedical drug use or the other behavioral expressions of addiction of interest to readers (e.g., gambling addiction, sex addiction).

Another element of this early formulation has likely been neglected, as conveyed in the following quotation, also from Rottenberg (1968), which sets up this section's appraisal of the importance of systems science in the evaluation of alternative governmental actions and policy instruments:

Suppose non-addicts experiment with drugs for a number of reasons, say: (a) they are told by an addict that it produces pleasurable experiences, or, by observing a user, they can see that it produces these experiences; (b) they admire another whom they know to be a user because of other qualities he possesses (he is an accomplished playwright, or poet, or musician); and (c) they desire to be a member of a community they believe to be congenial, and the consumption of drugs is a necessary condition of entry into this community.

Of this small catalogue of motivations for experimentation, (a) and (b) would be operative whether the observed addict is isolated or one of a cluster of addicts. However, (c) would be operative only when there were clusters. As long as there is one additional incentive associated with a cluster which is absent in the case of the isolated addict, the cluster will be a stronger focus of infection than the individual; and a cluster will have stronger infecting consequences than an equal number of isolated individuals.

This suggests that the quantity of enforcement per addict should be larger where clusters occur than where there are no clusters and addicts are isolated; that clusters should be broken up and their members diffused more thinly in the population and in space; that clusters, where they are not broken, should be isolated; that devices should be employed to cause that segment of the non-addict population which is on the margin of experimentation to believe, given the values they presently hold, that the addict cluster community is not congenial (this may require either the exposition of the truth, or systematic misrepresentation of fact, or some combination of them); and that devices be employed to alter the values of the sector that is on the margin of experimental drug consumption so that the probability diminishes that experiments occur. (p. 89)

Rottenberg was an early contributor, but he was not alone during the 1960s and 1970s in his efforts to create a rational conceptual framework for evaluating alternative public policy investments (e.g., where to focus intervention resources). Readers will benefit from a review of contemporaneous contributions by Erickson (1969), Greenwood (1971), Koch and Grupp (1971), Nisbet and Vakil (1972), Phares (1973), Eatherly (1974), Yeager (1975), Votey and Phillips (1976), and Bernard (1983) as well as more recent work from Jonathan Caulkins and his colleagues (e.g., Caulkins, Feichtinger, Tragler, & Walner, 2010). Some of these contributions were
fairly narrow in scope, as with Greenwood’s attempt to estimate prevalence of drug use. Other contributions were oriented toward selection of policy instruments, as manifest in Eatherly’s attempt to decide whether intervention resources should be focused on drug pushers or on extramedical drug users.

Within this context, I introduce medical and public health approaches to the management of the dynamically changing epidemics of the addiction syndrome. As might be forecast on the basis of what has been covered in prior sections of this chapter, these approaches have been worked out primarily for communicable diseases with an infective agent as the necessary cause. Nonetheless, from the 1960s onward, refinements of these approaches were made on the basis of observations made during and after drug epidemics, with some observations favoring a punitive approach (e.g., see the descriptions of experience in Japan and Sweden from Brill & Hirose, 1969, and from Bejerot, 1975) and other observations favoring a medical and public health approach (e.g., Hughes & Crawford, 1972; Hughes et al., 1972; Lindesmith, 1973). Regrettably, definitive evidence about the value of these alternative approaches remains just beyond researchers’ grasp (e.g., see Manski et al., 2001).

Peer Contagion in Relation to This Facet of Epidemiological Dynamics

In relation to this facet of epidemiological dynamics, the topic of peer contagion merits an introduction. Previously in this chapter, I alluded to the possibility that parents might be influential in the spread of drug use via their supervision and monitoring of children. This monitoring behavior of parents can function as an individual-level shield that might protect young people against drug exposure opportunities and the transition to drug use, conditional on the first chance to try, which is called behavioral autarcesis (Chen et al., 2004). The literature on influential sources of variation in youthful drug involvement is focused on peer-to-peer influence. Often, oversimplified multiple regression models have been used to estimate the relative influence of peers and parents on the occurrence of youthful drug use, generally with a result that peer influence is found to be stronger than parental influence. This approach neglects what can be observed when the models allow parents’ supervision and monitoring of their children to influence the selection of peers with whom the children affiliate (e.g., see Lloyd & Anthony, 2003). Be that as it may, a very large body of evidence has supported the importance of peer influence in the context of behavior problems generally and extramedical drug use specifically, with the earliest studies conducted more than 50 years ago (e.g., see Brook et al., 2008; Chein, Gerard, Lee, & Rosenfeld, 1964; Dodge, Dishion, & Lansford, 2006; O’Donnell et al., 1976; Thornberry & Krohn, 1997).

Within this body of evidence, there is some discussion of a peer contagion effect, including an expression of concern that the epidemiological concept of contagion necessarily requires focus on quite fine-grained, distinctive, specific facets of experience to be useful in research on the epidemiological dynamics of extramedical drug use and related problem behaviors (Anthony, 2006). Nonetheless, the published peer contagion literature includes two exceptionally useful studies that merit attention in relation to epidemiological dynamics research on extramedical drug use and its consequences.

The first of these two studies (Dishion & Andrews, 1995) originated in the Adolescent Transitions experiment to compare and contrast the effects of interventions intended to reduce youthful drug taking, among other problem behaviors. One of the interventions involved structural aggregation of high-risk youths into groups, which made it efficient to deliver the peer focus intervention intended to improve these behaviors. Random assignment of high-risk youths to this peer-focus condition was followed by greater increases in tobacco smoking and teacher-reported delinquent behavior, compared with expected values from the contrast condition of youths who had received minimal intervention and no structural aggregation into groups of high-risk youths (Dishion & Andrews, 1995). This unintended adverse consequence of the peer-focus intervention has prompted expressions of concern about the potentially harmful and iatrogenic effects of interventions that include structural aggregation of troubled young people and discovery of other related effects in peer-group intervention programs with structural aggregations of the type.
applied in the peer-focus condition of the randomized trial (e.g., see Dishion, McCord, & Poulin, 1999; Dodge et al., 2006).

The second of the two studies pertinent to peer contagion involved random assignment of matriculating freshman to roommates as they started the 1st year of postsecondary school. Duncan et al. (2005) described randomly assigning 279 men and 435 women to their college dormitory roommates after prematriculation surveys had assessed heavy episodic (binge) drinking (HED) and other characteristics of their high school and prior history. Estimating the effects of high school HED on levels of drinking during the postsecondary years (after random assignment), Duncan et al. observed a multiplier effect such that men with HED in high school had much higher drinking levels during their university years if randomization had assigned them a roommate who had also experienced HED during high school than if randomization had assigned them a roommate who had not. Moreover, when a random pairing of a roommate with an HED history and a roommate without an HED history occurred, there was no apparent adverse effect on either roommate.

Engaging in a thorough examination and discussion of these apparent examples of a peer contagion effect for extramedical drug use is beyond the scope of this chapter. Interested readers are referred to the original papers and to the monograph that was produced after several years of examining the evidence and pondering its implications for the future (Dodge et al., 2006). For the present purposes, these experimental results have a clear implication for and importance to an understanding of the epidemiological dynamics of extramedical drug use and the sequelae of such use. I am skeptical that the evidence indicates a peer contagion effect and have offered other explanations (Anthony, 2006). Nonetheless, the evidence is important as epidemiologists seek to design future research to investigate this facet of the epidemiological dynamics of extramedical drug use and its spread through human populations.

**Upstream Interventions, Downstream Effects**

Finally, borrowing the upstream and downstream concepts introduced earlier and returning to the primary focus of public health epidemiology research, I report on some recent findings from an evaluation of an upstream population-level social marketing campaign that was intended to alter the epidemiological dynamics of extramedical drug use at both the population and the individual levels (i.e., via effects on downstream attributes of the individual). Specifically, Scheier and Grenard (2010) described the implementation and research approach used to evaluate the National Antidrug Media Campaign, which attempted to saturate television and radio with antidrug public service announcements. Implemented at a national level, the campaign is now being evaluated at the individual level in terms of growth trajectory models. Early evaluations of the campaign suggested no major effects on subgroup incidence rates or individual-level probabilities of initiating drug use, although it must be said that the design did not include no-exposure control groups; the only control groups were low-level-awareness groups, whose drug experience was contrasted with that of those in high-level-awareness groups (i.e., where awareness refers to recollection and awareness of the campaign over a 4-year span of time). Consistent with previously reported evaluation results (Hornik & Jacobsohn, 2008), these longer term results indicated that higher levels of campaign awareness were associated with quicker acquisition of drug use behaviors and acceleration of these behaviors (i.e., the opposite effect from what was intended). Later during the 4-year evaluation interval, an increasing awareness of the campaign was associated with deceleration of heavy episodic drinking and tobacco smoking, but no intended or beneficial effects were found for the individual-level trajectories of cannabis smoking (Scheier & Grenard, 2010). In future research, it will be interesting to investigate the possible heterogeneity of these effect estimates with respect to subgrouping defined by drug exposure opportunity; completely different conclusions might be drawn if the analyses focused on individuals for whom cannabis exposure opportunities had occurred. That is, the antidrug campaign's effect on the probability of transitioning to cannabis use once the chance to try cannabis has occurred has not yet been evaluated and might show more sensitivity to the public
service announcement messages than the unconditional occurrence of cannabis smoking, irrespective of whether the young people even had the chance to try cannabis.

In a review by Wakefield, Loken, and Hornik (2010) as well as a prior contribution by Pechmann and Reibling (2000), I should note that, with respect to legal drugs, the evidence tends to support very tangible effects of antismoking campaigns on the prevalence of tobacco smoking in the United States and Canada, and possibly stronger effects on the initiation of smoking (vs. duration of smoking). There is no clear explanation for the discrepancy of results when the outcomes of interest involve legal drugs versus the generally illegal and internationally regulated drugs. However, the legal products are more widely available than the other drugs, which public health workers would expect to translate into greater drug exposure opportunities for tobacco than for cannabis and the less commonly used drugs regulated under CSA. So the more prevalent patterns of drug use might invite more “anti” campaign impact than drug use patterns that are less common.

Reflecting on a unitary syndrome model, there are some unanswered questions about epidemiological dynamics, some of which might prove quite difficult to answer, unless one pays attention to the specific behaviors yoked to the syndrome concept. For example, consider the epidemic spread or contagion of unbidden behaviors, a category within which one might wish to include an array of items. In a unitary model of these unbidden behaviors, one might wish to include yawning, explosive sneezing, and an abrupt facial grimace. With the idea of contagion in the background as well as the unitary model of unbidden behaviors, imagine a panel of scientists sitting on stage at the front of a large auditorium, all facing the audience during a series of symposium talks. Next, imagine that one panelist closest to the lectern emits an unbidden yawn. Moments later, another panelist, sitting next to the first, sneezes explosively. Then, a third panelist, sitting next in the sequence, grimaces (perhaps because he or she has just experienced a “charley horse” leg spasm), and so on down the line of panelists sitting on the stage, demonstrating one unbidden behavior after another during a colleague’s talk. Under the unitary model, it would be difficult to avoid the thought of behavioral contagion and an interpretation that each emitted behavior in the sequence might be an after-effect of the prior effect: unbidden yawn followed by unbidden sneeze followed by unbidden grimace, and so on. Nonetheless, the investigation of contagion in the mechanism of spread of behaviors does not generally admit this degree of nonspecificity, and most contagion researchers would be skeptical about the idea that a yawn by one panelist would cause an explosive sneeze in the next panelist and a grimace in a third panelist (e.g., see Anthony, 2006).

Contrast the credibility of the evidence if one was to substitute a sequence of yawns in lieu of the just-described sequence. That is, the first panelist yawns during the talk, the one next to him yawns, the next one then yawns, and so on down the line. Here, the degree of specificity in the unbidden behavior (i.e., a sequence of visible yawns, one after the other) makes the idea of behavioral contagion much more credible. It would seem that a unitary model of unbidden behaviors might not serve well in this particular context of research on epidemiological dynamics, even though it might serve well in other contexts. Under this second facet of epidemiological dynamics, one might expect to encounter similar difficulties during the application of the unitary syndrome model for the addictions (i.e., in research on the familial aggregation of the addictions or in their person-to-person spread).

**Epidemiological Dynamics: The Third Facet**

The third facet of epidemiological dynamics involves modeling the persistence and rates of drug taking by affected individuals. Here, persistence refers to whether extramedical drug use occurs just once, twice, or a few times and then stops, which seems to be a modal pattern for self-administration of internationally regulated drugs. Alternately, once such drug use starts, its persistence can be manifest in a measurable rate of drug use. Two published articles and one article now under review (Anthony, 2010; Barondess, Meyer, Boinapally, Fairman, & Anthony, 2010; Barondess, Troost, Fairman, & Anthony, 2010) have described aspects of this facet of
epidemiological dynamics. What follows is an elaboration on what has been presented in those articles.

At the outset, knowledgeable readers will appreciate that one can estimate the rates of drug use for subgroups of the population; one can also estimate the variation among the rates of drug use for individuals in a population. In keeping with the themes of prior sections of this chapter, the following discussion is focused on the estimation of rates for population subgroups.

Nevertheless, whether individuals or subgroups are being studied, one of the most convenient rates to estimate involves an assessment of the number of days of drug use during the 30 days before the date of a standardized field survey assessment. For an individual, this monthly rate is formed as a ratio of the number of days of use divided by 30 person-days. For the subgroup, the estimated rate involves aggregation of the individual-level rates.

One of the difficult conceptual features in the formulation of this type of rate is that in any given month a persistent user might be completely abstinent. For that persistent user, the rate is zero days per 30 person-days. If these zero-day persistent users are ignored in the estimation of a population subgroup's estimated rate of drug use, conditional on persistence, the resulting calculation has an upward bias because the zero counts are eliminated from the count distribution for that month. Unless the explicit goal is to estimate the rate on the basis of a count distribution with a truncated zero, proper estimation of the rate of drug use, conditional on persistence, makes an allowance for the fact that some persistent users will pass through a month of abstinence from time to time.

In this section, I describe an approach that can be used to estimate the monthly rate of drug use in a fashion that accommodates the possibility of subgroups of persistent users who might abstain during a given month. Of course, if the goal is to estimate the quantity of drugs consumed by a subgroup in a given month, as in some of the systems science research on drug taking, then the persistent users with zero days of use are irrelevant, but following is an estimation problem with a goal that is quite different from the estimation of the mean monthly rate of drug use for various population subgroups.

Cross-Sectional Survey Solution to Estimation of Monthly Rates of Drug Use

Under ideal conditions, and with the kind of thinking that is encouraged in the introductory textbooks of the field, one might be inclined to presume that prospectively gathered longitudinal study data are required to produce monthly rate estimates of drug taking. This kind of thinking is faulty for several reasons. For example, planning for such a longitudinal study without some degree of certainty in a forecast of the size of the resulting estimates would be foolish. With no way to forecast the size of estimates to be observed in the longitudinal context, a research team is hamstrung, because this type of forecast is a crucial element in sample size calculations. This situation might seem paradoxical at first, but the size of the estimate to be observed in a longitudinal study must be forecast with some degree of accuracy before the sample size required for a longitudinal study can be specified. For this reason, it makes sense to approximate the size of these estimates before undertaking any longitudinal research on these topics. The approximation can be accomplished via purely theoretical simulations in relation to institutional knowledge about plausible size estimates, but the institutional knowledge might be wrong. If the simulated estimates are too small, then the longitudinal research will be underpowered and will yield too little information to produce a statistically reliable result—a waste of precious research dollars. If the simulated estimates are too large, then the longitudinal sample size will be larger than it should have been under more optimal conditions—again, a waste of precious research dollars allocated to recruit participants and gather data longitudinally, dollars that might be used in other projects.

One way to challenge the institutional knowledge and the theoretical simulation approach involves an initial completion of a cross-sectional survey, with a measurement plan and analyses carefully designed to forecast the estimates that might be obtained if a longitudinal study were to be completed. When these analyses can be completed in advance of the longitudinal research project, then the empirically based estimates from cross-sectional research can be used to help constrain errors that might otherwise distort sample size considerations.
for subsequent longitudinal research. Indeed, what would be superior to any single cross-sectional estimate is a family of estimates from a set of independent cross-sectional samples, as might have been completed in the years before the longitudinal research.

Extending the argument that it makes sense to derive one or more cross-sectional estimates in advance of any longitudinal research project as an aid to forecast the longitudinal study's sample size, in this specific instance, it is the cross-sectional survey and the careful analysis of the cross-sectional survey data that can drive the field toward completion of the longitudinal study. This interesting example of a cross-sectional study as field-driving research can lead to a reappraisal of the limitations that constrain inferences from longitudinal studies but do not constrain inferences from cross-sectional studies. This is the problem of sample attrition, over time, in virtually all longitudinal research projects that examine nonfatal outcomes. This kind of sample attrition can be a major limitation in longitudinal research generally, and in longitudinal research on drug involvement, drug dependence, and related outcomes specifically—for example, because of the substantial likelihood that serious drug users are more likely to be lost from the sample during the follow-up interval than are other drug users. It is noteworthy that this limitation does not plague the cross-sectional study sample often used to start-kick a longitudinal research project (i.e., the baseline sample). This problem also does not plague the cross-sectional survey that is not designed to be longitudinal. Indeed, analyzed from the total survey error perspective, both types of samples, at cross-section, must face potential limitations similar in quality and quantity, for the most part.

To illustrate, when these studies are guided by epidemiological expertise, a predefined source population must be recruited and sampled. In most surveys, the concept of the source population is manifest in a sampling frame, and one source of potential error involves imperfections in the sampling frame with respect to the source population. In the context of a typical design of a multistage area probability sample community survey with a longitudinal elaboration, there is sampling of dwelling units (DUs). Whereas there might be no sampling frame errors up to the DU, it is within the DU that frame error can be introduced in a fashion that yields estimate bias compared with what might have been found if there had been sampling from a complete list of all members of the source population (e.g., in Denmark, where there is a registry of all citizens). Namely, when the survey team visits the DU and asks the head of household for a roster of the DU inhabitants, the father or mother might list a young adult daughter, still living at home, but might not mention the daughter's live-in boyfriend, who spends some but not necessarily all of his nights in their home. Should the survey team also sample and seek a roster of the household of the boyfriend's family of origin, the boy's mother or father might think, "He spends most of his nights over at his girlfriend's house, and he really doesn't live here any more." Hence, this young man is a member of the source population, in theory, but this type of sampling frame error leads to his omission from the DU rosters from which designated survey respondents are sampled. Anyone familiar with the epidemiology of drug use and dependence will appreciate that young never-married men are at higher risk of becoming a case than are other population subgroups. The implication is that this type of error in the sampling frame is present in this type of cross-sectional sampling approach, whether the sampling is for the baseline of a longitudinal study or for a stand-alone cross-sectional survey with no plan for longitudinal follow-up (Chen & Anthony, 2004).

Possible Superiority of Cross-Sectional Estimates Compared With Longitudinal Study Estimates

Having established that study estimate and inference limitations can be equally present in baseline samples of longitudinal follow-up studies and in stand-alone cross-sectional surveys without longitudinal follow-up, it should be noted that in the modern era, the stand-alone cross-sectional survey sample might actually be stronger than the baseline sample with respect to errors involving nonparticipation is appropriate. Formerly, recruiting participants for the cross-sectional baseline sample of a longitudinal research project was possible without disclosing the
fact that a request for follow-up participation might be forthcoming. In the modern era, committees for the protection of human subjects typically require full disclosure at the time of baseline sampling and recruitment. That is, in the baseline survey's disclosure and informed consent statement, the research team is usually obliged to disclose its intent for sustained follow-up and its need to secure personal identifiers and tracing information. When this is the case, participation in the baseline survey sample can suffer because some participants who would be willing to participate in an initial anonymous cross-sectional assessment might be unwilling to consent when follow-up participation and associated needs for personal identification and tracing information are stipulated. Under these circumstances, the baseline sample has limitations that the stand-alone cross-sectional survey sample does not possess.

One is left with a strong rationale and justification for harnessing the more readily available stand-alone cross-sectional sample survey data to produce estimates needed to design longitudinal research projects and, in the process, drive the field forward, perhaps with the cross-sectionally derived estimates being stronger than those observed in the baseline or follow-up data of longitudinal projects. By way of illustrating some substantive examples of this type of research, I can start with a thought experiment of a very large cross-sectional sample of newly incident drinkers of alcoholic beverages—that is, drinkers assessed within 3 months of starting to drink. Figure 4.1 is a generalized adaptation of a drug dependence count process figure in Anthony (2010); this figure illustrates the onset of human drug self-administration, followed by a series of repetitions of drug-using behavior during an interval before emergence of any of the now-measurable features of the addiction or dependence process. Here, I have recast Figure 4.1 as a more generalized depiction of the syndrome coalescence process, with a feedback loop as clinical features of the syndrome emerge (e.g., obsession-like craving) and recursively begin to feed back and drive up the count of repetitive experiences. As shown, the syndrome may or may not coalesce, and the secondary complications of the syndrome (e.g., socially maladaptive behavior, premature mortality) may or may not occur.

Possible Reciprocities Linking Drug Dependence and Persistence to Rates of Drug Use
Here, in light of what is known about the generally insidious time course from the onset of drinking to the onset of alcohol dependence syndromes, one might expect very few of the newly incident drinkers in the earlier thought experiment to develop serious clinical features of alcohol dependence by any date of assessment occurring within the first 3 months after onset of drinking (see Wagner & Anthony, 2002a). Nonetheless, even in this very early stage of drinking, it should be possible to make some predictions about population subgroups who might drink once or twice and then never drink again, versus subgroups for whom drinking is persistent and for whom there is a tangible excess monthly rate of drinking, as measured by the number
of drinking days during the 30 days before assessment. In addition, for at least some of the individuals who persist, a tangible expression of a liability to become alcohol dependent quite quickly might exist, perhaps observable in the form of one, two, three, or possibly more of the individual clinical features of dependence, even when no fully fledged clinical syndrome of alcohol dependence has formed. If it is correct that few or no cases of alcohol dependence have developed within 3 months after the onset of drinking, claiming that the persistence or rate of drinking during that interval has been caused by alcohol dependence would be difficult, but it would be logical and feasible to estimate the degree to which the number or level of individual clinical features might be determined by the persistence and rate of drinking (e.g., see Anthony, 2010).

Figure 4.1 illustrates the possibility of emergence of clinical features and eventual formation of a syndrome, as might occur over a span of months or years after the onset of drinking.

Conceptual Model for Relations Linking Drinking Persistence and Rates to Dependence Levels

Figure 4.2 depicts a cartoonlike amendment of the conceptual model presented in the earlier sections of this chapter. The circle labeled with the Roman numerals I and II depicts a sorting of the newly incident drinkers into one of two latent classes, with Class II being the newly incident drinkers for whom it makes sense to estimate a monthly rate of drinking and with Class I being the newly incident drinkers who might have tried alcohol once, twice, or a few times, then stopped, and for whom the expectation is no future drinking whatsoever. Here, the concept of expectation is important because there is no deterministically certain assignment of newly incident drinkers to these two classes. Instead, there is an expectation—but not a certainty—that members of Class II will persist in drinking, at least for a time, even when there might have been zero drinking days in the 30 days before assessment. It is an expectation—but not a certainty—that members of Class I will never again drink. Conditional on this persistence outcome, it is possible to estimate the mean rate of drinking, with an inclusion of zero values as might be observed for members of this class for whom there is persistence of drinking even when the 30 days before assessment have not included drinking. I mentioned this possibility of zero drinking days for persistent drinkers in the introduction to this section. To illustrate, for ordinarlly persistent drinkers, the study assessment might occur midway through Lent or during some other interval such as a high school or collegiate sports season when the newly incident persistent drinker has abstained for a time for reasons of religion or the alcohol control policy governing sports eligibility during the season.

The solid-line rectangle in Figure 4.2 stands for the rate of drinking, conditional on the persistence outcome, as manifest by the number of drinking days during the 30 days before the date of survey assessment. It is allowed to be correlated with the just-described class assignments.

The second circle in Figure 4.2 stands for the possibility that there might be a tangible expression of a liability to become alcohol dependent observed quite quickly after onset of drinking, perhaps observable by the number of individual clinical features of dependence that have been experienced within the first 3 months of newly incident drinking.

FIGURE 4.2. The syndrome coalescence process as applied to drinking of alcoholic beverages. The formation of a count process may be a novel phenotype manifestation of underlying genetic susceptibility traits of the East Asian population subgroup, with the coalescence of an alcohol dependence syndrome in a feedback loop with the count process once drinking has started. Copyright 2011 by James C. Anthony. Reprinted with permission.
(i.e., since drinking onset). Nonetheless, because of the possibility of false-positive reporting of those clinical features (e.g., misunderstanding what is meant by feeling tolerant), the circle is labeled "Coalescence of Dependence?" Here, the question mark reflects uncertainty about what is being measured. In practice, scientists can measure the variable under study by asking standard survey items about the individual clinical features of alcohol dependence and either counting them up or fitting a latent trait model as depicted, with each item standing as a manifestation of the underlying concept. In relation to the theory, this concept might be the liability just described, or it might be the level of dependence observed soon after drinking onset, or it might be best positioned as a count of early clinical features.

Conceptualized in any of these ways, estimating the degree to which this dependence variable might be determined by persistence class membership, conditional drinking rate, or both will be of interest. Alternately, it might be of interest to estimate the degree to which these three variables are intercorrelated; this estimate, to satisfy readers who might think that a liability or level variable, as manifest in these survey items on alcohol dependence soon after drinking onset, is already potent enough to influence drinking persistence and rates. Nonetheless, the theory is that in the first 3 months after drinking onset, the model makes most sense as depicted with a regression of the dependence variable on the persistence class and conditional rate variables.

On the left side of Figure 4.2 is a square that stands for whether the newly incident drinker is of East Asian heritage. This population subgroup has interesting characteristics in relation to the occurrence of alcohol dependence because, compared with other population subgroups, the null variants of alleles that govern the metabolism of acetate and acetaldehydes formed after ethanol self-administration are overabundant. In theory, the null allelic variants confer genetic susceptibility traits for a flushing reaction after drinking, sometimes experienced as an aversive effect of drinking, with redness, itching, and even palpitations and other untoward responses (e.g., see Anthony, 2010; Barondess, Troost, et al., 2010). The value of this variable can be thought to be equal to 1 if the newly incident drinker is of East Asian heritage (otherwise, it is equal to 0), but for a variety of reasons the contrast typically is set up so as to compare the subgroup of East Asian heritage with the subgroup of non-Hispanic White heritage because the nub of the theory is that even within the first months after the onset of newly incident drinking, there might be manifestations of genetic protection with respect to the ultimate risk of alcohol dependence—namely, as manifest among the East Asian subgroup of newly incident drinkers being overrepresented in the never-again-drinker Class I and underrepresented in the subsequently-persistent-drinker Class II, and as manifest in the East Asian newly incident drinkers having a tangibly lower rate of drinking, conditional on the drinking outcome. The model conveys the hypothesized possibility of a direct influence on the dependence outcome as well as an indirect influence via an indirect effect on class membership with respect to never-again-drinkers (Class I) versus subsequently-persistent-drinkers (Class II).

In steps toward fitting and testing the model depicted in Figure 4.2, the goal of estimating all of these relationships within the first 3 months after onset of drinking has not yet been achieved. Nonetheless, an attempt was made to estimate the representation of the East Asian subgroup of newly incident drinkers within the two classes depicted in Figure 4.2 and to estimate relative drinking rates, with newly incident drinking respecified to encompass drinking onsets within 24 months after onset of drinking. In this research, because as many as 24 months have passed from the month of onset of drinking to the month of survey assessment in the cross-sectional sample, the likelihood of reciprocities linking persistence and conditional drinking rates with level of alcohol dependence is greater. For this reason, the research to date has dropped the dependence construct from the model. The resulting model estimates apply to the one enclosed within the dashed-line rectangle in Figure 4.2.

Empirical Illustration of the Value of the Drinking Persistence and Rate Model

I close this illustration with a presentation of estimates from a cross-sectional survey sample and indicate more clearly how researchers can use these
estimates to drive forward a line of longitudinal studies that investigate this model. For example, Barondess, Meyer, et al. (2010) initially applied the Figure 4.2 model (within the dashed-line rectangle) to newly incident tobacco smoking, identified 8,816 newly incident tobacco smokers found in nationally representative sample surveys as observed and assessed within 24 months after onset of smoking, and fit the data using a zero-inflated Poisson regression model. The research team found some relationships of interest, such as overrepresentation of 18- to 25-year-old newly incident smokers in the class of persistent smokers compared with younger adolescent-onset smokers—perhaps reflecting the legality of tobacco cigarette purchase and smoking for the 18- to 25-year-olds and not for newly incident smokers younger than age 17. Consistent with this interpretation, the 18- to 25-year-old newly incident smokers assigned to the persistent smoker class also had a higher estimated monthly rate of smoking than their younger counterparts in the population (all ps < .05).

After working through the details of this modeling process in exploratory-developmental research targeting tobacco cigarette smoking, this research group turned to a test of the specific hypothesis that East Asian newly incident drinkers might be underrepresented in the subsequently-persistent-drinker class and might have a lower monthly drinking rate conditional on the persistence outcome. As it happens, to protect the identities of survey participants, the national survey public use dataset did not release the measured variable to allow East Asian heritage assignment. Nonetheless, the general Asian category was thought to be useful for the purpose of analysis, with an appreciation that the heterogeneity of allelic variants within this larger Asian subgroup might yield an underestimation of the persistence parameter as well the conditional monthly drinking rate estimate. Again, the zero-inflated Poisson model was used to model the two equations, using data from 20,996 newly incident drinkers of alcoholic beverages from nationally representative samples, with one equation for membership in the never-again-drinking class versus the subsequently-persistent-drinking class, and the other equation for estimation of the monthly drinking rate, conditional on the persistence outcome. As predicted, estimates for drinking persistence and the conditional drinking rate were consistent with greater protection of members of the Asian subgroup during the 24-month interval after onset of drinking, well before clinical features of alcohol dependence had developed in most members of this newly incident drinking sample (Barondess, Troost, et al., 2010).

**Future Extensions of This Line of Research**

Efforts to extend this line of research to other psychoactive drug compounds are underway. Analyses have already been completed for cocaine use and for cannabis smoking; the next steps include coverage of other drug compounds such as the prescription analgesic medicines. Hypotheses about excess persistence and higher rates of cocaine use have been framed in relation to crack smoking and cocaine use by injection, which might be expected to accelerate drug involvement relative to the nasal insufflation of cocaine hydrochloride powder. In cannabis and analgesics drug research, the primary hypotheses involve male–female differences and variations in relation to early age of onset as well as history of prior heavy episodic drinking.

Ahmedani and Anthony have proposed an extension of this line of research into the domain of drug treatment services research (the proposal is currently under review). Here, the effort is to study newly incident drug users who are quite rapidly enrolled in drug treatment services or who receive counseling from primary care practitioners, social workers, or others within 24 months after onset of their initial extramedical drug use. Ultimately, the goal of this line of research is the improvement of early outreach and intervention services to prevent, delay, or reduce the duration of drug dependence syndromes that emerge within the first few years after onset of extramedical drug use.

Returning to my reflections on the unitary syndrome model, it is possible to envision the use of these epidemiological dynamics models in relation to other behaviors that eventually take shape in the form of a dependence or addiction syndrome process. Slutske, Jackson, and Sher (2003) have characterized longitudinal trajectories of gambling behavior of college students along these lines, but
the problem of zero inflation has not been addressed (e.g., via use of the zero-inflated negative binomial or zero-inflated Poisson models). I have been unable to find attention to the zero-inflation problem or to zero-inflated binomial or zero-inflated Poisson regression modeling of any of the behaviors typically associated with the syndrome model of addiction. Whether the zero-inflated negative binomial or zero-inflated Poisson regression models can be converted to multivariate models that would accommodate the multiple behaviors and behavioral disturbances encompassed by the unitary syndrome model is an area of active research in new statistical methods.

EPIDEMIOLOGICAL DYNAMICS: THE FOURTH FACET

The final of four facets of epidemiological dynamics might involve the most difficult and challenging topic under investigation—namely, the estimated probability of transitioning from use to a dependence syndrome. This facet's complexity includes some general technical difficulties faced in any contemporary examination of population health experience. Here, when the goal is to estimate the transition from drug use to drug dependence, additional complexities include secular changes in diagnostic approaches and diagnostic criteria for drug dependence, variations in diagnostic criteria across drug compounds, and experience-determined skipouts in the assessment of drug dependence as well as gated assessment protocols.

Before presenting evidence about this facet of epidemiological dynamics, including estimates of the conditional probability of becoming a case of drug dependence once drug use starts, I review several of these complexities. The first issue relates to total survey error, a topic discussed earlier in the Cross-Sectional Survey Solution to Estimation of Monthly Rates of Drug Use section.

Total Survey Error as a Complexity in Relation to the Likelihood of Transitioning From Drug Use to Dependence

From time to time throughout the relatively short history of epidemiological research on extramedical drug use (i.e., since population census surveys in the late 19th century), there have been registries of officially recognized drug users and of officially recognized cases of drug dependence and related problems, including registries in the United States. For example, McNamara and Starr (1973) asserted that most U.S. states have kept registries of people officially recognized as addicted to narcotics, despite concerns about patient confidentiality and protection of their rights to privacy. In Denmark and elsewhere, there continue to be central registries for the total citizen population of the jurisdiction as well as registration of treatment histories, including treatment for drug-related problems, and deaths resulting from drug-related causes, which can be put to work in especially informative epidemiological research on suspected effects of drug use (e.g., see Sjøgren, Grønbæk, Peuckmann, & Ekholm, 2010).

Estimating the probability of transitioning from extramedical drug use to drug dependence-associated outcomes would seem to be relatively straightforward where clinical registries exist. Nonetheless, the complexity in this context is that official statistics and registration can be expected to have no more than partial coverage of the entire distribution of drug experience and that coverage might involve an overrepresentation of the most severely affected and perhaps the most socially maladaptive cases of drug involvement. This argument rests on an appreciation that many drug users come to be officially recognized because of public intoxication, socially maladaptive behavior, or extraneous law violations that might be related to drug taking but that are not necessarily the result of drug taking per se (e.g., see Golub, Johnson, & Dunlap, 2007).

At first blush, general community surveys of drug taking might seem to overcome these deficiencies. Nevertheless, as noted earlier, analyzed from the perspective of total survey error, there are reasons to believe that estimates of this transition probability on the basis of population surveys might also be subject to biases. Sampling frame error has been introduced as a potential source of bias. Information or misclassification biases resulting from shortfalls in memory or accuracy and completeness of reporting about drug experiences qualify as additional sources of potential error.
Secular Changes in Diagnostic Approaches and Diagnostic Criteria

When one considers secular changes in diagnostic approaches and diagnostic criteria, additional complexity is introduced. As evaluated at any single point in time, these changes might be ignored; however, the estimates of transition probabilities from drug use to drug dependence are what they are, subject to the diagnostic approach and criteria in place at that point in time. Nonetheless, if the goal is to develop more generalizable estimates that might hold from time to time, as is required for any study of epidemiological dynamics, the secular shifts in diagnostic approaches and criteria introduce insurmountable difficulties.

One illustration of this difficulty involves a comparison of estimates for a rapid transition from onset of extramedical use of cocaine to onset of a cocaine dependence syndrome, initially derived from the U.S. National Comorbidity Survey (NCS) from 1990 to 1992 (Anthony et al., 1994) and subsequently derived from the NSDUH from 1995 to 1998 and 2000 to 2001 (O’Brien & Anthony, 2005; Reboussin & Anthony, 2006). When comparing estimates from these two epidemiological studies, conducted almost 10 years apart, the reassuring feature is that roughly 5% to 6% of newly incident cocaine users were found to have developed a DSM-IV-TR-type syndrome of cocaine dependence within 24 months after onset of cocaine use. Notwithstanding this similarity of estimates, and the sobering information that one in 16 to one in 20 of U.S. cocaine users quite rapidly progress to fully fledged cocaine dependence, it is noteworthy that the NCS in 1990 to 1992 required face-to-face personal interview methods, with the trained field interviewers reading out the pre-coded standardized survey questions and the respondents giving oral responses that were transcribed by the interviewers. In contrast, by the early 2000s the NSDUH had shifted over entirely to computer-assisted self-interviewing with an audio interface (ACASI) so that the respondent used laptop computer headphones to listen to the survey question and a keyboard to log an answer without aid of the field staff intermediary—an apparent improvement in survey methods, with the new ACASI approach designed to promote accuracy and completeness of survey responding about sensitive topics. Therefore, despite the similarity in survey estimates, one is left with uncertainty about the possibility that the risk of making a rapid transition from onset of cocaine use to onset of cocaine dependence has remained the same. If the shift in NSDUH survey methods was accompanied by increases in the accuracy and completeness of responding, the transition probability of interest across the time span from 1990 to 1992 to 2001 to 2002 might actually have been a decline.

A similar complexity has surfaced in relation to the issue of rapid transition from onset of cannabis use to cannabis dependence (i.e., within a span of 24 months after onset of cannabis use). As estimated in the NCS data from 1990 to 1992, roughly 3% to 4% of cannabis users made this transition within the first 24 months after onset of cannabis smoking (Wagner & Anthony, 2002a). On the basis of the NSDUH estimates from 2000 to 2001, here again using the ACASI approach rather than the NCS interviewer approach, roughly 3% to 4% of newly incident cannabis smokers had developed cannabis dependence by the date of assessment. The similarity of these estimates, despite the shift from interviewer-mediated to computer-assisted self-interviews, makes it difficult to claim that an increasing Δ-9-tetrahydrocannabinol content of cannabis over that period of time has resulted in greater probability of cannabis dependence, notwithstanding claims that cannabis dependence prevalence has increased over that same interval of time (Compton, Grant, Koliver, Glantz, & Stinson, 2004).

Secular change in diagnostic criteria might be a more pernicious source of difficulty or complexity in research on epidemiological dynamics of extramedical drug use and its consequences, such as the drug dependence syndromes. Initial complexity was introduced when the World Health Organization expert panels observed that the form or clinical features of drug addiction or dependence might vary from one type of drug compound to another. This observation prompted their recommendation in favor of a typology—for example, with drug dependence of the morphine type distinguished from drug dependence involving other types of drug compounds.
This type of approach is anathema to the epidemiological researcher interested in developing a comparative epidemiology within and for each type of drug compound. A comparative epidemiological approach to research on a syndrome necessarily requires a stable case definition or set of diagnostic criteria irrespective of the agent under study. That is, if one is to make the most sensible comparison of epidemiological estimates for the risk of transitioning from initial drug use to onset of drug dependence within 24 months after onset of use, drug by drug, then it is important to set up the same type of case definition or diagnostic criteria for each drug—even if it means forcing the clinical features of drug dependence to rest on a Procrustean bed.

The specification of differing diagnostic criteria for drug dependence made it virtually impossible to make this type of comparison across drugs in the original Epidemiologic Catchment Area survey data from the early 1980s (Anthony & Helzer, 1991). The revision of the third edition of the DSM (DSM–III–R; American Psychiatric Association, 1987) fortunately made it possible to create a partial alignment (except for tobacco) so as to bring the diagnostic criteria into rough conformity (e.g., as in Anthony et al., 1994). The DSM–IV (American Psychiatric Association, 1994) also retained the same general configuration, making it possible to develop a comparative epidemiology of dependence conditional on onset of drug use on the basis of the NSDUH and other surveys completed since its publication in 1994 (e.g., Stone et al., 2007).

Whether congruent diagnostic criteria will be specified for the various drug types in the DSM–5 is unknown at present. In the meantime, my recommendation is to specify a set of core diagnostic criteria that will apply to all forms of the addiction syndrome model, without respect to their specific neuropsychopharmacological characteristics, so as to permit comparative research untrammeled by complexities associated with drug-by-drug or behavior-by-behavior variations in diagnostic criteria. This set of core diagnostic criteria might have a complement in a set of drug-specific diagnostic criteria, such as might be required in clinical practice and the care and management of drug-dependent patients. Shaffer (in the Introduction to this handbook) has offered specifications along these lines.

Implementing this recommendation might also solve another problem that currently plagues epidemiological research focusing on the causes of drug dependence. Namely, for the past 30-plus years, the diagnostic criteria for drug dependence have referenced maladaptive behavior such as getting into trouble with family, friends, or the law among the criteria specified for drug dependence, with the possible exception of tobacco–nicotine dependence. Consequently, the DSM approach has created a built-in bias that ensures statistical associations that link drug dependence with neuropsychiatric and behavioral disturbances in which socially maladaptive behavior are central (e.g., childhood conduct disorder, adult antisocial personality disorder). Therefore, it is difficult to make sense of research that seeks to estimate the associations linking earlier childhood conduct disorder to later drug dependence or that link concurrent adult antisocial personality disorder with drug dependence. There is a tautology or built-in bias here that yields an inevitable association, and the solution is to disengage from the diagnostic criteria for drug dependence (or addiction) any of the clinical features that involve violations of social norms or failures to live up to the expectations of individuals in the drug user’s social fields.

This is not to say that maladaptive failures to live up to expectations of family, friends, or society at large are unimportant consequences of becoming drug dependent (or developing drug addiction). Rather, it is to say that these are potentially causal relationships that might be estimated but that are not necessary in relation to the defining diagnostic criteria for the drug dependence syndrome or in other applications of the addiction syndrome model.

If DSM–5 sustains the DSM–III (American Psychiatric Association, 1980), DSM–III–R, and DSM–IV tradition of including socially maladaptive behavior as clinical features of drug dependence (addiction) in its lists of diagnostic criteria, then one can expect a sustained impression that drug dependence (addiction) is strongly associated with childhood conduct disorder and adult antisocial personality
disorder—in part because of the diagnostic conven­
tions. An alternative set of diagnostic criteria for epi­
demiological and causal research may be necessary
if the DSM-5 sustains this tradition.

Experience-Determined Skip-Outs
and Gated Assessments
A technical detail of field survey research must
regrettably be explained as a complexity that inter­
feres with the task of estimating the probability of
transitioning from the onset of extramedical drug
use and developing a drug dependence syndrome.
Specifically, for the sake of efficiency and to reduce
respondent burden, the designers of field survey
assessments have been motivated to streamline the
assessment of drug dependence in several different
ways. I describe two approaches in this section.
The first approach is often called a *skip-out*, and it
is based on a logical or probabilistic inference
about whether an individual respondent will qual­
ify as being a case of drug dependence. In general,
the skip-out approach is based on the idea that
drug dependence will not develop until after a
certain number of occasions of drug use have
occurred. For example, with respect to tobacco
cigarette smoking, the standard skip-out rule has
involved 100 cigarettes. According to this skip-out
rule, anyone who has not smoked at least 100 ciga­
rettes is not assessed for tobacco-nicotine
dependence.

The second approach has been called the *gated*
assessment. This approach is based on a definitional
feature of the contemporary diagnostic criteria. That
is, as mentioned previously in the Secular Changes
in Diagnostic Approaches and Diagnostic Criteria
Section, the current diagnostic criteria now refer to
occurrence of socially maladaptive consequences of
drug use, hazard-laden drug use (e.g., repeatedly
getting into trouble with the law by driving under
the influence), or both. Indeed, these manifestations
of a drug dependence syndrome are often regarded
as aspects of its clinical significance and without
them, there could be a syndrome of no clinical sig­
nificance (e.g., see Degenhardt, Bohnert, &
Anthony, 2007, 2008). It follows that, in the absence
of evidence that these socially maladaptive conse­
quences of drug use or hazard-laden drug use have
occurred, there is no reason to ask about the other
clinical features of drug dependence (e.g., craving,
withdrawal). Of course, to the degree that epidemi­
ological studies have differed in their implementation
of these gated assessment approaches, it has been
difficult to make comparisons across studies and to
develop more generalizable estimates that might
hold from time to time, as is required for any study
of epidemiological dynamics.

Starting Estimates
Notwithstanding these methodological difficulties,
as a series of publications is now available with start­
ing estimates that can help to guide discussions
about the probability that a user of this or that drug
might become dependent on the drug. For illustra­
tion, Anthony et al. (1994) estimated that when
people start smoking tobacco, about one in three of
them go on to develop a tobacco dependence syn­
drome. For heroin users, about one in four develop
dependence. For intranasal cocaine powder users,
about one in six develop cocaine dependence; for
users of crack cocaine, the estimate is closer to one
in five. For those who start drinking alcohol, drink­
ing on at least one occasion, about one in six to
seven become dependent on alcohol; roughly one
in nine to 10 cannabis smokers develop cannabis
dependence. With respect to the other drug com­
pounds under study (e.g., LSD and related psyche­
delic drugs), Anthony et al. estimated that the
chance of transitioning from onset of drug use
onset of drug dependence was lower than one
in 10, and sometimes as low as one in 20. Anthony
et al. as well as Wagner and Anthony (2002a)
presented these and more detailed estimates for
alcohol, cocaine, and cannabis. In addition, drug­
specific estimates based on more recently gathered
NSDUH data, with a focus on the first 12 to 24
months after onset of drug use, have been published
in a series of more recent articles (e.g., Chen, Storr,
Stone et al., 2007).

A January 2011 contribution from the recently
completed National Epidemiologic Study of Alchoholism
and Related Conditions (NESARC) is noteworthy
(Lopez-Quintero et al., 2011). Nonetheless, compari­
on of these NESARC estimates with the prior NCS
and NSDUH estimates has been made complicated because the skip-out and gating assessment approaches used in the NESARC have not been disclosed in the published article. For this reason, at this time, there is no basis for a direct comparison. For example, the NESARC estimate for transition from tobacco smoking to tobacco–nicotine dependence appears to be based on the traditional skip-out (100-plus cigarettes), but this information is not stated in the published article. In contrast, the NCS and NSDUH estimates are not based on the 100-plus cigarette skip-out rule.

In sum, what researchers now have to work with in research on the epidemiological dynamics of the transition from onset of drug use to the onset of drug dependence is a set of starting estimates, based largely on the 1990 to 1992 NCS and also based on more recent NSDUH estimates of the probability of a rapid transition from onset of extramedical drug use to onset of the corresponding drug dependence syndrome. A more thorough exploration of the epidemiological dynamics of this particular facet of drug dependence epidemiology will require new data or clarification of the published estimates from more recent surveys, such as the one so carefully completed by the NESARC research team.

If investigators pursue these lines of epidemiological research for other expressions of the addiction syndrome, one must ask whether gambling, sex, or Internet addictionologists ever observe specific forms of gambling, sexual behavior, or Internet behavior that are never or rarely followed by the development of this syndrome, as seems to be true for some psychoactive drug compounds. For example, possibly as a result of the very rapid development of tolerance, the conditional probability of formation of an LSD dependence syndrome is remarkably small, with compound-specific variations that can in theory be traced back to differences in signal processing or in receptor activity (Halberstadt & Geyer, 2011; Stone et al., 2007). It would be of interest to know whether any analogous neuroadaptative mechanism might exist that dampens persistence and rates of gambling, sex, or Internet behavior.

As it happens, in a comparison with the substantial and growing body of published evidence about the transition from first drug use to drug dependence, specific for each drug compound or route of administration (e.g., see Strain & Anthony, 2009), the body of epidemiological evidence bearing on these facets of the epidemiological dynamics for the other allied syndromes is relatively sparse, with the possible exception of the recovery process. As noted by Shaffer (in the Introduction to this handbook), W. A. Hunt, Barnett, and Branch (1971) advanced a thesis about resemblance of the recovery process curves for the syndrome of interest, irrespective of the central drug taking or other behavior under study, but an epidemiologist's eye will note that all of the case material in that empirical summary is based on patients in treatment. Epidemiological sampling of college-age populations seems to lead to somewhat different conclusions, although to date the epidemiological studies have largely been restricted to college student samples. For example, on the basis of their college student panel surveys, Slutske (2003) characterized problem gambling as a quite transitory and episodic experience rather than one that has the enduring and chronic nature of the drug dependence syndrome. Of course, it is possible that issues of diagnostic criteria and measurement complicate this evidence, or perhaps there is a more severe form of the gambling syndrome among the treated cases studied characterized by W. A. Hunt et al., Shaffer (in the Introduction to this handbook), and in other chapters of this volume, as compared with a more limited, mild, and possibly not as well-measured gambling syndrome in epidemiological field studies of the type conducted by Slutske et al.

Returning to the overall conceptual model of a unitary syndrome, the cumulative research experience of the drug dependence syndrome has prompted me to recommend the creation of a new set of research diagnostic criteria akin to those developed by Robert Spitzer, Jean Endicott, and colleagues almost a half-century ago for use in their clinical research on psychiatric disturbances. These new criteria should reflect insights developed over the years, with specifications that allow a core concept of a drug dependence syndrome to be developed irrespective of the drug-specific variations in clinical manifestations of neuroadaptational processes that occur after drug taking starts. For
example, the tobacco dependence withdrawal syndrome is not the same as the opioid dependence withdrawal syndrome. As noted earlier, these nuances of differences must be set aside if epidemiologists are to be able to calibrate cross-drug comparisons (e.g., to gauge whether the probability of becoming dependent on tobacco after one self-administration of that drug product is the same as or different from the probability of becoming dependent on crack cocaine after one self-administration of that drug).

The unitary syndrome model for all addictions might prompt others to recommend development of new research diagnostic criteria for the unitary syndrome. In the Introduction to this handbook, Shaffer provides an initial specification of the core clinical features that can be expressed in a set of diagnostic criteria that could guide development of new clinical and field survey assessment tools. Once developed, these tools would make it possible to draw cross-behavior comparisons analogous to those just mentioned for tobacco versus crack cocaine. For example, it would be useful to estimate the probability of developing a gambling addiction syndrome after the first gambling occasion, for comparison with the probability of developing a sex addiction syndrome after the first sexual experience, and onward for comparison to other expressions of the syndrome (e.g., Internet, competitive running), including the drug dependence syndromes. Welte, Barnes, Tidwell, and Hoffman (2009) have attempted to estimate these associations from cross-sectional survey data that confound the gambling syndrome process with the type of game. That is, there is no attempt to address temporal sequencing from the first gambling experience of a specific type to later emergence of the syndrome. Temporally sequenced cross-sectional research and, one hopes, prospective and longitudinal research can give a more complete view of this process, helping to clarify which game types launch the syndrome more rapidly or more languorously. Completion of this type of work for gambling, sex, Internet, and other forms of the addiction syndrome would represent a practical and forward-looking implication of the unitary syndrome model advanced by Shaffer.

CONCLUSION

In conclusion, a general conceptual framework for research focusing on the epidemiological dynamics of extramedical drug use and its associated syndromes and hazards has been derived from a long tradition of research on the epidemiological dynamics of communicable diseases. In some instances, the concepts, principles, and research approaches used to study the epidemiological dynamics of the communicable diseases are directly portable. In other instances, as in the congruency of incubation periods and induction periods, there is a need for adaptation to the context of the research problems.

In its scope, epidemiological dynamics covers a lot of territory. In epidemiology generally, the topic is oriented largely toward population-level dynamics such as macro-level social influences on epidemics. In contrast, the notion of an addiction syndrome model as put forward in this text appeals more to aspects of individual-level variation. Accordingly, in this chapter I have focused on a selection of facets of epidemiological dynamics that are prominent in research on extramedical drug use when studied at the individual level, the persistence or cessation and rates of drug use, and the transition from drug use to drug dependence. The exception has been the work on systems science, the medical and public health approaches to management of epidemics, and the operations research modeling outlined earlier (e.g., Caulkins et al., 2010).

If this chapter achieves its goal, readers will become more highly motivated to investigate these facets and the other neglected facets of epidemiological dynamics in more detail, producing future advances in the understanding of this important area of public health investigation. As stated in the introductory sections of this chapter, the coming decades of the 21st century are promising and should yield secrets about the epidemiological dynamics of extramedical drug use and the development of drug dependence that now remain hidden from view. The future will tell whether the same will be done for other forms of behavior that are encompassed by the addiction syndrome model. Researchers now know how to answer epidemiological questions to discover these secrets. We should answer those questions.
In this chapter, I have provided readers with some epidemiological perspectives that might be said to challenge the validity of the unitary syndrome model advanced by Shaffer (in the Introduction to this handbook). I would like to leave the reader with my personal perspective that the evaluation of a model of this type actually is not a question of whether it is or is not the correct model. Within this context, one must evaluate a model by whether it provokes scientists to answer questions that they might otherwise not attempt to answer. For example, until the middle of the 20th century, there were no epidemiological studies of the probability of developing dependence on drug compounds; no one asked for comparisons across drug compounds of this type. These questions emerged in a post-DSM-III era and the Epidemiologic Catchment Area study; however, it became clear that diagnostic criteria differences for various forms of drug compounds thwarted a calibration of the resulting estimates. One of the advantages of a unitary syndrome model is that it sets forth a common core of clinical features that can be expressed in diagnostic criteria and associated measurements. Once the associated measurements are worked out, it will become possible to ask and answer questions of the type sketched earlier (e.g., regarding the probability of becoming addicted to gambling soon after the onset of gambling relative to the probability of becoming addicted to sex soon after the onset of sexual behavior). As such, the unitary syndrome model provokes researchers to ask new questions and seek answers to epidemiological questions that they might otherwise not contemplate or attempt to answer, irrespective of the ultimate correctness of the underlying theory.

References


Probability and predictors of transition from first use to dependence on nicotine, alcohol, cannabis, and cocaine: Results of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). *Drug and Alcohol Dependence*, 115, 120–130. doi:10.1016/j.drugalcdep.2010.11.004


The syndrome model of addiction emphasizes that very different expressions of addiction have common etiologies, common functions, and common treatments. The transtheoretical model (TTM) rests on the assumption that there are common pathways to change, not only for different expressions of addiction but also across a much broader range of behaviors. In this chapter, I first present the conceptual development of the TTM, then empirical tests of the model, and finally practical applications of the TTM. I make the case that a more comprehensive model of behavior change, one that is based on common dynamics or change constructs, can complement the syndrome model of addiction to produce greater impacts not only across addiction expressions specifically, but also across health risk behaviors generally.

More than 35 years of comparative therapy research meta-analyses have demonstrated that differences between psychotherapies are small and nonsignificant (Luborsky, Singer, & Luborsky, 1975; Smith & Glass, 1977). This is particularly evident when analyses control for the researcher's allegiance to a specific therapy, because effect sizes related to the researcher's allegiance are much larger than effect sizes related to specific therapies (Wampold, 2008). Therefore, the development of the TTM began with the dodo bird question: Is it true that all therapies have won, and all must be given prizes? Is it true that very different therapies—such as motivational interviewing, cognitive-behavioral therapy, and 12-step therapy—produce very common outcomes with addiction-related problems with alcohol (Project MATCH Research Group, 1997)?

If very different therapies produce very common outcomes, then common dynamics (or change constructs) likely account for the outcomes. The initial conceptual analysis for the TTM focused on the theoretical change processes emphasized in 25 of the leading systems of psychotherapy (J. O. Prochaska, 1979). The first finding was that these systems had more to say about why people do not change than about how people can change. These systems were more theories of personality and psychopathology than theories of psychotherapy. Nevertheless, 10 processes, such as consciousness raising from Freud, reinforcement management from Skinner, and helping relationships from Rogers, provided guidance for the TTM.

INTEGRATING STAGES AND PROCESSES OF CHANGE

The next development stage for the TTM included a theoretical search for how these processes of change might be integrated into a more systematic and comprehensive approach to change and to psychotherapy. This search was part of the Zeitgeist in which representatives of competing therapies were collaborating to find methods for integrating the best ideas from leading therapies. Goldfried (1980) proposed that theoretical integration would likely not occur at the conceptual level of concrete therapeutic techniques, in part because the number of techniques are potentially unlimited. He also suggested that integration would not be at the most abstract level of
assumptions about humanity because at this level, there are irreconcilable differences, such as behavior being controlled by environmental contingencies (Skinner) versus behavior being driven by biological forces (Freud). Goldfried recommended that the research related to change processes should be at the mid-level of abstraction of change principles, or what the TTM labeled change processes.

The initial empirical research on the TTM rested on the assumption that if common principles or processes drive change across different therapies, then they should also drive change that occurs without therapeutic assistance. This research sought to compare how much each change process was used by individuals in professional therapies versus self-changers without any professional help. Smoking was the initial target behavior because it is arguably the most deadly expression of addiction that is also very difficult to overcome, with or without professional therapies. This process led to the discovery of a dynamic construct that was not in any of the more than 150 therapies then in existence. The dynamic construct was the stages of change:

1. **Precontemplation**, in which individuals are not intending to change in the foreseeable future;
2. **Contemplation**, in which individuals are intending to change, but not immediately;
3. **Preparation** (originally called determination), in which individuals are ready to take immediate action in the next month;
4. **Action**, which was estimated to be the first 6 months after reaching criteria (such as quitting) and in which the risk for relapse is greatest; and
5. **Maintenance**, in which there are still risks for relapse but individuals do not have to apply the change processes very often to prevent relapse.

Researchers and clinicians immediately recognized the stages of change as a missing link that they could use to integrate the processes of change. With a grant from the National Cancer Institute, J. O. Prochaska and DiClemente (1983) assessed about 1,000 smokers in different stages of change who were not in any professional therapy. They found systematic relationships between the stages and nine processes of change. A process called *social liberation* did not differ by stage, probably because people apply it more at the social level than at the individual level and because it involves giving people more choices that can help them change, such as smoke-free restaurants and alcohol-free parties. Figure 5.1 presents the empirical integration found between processes and stages of change. Guided by this integration, the TTM emphasizes the first three processes in the following list for individuals in the precontemplation stage:

1. **Consciousness raising** involves increased awareness of the causes, consequences, and responses to a particular problem. Interventions that can increase awareness include observations, confrontations, interpretations, feedback, and education. Some techniques, such as confrontation, pose considerable risk in terms of retention, and motivational enhancement methods, such as personal feedback on the current and long-term consequences of continuing the addiction-related

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<td>Consciousness Raising</td>
<td>Environmental Reevaluation</td>
<td>Dramatic Relief</td>
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**FIGURE 5.1** Stages by principles and processes of change.
behavior, might have better long-term success. Increasing the costs of not changing is the corollary of raising the rewards for changing. So, consciousness raising should attempt to increase the perceived rewards for changing.

2. **Dramatic relief** involves emotional arousal about one’s current behavior and the relief that can come from changing. Fear, inspiration, guilt, and hope are some of the emotions that can move people to contemplate changing. Psychodrama, role-playing, grieving, and personal testimonies are examples of techniques that can move people emotionally. It is important to note that earlier literature on behavior change concluded that interventions such as education and fear arousal do not motivate behavior change (J. O. Prochaska, 1979). Unfortunately, researchers evaluate many interventions in terms of their ability to move people to immediate action (J. O. Prochaska, 1979). However, processes such as consciousness raising and dramatic relief should move people to the contemplation stage rather than the action stage. Therefore, researchers should evaluate their effectiveness according to whether they lead to the expected progress.

3. **Environmental reevaluation** combines both affective and cognitive assessments of how an addiction affects one’s social environment and how changing would affect that environment. Empathy training, values clarification, and family or network interventions can facilitate such reevaluation. For example, a brief media intervention aimed at a precontemplative smoker might involve an image of a man clearly in grief saying, “I always feared that my smoking would lead to an early death. I always worried that my smoking would cause lung cancer. But I never imagined it would happen to my wife.” Beneath his grieving face appears this statistic: “50,000 deaths per year are caused by passive smoking (California Department of Health).” In 30 seconds, this message applies consciousness raising, dramatic relief, and environmental reevaluation.

4. **Self-reevaluation** first occurs during the contemplation stage and combines both cognitive and affective assessments of a self-image free from addiction. Imagery, healthier role models, and values clarification are techniques that can move individuals in this type of intervention. Clinically, patients first look back and reevaluate how they have lived as individuals with addiction. As individuals progress into the preparation stage, they begin to apply self-reevaluation with a focus on the future as they imagine how life could be if they were free from addiction.

5. **Self-liberation** occurs from preparation to action and involves both the belief that one can change and the commitment and recommitment to act on that belief. Techniques that can enhance such willpower include public rather than private commitments. Motivational research has also suggested that when individuals have only one choice, they are not as motivated as when they have two choices (Miller, 1985). Three choices are even better, but four choices do not seem to enhance motivation. Wherever possible, then, clinicians should help patients recognize three of the best choices for applying each process. With smoking cessation, for example, there are at least three good choices: (a) quitting cold turkey, (b) using nicotine replacement therapy, and (c) using nicotine fading. Asking clients to choose which alternative they believe would be most effective for them and to which they would be most committed can enhance their motivation and their self-liberation.

6. **Helping relationships** advance most during the action stage. This process combines caring, openness, trust, and acceptance as well as support for changing. Rapport building, a therapeutic alliance, counselor calls, buddy systems, sponsors, and self-help groups can be excellent resources for social support. If patients become dependent on such support to maintain change, the support will need to be carefully faded, lest termination of therapy becomes a condition for relapse.

7. **Contingency management** is appropriate in the action stage and involves the systematic use of reinforcements and punishments for taking steps in a particular direction. Because successful self-changers rely much more on reinforcement than punishment, emphasizing reinforcements for progressing rather than punishments for regress is useful. Contingency contracts, overt and
covert reinforcements, and group recognition are methods of increasing reinforcement and incentives that increase the probability that healthier responses will be repeated. To prepare patients for the longer term, providers should teach help seekers to rely more on self-reinforcements than social reinforcements. Clinical experiences have shown that many patients expect much more reinforcement and recognition from others than they actually receive. Relatives and friends might take action for granted. Average acquaintances typically provide only a few positive consequences early in the action stage. Self-reinforcements are obviously much more under self-control and can be given more quickly and consistently when temptations to lapse or relapse are resisted.

8. **Counterconditioning** is appropriate during the action and maintenance stages and requires learning healthier behaviors that can substitute for addiction-related behaviors. Counterconditioning techniques tend to be quite specific to a particular behavior. They include desensitization, assertion, and cognitive counters to irrational self-statements that can elicit distress. For example, individuals who are self-medicating for depression with a depressant, such as alcohol, could substitute an antidepressant or exercise as a more appropriate technique for countering depression.

9. **Stimulus control** is used most during action and maintenance and involves modifying the environment to increase cues that prompt healthy responses and decrease cues that lead to relapse. Avoidance, environmental reengineering (such as removing addiction-related substances and paraphernalia), and attending self-help groups can provide stimuli that elicit healthy responses and reduce the risk of relapse.

J. O. Prochaska and DiClemente's (1983) article on the integration between the processes and stages of change became the most cited empirical publication of nearly 10,000 articles on tobacco, indicating the impact this integration has had on the field (Byrne & Chapman, 2005). A conceptual and clinical extension of this integration to addiction in general (J. O. Prochaska, DiClemente, & Norcross, 1992) became the most cited article out of the same body of literature, indicating how much key stakeholders have applied this integration of dynamic constructs across expressions of addiction.

**INTEGRATING STAGES AND PROS AND CONS OF CHANGING**

The expansion of the TTM to include two additional dynamic constructs indicates how these individual dynamics apply to a much broader range of behaviors beyond addiction. These dynamic constructs are the pros and cons of changing and reflect decision-making principles that are critical across the stages of change. When considering whether they should struggle to get free from addiction, for example, people will consider some pros of changing, such as better health and functioning, and some cons, such as increased stress and giving up a source of pleasure. Velicer, DiClemente, Prochaska, and Brandenburg's (1985) initial research on decision making drew on the model Janis and Mann (1977) presented in their work titled *Decision-Making: A Psychological Analysis of Conflict, Choice, and Commitment*. Their decision-making model included eight constructs: (a) instrumental benefits to self, (b) instrumental benefits to others, (c) approval from self, (d) approval from others, (e) instrumental costs to self, (f) instrumental costs to others, (g) disapproval from self, and (h) disapproval from others. Janis and Mann based their work on clinical interviews. Velicer et al. created a survey with items designed to represent each of these eight constructs. A principal-components analysis of the responses of 960 smokers (representing the stages of change) to this questionnaire reduced the results to only two components or constructs, the pros and cons of changing (Velicer et al., 1985). When Velicer et al. extended this research to other addiction-related behaviors, such as cocaine abuse, the items written to represent eight constructs were again reduced to just the pros and cons of changing. J. O. Prochaska et al. (1994) also determined that when these dynamics were extended to non-addiction-related behaviors such as diet, sun exposure, and exercise, the same results were found.
More important was the consistent patterns of relationships between these decisional balance constructs and the stages of change. Although some might construe the pros and cons of change as polar opposites, these constructs have practically no correlation with each other. However, as Figure 5.2 illustrates, a very clear relationship emerges from graphing the pros and cons. During the precontemplation stage, the cons of changing are clearly higher than the pros. During the action stage, the opposite occurs, with the pros higher than the cons. These patterns emerge, however, only when raw scores are converted to standardized scores (Velicer et al., 1985). With smoking, for example, smokers in every stage would rate the pros of quitting smoking as more important than the cons. Standardized scores control for such response tendencies and highlight differences. We interpret the differences between raw scores and standardized scores as indicating that decision making about changing behaviors is not a fully conscious and rational process. Part of treatment is to help make decision making be a more conscious, rational, and empirical process, typically beginning by giving clients feedback about how their ratings of the pros and cons relate to smokers in their stage of change who progressed the most.

In J. O. Prochaska et al.’s (1994) meta-analysis across 12 behaviors, other clear patterns emerged. For all 12 behaviors, the pros of changing were higher in the action stage than in the precontemplation stage. The consistent pattern for the cons occurred between the contemplation and action stages, with the cons being lower in action than in contemplation for 11 of 12 behaviors. Even more striking was that when averaging across all 12 behaviors, the pros of changing were 1 standard deviation higher in the action than in the precontemplation stage. The cons, however, were only 0.5 standard deviation lower in action than in contemplation. These results suggest that, across a variety of expressions of addiction-related and non-addiction-related behaviors, the pros increase twice as much as the cons decrease when individuals progress to action. J. O. Prochaska et al.’s article on the integration between the pros and cons and stages of change for 12 behaviors became the 14th most-cited article in the tobacco literature and the highest impact article published in the American Psychological Association’s Health Psychology journal, indicating how much researchers have applied this integration of dynamic constructs across a broad range of behaviors.

Researchers have recently replicated these results in a much larger meta-analysis of 48 health-related behaviors, with data from 125 studies carried out in nine languages in 10 countries (Hall & Rossi, 2008). Historically, one might assume that such heterogeneity of behaviors and populations would produce so much noise that there would be no clear signals. Instead, across behaviors, the cons were much higher than the pros in the precontemplation stage. The pros and cons were essentially equal in the contemplation stage, reflecting the profound ambivalence described by Shaffer and Robbins (1991) in the love–hate relationship that people with addiction have with their substance or behavior of choice. Miller and Rollnick (2002) also identified ambivalence as a major barrier to change. Motivational interviewing is designed, in part, to help clients resolve their ambivalence. In the Hall and Rossi (2008) meta-analysis, such ambivalence was characteristic of individuals in the contemplation stage across 48 different behaviors.

At least as striking as the research focusing on pros and cons are the results of research focusing on the strong and weak principles (J. O. Prochaska, 1994). On the basis of research with 12 behaviors, the strong principle predicts that the pros would be 1 standard deviation higher in the action stage than in the precontemplation stage. Across 48 behaviors, the pros were 1.00 standard deviation...
higher (Hall & Rossi, 2008). Here the magnitude of the difference was evident to the second decimal point. The weak principle predicts the cons would be 0.5 standard deviation lower in the action than in the contemplation stage, but the prediction was not as precise, with the cons being 0.56 standard deviation lower.

These same patterns of dynamics might hold for similar constructs from different theories. Researchers conducted two studies in the Netherlands relating positive and negative expectancies from social cognitive theory to the stages of change from the TTM (Dijkstra, Conijn, & DeVries, 2006; Dijkstra, DeVries, & Bakker, 1996; O. Prochaska, 2006). With large samples of smokers, both studies found patterns for positive and negative expectancies that paralleled the patterns for the pros and cons. These patterns included the strong and weak principles, with positive expectancies about 1 standard deviation higher in the action than in the precontemplation stage and negative expectancies about 0.5 standard deviation lower in the action than in the contemplation stage. These results support the original assumption that there are common dynamic constructs that people treat as separate constructs across different theories, such as pros and cons, positive and negative expectancies, advantages and disadvantages, facilitators and barriers, and benefits and costs.

What does the strong principle imply for changing multiple behaviors? Using IQ as a metaphor, 1 standard deviation equals 15 IQ points. Average college graduates have an IQ of 115. If they were able to raise their IQ to 130, they would be geniuses, so this is a big difference that can have important impacts. However, an effect size of 1 standard deviation is much too large for the best interventions to produce when changing behavior on a population basis. This is one reason why researchers construe intentional behavior change as a process that unfolds over time and involves progress through a series of stages. If one tries to pressure patients to take immediate action, one is likely to produce resistance and reactance, such as terminating therapy prematurely, which happens about 75% of the time with addiction (Connors, Walitzer, & Derman, 2002).

Another implication is that the behavior change IQ of patients and providers needs to increase by 15 points. High school graduates have an IQ of 100; if they could increase it to 115, they could have the mental ability of college graduates. When it comes to behavior change, individuals do not need to be geniuses, but they do need to be smarter. One way to increase behavior change intelligence is to teach patients and providers about dynamic change principles, such as the pros needed to increase progress from the precontemplation to the contemplation stage. They can learn that this principle can be applied to at least 48 different behaviors, and they do not have to learn a new principle of progress for each separate behavior.

Here is an example of how applying one single dynamic principle such as this can have big population impacts. The U.S. military wants to be smoke free, in part for fitness and in part because each smoker produces more than $4,000 per year in excess costs resulting from factors such as health, lost productivity, and fires. The Air Force adopted a policy that required total abstinence of enlisted people during the 6 weeks of basic training. The Air Force monitored abstinence through random blood draws for detecting cotinine. The consequences of finding cotinine were severe: Recruits had to repeat basic training. Needless to say, there was total abstinence.

As with other objects of addiction, the first 6 weeks of stopping smoking represent the steepest part of the relapse curve (Hunt, Barnett, & Branch, 1971). What was the prevalence rate of smoking in enlisted people 12 months after basic training? It was 28% higher (Klesges et al., 2006). I concluded the Air Force policy was a major smoking risk factor for the health of enlisted people. The Air Force also did a population intervention to prevent relapse after basic training. They had one 45-minute session during which they reviewed the pros of staying quit. Such a review is not, however, a principle of relapse prevention: It is a principle for progression from the precontemplation stage. The relapse prevention project had sufficient statistical power with a treatment group of about 7,000 and a control group of 30,000. Nevertheless, there was no statistically significant effect on the full samples. At
the 12-month follow-up, however, precontemplators in the treatment group had quit rates that were 4 times greater than those of the control cadets. With cadets of color, the quit rates were 5 times greater (Klesges et al., 2006). I should note that some pros were pretty powerful. Smoking costs the average cadet 1 month’s income, and not smoking is an important predictor of promotion. These results suggest that applying stage-appropriate dynamic principles can help transform social controls, such as enforced abstinence, into self-controls, such as being quit 12 months later when there were no random blood draws and no threat of repeating basic training.

These results can also have important implications for the treatment of addiction, especially for mandated treatments. Tsoh (1995) assessed the pros and cons of psychotherapy for patients in drug treatment who were in different stages of changing their addiction-related behavior. She not surprisingly found the same type of pattern as in 48 different behaviors, such as the cons of therapy being higher for patients in the precontemplation stage than the pros. More striking was the assessment of whether patients were in therapy out of choice or coercion, depending on their stage for changing their addiction-related behavior. She found no significant differences between stages for actual coercion, such as being mandated by the courts, social services, or employers. She did find, however, significant differences between stages for perceived coercion and choice, with patterns similar to pros and cons.

Patients in the precontemplation stage, for example, perceived that they were in therapy more out of coercion than choice, with the opposite pattern for patients in the preparation stage and later. However, these patterns were found only when standardized scores were used, suggesting that the dynamic of coercion and choice might not be as conscious and rational as raw scores would suggest (Tsoh, 1995).

These results also suggest that helping patients progress through the stages of change can reduce their sense of coercion and increase their experience of choice. A study of mandated weekly group therapy for partner abuse reflected the impacts that such dynamics might produce (Levesque, Driskell, Prochaska, & Prochaska, 2011). The researchers randomly assigned men in treatment to best practice alone or best practice plus three 20-minute interactions with a computer program tailored to participants’ stage of change, pros and cons, and other transtheoretical variables relevant to helping them progress from one stage to the next. If they progressed, the program was tailored to dynamic constructs matched to their current stage. The program assessed each relevant variable and provided normative feedback on participants’ use of each variable compared with peers who progressed the most and gave feedback over time on any progress made on dynamic constructs, such as progressing two stages or increasing in their pros of changing. At 6 months, the enhanced treatment group had significantly fewer men in the precontemplation stage and significantly more in the action stage with low risk for relapse. Their partners reported significantly lower rates of physical and emotional abuse. Of special interest was that the treatment group voluntarily sought about twice as much individual, couples, and group therapy, with about 50% seeking more help. These men had complex conditions, including addiction and personality or affect disorders, besides problems with violence. Nevertheless, about half had progressed from being coerced into therapy to seeking treatment by choice.

**DYNAMIC CONSTRUCTS: PREDICTING ENGAGEMENT IN TREATMENT AND THE CHANGE PROCESS**

Engagement might well be the number one challenge for behavior change programs. In informal discussions, program consultants reported that engagement was the number one concern of employers providing proactive outreach programs for management of chronic conditions. About 85% of the effort in effective programs needs to be lifestyle management involving major behavior changes. Yet only about 8% of eligible employees participate in such free programs provided by telephone to their homes by trained counselors. Primary care practices now identify about 50% of their patients as having mental illness, but only 8% of these patients follow through on referrals to appropriate treatment programs (Cummings, 2003).
When health plans offer free smoking-cessation clinics, only about 1% of smokers participate (Lichtenstein & Hollis, 1992). Our unpublished analyses of requests for proposals for free quit lines have shown that state public health departments typically budget for only one quarter of 1% of smokers participating. Clinics for problem gamblers in Windsor, Ontario, Canada, receive 2% of casino earnings to serve such gamblers, but they have had many more resources than they have had clients (Robert McDonald, personal communication, 2006).

Lichtenstein and Hollis (1992) reported on one health care plan that decided to be very proactive about engaging many more smokers in their free smoking-cessation clinics. Physicians spent time with individual smokers just to get them to sign up; if that did not work, nurses took up to 12 minutes to get them to sign up; if that did not work, health educators took up to 15 minutes; and if that did not work, counselors called them at home. This recruitment effort is the most intensive proactive one described in the literature, and it resulted in 35% of smokers in the precontemplation stage and 64% in the contemplation and preparation stages signing up. However, only 3% of the smokers in the precontemplation stage showed up, 2% finished the program, and none ended up quitting. With the other group, 15% showed up, 11% finished, and some unreported percentage ended up quitting (Lichtenstein & Hollis, 1992). In one population, the stages of change alone predicted differential rates of signing up, showing up, finishing up, and ending up taking action.

At a workshop that I gave in Windsor, Ontario, Canada, clinicians from one clinic told the audience how they decided to be creative in treating problem gamblers. They used a traffic light as a metaphor for the stages of change. They placed ads on the backs of city buses with a traffic light that said, "Red light not ready; yellow light getting ready; green light ready. Ready or not, give us a call, we can be of help with your gambling. Wherever you are at, we can work with that." They were flooded with calls, some of which included questions such as "Is it really true that wherever I am at you can work with that?" The answer was, "Come in and give us a try!" Of special interest was a story of two problem gamblers standing on a street corner, pointing to a bus with a traffic light on the back. "Hey, there goes my bus. That's what brought me here," one said, suggesting he had identified the bus in the metaphor as what helped him to engage in therapy.

What happens when people do show up for treatment? Historically, about 50% of patients terminate therapy prematurely, as judged by their therapists (O'Donnell, 2009). The best predictors of dropout include static variables, such as demographics (minority status and lower education) and presenting problem (addiction). Brogan, Prochaska, and Prochaska (1999) used dynamic constructs, such as stage of change, and predicted more than 90% of the premature terminators. The baseline profile of the whole group of premature terminators was one of people in precontemplation. Patients who terminated quickly but appropriately tended to be in the action stage when they first engaged in therapy. A recent meta-analysis (Norcross, Krebs, & Prochaska, 2011) found that across studies including addiction, general mental health, obesity, and other health risk behaviors, stages of change produced significant and moderate effect sizes for predicting premature termination.

Understanding premature termination is especially important in the treatment of addiction, where dropout rates are about 75% for such problems as alcohol abuse (Connors et al., 2002), overweight and obesity (J. O. Prochaska, Norcross, Fowler, Follick, & Abrams, 1992), and illicit drug use (National Institute on Drug Abuse, 1999). Yet the National Institute on Drug Abuse (1999) stated that the best predictor of successful recovery from the abuse of illicit drugs is the length of time spent in drug treatment. One leading treatment center for alcohol problems had about a 75% dropout rate and compared two interventions for reducing the rate. One approach was the role induction method that has been most widely used and involves preparing patients for what to expect in the unusual experience of therapy and what client and therapist roles and responsibilities were. This approach had no effect. The other approach was one session of motivational interviewing, which significantly reduced the dropout rate from 75% to 50% (Connors et al., 2002).
Many people consider the dynamic construct of the working alliance or therapeutic alliance to be the best measure of engagement and one of the best predictors of therapy dropouts and outcomes (Horvath & Bedi, 2002). The data are mixed, and Barber (2009) has concluded that accepting that the working alliance is critical to clinical outcomes is premature. In Project MATCH, the therapeutic alliance was a significant predictor of treatment participation and treatment outcomes for the outpatient sample but not for the aftercare sample (Connors, Carroll, DiClemente, Longabaugh, & Donovan, 1997). The best predictor of the therapeutic alliance in both samples, however, was readiness to change, on the basis of one of the measures for stages of change (Connors et al., 2000). More recently, Norcross et al. (2011) found in a meta-analysis that empathy, rather than working alliance, was the stronger predictor of outcomes. The therapeutic relationship might be the more fundamental dynamic for effective therapy. For example, it might include therapists who can show empathy for patients in the early stages of change and who perceive the cons of therapy as outweighing the pros, perceive treatment for addiction as more coercion than choice, or have profound ambivalence or serious resistance to changing.

Patterns of Progress on Dynamic Constructs

The empirical investigations of relationship patterns between stages of change and the pros and cons of changing and the processes of change discussed earlier relied on cross-sectional data. In this section, I describe patterns of longitudinal progress on these same variables for three types of smokers who were assessed every 6 months for 2 years. The three types represented dynatypes, which are patterns of change over time. Sun, Prochaska, Velicer, and Laforge (2007) labeled the first dynatype maintainers, who were in the action stage and quit smoking at one follow-up and remained quit at every subsequent follow-up. The second dynatype is relapers, who took action and quit smoking at one follow-up but were back to smoking at a subsequent follow-up. The third was stable smokers, who did not progress to the action stage at any follow-up.

The stable smoker dynatype had a very clear and consistent pattern in its application of 14 dynamic constructs (i.e., 10 processes of change, pros of changing, balance between pros and cons, and self-efficacy). This pattern was essentially flat lines over 24 months, with minimal increases or decreases on any of the 14 constructs. This pattern reflects excellent test–retest reliability of the 14 dynamic constructs in groups that failed to progress from smoking to not smoking at any assessment. This unchanging path on dynamic or change constructs was replicated in two groups of stable smokers, one a treatment group and one a control group, indicating that smokers who fail to take action follow essentially the same path whether they receive treatment or not (Sun et al., 2007).

The maintainer dynatype exhibited a dramatically different pattern. Here the dynamic constructs increased or decreased considerably depending on which direction yielded successful action. The pros of quitting smoking and self-efficacy for quitting increased by about 0.4 standard deviation and 0.2 standard deviation, respectively. The cons of quitting, however, decreased significantly. Some of the processes of change, such as consciousness raising and self-reevaluation, decreased over time; others, such as counterconditioning, increased (Sun et al., 2007).

What was especially striking was that both the treatment and the control maintainers followed the same pattern for each dynamic construct. These patterns indicate that smokers who succeed in taking and maintaining action follow the same path, whether they received treatment or not. This is not to say that the two groups successfully quit at the same rates. Outcome analyses consistently demonstrate across at least 10 of our center’s studies that the treatment groups quit at significantly higher rates ($M = 25.6\%$) than the control groups ($M = 19.7\%$; Sun et al., 2007). These outcome data indicate that treatment helps a higher percentage of smokers to find and follow successful paths at a higher rate than control participants acting on their own.

These data are relevant to the dodo bird question as to how very different therapies produce very common outcomes. Treatment and control groups would appear to represent intervention conditions that signify greater differences than two or three types of psychotherapies that are professionally and
scientically trying to help clients take effective action on chronic conditions such as addiction. If successful treatment and control smokers follow common pathways to quitting smoking, one might predict that the same patterns would exist in different types of psychotherapies and thus that different therapies would produce common outcomes, as with the percentage of patients in the maintenance dynatype, in addition to common pathways to change. What happened to the relapse dynatype? At baseline, they mirrored maintainers on the 14 dynamic constructs, and both groups consistently made better efforts than the stable smoker dynatype. Over the first two follow-ups, the relapers paralleled the paths of the maintainers. As they relapsed over time, however, their use of dynamic constructs regressed toward the pattern of the stable smokers. The two dynatypes did not converge, however, because the relapers' application of the dynamic constructs ended up being between that of the maintainers and stable smokers, but generally closer to the maintainers. These patterns would predict that the relapers who took action would be more likely to quit smoking over the next 12 to 24 months at higher rates than the stable dynatype, who were not taking action at any assessment point. Longitudinal research has confirmed this prediction (e.g., J. O. Prochaska, DiClemente, Velicer, Ginpil, & Norcross, 1985).

The three dynatypes and their patterns of progress and applications of dynamic constructs have essentially been replicated across different types of behaviors, including affective behaviors (stress and depression), energy balance behaviors (diet and exercise), adherence behaviors (cholesterol management medications), and appearance behavior (sun exposure; J. O. Prochaska, 2011). Measures of use of dynamic constructs in control group dynatypes were only available for smoking, diet, and sun exposure.

### Dynamic Predictors of Progress

The next type of comparative behavior change research across multiple behaviors has been our studies on baseline predictors of progress to the action and maintenance stages at mid- and final follow-up. From our dynatype research on patterns of progress (Sun et al., 2007), we could predict that smokers who were taking effective action at follow-up would make significantly better efforts on dynamic constructs at baseline. Blissmer et al. (2010) found this, not only with an addiction-related behavior (smoking) but also for energy balance behaviors (diet and exercise) and for an appearance behavior (sun exposure). Blissmer et al. also confirmed these predictions with affective behaviors (stress and depression) and adherence behavior (cholesterol medication).

Our outcome analyses (e.g., J. O. Prochaska et al., 2004, 2005, 2007, 2008) suggest that groups who were taking effective action at follow-up would have been significantly more likely to have been randomly assigned to the treatment than to the control condition. Research (e.g., Blissmer et al., 2010) has confirmed this prediction for six behaviors: smoking, diet, exercise, sun exposure, stress, and medication adherence. The TTM model would also predict that groups who were taking effective action at follow-up would have been significantly further into the preaction stages of change at baseline than those who were not. Research (e.g., Blissmer et al., 2010) has also confirmed these predictions for six behaviors. Finally, from the psychotherapy research, we were able to predict that groups who were taking effective action at follow-up would have significantly more severe target behaviors at baseline than those who were not. Research has confirmed these predictors with all target behaviors, except effective stress management, for which the successful group had greater stress at baseline (e.g., Blissmer et al., 2010).

These four effects (i.e., treatment, stage, effort, and severity) are all dynamic constructs that can be open to change. Blissmer et al. (2010) did not find any consistent pattern for static demographic variables predicting effective action across multiple behaviors. Typically, with demographic variables no significant effect sizes were found or effect sizes were very small. These results are important, because treatment providers can use the four effects to create smarter goals, such as helping people stay in treatment by progressing one stage at a time and by making smarter efforts that can reduce severity of the problem. This set of smarter goals might produce significantly greater action at long-term follow-up.
Simultaneously Changing Multiple Behaviors

If the aforementioned dynamics hold across multiple behaviors, then providers should be able to change multiple behaviors simultaneously. The recent National Institutes of Health (2009) report *NIH Science of Behavior Change* recommended simultaneously changing multiple behaviors as one of the top research priorities across the National Institutes of Health. The report also suggested that this type of research is one of the riskiest. The Heart Health Projects of Minnesota; Stanford, California; and Pawtucket, Rhode Island, represent the difficulties of such multibehavior change. These projects turned out to be major disappointments. Leaders of the Minnesota Heart Health Projects even concluded that they might have been better off if they had put all of their intervention resources into changing a single behavior, such as smoking (Luepker et al., 1994).

The common clinical wisdom suggests that changing a single behavior such as smoking is hard enough that researchers should not risk overwhelming individual patients or entire populations with simultaneously trying to change multiple behaviors (Patterson, 2001). Research supported by the Robert Wood Johnson Foundation could not find, as of 2003, any programs of research that were successful in such simultaneous changes (J. O. Prochaska, Redding, & Evers, 2007).

Since then, however, a series of important breakthroughs have occurred. First, my colleagues and I were able to demonstrate that we could simultaneously change three (Johnson et al., 2006, 2008; Jones et al., 2003; J. O. Prochaska et al., 2004, 2008) or four (J. O. Prochaska et al., 2005) behaviors for disease management and prevention. Moreover, we were able to demonstrate that individuals treated for two behaviors (such as smoking and diet) were as successful as those treated just for smoking, and those treated for three behaviors were as successful as those treated for two (J. J. Prochaska, Velicer, Nigg, & Prochaska, 2008). Across such studies, multiple behavior changes included addiction-related behaviors (smoking), affective behaviors (stress and depression), energy balance behaviors (diet, emotional eating, and exercise), adherence behaviors (medication for cholesterol management), and appearance behaviors (sun exposure).

Another important discovery was labeled *coaction*, which is the probability that if individuals take effective action on one behavior (e.g., smoking), they are more or less likely to take action on a second behavior (e.g., diet). In a large control group, we (Johnson et al., 2008; Mauriello et al., 2010; J. O. Prochaska et al., 2008) found they were less likely. In treatment groups, they were 1.5 to 3.5 times more likely. Coaction appears to be much more of a treatment phenomenon than a naturally occurring phenomenon.

We have applied the coaction phenomenon to produce greater treatment impacts with fewer demands on clients and clinicians. In one study (Johnson et al., 2006), we had a primary behavior (i.e., adherence to cholesterol medication) that we treated with a module that was based on computer-tailored interventions that targeted each of the TTM dynamic constructs. We provided minimal treatment tailored to stage of change for the secondary behaviors of exercise and diet. We also provided all treatment participants a stage-based interactive workbook that taught them the principles and processes of progressing across the stages of change for all three behaviors. The outcomes were nonadherence rates in the control group that were three times as great at 18 months as those of the treatment group. For exercise and diet, twice as many members of the treatment group as of the control group were taking effective action.

What treatment process can produce multiple behavior change with reduced treatment demands? One hypothesis is that treatment participants are learning common principles and processes that can be applied across each behavior. To progress from the precontemplation to the contemplation stage, for example, they can learn that their pros for changing must increase. The workbooks provide examples of pros for changing each of the three target behaviors. Such workbooks have been a standard part of our center's treatment programs for adults for both single and multiple behavior change.

The real world can, however, create challenges to such interventions. For example, with children and...
youth, schools only permit about three 30-minute individualized and interactive sessions with computer-tailored interventions. This was the case with Mauriello et al.’s (2010) High School Healthy Weight Management project, in which they targeted exercise (the primary target), diet, and television watching as risk behaviors. In this program, Mauriello et al. used full TTM tailoring with the dynamic constructs (e.g., pros and cons of changing). Diet and TV watching rotated from session to session in receiving optimal constructs (i.e., stage, pros and cons, self-efficacy) that account for the most variance in behavior change or minimal (stage-only) tailoring. The theme of the program was acquiring or maintaining a healthy lifestyle. Mauriello et al. linked this higher order construct to each of the three target behaviors.

In a second study (Velicer et al., 2011), we assessed whether this healthy lifestyle program could produce changes not only in the treated behaviors of exercise, diet, and TV watching but also in the untreated behaviors of preventing the use of smoking and alcohol at a young age. For this to occur, coaction would have to drive such prevention. We did this study with younger adolescents in middle schools.

As predicted, at 24 months this treatment produced significantly greater percentages of adolescents who progressed from being at risk for exercise at baseline to not at risk at 24 months (48.3% vs. 37.4%, respectively; Velicer et al., 2011). For those who were at criteria for physical activity at baseline, significantly more in the treatment group than in the comparison group maintained their low-risk status (73.5% vs. 45.9%, respectively). For those at risk for diet (fruits and vegetables) at baseline, significantly more in the treatment group than in the comparison group were taking action or in maintenance (and were not at risk) at 24 months (27.9% vs. 11.6%, respectively). For those at criteria at baseline for fruits and vegetables, significantly more in the treatment group maintained their low-risk status at 24 months (58.5% vs. 15.8%, respectively). We found similar patterns for TV time, with a greater percentage in the treatment group at risk at baseline in action or maintenance at 24 months (48.4% vs. 25.1%, respectively). Of those not at risk at baseline, significantly more in the treatment group maintained their low-risk status at 24 months (78.8% vs. 67.2%, respectively).

Especially striking was that this treatment, which was directly treating energy balance behaviors, also produced significantly greater effects than those in the comparison group on preventing acquisition of two expressions of addiction. That is, with smoking acquisition in adolescents who reported not intending ever to smoke, a significantly smaller percentage of the treatment group than of the comparison group were smoking at 24 months (3.4% vs. 6%, respectively; Velicer et al., 2011). Similarly, with alcohol acquisition in adolescents who reported not intending to drink, a significantly smaller percentage in the treatment group than in the comparison group reported drinking at 24 months (4.4% vs. 7.1%, respectively). Our hypothesis is that the treatment’s theme of acquiring or maintaining a healthy lifestyle drove this coaction.

In another study, we (Evers, Prochaska, Van Marter, Johnson, & Prochaska, 2007; J. O. Prochaska, Evers, Prochaska, Van Marter, & Johnson, 2007) tested the same hypothesis, that a thematic approach might be able to produce coaction of behaviors that are linked to the theme. With bullying, the number one daily health and mental health worry of children in the United States, we again had very limited time for treatment in schools. Current perspectives on bullying include multiple behaviors, such as physical (hitting, beating), emotional (stealing or damaging goods), and social (malicious gossip, ostracizing) behaviors. What higher order construct or theme could produce changes across each of these multiple behaviors? The theme we chose was relating with respect. Our computer-tailored interventions used TTM tailored to this theme and included not only bullies but also passive bystanders, who could be part of the solution, and victims, who could be guided to receive more effective help. Across high schools, middle schools, and elementary schools in which about half of students were eligible for free lunch, these programs were able to produce 30% to 40% effective action in bullies, passive bystanders, and victims, which about doubled the changes seen in the control schools.
TOWARD A UNIFIED MODEL OF MULTIPLE BEHAVIOR CHANGE

I began my search with the common dynamics or change constructs that could account for how very different therapies could produce such common outcomes. This search led to the discovery of the stages of change, which could be used to integrate processes and principles of change that were identified in leading theories of psychotherapy and behavior change. People have used these TTM variables to predict who signs up for treatment, who shows up, who finishes, and who ends up taking more effective action.

These dynamic constructs can help therapists to understand how they can help clients make the behavior change to engaging in greater adherence with biological therapies and psychological therapies. Cross-sectional analyses of three of these dynamic constructs (stages, pros and cons of changing) have produced predictable patterns across 48 different behaviors. Longitudinal analyses (Sun et al., 2007) of three behavior change dynatypes (i.e., maintainers, relapsers, and nonchangers) have found predictable patterns of applications of these dynamic constructs across multiple types of behaviors (i.e., addiction-related, affective, energy balance, adherence, and appearance behaviors). Baseline studies of these dynamic constructs and of treatment and severity status have illustrated significant predictors of long-term outcomes in these same types of behaviors.

Applying these dynamic constructs in TTM-tailored interventions has demonstrated simultaneous multiple behavior change across these same types of behaviors as well as in bullying behaviors, which has lead to three strategies for simultaneous multiple behavior change. The first is a separate treatment module or manual for each target behavior. The second is coaction, driven in part by patients learning common principles and processes of change that they can apply across different types of behavior. The third is a thematic approach that uses higher order dynamic constructs, such as relating with respect or having a healthy lifestyle.

Comparing these three strategies of multiple behavior change can help the psychotherapy and behavior change field progress toward a unified model of treatment and change. Analyses of treatment mediators across different types of behaviors and different types of treatments applying common dynamics can further advance the field. At this time, my cursory causal model is that the combination of higher order dynamic constructs (themes) can help move individuals to apply a common set of dynamic processes of change that can produce coaction across behaviors that a provider is treating fully, optimally, or minimally.

Given the knowledge that already is available, the science of behavior change should be adopting simultaneous multiple behavior change as the norm rather than the exception. In the clinical world of changing addiction-related behaviors in complex cases, multiple behavior change is already the rule, implicitly or explicitly. Few clients with an addiction-related behavior do not face problems with affective behaviors, such as anger, anxiety, depression, distress, or stress. When faced with times of psychological distress, Americans most often treat their distress by smoking more cigarettes, drinking more alcohol, eating more comfort foods, or taking more over-the-counter or under-the-counter drugs (Mellinger, Balter, Manheimer, Cisin, & Parry, 1978). Most patients have treatment adherence problems with their biological medicines, behavioral medicines, or both. Too many smokers have relied on their nicotine addiction to help manage their weight, and they could benefit from healthy energy balance behaviors to manage not only their weight but also their moods and the times when they are most tempted to relapse.

The problem is that clients and clinicians have not had the support of a science of simultaneous multiple behavior change. Today, a growing body of knowledge is pointing toward a unified model that can generate strategies for changing both multiple expressions of addiction and multiple types of behavior that are central to enhancing emotional, mental, physical, social, and even vocational health and well-being.

The syndrome model of addiction provides an integrative framework for producing simultaneous change across multiple expressions of addiction. It emphasizes that very different expressions of
addiction have common etiologies, common functions, and common treatments. This model would support the prediction that therapists should be able to produce significant treatment-induced coaction across multiple expressions of addiction. An open question is whether a dynamic theme, such as becoming free from addiction, or the application of change constructs, such as the pros and cons of changing that are common across different expressions, would best drive such coaction.

One innovation that research on simultaneous multiple behavior change could bring the syndrome model is that the model could apply coaction strategies across an even broader range of behaviors, including affective, adherence, and energy balance behaviors as well as expressions of addiction (e.g., Johnson et al., 2006; Mauriello et al., 2010). More research on the dynamic constructs that can link such different types of behavior and that can drive changes across such different behaviors would improve understanding of behavior change. The syndrome model encourages researchers, providers, and other key public health stakeholders to search for commonalities in areas in which society has too often been preoccupied with differences.

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PART II

DISTAL AND PROXIMAL INFLUENCES ON ADDICTION
Addiction to a substance or behavior describes a compulsive pattern of use or behavior, characterized by impaired control and continued use of a substance or engagement in a behavior in the face of negative consequences (Leeman, Grant, & Potenza, 2009; O'Brien, Volkow, & Li, 2006). Core components of various expressions of addiction, therefore, include (a) continued engagement in a behavior despite adverse consequences, (b) diminished self-control over engagement in the behavior, (c) compulsive engagement in the behavior, and (d) an appetitive urge or craving state before engagement in the behavior (J. E. Grant, Brewer, & Potenza, 2006). In addition to similarities across different expressions of addiction, there are also differences among addictive behaviors. For example, tolerance and withdrawal have different neurobiologies depending on the addictive behavior (Dalley, Everitt, & Robbins, 2011).

Drug experimentation, or initiation of addictive behaviors, typically begins during adolescence, initially resulting in hedonic experiences that produce immediate positive reinforcement (Rutherford, Mayes, & Potenza, 2010; Wagner & Anthony, 2002). As drug use continues, neuroadaptations occur relating to the development of drug tolerance, resulting in a reduction of the pleasurable sensations achieved from the initial level of drug use. Motivational neurocircuitry and multiple neurotransmitter systems are implicated. As the cycle continues, subjects increase the frequency and amount of drug use to achieve the same rewarding experience. For many drugs, increased use also leads to withdrawal symptoms when drug use is reduced or stopped. These withdrawal symptoms can be at least temporarily relieved by drug use; consequently, a vicious cycle is established. Over time, hedonic motivations for substance use diminish, and negative reinforcement motivations increase. The result is that drug-taking behaviors become less rewarding and more compulsive or habitual. This shift likely reflects a progression of involvement of ventral to dorsal cortico–striato–pallido–thalamic circuitry (Everitt & Robbins, 2005; Haber & Knutson, 2010).

Multiple brain structures are involved in addiction. The structures most often implicated in addiction and vulnerability for addiction therefore appear to reflect dysfunction in motivational neurocircuitry (Everitt & Robbins, 2005; Volkow & Li, 2004). A central feature of motivated behaviors involves cortico–striato–pallido–thalamic circuits (Alexander & Crutcher, 1990; Alexander, DeLong, & Strick, 1986), along with the amygdala (providing affective information), hippocampus (contextual memory), hypothalamus (homeostasis), insula (interoceptive processing; George & Koob, 2010; Naqvi & Bechara, 2009), and cingulate cortex (emotional regulation and cognitive control; Bush et al., 2002).

As we demonstrate in this chapter, research focusing on the neurobiology of addiction has suggested that both substance and behavioral expressions of addiction share common etiologies, consistent with a syndrome model of addiction (Shaffer et al., 2004). Therefore, the objectives of this chapter are to present the neurobiology, cognition, and genetics underlying substance and...
behavioral addictions and to highlight the shared biological antecedents to multiple patterns of addictive behavior.

NEUROBIOLOGICAL PROCESSES

Multiple neurotransmitter systems (e.g., serotonergic, dopaminergic, noradrenergic, opioidergic, glutamatergic) have been implicated in the pathophysiology of addiction (see Potenza, Sofuoglu, Carroll, & Rounsaville, 2011, for review). These neurotransmitters appear to play roles in both substance and behavioral expressions of addiction and thereby suggest shared neurobiological systems.

Serotonin

Serotonin has also been associated with heavy alcohol use and addiction (Herman, Philbeck, Vasilopoulos, & Depetrillo, 2003; Kreek, Nielsen, Butelman, & LaForge, 2005), and the association of serotonin and addictions might be the result of serotonin's role in mediating impulse control. Evidence for serotonergic involvement with addiction comes in part from studies of platelet monoamine oxidase B activity, which correlates with cerebrospinal fluid (CSF) levels of 5-hydroxyindole acetic acid (5-HIAA; a metabolite of serotonin) and is considered a peripheral marker of serotonin function. Low CSF 5-HIAA levels correlate with high levels of impulsivity and sensation seeking and have been found in pathological gambling and substance use disorders (Roy & Markku, 1989).

LeMarquand, Benkelfat, Pihl, Palmour, and Young (1999) found that a sample of nonalcoholic young men (ages 18–25) with family histories of alcoholism responded differently to a tryptophan (amino acid precursor to serotonin) depletion procedure than did a sample of young men of similar age with a negative family history of alcoholism. Although in a tryptophan-reduced state, family-history-positive young men made more commission errors on a go/no-go behavioral task—suggesting difficulty in inhibiting responses—than did family-history-negative young men. These findings suggest associations between impulsive behavior and low levels of serotonin in individuals with a greater propensity for developing substance addictions.

Pharmacologic challenge studies that measure hormonal response after administration of serotonergic drugs have also provided evidence for serotonergic dysfunction in substance use disorders. Individuals with addiction report a high after administration of meta-chlorophenylpiperazine, a partial serotonin agonist that binds to multiple serotonin receptors (5HT1 and 5HT2) with particularly high affinity for the 5HT2c receptor (DeCaria et al., 1997; Pallanti, Bernardi, Quercioli, DeCaria, & Hollander, 2006). Furthermore, serotonergic probes have been used in conjunction with brain imaging for individuals with impaired impulse control. Individuals with alcohol addiction demonstrate a blunted response in the basal ganglia circuits involving orbital and prefrontal cortices when given meta-chlorophenylpiperazine (Hommer et al., 1997).

Although serotonin might be involved with the onset and maintenance of addiction, it is unlikely that dysfunction in serotonin neurotransmission is solely or predominately responsible for addictive behaviors. Clinical trials of selective serotonin reuptake inhibitors during the treatment of alcohol or gambling problems have demonstrated limited efficacy (J. E. Grant & Kim, 2006; Kenna, 2010), suggesting limitations with respect to a central or solitary role for serotonin in addictive behaviors.

Dopamine

Research has implicated dopamine in rewarding and reinforcing drug-using behaviors (Goldstein & Volkow, 2002; Nestler & Malenka, 2004). More specifically, dopamine appears to be related to the prediction of reward and salience (Lu, Grimm, Shaham, & Hope, 2003; Schultz, Tremblay, & Hollerman, 2000). Repetitive addictive behaviors after an urge might reflect a unitary process. Preclinical and clinical studies have suggested that an underlying biological mechanism for urge-driven disorders might involve the processing of incoming reward input by the ventral tegmental area–nucleus accumbens–orbitofrontal cortex circuit (Volkow, Fowler, & Wang, 2003). The ventral tegmental area contains neurons that release dopamine to the nucleus accumbens and orbitofrontal cortex. Alterations in dopaminergic pathways have been proposed to underlie the seeking of rewards (e.g., drugs,
gambling) that trigger the release of dopamine and produce feelings of pleasure (Volkow et al., 2003). Continued seeking and taking of substances can be perpetuated, at least in part, by reduced numbers of dopamine D2 receptors in the brain (Volkow et al., 2003). The reduced number of receptors leads to altered dopamine signaling, which could lead vulnerable individuals to be inadequately reinforced by everyday activities considered by most people to be enjoyable (Volkow et al., 2003).

On a molecular level, dopamine function, particularly striatal D2–D3 receptor function, appears relevant to this process and has been implicated across a variety of expressions of addiction (Johnson & Kenny, 2010; Steeves et al., 2009; Wang, Volkow, Thanos, & Fowler, 2009). For example, one early molecular genetic study implicated the TaqA1 allele of the dopamine receptor gene DRD2 similarly across substance and behavioral expressions of addiction (Comings, 1998). Multiple other neurotransmitter systems, however, contribute and may represent better treatment targets, particularly because D2–D3 receptor antagonists have not demonstrated clinical efficacy for addiction.

Dopamine involvement with addiction, particularly behavioral expressions of addiction, is further suggested by studies of medicated patients with Parkinson's disease (Abosch et al., 2011; Voon et al., 2006; Weintraub et al., 2006), which found that 6% to 30% experienced the new onset of an addictive behavior after starting dopamine agonist medication. Additionally, psychomotor stimulant drugs such as d-amphetamine are known to increase impulsive responding, an effect that depends on dopamine release in the nucleus accumbens (Cole & Robbins, 1987), probably in the shell subregion (Murphy, Robinson, Theobald, Dalley, & Robbins, 2008).

Dopamine levels, however, are not sufficient to account for the onset of addiction because dopamine increases can be observed among addicted and nonaddicted individuals after the introduction of a drug (Goldstein & Volkow, 2002). In fact, the dopamine release observed in response to cocaine administration is typically larger among nonaddicted individuals than addicted individuals (Volkow et al., 1997). Although multiple studies have implicated dopamine systems in drug addiction, drugs influencing dopamine neurotransmission (e.g., dopamine antagonists like haloperidol) have not demonstrated much success in the treatment of addictions.

**Norepinephrine**

Studies have found higher levels of noradrenaline and its metabolites in urine, blood, and CSF samples obtained from pathological gamblers than from nongamblers, and noradrenergic measures correlate with measures of extraversion (Roy, Adinoff, Roehrich, & Lamparski, 1988; Roy, De Jong, & Linnoila, 1989). Gambling has also been associated with autonomic arousal such as increases in heart rate (Meyer et al., 2004). Noradrenergic activity influences prefrontal cortical function and posterior attention network, and adrenergic drugs have been shown to influence specific aspects of impulse control in animal and human studies (Chamberlain & Sahakian, 2007).

**Opioids**

Research has implicated opioids in pleasurable and rewarding processes; opioid function can influence neurotransmission in the mesolimbic pathway that extends from the ventral tegmental area to the nucleus accumbens or ventral striatum (Spanagel, Herz, & Shippenberg, 1992). Placebo-controlled, double-blind, randomized trials have evaluated the efficacies and tolerabilities of naltrexone and nalmefene with alcohol dependence, opiate addiction, gambling addiction, and cocaine addiction (Fareed, Casarella, Amar, Vayalapalli, & Drexler, 2010; Leung & Cottler, 2009; Rodriguez-Arias, Aguilar, Manzanedo, & Miñarro, 2010; Rösner et al., 2010). Although opioid antagonists (e.g., naltrexone) have consistently demonstrated benefit for the treatment of addiction, other factors also appear to be associated with treatment response to opioid antagonists (e.g., allelic variants of the gene encoding the µ-opioid receptor; Oslin et al., 2003).
Glutamate
Glutamate, the most abundant excitatory neurotransmitter, has been implicated in motivational processes and drug addiction (Kalivas & Volkow, 2005). On the basis of data suggesting a role for glutamatergic therapies with addiction, the glutamatergic modulating agent N-acetyl cysteine has been investigated in the treatment of cocaine dependence (LaRowe et al., 2007), nicotine dependence (Knackstedt et al., 2009), and gambling addiction (J. E. Grant, Kim, & Odlaug, 2007). The preliminary evidence for N-acetyl cysteine’s ability to reduce cravings across expressions of addiction has been positive, but there is a need for additional investigations into glutamatergic contributions to addiction and glutamatergic therapies for the treatment of addiction.

Stress Hormones
Abnormalities in the hypothalamic–pituitary–adrenocortical system have been implicated in a range of addictive behaviors. Acute intake of both nicotine and alcohol causes stresslike cortisol responses (Lovallo, 2006), and their persistent use might dysregulate the hypothalamic–pituitary–adrenocortical system. The risk for dependence and for relapse after quitting a pattern of addiction might be associated with deficient cortisol reactivity to stress. In addition, preexisting alterations in frontal–limbic interactions with the hypothalamic–pituitary–adrenocortical system can reflect addiction proneness, as shown in studies of offspring of alcohol- and drug-abusing parents (Lovallo, 2006; Sher, Cooper, Mann, & Oquendo, 2006).

The evidence from neurobiological research has suggested that both substances and behaviors (e.g., gambling) have the capacity to stimulate similar neurobiological systems. Disparate objects stimulating similar neurobiological pathways suggests at least some common overlap across addiction pathways and provides additional support for the syndrome model of addiction.

NEUROIMAGING
In addition to findings regarding the roles of specific neurotransmitters, another valuable line of research has sought to localize particular areas of the brain in which people with addiction have evidenced altered activation (for a review, please see Hyman & Malenka, 2001). Although imaging studies have been performed with multiple addictive behaviors, there is evidence of shared neurocircuitry among the various expressions of addiction.

Frontal cortical function influences higher order, executive functions (Tancredi, 2005) through cortical–striatal circuits (Robbins, 2007). Frontal lobe volume losses have been observed among individuals who are dependent on multiple drugs, suggesting the possibility of compromised executive functioning (Franklin et al., 2002; Pfefferbaum, Sullivan, Mathalon, & Lim, 1997). The orbitofrontal cortex and anterior cingulate gyrus appear particularly relevant to addiction because they are neuroanatomically connected to “reward-related” limbic areas. For people with addiction, these areas tend to be activated during the processes of intoxication, craving, and heavy episodic use (Goldstein & Volkow, 2002).

The brain regions implicated for those with addiction include regions that contribute to emotional and motivational processing, reward evaluation and decision making, and response inhibition. Relatively diminished activation of the ventral striatum has been observed in individuals with addiction, consistent with the findings on tasks involving reward processing (Pearlson & Calhoun, 2007; Reuter et al., 2005). Positron emission tomography studies have demonstrated reduced striatal dopamine D2 receptor binding in people with cocaine addiction, people who use heroin, and people with alcoholism (Volkow et al., 1995; Volkow, Fowler, & Wang, 1996; Wang et al., 1997).

The rush from drug use and the resultant cravings correspond to increased activity in the nucleus accumbens core, ventral tegmental area, caudate nuclei, cingulate cortex, and basal forebrain and decreased activity in the amygdala (Breiter et al., 1997). Ventral components of the prefrontal cortex, notably the orbitofrontal cortex, have been implicated in the processing of rewards (Knutson, Fong, Bennett, Adams, & Hommer, 2003; McClure, Laibson, Loewenstein, & Cohen, 2004; Schultz et al., 2000), and the lateral region activates when
additional information is needed to guide behavioral actions or when decision making involves the suppression of previously rewarded responses (Chamberlain & Sahakian, 2007; Elliott, Dolan, & Frith, 2000). In fact, drug cues can stimulate drug craving before conscious awareness of the cues (Childress et al., 2008). The failure of addicted subjects to activate certain brain regions during the early stages of response to cues that serve as triggers could contribute to poor self-control and subsequent drug use.

**Structural Findings for Specific Addictions**

Individuals with alcohol use disorders have demonstrated global cerebral changes. Scientists have observed decreases in gray and white matter volume among older drinkers compared with their younger counterparts (Paul et al., 2008; Pfefferbaum, Sullivan, Mathalon, & Lim, 1997; Torvik, Lindboe, & Rogde, 1982) as well as overall volumetric reductions in the reward neurocircuitry (Makris et al., 2008). Age-correlated CSF increases have also been noted among people with chronic alcoholism (Pfefferbaum et al., 1993), although after periods of abstinence, CSF reductions as well as volumetric white matter increases have been reported (Shear, Jernigan, & Butters, 1994). Research has observed a reduction in the volume of the corpus callosum among men and women with chronic alcoholism (Hommer et al., 1996; Pfefferbaum, Lim, Desmond, & Sullivan, 1996) as well as decreased D2 receptor availability in striatal areas (Heinz et al., 2004; Volkow, Wang, et al., 1996).

Structural changes associated with cocaine use disorders include decreases of gray matter in prefrontal, thalamic, and temporal areas (Franklin et al., 2002; Sim et al., 2007; Yuan et al., 2009) as well as an accelerated age-related decline of temporal lobe gray matter volume and smaller temporal lobe volumes than in normal control individuals (Bartzokis et al., 2000). Individuals with cocaine dependence have also demonstrated atypical anisotropy within the corpus callosum (Moeller et al., 2005) and orbitofrontal white matter (Lim, Choi, Pomara, Wolkin, & Rotrosen, 2002) using diffusion tensor imaging. Significantly more white matter hyperintensities have been found among cocaine-dependent individuals and, to a lesser but still significant degree, opiate users (Lyoo et al., 2004).

For opiate dependence, structural gray matter density deficits have been seen in the prefrontal, temporal, and cingulate cortices (Lyoo et al., 2006; Yuan et al., 2009). Significant ventricular and cortical volume loss (Pezawas et al., 1998) and changes in the rostral anterior cingulate cortex have also been demonstrated (Yuan et al., 2009).

Among male methamphetamine abusers, researchers have observed a greater occurrence of white matter hyperintensities (Bae et al., 2006). For both genders, severe gray matter decreases in the cingulate, limbic, and paralimbic cortices as well as decreased hippocampal volumes and significant white matter hypertrophy have been observed (Kim et al., 2006; Thompson et al., 2004).

Volumetric analysis with nicotine-addicted individuals has revealed reductions in gray matter volume and density (Gallinat et al., 2006). Increased amounts of nicotine receptors in the striatum have been correlated with smoking frequency (Paterson & Nordberg, 2000).

**Functional Findings in Specific Addictions**

Individuals with alcohol use disorders have demonstrated increased activation of the medial prefrontal cortex and striatum when presented with alcohol-related stimuli (Braus et al., 2001; Heinz et al., 2004; Tapert, Brown, Baratta, & Brown, 2004). Dopamine transporter quantity has also been associated with misuse of alcohol. Initially detoxified people with alcoholism have shown significantly decreased dopamine transporter presence that recovers over a period of abstinence (Laine et al., 1999). The interplay between dopamine transporters and D2 receptor quantity might be implicated in control and craving of alcohol use (Heinz et al., 2004).

Using positron emission tomography and functional MRI with cocaine-dependent individuals, scientists have demonstrated increased limbic system, anterior cingulate, dorsolateral prefrontal cortex, and dorsal striatum activity after presentation of a cocaine-related stimulus (Childress et al., 1999; Lim et al., 2002; Volkow et al., 2006). Positron emission tomography studies have also shown reduced dopamine transporter density and reduced dopamine D2
receptors in the striatum of subjects using methamphetamine. Decreased serotonin receptor levels and severity-dependent altered limbic and orbitofrontal activity have been shown for methamphetamine-dependent individuals (Chang, Alicata, Ernst, & Volkow, 2007).

Heroin-addicted individuals have evidenced reduced blood flow in frontal areas of the brain (Botelho et al., 2006) and altered prefrontal cortical activity (Yuan et al., 2009). Furthermore, animal model evidence has suggested that decision making related to opiate abuse involves circuitry in the limbic system (Frenois, Stinus, Di Blasi, Cador, & Le Moine, 2005).

Nicotine has been shown to modulate limbic system and prefrontal cortex activity (Stein et al., 1998; Zhang et al., 2009). Nicotine dependence has also been associated with altered D2 dopamine receptor availability in the striatum (Erblich, Lerman, Self, Diaz, & Bovbjerg, 2005; Fehr et al., 2008). During presentation of nicotine-related stimuli, nicotine-dependent individuals display heightened activation in the prefrontal cortex (McBride, Barrett, Kelly, Aw, & Dagher, 2006), ventral striatum, amygdala, orbitofrontal cortex, hippocampus, medial thalamus, left insula (Franklin, 2007; McClernon, Kozink, & Rose, 2008), and mesocorticollimbic system in general (Bühler et al., 2010). Smoking to enhance pleasurable relaxation has been associated with an increase in metabolic activity of the dorsal striatum (caudate, putamen), whereas smoking for calming effects has been associated with a decrease in thalamic activity (bilaterally) and with an increase in amygdala activity (Rose et al., 2007).

An increasing number of neuroimaging studies have been performed on subjects with pathological gambling. The ventromedial prefrontal cortex has been implicated as a critical component of decision-making circuitry in risk–reward assessment for pathological gambling (Bechara, 2003). Decreased activation of ventromedial prefrontal cortex has been observed among subjects with pathological gambling during presentation of gambling cues (Potenza, Steinberg, et al., 2003), performance of the Stroop Color–Word Interference Task (Potenza, Leung, et al., 2003), and simulated gambling (Reuter et al., 2005).

In a study using [18F]-deoxyglucose in positron emission tomography imaging, people with pathological gambling displayed significantly higher relative metabolic rates in the primary visual cortex, cingulate gyrus, putamen, and prefrontal cortex in the monetary condition than in the points-only condition, suggesting heightened sensory and limbic activation with increased valence or risk (Hollander et al., 2005). Other imaging studies have implicated brain regions involved in attentional processing for people with pathological gambling when they view gambling-related material (Crockford, Goodyear, Edwards, Quickfall, & el-Guebaly, 2005).

The issue of causation is difficult to address in human neuroimaging studies. First, in addition to regular drug use and abuse, we should note that the lifestyles and common nutritional deficiencies concurrent in various expressions of addiction likely play some role in the structural changes observed (Charness, 1993). Second, the imaging findings might reflect neural dysfunction that results from continued engagement in addiction or possibly dysfunction that predates addiction. Animal studies have allowed the opportunity to control exposure to drugs and to observe the influences of this exposure on the animal's neural circuitry (Tabakoff & Hoffman, 2000). In a study of nonhuman primates exposed to cocaine, Beveridge, Gill, Hanlon, and Porrino (2008) found neural changes in the ventromedial prefrontal cortex as well as the orbital and dorsolateral prefrontal cortices after prolonged drug exposure.

FAMILY HISTORY AND GENETICS

The development of addiction reflects a complex interaction between genes and environment. Low socioeconomic class, drug and alcohol availability, and poor social supports have all been associated with increased risk for addiction. Drug exposure during adolescence might also result in unique neuroadaptations that increase the reinforcement value of drugs (Adriani et al., 2003).

Researchers have estimated that 30% to 60% of addiction vulnerability is attributable to genetic factors (Kreek et al., 2005; Uhl & Grow, 2004). The genetic propensity for addiction appears two
pronged: There are contributions common across expressions of addiction, and specific expressions appear to be associated with particular genetic variants (Kreek et al., 2005). One can estimate the genetic versus environmental contributions to specific behaviors and disorders by comparing their concordance between identical (monozygotic) and fraternal ( dizygotic) twin pairs. In a study of male twins using the Vietnam Era Twin Registry, 12% to 20% of the genetic variation in risk for pathological gambling and 3% to 8% of the nonshared environmental variation in risk for pathological gambling was accounted for by risk for alcohol use disorders (Shah, Eisen, Xian, & Potenza, 2005). Two thirds (64%) of the co-occurrence between pathological gambling and alcohol use disorders was attributable to genes that influence both disorders, suggesting overlap in the genetically transmitted underpinnings of both conditions.

Family, twin, and adoption studies have demonstrated a substantial genetic contribution to the development of nicotine, alcohol, and drug addiction (with heritability estimates ranging from 50% to 60%; Bierut, 2011). Environmental factors may play a stronger role in the initiation of drug use, and genetic factors may contribute more to the transition from regular use to the development of addiction (Vink, Willemsen, & Boomsma, 2005).

One possible conceptualization of the genetics of addiction is that impulsivity, not addiction, is inherited. In a study by Ersche, Turton, Pradhan, Bullmore, and Robbins (2010), the siblings of stimulant abusers demonstrated increased impulsivity compared with control subjects. These findings suggest that impulsivity, not a specific addiction, might be inherited.

Only a few specific genes have been identified with polymorphisms that either predispose or protect a person from drug addiction. Specific alleles for genes that encode alcohol dehydrogenases seem to be protective against alcohol use disorders (Chen et al., 1999). Certain polymorphisms in the gene-encoding cytochrome P450 2A6 appear to be protective against nicotine addiction (Rao et al., 2000). Conversely, certain gene clusters may be associated with a high risk of nicotine dependence (Berrettini, 2008; Bierut et al., 2008), and certain GABA receptor genes appear to increase the risk for developing an alcohol use disorder (Dick et al., 2004). Although evidence has suggested that the D2 receptor is involved in addiction, particularly in relation to stimulant drugs (Le Foll, Gallo, Strat, Lu, & Gorwood, 2009), potential candidate genes regulating the dopamine or opioid systems have produced positive and negative results (Arias, Feinn, & Kranzler, 2006; Gelernter et al., 1991).

Although the genetic vulnerability to addiction likely represents the combination of hundreds of genes of modest effect (Bierut, 2011), genetic risk factors do not distinguish potential objects of addiction. Much of the research evidence has suggested genetic and environmental nonspecificity across a range of addictive behaviors (Shaffer et al., 2004).

**PSYCHOLOGICAL PROCESSES**

In addition to neurobiology, behavioral, cognitive, and dispositional attitudes can also play a role in the etiology of addictions. Behavioral and social learning theorists have focused on the role of direct and vicarious reinforcement in the development and maintenance of behaviors. Cognitive theorists have focused on information-processing biases that inflate subjective estimates of succeeding in various behaviors (e.g., getting high and being able to control use) or that otherwise promote addictive behavior persistence. Dispositional traits such as impulsiveness, sensation seeking, neuroticism, and extraversion as well as antisocial personality traits have also been postulated to be significant in the development of addictions. These psychological processes supplement the underlying neurobiology and might account for ways in which individuals express the addiction syndrome (Shaffer et al., 2004).

**Positive reinforcement** refers to the introduction of a hedonically positive consequence that strengthens a preceding response. There might be a range of reinforcers—other than the item of reward—available to people with addiction that might serve to initiate and perpetuate the behavior (e.g., Ocean & Smith, 1993). These reinforcers include social, material, ambient, and cognitive reinforcers as well as physiological arousal.
A negative reinforcement theory (i.e., involving the removal of a punishing stimulus) hypothesizes that initiating but not completing a habitual behavior leads to uncomfortable states of arousal. Note that this theory shares features with drive-reduction theory and could account for why an addiction continues despite continuous negative consequences. Another negative reinforcement-based model argues that addictions in general may allow individuals who are chronically either over- or underaroused to achieve optimal arousal level (Jacobs, 1986).

Another negative reinforcement model is based on the self-medication model. A number of studies have found elevated rates of depression and anxiety disorders in individuals with addictions. The lifetime prevalence rates for mood and anxiety disorders, depending on the addiction, range from 12.4% to 40.9% (Boschloo et al., 2011; Conway, Compton, Stinson, & Grant, 2006; B. F. Grant et al., 2004; Griesler, Hu, Schaffran, & Kandel, 2011; Lorains, Cowlishaw, & Thomas, 2011; Schuckit et al., 1997). Depressed or anxious individuals might engage in addictive behaviors to distract themselves from life stressors and unpleasant cognitions. Ironically, problems commonly resulting directly from addiction (e.g., financial distress, relationship problems, criminal activity) can, in turn, lead to even more variants of addiction expression as a misguided attempt at symptom management.

Certain personality characteristics can promote addiction. For example, being extremely extraverted, neurotic, or impulsive increases one's risk for addiction in general. One study found that individuals with multiple expressions of addiction were more impulsive than those with only one expression of addiction (Vitaro, Arseneault, & Tremblay, 1997). Individuals with multiple addiction expressions tend to share two common features: (a) being chronically over- or underaroused and (b) having had unfortunate experiences during childhood that led to feelings of inadequacy, rejection, or guilt (Jacobs, 1986).

Impulsiveness can be defined as acting with little forethought, self-control, or regard for consequences. Trait impulsiveness might increase the likelihood of addiction; research has shown that people with addictive behaviors score high on measures of impulsiveness (Castellani & Rugle, 1995; Dalley et al., 2007; Hayaki, Anderson, & Stein, 2006). Impulsive individuals might also be highly responsive to positive reinforcement but rather insensitive to punishment. Impulsive individuals often have to struggle to imagine negative outcomes. They have difficulty dividing attention among competing stimuli and thus might be insensitive to internally generated cognitions focusing on restraint (Abrams & Kushner, 2004). As such, the initiation of an addictive behavior can quickly lead to a loss of control.

Individuals high in sensation seeking search for novel, exciting experiences that can entail an element of physical or social risk. Although some question exists as to whether high levels of sensation seeking result in addictive behaviors or are an effect of drug use (Ersche, Turton, Pradhan, Bullmore, & Robbins, 2010), individuals high in sensation seeking might seek behaviors that provide excitement and produce high arousal but avoid others that do not.

One explanation for why all individuals who engage in addictive behaviors do not succumb to a substance use disorder could be individual differences in biological constraints surrounding reinforcement sensitivity. For some individuals, negative or positive reinforcement from an addictive behavior might have a more powerful influence on future behavior. Alternatively, some individuals might be more or less sensitive or responsive to the punishment associated with risks, losses, or social opprobrium related to the behavior. Searching for such individual-difference moderating variables can ultimately help to refine both psychological and biological understanding of operant processes that influence the etiology and maintenance of addictions.

COGNITIVE PROCESSES

Does disordered thinking cause individuals to make irrational decisions and, on the basis of these irrational decisions, engage pathologically in some form of addiction? By contrast, perhaps those who are pathologically driven to a behavior have cognitive vulnerabilities that make drug use or other
experience-shifting behavior appealing. Additionally, exposure to drugs of abuse can result in pathological cognitive processes that then further the likelihood of continuing drug use.

Supporting the causal status of irrational thinking in promoting problematic addictive behavior are data showing that treatments aimed primarily at changing inaccurate beliefs about the addictive behavior are effective in reducing the behavior (Aharonovich, Amrhein, Bisaga, Nunes, & Hasin, 2008; Carroll, Nich, Ball, McCance, & Rounsaville, 1998; McKee et al., 2007). Addiction-related cognitions provide a starting point in the search for a biologically grounded formulation of addictive behavior in which cognition plays a central role. For example, research has suggested that cognitive dysfunction might be an underlying factor that can influence the development and maintenance of various expressions of addiction (e.g., including substance and alcohol dependence and pathological gambling; Bates, Bowden, & Barry, 2002; Kalivas & Volkow, 2005). Past research has found cognitive dysfunction common across a variety of substance dependence domains (van der Plas, Crone, van den Wildenberg, Tranel, & Bechara, 2009; Vik, Cellucci, Jarchow, & Hedt, 2004), alcohol dependence (Bates et al., 2002; Green et al., 2010), and pathological gambling (Goudriaan, Oosterlaan, De Beurs, & Van Den Brink, 2006; van Holst, van den Brink, Veltman, & Goudriaan, 2010). In this section, we concentrate on the scientific literature concerning the cognitive functioning of individuals with addiction. Although the addiction syndrome model accounts for the shared cognitive dysfunction seen across multiple expressions of addiction, we focus on alcohol dependence, nicotine dependence, cocaine dependence, marijuana dependence, and pathological gambling because these expressions of addiction have the most supporting research; nonetheless, other expressions of addiction might have similar associated cognitive dysfunction.

**Alcohol**

Approximately 50% to 80% of people with alcohol dependence incur significant cognitive deficits that affect daily functioning (Bates et al., 2002; Rourke & Grant, 2009). Although some cognitive decline is reversible, it is not uncommon to see permanent impairment (Green et al., 2010). Alcohol dependence does not, however, affect all cognitive functions equally. Some research has suggested that alcohol dependence does not alter general intelligence, learned knowledge, and automatic processes (Eckardt & Martin, 1986; Evert & Oscar-Berman, 1995; Smith & Oscar-Berman, 1992). Nevertheless, alcohol-related cognitive impairment has been consistently noted across a variety of domains, including cognitive efficiency (Glenn & Parsons, 1991, 1992; Lawton-Craddock, Nixon, & Tivis, 2003; Nixon & Parsons, 1991; Nixon, Tivis, & Parsons, 1995), executive skills (Bechara et al., 2001; Ihara, Berrios, & London, 2000; Moselhy, Georgiou, & Kahn, 2001; Sullivan, Rosenbloom, & Pfefferbaum, 2000), learning and memory (Beatty, Hames, Blanco, Nixon, & Tivis, 1996; Brandt, Butters, Ryan, & Bayog, 1983; Di Sclafani et al., 1995; Fama, Pfefferbaum, & Sullivan, 2004; Munro, Saxton, & Butters, 2000; Rourke & Grant, 1999), inhibitory control (Abrons, Fillmore, & Marczinski, 2003; Abrons, Gottlob, & Fillmore, 2006; Bobova, Finn, Rickert, & Lucas, 2009; de Wit, Crean, & Richards, 2000; Marczinski, Abrons, Van Selst, & Fillmore, 2005; Weafer & Fillmore, 2008), spatial processing (Fein, Torres, Price, & Di Sclafani, 2006), processing speed (Alonso, Cardellach, Casademont, & Miro, 2004), visuospatial skills (Beatty et al., 1996; Munro et al., 2000; Sullivan et al., 2000), and working memory (Ratti et al., 1999). Alcohol intoxication has also been found to increase attention bias for alcohol-related cues (Duka & Townshend, 2004; Field & Cox, 2008; Schoenmakers, Wiers, & Field, 2008).

After a period of alcohol abstinence, people with a history of alcohol use disorders can recover some cognitive deficits. Some cognitive areas can recover as soon as 1 week after drinking stops, and others might improve over the course of several years (Bartels et al., 2007; Cocchi & Chiavarini, 1997; Mann, Günther, Stetter, & Ackermann, 1999; Munro et al., 2000; Reed, Grant, & Rourke, 1992; Rourke & Grant, 1999; Sullivan et al., 2000); still, some cognitive impairment can be permanent (Bates et al., 2002; Wegner, Günthner, & Fahlé, 2001).
Nicotine
Although controversial, acute nicotine use might enhance cognitive functioning (Azizian, Monterosso, O’Neill, & London, 2009; Heishman, 1998; Heishman, Kleykamp, & Singleton, 2010), such as memory, attention, and motor abilities (Bar et al., 2008; Foulds et al., 1996; Froeliger, Gilbert, & McClernon, 2009; Giessing, Thiel, Rösl, & Fink, 2006; Hahn et al., 2007; Holmes, Chenery, & Copland, 2008; Lawrence, Ross, & Stein, 2002; Meinke, Thiel, & Fink, 2006; Perkins et al., 1994, 2001, 2008; Sharma & Brody, 2009; Tucha & Lange, 2004). Moreover, in several large community samples, some research has found no to little association between chronic smoking and cognitive dysfunction (Schinka, Belanger, Mortimer, & Graves, 2003; Schinka et al., 2002).

Most research concerning the association between chronic cigarette smoking and cognitive dysfunction has used middle-aged and older adult participants in cross-sectional designs (Durazzo, Meyerhoff, & Nixon, 2010). For current, chronic smokers, research has identified cognitive dysfunction across several domains, including learning and memory (Ernst, Heishman, Spurgeon, & London, 2001; Heffernan et al., 2005; Hill, Nilsson, Nyberg, & Bäckman, 2003; Schinka et al., 2003; Spilich, June, & Renner, 1992), executive functions (Paul et al., 2006; Razani, Boone, Lesser, & Weiss, 2004), processing speed and cognitive flexibility (Kalmijn, van Boxtel, Verschuren, Jolles, & Launer, 2002; Starr, Deary, Fox, & Whalley, 2007), visual search speed (Richards, Jarvis, Thompson, & Wadsworth, 2003), and general intellectual abilities (Deary et al., 2003).

Nicotine’s effects on cognitive function are modified on the basis of the user’s age. Among adolescent and young adulthood population segments, smoking has been associated with deficits in working and auditory memory, vocabulary, and arithmetic (Fried, Watkinson, & Gray, 2006; Jacobsen et al., 2005). Associations have also been found linking smoking, cognitive dysfunction, and an overall increased decline of cognition among populations of older adults (Berkman et al., 1993; Collins, Sachs-Ericsson, Preacher, Sheffield, & Markides, 2009; Deary et al., 2003; Edelstein, Kritz-Silverstein, & Barette-Connor, 1998; Elias et al., 2000; Huang, Dong, Zhang, Wu, & Liu, 2009; Launer, Feskens, Kalmijn, & Kromhout, 1996; Yaffe et al., 2009).

Cocaine
Neurocognitive studies of cocaine use have demonstrated inconsistent results. Nevertheless, chronic cocaine use has been associated with a variety of neurocognitive deficits, such as visual, spatial, memory, perceptual–motor speed, mental flexibility, verbal generation, attention, and concentration. These findings are consistent with neuroimaging and neurochemical studies that associate anterior cingulate gyrus and orbitofrontal cortex dysfunction with cocaine use (Ardila, Rosselli, & Strumwasser, 1991; Cunha, Bechara, De Andrade, & Nicastri, 2011; Gil- len et al., 1998; Hester & Garavan, 2004; Hoff et al., 1996; Kjome et al., 2010; Mittenberg & Motta, 1993; Moeller et al., 2010; O’Malley, Adamse, Heaton, & Gawin, 1992; Rosselli, Ardila, Lubomski, Murray, & King, 2001; Smelson, Roy, Santana, & Engelhart, 1999).

However, alcohol use is often a confounding factor in attempts to understand the neurocognition of cocaine users, because the co-occurring use of alcohol and cocaine is common. Despite this, researchers have found that cognitive deficits still exist among cocaine users after accounting for alcohol use (Bolla, Funderburk, & Cadet, 2000; Brown, Seraganian, & Tremblay, 1994; Di Scalfani, Tolou-Shams, Price, & Fein, 2002; Robinson, Heaton, & O’Malley, 1999).

Cannabis
There is no overall consensus about the impact of cannabis on neuropsychological functioning (Hart et al., 2010; Iversen, 2003, 2005). Although some research has found that cannabis affects multiple cognitive areas, such as executive functioning, verbal recall, attention, motor control, memory, and cognitive flexibility (Almeida, Novaes, Bressan, & Lacerda, 2008; D’Souza et al., 2008; Hunault et al., 2009; Lamers, Bechara, Rizzo, & Ramaekers, 2006; Lane, Cherek, Tcheremissine, Lieving, & Pietras, 2005; Lane, Cherek, Tcheremissine, Steinberg, & Sharon, 2007; Makela et al., 2006; Ramaekers et al., 2006), others have found no such effect (D’Souza et al., 2004; Hart et al., 2010; Hart, Van Gorp,

Research with healthy individuals and cannabis users has found that the acute effects of cannabis often result in cognitive deficits, including impairments in memory, attention, inhibitory control, and decision making, and that these effects can dissipate as the drug is metabolized and expelled from the body (Fried, Watkinson, & Gray, 2005; Kalant, 2004; Lane et al., 2005; Ramaekers et al., 2006, 2009; Solowij & Pesa, 2010). Less certain is how chronic cannabis use affects neurocognitive functioning. Although some investigators have found no major cognitive dysfunction after a lengthy abstinence (Harrison, Gruber, Hudson, Huestis, & Yurgelun-Todd, 2002; Lyketsos, Garrett, Liang, & Anthony, 1999; Solowij, 1998; Solowij & Grenyer, 2001), others have found that chronic cannabis use is associated with memory, attention, inhibitory control, abstract reasoning, and executive planning deficits (Almeida et al., 2008; Block, Farnham, Braverman, Noyes, & Ghoneim, 1990; Block & Ghoneim, 1993; Bolla, Brown, Eldreth, Tate, & Cadet, 2002; Bolla, Rothman, & Cadet, 1999; Ehrenreich et al., 1999; I. Grant, Gonzalez, Carey, Natarajan, & Wolfson, 2003; Kempel, Lampe, Parnefjord, Hennig, & Kunert, 2000; Pope, 2003; Pope, Gruber, Hudson, Huestis, & Yurgelun-Todd, 2001, 2002; Solowij & Pesa, 2010; Whitlow et al., 2004).

**Pathological Gambling**

Gambling research has identified impaired performance on a variety of neurocognitive measures (van Holst et al., 2010). Pathological gamblers have displayed increased motor inhibition on a variety of tasks, including the go/no-go task, card-playing task, stop-signal task, circle tracing time, and Stroop Color–Word Interference Test (Fuentes, Tavares, Artes, & Gorenstein, 2006; Goudriaan, Oosterlaan, De Beurs, & Van Den Brink, 2004; Goudriaan et al., 2006; Kertzman et al., 2008; Odlaug, Chamberlain, Kim, Schreiber, & Grant, 2011; Roca et al., 2008). People with pathological gambling also evidence higher rates of delay discounting than those who do not gamble (Dixon, Marley, & Jacobs, 2003; MacKillop, Anderson, Castelda, Mattson, & Donovick, 2006; Madden, Francisco, Brewer, & Stein, 2011; Petry, 2001a). Using neuroimaging measures, subjects with problem gambling evidenced less ventrolateral prefrontal cortex activation during the Stroop Color–Word Interference Task than healthy control subjects (Potenza, Leung, et al., 2003). Researchers have also identified deficiencies for time estimation, cognitive flexibility, planning tasks, decision making, and worse memory performance (Goudriaan et al., 2006; Leiserson & Pihl, 2007; Odlaug, Chamberlain, Kim, Schreiber, & Grant, 2011; Roca et al., 2008).

One of the most commonly used gambling-related research measures is the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994); in this task, subjects choose cards from one of multiple card decks to win as many points as possible. Decks have different proportions of reward and penalty cards. Numerous studies have reported decision-making deficits for subjects with pathological gambling compared with control subjects; those with pathological gambling had particular problems of too often choosing the disadvantageous decks (Forbush et al., 2008; Goudriaan et al., 2006; Petry, 2001b; Roca et al., 2008). Collectively, these studies have suggested that people with pathological gambling can be characterized as having decreased executive functioning and increased impulsivity.

Overall, cognitive dysfunction has been identified as a correlate across a wide range of expressions of addiction; however, this observation does not imply that cognitive dysfunction directly causes addiction. These deficits may possibly be present among individuals at risk for addiction, be secondary to an expression of addiction, or emerge from a combination of these two circumstances.

**IMPULSIVITY AS A COGNITIVE ENDOPHENOTYPE FOR ADDICTION**

*Impulsivity* is the predisposition toward “rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others” (Moeller et al., 2001, p. 1784). Impulsivity is therefore a candidate endophenotype for a variety of
neuropsychiatric disorders, including addiction. Impulsivity has at least three distinct psychological processes: (a) motor disinhibition, (b) overzealous motivation to attain a goal, and (c) inadequate reflection or cognitive analysis (Dalley et al., 2011). These different facets of impulsivity might reflect altered functioning of different underlying neural systems.

Impulsivity is associated with many forms of drug abuse and behavioral expressions of addiction; it likely results from multiple dysfunctions in cortico-striatal pathways (Dalley et al., 2011). This circumstance suggests that impulsivity and addictive behaviors might have common underlying causes (Chambers, Taylor, & Potenza, 2003; Kreek et al., 2005) or that cognitive dysfunctions (e.g., impulsivity) might preexist drug addiction (Audrain-McGovern et al., 2009; Nigg et al., 2006). Conversely, impulsivity might be a consequence of drug addiction (Beveridge et al., 2008).

Impulsivity has been linked to many drug and behavioral expressions of addiction, as evidenced by steeper delayed discounting and impulsive responding on the stop–reaction-time test or go/no-go tasks; for example, with opiate (Kirby & Petry, 2004), cocaine (Fillmore & Rush, 2002; Kirby & Petry, 2004), alcohol (Noël et al., 2007; Vuchinich & Simpson, 1998), and amphetamine (Monterosso et al., 2007; Monterosso, Aron, Cordova, Xu, & London, 2005) use disorders as well as pathological gambling (van Holst et al., 2010) and cigarette smoking (Bickel, Odum, & Madden, 1999). The acute and chronic administration of drugs can produce different cognitive effects. One theory is that the chronic use of drugs causes neurotoxic effects on the top-down control regions such as the prefrontal cortex (Everitt & Robbins, 2005; Kalivas & Volkow, 2005). On a cognitive level, impulsivity represents a shared cognitive antecedent or risk factor to the development and maintenance of both substance and behavioral expressions of addiction.

CONCLUSION

Although significant advances have been made in the understanding of addiction during the past 2 decades, addictive behaviors remain a substantial public health concern. Understanding the neurobiology of addiction provides an exciting entry into the development of more effective treatments. In addition to building on and advancing researchers' understanding of addiction, research efforts need to use what is known about the neurobiology, genetics, and cognitive processes of addiction to guide and refine the selection of therapies.

Neurobiological, cognitive, and genetic data support a broad conceptualization of addiction, moving past an understanding of addiction as simply taking a substance into the body. Although different drugs and behaviors can manifest differently in terms of clinical symptoms, research has suggested that substance and behavioral expressions of addiction appear to share a common neurobiology. Addictions are not independent, with each reflecting a unique pathophysiology. Recognizing addiction as a syndrome can further the neurobiological and genetic knowledge of addiction by improving early diagnosis and more effectively targeting treatment options.

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Comorbidity refers to the co-occurrence of two disorders, and this concept is very relevant to the construct of addiction. The addiction syndrome approach suggests that addiction is a singular, monolithic phenomenon that can present as different co-occurring objects of excessive behavior (e.g., misuse of nicotine and alcohol; problematic gambling and misuse of cocaine). Under the rubric of the addiction syndrome, these disorders would probably not be considered comorbid. For example, one would not talk in terms of an addiction syndrome of nicotine being comorbid with an addiction syndrome of alcohol. Instead, the addiction syndrome model would suggest that there are multiple co-occurring expressions of addiction. However, under the current Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision; DSM-IV-TR; American Psychiatric Association, 2000), it would be reasonable to think of nicotine dependence as being comorbid with alcohol dependence. A very large body of research has demonstrated that if an individual is addicted to one substance (or excessive pattern of behavior), then he or she is at a dramatically higher risk for addiction to additional substances (and excessive patterns of behavior). A second type of comorbidity relevant to the addiction syndrome is the co-occurrence of the addiction syndrome with other forms of psychopathology, which is the focus of this chapter. Our primary focus is on empirical observations regarding the comorbidity between disorders that fit within the addiction syndrome. In this chapter, we also review other types of psychopathology and the phenomena or mechanisms that might be responsible for the observed co-occurrence.

MODELS OF COMORBIDITY

Researchers have attempted to explain the comorbidity of multifactorial disorders (i.e., disorders caused by many contributing factors) in a variety of ways. For example, Neale and Kendler (1995) proposed a series of models, such as alternate forms, in which two disorders share an underlying continuum of liability; multiformity, in which having one disorder abruptly increases risk for the second; three independent disorders, in which comorbid individuals actually have a different disorder from individuals who only have one or the other disorder; correlated liabilities, in which risk factors for the two disorders are correlated; and the direct causal model, in which liability for one disorder causes the other disorder.

Alternatively, Lyons, Tyrer, Gunderson, and Tohen (1997) described a series of heuristic models to address comorbidity. The Lyons et al. models divide psychopathological phenomena into three levels: (a) the etiological level (including genetic and environmental factors), (b) the pathophysiological level (including both biological and psychological mechanisms), and (c) the behavioral or phenotypic level (including observable clinical phenomena). The described models...
include the pleiotropy model, in which the two clinical phenotypes reflect the same underlying etiological and pathophysiological mechanisms. The symptom nonspecificity model describes a situation in which the etiological and pathophysiological factors that produce the symptoms of Disorder A also produce some of the symptoms of Disorder B and the etiological and pathophysiological factors that produce the symptoms of Disorder B also produce some of the symptoms of Disorder A. Therefore, the risk of each of the two disorders is increased when the other disorder is present. The risk factor model refers to a situation in which the presence of one disorder "causes" the other disorder to occur.

The models proposed by Neale and Kendler (1995) and Lyons et al. (1997) are two examples of multiple approaches used to conceptualize comorbidity. The comorbidity observed between the addiction syndrome and other forms of psychopathology very likely reflects a number of different mechanisms or models of comorbidity. For example, the relationship between disorders of addiction and internalizing disorders might be etiologically distinct from disorders of addiction and other externalizing disorders, which are explored in this chapter.

INTERNALIZING AND EXTERNALIZING PSYCHOPATHOLOGY AND ADDICTION

Researchers have suggested that two pathways lead to addiction (Cloninger, 1988; Cloninger, Bohman, & Sigvardsson, 1981; Sher & Trull, 1994; Zucker, 1994). One pathway is through negative emotionality, neuroticism, or internalizing psychopathology, and the second pathway is through acting out or deviant behavior or externalizing psychopathology. Krueger, Caspi, Moffitt, and Silva (1998) have consistently made important contributions to characterizing the psychometric properties underlying dimensions that can explain comorbidity of the most commonly occurring Axis I disorders. They identified two underlying dimensions of variation: internalizing and externalizing pathology. Internalizing disorders are characterized by a tendency toward an inward expression of distress, whereas externalizing disorders are characterized by an outward expression of distress. Krueger et al.'s model of internalizing disorders initially included unipolar mood (i.e., major depressive episode and dysthymia) and anxiety disorders (i.e., generalized anxiety disorder, agoraphobia, social phobia, simple phobia, and obsessive–compulsive disorder), whereas externalizing disorders included substance use disorders (SUDs; substance abuse and dependence) and antisocial behavior disorders (conduct disorder [CD] and antisocial personality disorder [ASPD]). More important, Krueger et al. conceptualized these two categories of psychopathology as independent dimensions, not opposite ends of the same spectrum. As such, the presence of externalizing pathology should not affect one's vulnerability for developing internalizing pathology (and vice versa). This conceptualization of psychopathology does not preclude the possibility that certain individuals might be at high risk for developing externalizing and internalizing pathology. In fact, a single-dimensional model of psychopathology, despite not being the best-fitting model, also represents an adequate model of comorbidity covariance (Krueger et al., 1998). The precise nature of psychiatric comorbidity continues to be a topic of debate within the field.

COMORBIDITY OF INTERNALIZING PATHOLOGY AND ADDICTION

Krueger et al. (1998) and others have characterized SUDs and internalizing disorders as falling on separate dimensions of psychopathology. However, research has suggested that there is extensive comorbidity between SUDs and unipolar depression and anxiety.

Epidemiology of Comorbidity of Internalizing Disorders

Two of the largest epidemiological studies including SUDs and depressive disorders, the National Comorbidity Study (NCS; Kessler et al., 1994, 1997) and the National Longitudinal Alcohol Epidemiologic Survey (NLEAES; B. F. Grant, 1995; B. F. Grant & Harford, 1995), reported that depression increases the odds ratio (OR) for being diagnosed with alcohol abuse (NCS OR = 0.9, NLEAES
OR = 1.7), alcohol dependence (NCS OR = 2.0, NLEAES OR = 3.8), drug abuse (NCS OR = 1.6, NLEAES OR = 3.3), and drug dependence (NCS OR = 2.0, NLEAES OR = 6.9). In additional, evidence from the NCS and the Epidemiological Catchment Area study (Eaton, Regier, Locke, & Taube, 1981) has suggested that when individuals are diagnosed with an alcohol use disorder and a depressive disorder, each disorder increases the severity of the other (.35 and .36 increase in alcoholic symptoms and .81 and .96 increase in depressive symptoms for the NCS and Epidemiological Catchment Area study, respectively).

Not only is depression often comorbid with SUDs, it also influences the degree and nature of substance abuse among users. In a 2009 meta-analysis, Conner, Pinquart, and Gamble found that among individuals suffering from alcohol use disorders, depression predicted both concurrent and future alcohol use and impairment, concurrent use and impairment as a result of additional substances, and an earlier age of onset for an alcohol use disorder. In another meta-analysis among cocaine users, Conner, Pinquart, and Holbrook (2008) found that depression was associated with concurrent cocaine, alcohol, and other illicit substance use or impairment but not future cocaine use or impairment.

In a similar meta-analysis of intravenous drug users, Conner, Pinquart, and Duberstein (2008) found that depression predicted concurrent drug use and impairment, alcohol use and impairment, and needle sharing.

Substance use and comorbid depression are also common among adolescents. Evidence from the Dunedin Birth Cohort Study (Anderson, Williams, McGee, & Silva, 1987; Feehan, McGee, Raja, & Williams, 1994; Krueger et al., 1998) showed that 28.2% of adolescents with an SUD concurrently met diagnostic criteria for depression. Results from the Methods for the Epidemiology of Child and Adolescent Mental Disorders study (Kandel et al., 1999) showed that among adolescents, having an SUD was associated with a 3.7 OR risk for having concurrent depression. Use of specific substances is associated with increased odds of having depression. Cannabis use (OR = 1.4; Christchurch Health and Development Study; Fergusson, Lynskey, & Horwood, 1996), alcohol use (OR = 2.1), nicotine use (OR = 1.4), and other illicit drug use (OR = 2.8; Methods for the Epidemiology of Child and Adolescent Mental Disorders; Goodman et al., 1998; Kandel et al., 1997) are all associated with increased risk for depression relative to adolescents who are not substance users.

The literature has also suggested extensive comorbidity between SUDs and anxiety disorders in adults. According to the International Consortium in Psychiatric Epidemiology (Merikangas et al., 1998), approximately 32% of individuals meeting diagnostic criteria for alcohol dependence and 45% of individuals meeting diagnostic criteria for drug dependence also met criteria for an anxiety disorder in their lifetime (note that these values are based on averages for reported data from Fresno, California; Germany; Mexico; the Netherlands; Ontario, Canada; and the United States). These comorbidity rates exceed those for mood disorders (26% for alcohol dependence and 35% for drug dependence). According to the National Epidemiologic Survey on Alcohol and Related Conditions (B. F. Grant, Stinson, Dawson, Chou, Dufour, et al., 2004), having a diagnosis of any anxiety disorder was associated with increased risk for having a diagnosis of any SUD (OR = 1.9), any substance dependence (OR = 2.8), any alcohol use disorder (OR = 1.7), alcohol dependence (OR = 2.6), any drug use disorder (OR = 2.8), any drug abuse (OR = 1.7), and any drug dependence (OR = 6.2). These odds, however, were generally lower than those associated with unipolar depressive disorders (see B. F. Grant, Stinson, Dawson, Chou, Ruan, & Pickering, 2004, for a full breakdown by SUD, specific mood disorder, and specific anxiety disorder).

Studies have suggested that the comorbidity between SUDs and anxiety among adolescents compared with adults is less robust. In their review, Armstrong and Costello (2002) concluded that although substance use is common among adolescents diagnosed with anxiety disorders, with prevalence rates ranging from 7% (Children in the Community Study; Brook, Cohen, & Brook, 1998; Cohen et al., 1993; Velez, Johnson, & Cohen, 1989) to 40% (Feehan et al., 1994) but
logical studies have demonstrated a stronger link between externalizing pathology and substance use problems than having an externalizing pathology and no anxiety disorder. Using data from the NCS Replication (NCS–R), researchers demonstrated that having an anxiety disorder might actually reduce the risk for developing an SUD among individuals with externalizing pathology. However, some evidence has suggested that among individuals without externalizing pathology, having a certain anxiety diagnosis (social phobia) is associated with 6.5 greater odds of developing cannabis dependence and 4.5 greater odds of alcohol dependence at 14-year follow-up (Buckner et al., 2008).

In addition to the comorbidity between internalizing disorders and SUDs, there is also some evidence for comorbidity between internalizing disorders and other expressions of addiction. Research on the emotional antecedents of compulsive shopping (Miltenberger et al., 2003) has suggested that many people experience feelings of anxiety and depression. In addition, McElroy, Keck, Pope, Smith, and Strakowski (1994) found that among 18 compulsive shoppers, 17 had one or more first-degree relatives with major depression, and three had relatives with an anxiety disorder. Whereas epidemiological research on compulsive shopping is limited, evidence from small-group studies of compulsive shoppers (reviewed by Black, 2007) showed that anywhere from 23% (Koran, Bullock, Hartston, Elliott, & D'Andrea, 2002) to 100% (Lejoyeux, Tassain, Solomon, & Adès, 1997) of individuals also met diagnostic criteria for a mood disorder and that 41% (McElroy et al., 1994) to 80% (Schlosser, Black, Repertinger, & Freet, 1994) met criteria for an anxiety disorder.

Evidence also exists of a link between pathological gambling and internalizing psychopathology. In a 5-year follow-up study of 101 individuals who had recently quit gambling, lifetime depression predicted a longer period of time necessary to establish a period of at least 3 consecutive months without relapse (Hodgins & el-Guebaly, 2010). According to results from the National Epidemiologic Survey on Alcohol and Related Conditions (Petrzak, Morasco, Blanco, Grant, & Petry, 2007), of older adults self-identified as recreational gamblers, 12.6% had a comorbid mood disorder and 15.0% had a comorbid anxiety disorder, compared with 11.0% and 11.6%, respectively, of individuals without a history of regular gambling. These values jumped dramatically to 39.5% and 34.5%, respectively, when examining people with pathological gambling (not simply individuals who gamble recreationally). These percentages were similar to those found when examining general prevalence (i.e., not exclusively for older adults) in the National Epidemiologic Survey on Alcohol and Related Conditions; 49.62% and 41.30% of pathological gamblers also met diagnostic criteria for mood and anxiety disorders, respectively (Petry, Stinson, & Grant, 2005). According to epidemiological findings from the NCS–R (Kessler et al., 2008), having a unipolar depressive disorder was associated with a 2.5 OR of having pathological gambling, whereas anxiety disorders had an associated OR of 3.1.

Evidence for comorbidity of internalizing disorders and other expressions of addiction, such as Internet addiction, binge eating, and sex addiction, is less compelling. Whereas many studies have demonstrated a link between Internet addiction and depression (Bernardi & Pallanti, 2009; Black, Belsare, & Schlosser, 1999; Ha et al., 2006; Kim et al., 2006; Ko, Yen, Chen, Yeh, & Yen, 2009; Kratt et al., 1998; Morrison & Gore, 2010; Yen, Ko, Yen, Wu, & Yang, 2007) as well as anxiety disorders (Bernardi & Pallanti, 2009; Ko et al., 2009; Yen et al., 2007)—particularly social phobia, generalized anxiety disorder, and obsessive–compulsive disorder—no epidemiological studies are available. Similarly, although many studies have associated
eating disorders, including the bingeing subtype of bulimia nervosa, with depression and anxiety (for reviews, see Godart et al., 2007, and Swinbourne & Touyz, 2007, respectively), the evidence does not unequivocally demonstrate a link between binge eating itself and internalizing disorders. Last, a review of the literature did not yield any clear relationships between sex addiction and internalizing disorders.

Across many expressions of addiction, the evidence for comorbidity with internalizing pathology is strong. The majority of research has focused on SUDs, which are significantly comorbid with internalizing pathology but typically not at the level of externalizing pathology. However, internalizing pathology is also consistently comorbid with other forms of addiction, including pathological gambling and compulsive shopping (and perhaps Internet addiction and binge eating to a lesser extent). The relationship between internalizing pathology and non–substance use expressions of addiction might be even stronger than it is for externalizing pathology. However, additional research, especially regarding Internet addiction, sex addiction, and binge eating (not simply the bingeing subtype of bulimia nervosa), is necessary to fully capture the nature of these comorbidities.

Models of Comorbidity of Internalizing Disorders

In this section, we consider some contributing factors to the comorbidity of internalizing disorders and addiction. Evidence from genetic studies has not strongly supported common factors for internalizing pathology and addiction. However, neuroimaging studies have suggested that some biological factors might underlie their comorbidity.

Additionally, evidence has also highlighted the contribution of personality and environmental factors in the explanation of the comorbidity of internalizing pathology and addiction.

Genetic factors. The evidence for genetic factors contributing to internalizing pathology and addiction has largely focused on SUDs and generally does not support common underlying genetic factors. Kendler, Prescott, Myers, and Neale (2003) found that one genetic factor contributes to phenotypic expression of internalizing disorders (major depression, generalized anxiety disorder, and specific phobia) and another contributes to externalizing pathology (alcohol dependence, drug abuse and dependence, adult antisocial behavior, CD). In addition, they found that alcohol dependence and drug abuse and dependence each had significant disorder-specific genetic factors. The findings of Low, Cui, and Merikangas's (2008) family aggregation study did not support familial transmission of alcoholism among individuals diagnosed with panic disorder or social phobia. There is, however, some evidence for a genetic factor contributing to internalizing pathology and SUDs. For example, Kendler et al. (1995) found some shared genetic factor contributing to major depression, panic disorder, generalized anxiety disorder, and alcoholism. In spite of this overlap, alcoholism had unique genetic variance (i.e., genetic influences not shared with the mood or anxiety disorders) of more than 75%. Similarly, Lin et al. (1996) demonstrated a significant relationship between depression and drug use disorders among monozygotic twins, even after controlling for confounding family factors, a technique that eliminated the significant association between depression and alcohol use in this sample.

Among addictive behaviors unrelated to substance use, the evidence for genetic overlap with internalizing pathology is sparse. To date, studies examining this relationship with compulsive shopping, sex addiction, and Internet addiction have not been completed. Although no research has specifically addressed the genetic overlap for internalizing pathology and binge eating, some researchers have examined its relationship with bulimia nervosa. Walters et al. (1992) demonstrated a genetic correlation of .46 for the heritability of depression and bulimia nervosa among 1,033 female twins. In addition, they found that for both depression and bulimia nervosa, approximately half of the phenotypic variance is explained by environmental factors, but that those factors are unique to each disorder. These findings supported those of Rowe, Pickles, Simonoff, Bulik, and Silberg (2002), who found significant genetic overlap (44%) between bulimia nervosa and internalizing pathology. It is important to keep in mind, however, that these studies did not demonstrate a clear link specifically between the addictive behavior of binge eating and internalizing pathology.
Last, researchers have directly studied the evidence for a genetic overlap between pathological gambling and internalizing pathology. For instance, Potenza, Xian, Shah, Scherrer, and Eisen (2005) demonstrated a significant overlap (34%) of genetic factors contributing to depression and pathological gambling among 7,869 male twins. In addition, Dannon, Lowengrub, Aizer, and Kotler (2006) found evidence for higher prevalence of depression and anxiety disorders in both pathological gamblers and their first-degree relatives, compared with control subjects. In sum, more research is necessary to explore the genetic overlap between non–substance use behavioral addiction and internalizing pathology.

**Neurological factors.** Models of comorbidity supported by neuroimaging studies are less common and deal almost exclusively with substance use and depression. For a review of potential neurobiological mechanisms that could explain the comorbidity of depression and substance use, see Rao (2006). These models typically involve explanations of how particular neurological–neurotransmitter systems affect or promote substance use. For example, the neurotransmission in the mesocorticolimbic dopamine reward system relates to the reinforcement of substance use, but depression is also associated with diminished mesostriatal and mesocorticolimbic dopamine function. Another model posits that depression is significantly influenced by serotonergic alterations, which also mediate the effects of substances that influence the dopamine reward system. Other explanations of comorbidity highlight a cholinergic hypersensitivity in depression and the effect of nicotinic receptors on cholinergic systems and feelings of depression. Finally, the changes in the limbic–hypothalamic–pituitary–adrenal system can lead to depression when triggered by stress. In substance use, this system mediates the effect of stress on drug seeking. Moreover, drug use influences neurotransmission in the amygdala, which activates the mesocorticolimbic dopamine pathways (Rao, 2006). In addition to these neurotransmitter models of addiction and depression, evidence has also suggested that marijuana use is associated with decreased cortical white matter during adolescence and that this decrease predicts the severity of depressive symptoms (Medina, Nagel, Park, McQueeney, & Tapert, 2007). To date, neuroimaging studies have not examined nonsubstance expressions of addiction and internalizing disorders.

**Psychosocial and personality factors.** In addition to biological models of comorbidity among addictive and internalizing pathology, there is some evidence of underlying psychosocial and personality factors influencing this relationship, at least for substance use. Evidence has suggested that comorbidity between substance use and depression is associated with self-medication (especially in men; Bolton, Robinson, & Sareen, 2009), impulsivity (Zilberman, Tavares, Hodgins, & el-Guebaly, 2007), psychosocial impairment and active involvement with boys (for young girls; King et al., 1996), older age and schoolwork problems (for young boys; King et al., 1996), and high neuroticism and poor social support (de Graaf, Bijl, Ten Have, Beekman, & Vollebergh, 2004). There is a paucity of research examining psychosocial and personality factors underlying the comorbidity between non–substance-use-related addictive behaviors and internalizing pathology.

Taken together, these studies demonstrate a contribution of many factors to the comorbidity of internalizing pathology and addiction. Whereas most research has focused on substance use, evidence exists for models of comorbidity that reflect the influence of genetics, neurotransmission, and psychosocial factors. Perhaps the most parsimonious explanation for this incomplete picture is that psychosocial and environmental factors interact with genetic predispositions toward internalizing and addictive pathology, influencing the neurobiology and neurochemistry of affected individuals. More research in this area, especially with respect to non–substance-use-related addictive behaviors, is necessary for a better understanding of the nature of such comorbidity.

**COMORBIDITY OF EXTERNALIZING PATHOLOGY AND ADDICTION**

Externalizing disorders, such as SUDs, CD, oppositional defiant disorder (ODD), attention-deficit/hyperactivity ...
disorder (ADHD), and ASPD, are characterized by disinhibited personality, manifested in low con-
straint and conscientiousness and elevated sensation seeking and impulsivity (Krueger & South, 2009).
The onset of externalizing disorders is typically in 
childhood or adolescence and the prevalence typi-
cally decreases with age, although many external-
ing disorders persist into adulthood.

Epidemiology of Comorbidity of 
Externalizing Disorders
Disorders of addiction are often comorbid with 
externalizing disorders. The majority of research 
has focused on SUDs, which are classified as a type 
of externalizing disorder. A meta-analysis of 4,930 
adolescents and 1,956 adults found that among 
substance-dependent individuals, 81.3% of those 
younger than age 15, 79.5% of those ages 15 to 17, 
66.6% of those ages 18 to 25, 45.5% of those ages 
26 to 39, and 44.0% of those age 40 and older had 
at least one externalizing problem within the past 
year (Chan, Dennis, & Funk, 2008). Among adoles-
cent smokers, individuals with an externalizing 
disorder consumed more tobacco in the first 2 years 
of smoking than those without an externalizing 
disorder (Aklin, Moolchan, Luckenbaugh, & Ernst, 
2009). According to the NCS–R, the prevalence of 
externalizing problems was associated with 
increased odds of having alcohol abuse (OR = 6.7), 
alcohol dependence (OR = 7.6), substance abuse 
(OR = 9.9), and substance dependence (OR = 13.1; 
Hofmann et al., 2009).

Numerous studies have found that CD very 
commonly co-occurs with SUDs (Clark et al., 
1997; Crowley & Riggs, 1995; Grilo, Becker, 
Fehon, Edell, & McGlashan, 1996). Chan et al.'s 
(2008) meta-analysis supported these findings; 
among adolescents with SUDs, 74.2% of individu-
als had met criteria for CD within the past year, 
which exceeded the prevalence rates of co-
occurring depression and other internalizing 
disorders in this sample. In a review of the comor-
bidity among substance use, abuse, and depen-
dence among adolescents, Armstrong and Costello 
(2002) also reported that CD and ODD (termed 
disruptive behavior disorders) were most commonly 
comorbid with SUDs, with a median OR of 4.0.

The median prevalence of disruptive behavior 
disorders among adolescents with substance 
dependence was 46%, whereas in the absence of 
any substance use, rates of disruptive behavior 
disorders were between 0% and 12%.

Longitudinal studies have found that the pre-
ience of CD significantly predicts later SUDs 
(Deykin, Levy, & Wells, 1987; Disney, Elkins, 
McGue, & Iacono, 1999; Lewinsohn, Hops, Roberts, 
Seeley, & Andrews, 1993; Wilson & Levin, 2001), 
although some findings have been mixed (Henry 
et al., 1993). One study demonstrated that a diagno-
sis of CD between ages 11 and 14 was a powerful 
predictor of SUDs by age 18 (OR = 4.27; Elkins, 
demonstrated that participants who met diagnostic 
criteria for CD or ODD at age 18 had higher concur-
rrent SUDs, more severe substance use, and a steeper 
trajectory of increasing substance use over time 
(from age 12 to age 18).

SUDs and ASPD are also often comorbid (B. F. 
Grant, Stinson, Dawson, Chou, Ruan, & Pickering, 
2004; Morgenstern, Langenbucher, Labouvie, & 
Miller, 1997). According to the DSM–IV–TR, ASPD 
is "a pervasive pattern of disregard for, and violation 
of, the rights of others that begins in childhood or 
early adolescence and continues into adulthood" 
Tobacco, alcohol, and cannabis use predicted higher 
rates of ASPD in young adulthood (Brook et al., 
1998), and the co-occurrence of ASPD and SUDs is 
associated with increased functional impairment 
and substance-related harm (Brooner, Schmidt, 

The comorbidity between ADHD and SUDs is 
also high (Clark et al., 1997; Crowley & Riggs, 
1995; Grilo et al., 1996), and research has suggested 
that the prevalence rate of ADHD among adoles-
cents with SUDs (63.6%) exceeds the prevalence 
rates of co-occurring internalizing disorders (Chan 
et al., 2008). Studies have found that between 17% 
and 45% have a history of alcohol abuse or depen-
dence and that 9% to 30% of adults with ADHD 
have a history of drug abuse or dependence (Wilens, 
2004). The prevalence of ADHD ranges from 35% to 
71% among those who abuse alcohol and from 15% 
to 25% in those who abuse substances (Wilens,
In a longitudinal study, Elkins et al. (2007) reported that hyperactivity–impulsivity symptoms predicted initiation of numerous types of substance use, even when controlling for CD. Inattention symptoms alone posed less risk, and a diagnosis of ADHD (as opposed to symptom counts) significantly predicted tobacco and illicit drug use only (Elkins et al., 2007). Furthermore, people with SUDs and ADHD had more severe SUDs, and adults with SUDs and ADHD had an earlier onset of SUDs than people without ADHD.

There is a paucity of research examining externalizing psychopathology and other types of addiction, such as binge eating, pathological gambling, and compulsive shopping. In terms of addiction to food and binge eating, one study reported that eating disturbances, including binging, purging, and restriction, were significantly associated with aggressive behavior in girls; participants who reported eating disturbances were two to four times more likely to demonstrate aggressive behavior (Thompson, Wonderlich, Crosby, & Mitchell, 1999). Furthermore, eating disturbances and aggressive behavior were significantly associated with both drug use and attempted suicide. One longitudinal study reported an association between ASPD and binging and obesity at age 33 (Johnson, Cohen, Kasen, & Brook, 2006). More recent research has highlighted a relationship between obesity and eating disorders, particularly binge eating and bulimia, and ADHD. For example, data from the Collaborative Psychiatric Epidemiology Surveys demonstrated that adult ADHD was associated with a greater likelihood of obesity (OR = 1.81; Pagoto et al., 2009). Another study found that adult ADHD symptoms were significantly higher among individuals with binge eating disorder and among obese individuals compared with normal-weight participants (Davis et al., 2009).

Although pathological gambling often co-occurs with externalizing disorders, studies have demonstrated that gambling problems tend to co-occur most often with internalizing disorders and other addictive behaviors, such as alcohol and drug use (Black & Moyer, 1998; Dannon et al., 2006; Winslow, Subramaniam, Qiu, & Lee, 2010). Because pathological gambling might be conceptualized as a disorder of impulse control, it is not surprising that studies have demonstrated a high prevalence of ADHD among individuals with gambling problems. One study reported that 20% of pathological gamblers met criteria for ADHD, and pathological gambling was also associated with higher rates of compulsive buying and compulsive sexual behavior (J. E. Grant & Kim, 2003; Specker, Carlson, Christenson, & Marcotte, 1995). Data from the National Epidemiologic Survey on Alcohol and Related Conditions suggest a high comorbidity between pathological gambling and fire setting (OR = 4.8), which reflects antisocial behavior and is associated with deficits in impulse control (Blanco et al., 2010). Pathological gambling is also associated with higher rates of ASPD (Black & Moyer, 1998).

Research on compulsive buying has also demonstrated a higher comorbidity with internalizing disorders compared with externalizing disorders (Mueller et al., 2010). Impulse control disorders are commonly associated with compulsive buying. Among patients with compulsive buying disorder, 21% had a comorbid impulse control disorder, most commonly intermittent explosive disorder (11%; Mueller et al., 2010). Studies have also found a strong association between compulsive buying and other forms of addiction, including exercise addiction (Lejoyeux, Avril, Richoux, Embouazza, & Nivoli, 2008), alcohol and drug use (Black, 2007; Roberts & Tanner, 2000), and sexual behavior (Roberts & Tanner, 2000).

Disorders of addiction, therefore, are highly comorbid with externalizing disorders. The majority of research has focused on SUDs, which have a stronger association with CD, ODD, and ADHD than do internalizing disorders. SUDs and ASPD are also highly comorbid. The literature has suggested an association between binge eating and externalizing pathology, including ADHD, ASPD, and aggressive behavior. Pathological gambling and compulsive buying seem to be less associated with the externalizing spectrum and more closely related to internalizing pathology, although these disorders are associated with measures of impulsivity and some measures of antisocial behavior. More research, however, is needed to understand the association between non–substance-related...
expressions of addiction and externalizing psychopathology.

Causes of Comorbidity of Externalizing Disorders

Understanding the nature of the relationships between addiction and comorbid externalizing psychopathology will help to elucidate the etiology, maintenance, and treatment of these disorders. Some evidence has suggested that the presence of an externalizing disorder might cause an addiction or make the manifestation of addiction more probable (Elkins et al., 2007). For example, individuals with ADHD might be more likely to develop nicotine dependence because nicotine improves attention (Whalen, Jamner, Henker, Gehricke, & King, 2003).

The impact of psychopathology on addiction, however, is very complex. An interesting finding by Hofman et al. (2009) was that anxiety disorders reduced the association between externalizing disorders and SUDs among participants in the NCS-R. Hofman et al. suggested that findings might reflect the fear of bodily symptoms among individuals with anxiety, which prevents individuals with externalizing problems from engaging in drug-seeking behaviors. Substance use or other types of addiction might also elicit the onset of other externalizing disorders (Wilson & Levin, 2001).

Numerous studies have consistently identified an externalizing spectrum of psychopathology, which might explain the comorbidity between disorders of addiction and other externalizing disorders. DSM-IV-TR externalizing disorders, including alcohol and drug dependence, CD, ADHD, and ASPD, form a single externalizing factor, which was clearly distinguished from a separate internalizing factor (Kendler, Davis, & Kessler, 1997; Krueger, 1999; Krueger et al., 1998; Krueger, Chentsova-Dutton, Markon, Goldberg, & Ormel, 2003; Vollebergh et al., 2001). The presence of a single externalizing factor might suggest that disorders of addiction and other externalizing disorders tend to co-occur as a result of common underlying vulnerability factors.

Twin studies have demonstrated that the externalizing spectrum is highly heritable. For example, genetic factors were responsible for 81% of the variation in individual differences in a single externalizing factor made up of antisocial behavior, CD, alcohol dependence, drug dependence, and unconstrained personality style (Krueger et al., 2002). Another study reported strong genetic contributions (85%) to an externalizing factor linking CD, substance use, ADHD, and novelty seeking (Young, Stallings, Corley, Krauter, & Hewitt, 2000). Furthermore, genetic influences on an externalizing factor were found to be separate from genetic influences on an internalizing factor (Kendler, Prescott, et al., 2003).

The syndrome model of addiction proposes that multiple expressions of addiction develop from a common etiology. As described in the introduction to this handbook, there are common genetic and molecular vulnerabilities for various forms of addiction, such as pathological gambling and alcohol dependence (Slutske et al., 2000) and drug addiction and compulsive running (Werme, Lindholm, Thoren, Franck, & Brene, 2002), and shared genetic and environmental risk factors for substance abuse are largely substance nonspecific (Kendler, Jacobson, Prescott, & Neale, 2003).

Consistent with the comorbidity model of addictive behaviors described earlier, genetic and environmental nonspecificity exists across externalizing pathology more generally, suggesting that various expressions of externalizing pathology might develop from shared vulnerability factors. For example, resemblance between parents and twin offspring is accounted for entirely by transmission of the general externalizing propensity as opposed to transmission of specific risks for specific outcomes (Hicks, Krueger, Iacono, McGue, & Patrick, 2004). Research has found a general vulnerability for externalizing disorders to reflect common underlying personality traits (Iacono, Malone, & McGue, 2008; Krueger et al., 1996; Slutske et al., 2002), deficits in cognitive functioning (Finn et al., 2009; Iacono et al., 2008), and neurobiological processes (Begleiter & Porjesz, 1999; Carlson, McLarnon, & Iacono, 2007; Frodl, 2010; Iacono, Carlson, Malone, & McGue, 2002; Yoon, Iacono, Malone, & McGue, 2006). Addictive behaviors share common symptoms and personality traits with other types of externalizing psychopathology, including impulsivity, novelty seeking, behavioral disinhibition, and...
negative emotionality or neuroticism (Krueger et al., 1996). In addition, deficits in reward processing that underlie disorders of addiction, as described throughout this handbook, have also been identified among individuals with externalizing disorders (Bjork, Chen, Smith, & Hommer, 2010), such as ADHD and CD (Gatzke-Kopp et al., 2009).

Numerous twin studies have examined the extent to which common genetic and environmental influences contribute to the co-occurrence of SUDs and other externalizing disorders. Many studies have reported a genetic correlation among alcohol and drug abuse or dependence and ASPD (Fu et al., 2002; Grove et al., 1990; Pickens, Svikis, McGue, & LaBuda, 1995). Findings regarding CD and alcohol and drug dependence are less clear; results from one study suggested common environmental influences, but not shared genes (True et al., 1999). Other studies have demonstrated that common genetic risk factors account for most of the correlation between these disorders (Slutske et al., 1998). Measures of social deviance, excitement seeking, and other disocial tendencies also share genetic liability with alcohol problems (Jang, Vernon, & Livesley, 2000; Mustanski, Viken, Kaprio, & Rose, 2003). More research is needed to examine the genetic correlation between other forms of addiction and externalizing disorders.

Research has demonstrated genetic and environmental nonspecificity across addictive behaviors (Betz, Mihalic, Pinto, & Raffa, 2000; Nestler, Barrot, & Self, 2001) and, more broadly, across externalizing psychopathology. Various forms of addiction share common etiologies with other externalizing disorders, with unique influences differentiating syndromes within the spectrum (Krueger, Markon, Patrick, & Iacono, 2005). The literature, therefore, supports a syndrome model of addiction that likely fits within an externalizing spectrum of psychopathology.

CONCLUSION

In this chapter, we have reviewed some of the extensive empirical findings demonstrating comorbidity between the addiction syndrome and other types of psychopathology. Ample evidence exists for an association between both internalizing and externalizing disorders and addictive disorders. The majority of research has focused on SUDs, which tend to co-occur more often with other externalizing disorders than with internalizing disorders. However, some expressions of addiction, such as pathological gambling and compulsive buying, might relate more closely to internalizing pathology.

We have addressed some of the putative models for explaining the observed comorbidity throughout this chapter, and research has suggested that a number of different mechanisms are responsible. Our own work with the Vietnam Era Twin Registry (VETR) over the past 20 years exemplifies several of the phenomena that might be responsible for the observed comorbidity.

Some of our research is most consistent with a model in which comorbid disorders share causal factors. We investigated the basis for the observed comorbidity between two disorders within the addiction syndrome, pathological gambling and alcohol dependence, using data from the VETR (Slutske et al., 2000). We found that risk for alcohol dependence accounts for a significant but modest proportion of the genetic (12%-20%) and environmental (3%-8%) risk for pathological gambling. In another study that used data from the VETR, we addressed the hypothesis that the observed comorbidity between pathological gambling and ASPD reflects a phenomenon in which being a pathological gambler causes the individual to engage in antisocial behavior, which qualifies him or her for a diagnosis of ASPD (Slutske et al., 2001). Our data suggest that rather than pathological gambling and ASPD having a causal relationship, the two disorders tend to co-occur because the same genetic factors impart a risk for both disorders. These results are consistent with numerous other studies described in the Comorbidity of Externalizing Pathology and Addiction section, demonstrating that common genetic influences largely account for the comorbidity between disorders of addiction and externalizing pathology.

Using data from our research with the VETR, we also investigated the mechanism responsible for the observed comorbidity between alcoholism and major depression (Lyons et al., 2006). The model
that provided the best fit to the data was one of reciprocal causation—that is, if an individual had major depression, he or she was at greater risk to develop alcoholism subsequently, and having alcoholism also predicted higher risk of subsequent major depression.

Some of our findings from research with the VETR are most consistent with self-medication as an explanation for the co-occurrence of addiction with other psychopathology. Specifically, we found that individuals with schizophrenia, as well as their unaffected cotwins, were more likely to be daily smokers and to complain of difficulty concentrating when they attempted to quit smoking (Lyons et al., 2002). This smoking behavior suggests that nicotine might be ameliorating some of the deficits in attention that are associated with genetic vulnerability for schizophrenia. Our findings also provided support for a phenomenon whereby the addiction syndrome itself increased the risk of subsequent development of psychopathology. Specifically, we found that individuals with nicotine dependence who were exposed to trauma had an increased risk of developing posttraumatic stress disorder compared with individuals without nicotine dependence who were exposed to trauma (Koenen et al., 2005).

Extensive research has documented very high rates of comorbidity between the addiction syndrome and numerous other forms of psychopathology, within both the externalizing spectrum and the internalizing spectrum. The use, misuse, abuse, and dependence on psychoactive substances were the manifestations of the addiction syndrome that were much more often the subject of empirical studies of comorbidity than other potential expressions of the addiction syndrome, such as gambling, shopping, and sexual behavior. Additional research addressing the comorbidity of these other expressions of addiction might also prove informative in terms of supporting (or not) combining them with expressions of addiction that involve the ingestion of psychoactive substances. Until the pathophysiology and neurobiology of these phenomena are better understood, patterns of relationships among them might be important sources of data to inform conclusions about what belongs in the addiction syndrome and what does not. Whereas considerable empirical work has described the relationship between the addiction syndrome and other psychopathology, far less work has explained the relationship. However, the extant work characterizing the mechanisms accounting for comorbidity has strongly indicated that it is multifactorial, that is, a number of different phenomena appear to influence comorbidity.

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Psychologists define self-regulation as the ability to override automatic tendencies and resist current temptations in favor of better outcomes in the future. Drug and alcohol addictions often involve an automatized pattern of behavior that comes with short-term benefits (e.g., pleasure, an escape from self-awareness) but long-term costs to one's health and relationships. In this chapter, we examine how self-regulation research can provide insight into how people develop, quit, and sometimes fail to quit addictive behaviors.

There is a common misperception that once someone is addicted, self-control becomes irrelevant. The individual has no ability to control his or her behavior. Many practitioners and some researchers have asserted that addiction is a disease just as is a physical ailment that cannot be treated with self-control alone. Perhaps the most famous version of this theory, at least in recent years, is Leshner's (1997, 1999) bold assertion that addiction is a brain disease. According to Leshner, use of mind-altering drugs begins as voluntary behavior, but at some point the brain changes so that continued use and abuse of drugs or other substances becomes involuntary.

The view of addiction as a physical disease might be an appealing theory for individuals with addiction (see Davis, 1997; Peele, 1998) and a lucrative concept for the medical community (Heyman, 2009; Russell, Davies, & Hunter, 2011). However, essentially nothing in the current scientific literature has established this view as correct. Leshner (1997, 1999) himself conceded that the idea of a switch in the brain that is thrown, thereby converting voluntary behavior into involuntary addiction, remains a metaphor. Other researchers have pointed out how inadequate the disease theory is to account for existing evidence (e.g., Heyman, 2009; Schaler, 2000). For example, it may be fine to assert that alcoholism is a disease akin to diabetes or cancer, but nobody copes with diabetes or cancer by attending meetings at which fellow sufferers encourage each other to stop having symptoms, whereas Alcoholics Anonymous is widely regarded as being at least somewhat effective in reducing alcoholism. Moreover, the evidence for brain changes associated with addiction has indicated that these changes occur in the reward centers of the brain, not the motor control centers; consequently, there is no sign that behavior becomes involuntary. Schaler (2000) has also pointed out that no known physical marker for addiction exists—for example, something an autopsy could identify that would distinguish a person with addiction from someone who simply consumed the same quantity of drugs without developing addiction.

Thus, although self-control is probably not that useful for dealing with genuine physical illnesses, it may be crucial in attempts to overcome addiction. Higher self-control predicts alcohol use in moderation (Cook, Young, Taylor, & Bedford, 1998) and less alcohol abuse (Peluso, Ricciardi, & Williams, 1998).
Self-regulation mediates the relationship between traits (activity level and mood) and tobacco, alcohol, and marijuana use. Impulsivity, a concept related to self-control, has been shown to be associated with the severity of cocaine use (Moeller et al., 2001). All addictive behaviors involve a failure of self-control, and poor self-control might be one of the underlying causes for all expressions of addiction. In this way, research focusing on self-control and addiction supports the syndrome model of addiction. A great deal of research in the self-control literature has suggested that self-control failure might play a major role in why people begin and continue with the variety of addiction expressions (e.g., substance misuse to excessive gambling).

**SELF-REGULATION**

According to Baumeister and Heatherton (1996), the three key components of self-regulation are standards, monitoring, and execution.

**Standards**

Self-regulation is not just random change but change guided by (and often toward) some goal. *Standards* are the goals that individuals are working toward when they self-regulate. It is easy to assume that if individuals want to avoid initiating drug and alcohol use or to quit using once they have started, then their standards are clear. However, people with addiction can run into a few problems with standards. If individuals are unclear or inconsistent about their standards, they might have difficulty executing self-regulation in pursuit of one standard (e.g., reducing substance use). Moreover, if individuals have multiple conflicting standards, self-control is much less likely to be successful.

Addicted individuals struggle with fluctuating commitment to their standards. Research by Sayette and Hufford (1997) studied people’s attitudes toward cigarettes while they were in high- and low-urge conditions. When individuals were in a high-urge state, they listed a greater number of positive things about cigarettes than when they were in a low-urge state. In another study, researchers had either abstaining or nonabstaining smokers evaluate the consequences of smoking while holding a lit cigarette (Sayette, Martin, Wertz, Shiffman, & Perrott, 2001). They found that participants who had been abstaining rated positive consequences of smoking to be slightly more probable than did participants who had not been abstaining. This research suggests that smoking may seem like a less risky, more appealing activity when people have been going without it. These fluctuations in individuals’ views toward smoking may make it more difficult to maintain consistent standards regarding the importance of quitting smoking.

In addition to fluctuations in their commitment, addicted individuals may struggle with conflicting standards. In fact, addiction can even be defined as a conflict over one’s attachment to a particular behavior (Orford, 2001). The desire to smoke or drink often conflicts with people’s other desires, such as the desire to live a long and healthy life. However, when an individual decides to quit, the desire to quit may also conflict with other goals he or she may have.

One such goal is the goal of social acceptance. The desire to form and maintain social relationships is a central part of individuals’ well-being (Baumeister & Leary, 1995), and alcohol and drugs are sometimes seen as ways to connect with others. Expected social benefits from drinking are associated with frequency of alcohol use (Brown, Goldman, & Christiansen, 1985; Cooper, 1994; Roehling & Goldman, 1987), and in a longitudinal study increases in alcohol use were predicted by the extent to which alcohol was expected to ease social interactions (Smith, Goldman, Greenbaum, & Christiansen, 1995). Participants assigned to write about a time when they were excluded self-reported a greater willingness to try cocaine than participants who were asked to write about a time when they experienced injury, but only if others were present when they tried the cocaine (Mead, Baumeister, Stillman, Rawn, & Vohs, 2011). In one study of smoking and social groups, students who wanted to be friends with a smoker were twice as likely to begin smoking as students who wanted to be friends with a nonsmoker (Aloise-Young, Graham, & Hansen, 1994). More impressive, these efforts were generally successful. Students outside the smoking-friend group who smoked a similar amount as their desired friend were more successful at solidifying the friendship.
than students who did not match their smoking to their desired friend's. Given that people expect social benefits from alcohol and cigarettes, and to some extent seem to garner social benefits from these behaviors, they may find that their goal of avoiding these substances is in conflict with their social goals. Trying to pursue both goals simultaneously may make one or both more likely to end in failure.

On the positive side, when an individual's social goals and goals to quit align, quitting is more likely to be successful. Christakis and Fowler (2008) found that people tend to quit smoking in groups. An individual becomes more likely to quit if his or her spouse, friend, or coworker quits. Although conflicts between one's social goals and one's goals to avoid drugs and alcohol may undermine self-regulation, having harmonious goals may make self-regulation even more likely to end in success.

As a side note, recent theoretical work has noted that self-control can be used to pursue as well as resist addictive behaviors (Rawn & Vohs, 2011). Individuals who do not find smoking intuitively appealing may use self-control to make themselves try cigarettes and alcohol to gain social relationships or social status. This is an important illustration of why standards are important to self-control. The ability to exercise self-control can only be as positive as the standards one is trying to regulate toward.

Another goal that might conflict with people's desire to avoid drugs and alcohol is the goal of emotion regulation. Individuals often strive to maintain positive moods and improve negative ones. Negative affect causes individuals to engage in impulsive behaviors when they believe those behaviors can improve their mood (Tice, Bratslavsky, & Baumeister, 2001). Research on smoking has shown that people smoke more when they are distressed (Ashton & Stepney, 1982; Schachter et al., 1977) and smokers feel better when they smoke (Gilbert & Spielberger, 1987). Alcohol is also viewed as a way to improve mood (Sayette, 1993; Stockwell, 1985). Although there is debate about the strength of mood effects on relapse, research has suggested that mood broadly plays some role in relapse (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Brownell, Marlatt, Lichtenstein, & Wilson, 1986). There is also some evidence that individual differences in emotional processing may predict people's likelihood of using alcohol as a means of mood improvement. Among people with intense negative emotions, individuals who are better able to differentiate their emotions use less alcohol than individuals who are less able to differentiate their emotions (Kashdan, Ferszidzis, Collins, & Muraven, 2010). In combination, this research suggests that people may use alcohol to improve a negative mood and that, in these moments, the goal of improving the mood often takes precedence over the goal of regulating their intake.

The use of alcohol and drugs to improve mood can also become cyclical. In one study, individuals who broke their self-imposed drinking limits were more likely to feel guilty the next day (even after controlling for actual amount consumed) than individuals who did not break their self-imposed drinking limits (Muraven, Collins, Morsheimer, Shiffman, & Paty, 2005a). That guilt, in turn, predicted more intake and more limit breaking in the future. In another study, participants who made more internal attributions for their drinking felt guiltier about drinking, which predicted more drinking in the future, than people who externalized responsibility for alcohol consumption (Muraven, Collins, Morshier, Shiffman, & Paty, 2005b). When people feel guilty about how much they have drunk, they may prioritize feeling better over holding to their standards.

**Monitoring**

The second component of self-control is monitoring. Monitoring involves keeping track of where one is in relation to the standard. It requires that an individual maintain awareness of the goal and his or her relationship to the goal (a sometimes unpleasant reality). Self-regulation can often break down when individuals stop keeping track of their progress or lose self-awareness. Some research has suggested that individuals are more likely to fail at self-regulation when they are faced with various distractions, such as social situations (Brownell et al., 1986). Recent research has shown that craving increases mind wandering (Sayette, Schooler, & Reichle, 2010). Smokers who were assigned to abstain from
smoking were both more likely to engage in mind wandering and less likely to notice that their mind had wandered, which suggests that smokers who are trying to quit may have the hardest time focusing on anything, including their goal to quit smoking. Attempts to quit smoking might be further impeded among people who drink alcohol and smoke (a common comorbidity; see Piasecki, McCarthy, Fiore, & Baker, 2008; Sayette, Martin, Wertz, Perrott, & Peters, 2005) because alcohol has been shown to reduce people's ability to monitor their performance (Sayette, Reichle, & Schooler, 2009). Moreover, some research has suggested that withdrawal increases people's attention to smoking-related information. In one study, participants going through withdrawal struggled more on a Stroop task of smoking-related words than participants who were smoking normally (Waters et al., 2003). Furthermore, poor performance on the smoking Stroop task predicted relapse, which suggests that when quitting, individuals have trouble thinking about things other than the forbidden substance. It may mean that these people would also have trouble keeping their minds on their smoking-related goals or their progress toward those goals.

Execution
The third and final component of self-regulation involves the capacity to bring about the desired change in one's behavior. Research on this final step has shown that acts of self-regulation draw on a limited resource (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Muraven, Tice, & Baumeister, 1998). Each act of self-regulation requires and depletes this resource. As a result, one act of self-control makes a successive act of self-control more likely to end in failure. For example, in one study, participants who were asked to resist a plate of cookies (which requires self-control for most people) subsequently spent less time persisting on a puzzle than participants who were asked to resist a plate of radishes (which one would expect requires less self-control than resisting cookies). The limited-resource model is supported by evidence that individuals who are trying to quit smoking while restricting eating are less likely to succeed than individuals who are not regulating another behavior (Mizes et al., 1998).

Underage social drinkers were more likely to violate their self-imposed drinking limit on days on which they experienced self-control demands, even when controlling for mood and urge to drink (Muraven, Collins, Shiffman, & Paty, 2005). The implication is that the capacity to change (akin to the folk concept of willpower) is limited, and if people use it for multiple tasks, there is less available for any particular one.

In a study of social drinkers, participants were given either a thought-suppression task or a list of arithmetic problems to solve (control condition; Muraven, Collins, & Nienhaus, 2002). They were then asked to taste test both alcoholic and nonalcoholic beer in the lab, keeping in mind that they would be doing a driving simulation later in the study. Consistent with the limited-resource model of self-control, participants in the thought-suppression condition achieved a higher blood alcohol level than participants in the control condition. The thought-suppression task had used up the self-control resources needed for participants to refrain from drinking. This main effect was qualified by an interaction between condition and temptation to drink. Among participants in the thought-suppression condition, participants who self-reported a larger temptation to drink on average achieved a higher blood alcohol level than participants who self-reported a lower temptation to drink, which suggests that having limited self-regulatory resources poses the greatest risk for individuals with the greatest temptation to engage in a potentially detrimental behavior.

Similar results have been found for smoking. In one study on smokers, participants were asked to refrain from eating either a plate of tempting sweets or a plate of vegetables (Shmueli & Prochaska, 2009). Researchers found that participants who had to refrain from eating the sweets were more likely to smoke during their break than participants who had to refrain from eating vegetables. These results suggest that individuals might have difficulty avoiding cigarettes and alcohol after experiencing other self-control demands.

The other implication of the limited-resource model is that any act that relies on self-control resources also consumes them, leaving less available for other activities. Individuals who are quitting smoking or drinking may have fewer self-regulatory resources to draw on.
resources available for other demands in their lives. A few studies have suggested this may be true. In one study, among individuals with a high temptation to drink, those who were assigned to smell alcohol performed worse on subsequent measures of self-control than those who were assigned to smell water (Muraven & Shmueli, 2006). In a study of chocolate lovers, participants high in chocolate craving who were assigned to abstain from eating chocolate showed impairment on working memory and reaction time (Kemps, Tiggeman, & Grigg, 2008). Overcoming a tempting stimulus may take self-control resources away from other activities.

Another important tenet of the limited-resource model is that individuals can conserve self-control resources for future demands (Muraven, Shmueli, & Burkley, 2006). In a series of studies, among individuals who had lowered self-control resources (because of a previous act of self-control), those who anticipated future demands on their self-control performed more poorly on a measure of self-control than those who did not anticipate future demands on their self-control. Moreover, those who conserved their self-control strength for the final task did better on the task than individuals who did not know about the final task. Thus, it seems that individuals are willing to make a trade-off between self-control performance now and self-control performance later. There might be times when individuals acquiesce to a relapse because they anticipate other self-control demands in the immediate future and do not want to use up all their self-regulatory resources on quitting. However, an individual who is quitting may deliberately cut back his or her self-regulation in other domains (i.e., procrastinate more at work, eat less healthily) to conserve resources that the individual needs to continue to avoid smoking or drinking. An important implication of this research is that an individual is very rarely, if ever, out of self-control resources. Instead, the individual's self-control may be low enough that it seems wise to conserve what remains in preparation for future demands.

In combination, this research has suggested that self-control demands can interfere with an individual's ability to quit and that quitting might interfere with a person's ability to engage in other kinds of self-regulation. Moreover, research focusing on conservation has suggested that individuals might relapse because they are saving their self-control resources for other anticipated self-control demands.

The limited-resource model provides additional information about a long-standing debate focused on whether individuals should quit multiple addictive behaviors simultaneously. Individuals who are addicted to one substance are often addicted to multiple substances, and whether individuals should attempt quitting both at once is unclear. Research has shown that between 88% and 90% of people with alcoholism smoke (Batel, Pessione, Maitre, & Rueff, 1995; Dreher & Fraser, 1967). An early review of the research concluded that treating co-occurring expressions of addiction simultaneously was effective, although most studies had not directly compared treating co-occurring expressions of addiction simultaneously with treating such expressions one at a time (Carroll & Malloy, 1977). Research since then has been inconclusive. In a study of individuals admitted to a substance abuse program, those admitted after a nonsmoking policy was implemented were no more likely to drop out of treatment than those admitted before the nonsmoking policy (Joseph, Nichol, Willenbring, Korn, & Lysaght, 1990). In addition, individuals admitted after the nonsmoking policy self-reported smoking less than individuals who were admitted before the nonsmoking policy. In this study, simultaneous regulation appeared to be effective (at least as self-reported).

A study of alcohol- and tobacco-dependent outpatients found different results (Stotts, Schmitz, & Grabowski, 2003). Researchers found that individuals who were high in both their motivation to quit smoking and their motivation to stop abusing alcohol dropped out of treatment earlier than individuals who were highly motivated only to stop abusing alcohol. These apparently conflicting results suggest that success in addressing multiple addictive behaviors might depend on the treatment context. In situations in which quitting one substance is externally enforced (e.g., an inpatient facility that bans smoking), quitting requires comparatively less self-control. In these cases, quitting multiple substances simultaneously may be no less effective than quitting one substance at a time. However, when individuals are
in less constrained environments, quitting multiple substances may require more self-regulation and therefore be less successful than quitting one substance at a time.

The good news, however, is that self-control practice over time can increase one's ability to engage in self-control (Oaten & Cheng, 2006, 2007). Individuals who were assigned to practice self-control (avoid sweets, hold a physically tiring handgrip for as long as possible each day) for 2 weeks before beginning a smoking cessation program went much longer before relapse than participants in control conditions (diary of times when they exerted self-control, math problems; Muraven, 2010). Practicing self-control before attempting to quit may be one way to increase the likelihood of success. In another study (O’Connell, Schwartz, & Shiffman, 2008), contrary to the authors’ predictions, the more often participants reported being tempted to smoke, the less likely they were to smoke, which suggests that practice at refraining from tempting behaviors can be beneficial in quitting.

Practicing and improving this self-regulation ability appears particularly important for substance users. Among people who use drugs and alcohol, self-control is a major predictor of life outcomes. Along with making individuals less likely to use and abuse potentially addictive substances, self-regulation can also reduce the negative effects of that use. One study found that self-regulation moderates the effect of drug, alcohol, and tobacco use on negative life outcomes. Among individuals who used drugs, alcohol, and tobacco, those with better self-regulation were less likely to self-report that these behaviors negatively affected their social life and school performance (Wills, Sandy, & Yaeger, 2002).

FREE WILL AND ADDICTION

Recently, self-regulation research has revived interest in the philosophical question of free will (e.g., Baumeister, 2008). People's perception of their own ability to freely choose and control their behaviors is a potentially fruitful area of future addiction research. Although there has been some debate regarding the amount of control people have over addiction, very little research has focused on how people’s beliefs about personal freedom affect their likelihood of forming and overcoming addiction. Burgeoning research about free will has shown that whether people believe in their ability to initiate and choose their own behaviors predicts a variety of behaviors, including aggression, helping, cheating, and counterfactual thinking (Baumeister, Masicampo, & DeWall, 2009; Vohs & Schooler, 2008). People's beliefs about free will might also predict addictive behaviors.

Research about free will has shown that determinism, or disbelief in free will, seems to increase the likelihood that people will give in to temptations around them. Individuals who were induced to believe in determinism were more likely to yield to a cheating opportunity and were more likely to take advantage of a chance to aggress against someone who had provoked them (Baumeister et al., 2009; Vohs & Schooler, 2008). This research suggests that in general people who believe they do not have control over their behavior will be more likely to give in to temptation. Future research may look into whether individuals who are highly tempted to drink or smoke are more likely to engage in these behaviors after having their belief in free will reduced.

Social influence is another way in which a belief in determinism may make people more vulnerable to the effects of drugs and alcohol. Research on free will and conformity has shown that people who are induced to believe in determinism are more likely to mimic others’ preferences when rating paintings than people who are induced to believe in free will (Alquist, Baumeister, & Tice, 2010). If others around them drink heavily, smoke, or try drugs, individuals who believe they cannot freely choose their behaviors may be more likely to follow the lead of those around them. However, if the norm of the group is to drink in moderation or to avoid drugs and alcohol entirely, individuals who believe in determinism may also be better at following those norms than individuals who believe in free will.

Individuals who believe in free will might also be better able to control their use of substances and use them in moderation. For example, those who believe that their behavior is within the bounds of
their control may have an easier time resisting alcohol the night before a big test or big date than individuals who believe it is outside of their control. Even if there are no differences in the amount of drugs and alcohol people consume on the basis of their belief in free will, there may be differences in the effects that usage has on their lives.

In some situations, inducing deterministic beliefs can reduce people’s drinking and smoking behaviors. Research has shown that individuals who are induced to believe in determinism feel less guilty about things they have done wrong than individuals who are induced to believe in free will (Stillman & Baumeister, 2008). As mentioned earlier, research has shown that the amount of guilt people feel for breaking their self-imposed drinking limit positively predicts the amount of alcohol they drink in the future (Muraven, Collins, Morsheimer, et al., 2005a). Individuals who excuse their excesses as being out of their control should feel less guilty, and as a result they should be less motivated to consume to make themselves feel better. Although individuals with a low belief in free will might generally be more likely to give in to drugs and alcohol, they might also feel guilty less often and thus use drugs or alcohol as a salve for guilt less often. That being said, if one never anticipates feeling guilty for excessive drinking or smoking, one may have no motivation to refrain from the behavior.

One other potentially positive effect on addiction of a belief in determinism involves situation selection. Individuals who believe they are largely products of their environment may be more inclined to choose their environment more carefully than individuals who believe their behaviors are less restrained. Research on the restraint bias has shown that overconfidence regarding one’s power to resist the situation can be detrimental. Among smokers who had recently quit, those who believed they had high impulse control were more likely to put themselves in tempting situations and were consequently more likely to relapse 4 months later than individuals who believed they had poor impulse control (Nordgren, van Herreveld, & van der Pligt, 2009).

In summary, a belief in free will likely has different effects on addiction depending on the situation. In general, a belief in free will should help people resist drugs and alcohol and the social pressure to engage in those behaviors. However, a belief in determinism may make people less likely to use alcohol and drugs when the addictive behaviors are motivated by guilt and might influence them to avoid tempting situations. This research on free will has suggested that people’s behavior is influenced by the amount of freedom they believe they have. Such evidence argues against the idea that addiction is outside of an individual’s control.

Along with free-will beliefs affecting people’s smoking and drinking behaviors, people’s behaviors may also affect their belief in free will. When people break their self-imposed drinking limits, or find themselves smoking after vowing to quit, they might begin to question their freedom to choose their own behaviors. Research focusing on free will should investigate the effect of addiction on people’s belief in free will. For example, researchers might find that people have reduced belief in free will on days when they have broken their self-imposed drinking limits or that smokers who have failed at quitting have a lower belief in free will than smokers who have never tried to quit. As a whole, research has shown that a belief in free will has largely positive effects. People who believe in free will perceive their life as more meaningful (Crescioni & Baumeister, 2010), perceive their career more positively (Stillman et al., 2010), and receive better job evaluations than people who do not believe in free will. As mentioned earlier, individuals who believe in free will also cheat less (Vohs & Schooler, 2008), help more, and aggress less (Baumeister et al., 2009) than individuals who do not believe in free will. It may be that failed attempts to quit cigarettes, for example, do more than harm people’s health. These failed attempts might also undermine their sense of personal freedom, making them more likely to engage in other self-destructive behaviors and reducing their overall sense of meaning in life.

CONCLUSION

Although it certainly is not the only underlying cause of addiction, self-control is one important predictor of addiction and recovery from addiction. Individuals facing conflicting goals or distractions
will have a harder time avoiding developing addiction than individuals with clear self-control goals and the ability to monitor their performance. Moreover, individuals with dispositionally low self-control or overly taxed self-control resources will be more vulnerable to addiction than individuals with strong self-control resources at their command.

Put another way, effective self-control might help buffer individuals from addiction. An individual with a high level of self-control might be exposed to objects of addiction without becoming addicted. Even more than attenuating the influence of exposure, effective self-control might even go so far as to help buffer people against genetic predispositions to addiction.

Research examining self-control practice has suggested that improving individuals' self-control can be one way to help already-addicted individuals overcome their addiction. Treatments that focus on self-control rather than a specific object of addiction hold the potential to keep recovering individuals from becoming addicted to other substances or behaviors. Poor self-control seems to be one antecedent of addiction, and improving self-control can be one way to buffer individuals against developing addiction.

References


At the time I write this, a prominent star golfer is in trouble because of what has been labeled a sexual addiction. He has been in treatment for this putative disorder at a “sexual addiction” clinic. Of course, sexual addiction is not a recognized disorder in any version of the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders* (DSM). When asked in an interview what was wrong with him, he did not use the term sexual addiction but referred to a failure to abide by his own moral values.

**ADDITION**

As pointed out by Shaffer (in the Introduction to this handbook), the term *addiction* was originally used in a broader sense than drug dependence. However, in the psychiatric nomenclature the term has been limited to substances. The first edition of the *DSM* (American Psychiatric Association, 1952) had a major category of addiction with two subcategories, alcoholism and drug addiction. The second edition of the *DSM* (American Psychiatric Association, 1968) included alcohol addiction and drug dependence, the distinction being that for some drugs dependence resulted in psychological rather than physiological withdrawal symptoms. It also included separate categories for the specific drugs (e.g., opiates, cocaine). Beginning with the third edition of the *DSM* (American Psychiatric Association, 1980), the term *addiction* was no longer used. Instead, substance abuse and dependence are distinguished from mere use and classified by drug, including alcohol.

*Substance abuse* refers to the use of alcohol or a specific drug despite adverse consequences in terms of impaired work, social relationships, legal punishment, or threats to health. Dependence includes, beyond abuse, the development of tolerance and withdrawal effects despite the recognition of adverse effects and the desire to quit or reduce use.

Shaffer (in the Introduction to this handbook) proposes a return to the broader definition of addiction, not restricted to substances but including the craving or compulsion in regard to the activity, loss of control, and continuation of the behavior despite adverse consequences. This definition could include sexual addiction, pathological gambling (now classified under impulse control disorders), and many other disorders of habit. The *DSM* classification is already considered to be too inclusive by many, and the advantage of new classifications under a broad addiction category is questionable.

However, the addiction syndrome approach raises questions that can be answered by research efforts. For instance, what is the evidence for an “addictive personality”? This question leads to several others. To what extent do the use of alcohol and different drugs covary within populations of users? To what extent does the use of substances in general correlate with other types of behavioral addictions such as gambling or seeking sexual variety? Is there a broader personality type associated with addictive behaviors as well as other behaviors involving pursuit of pleasure with little regard for the consequences? Sensation or novelty seeking has been suggested as a personality trait predisposing individuals to addictions.
of various sorts. Different personality types have also been suggested to underlie different motivations for alcohol or drug abuse.

RELATIONSHIPS AMONG RISK-TAKING BEHAVIORS AND PERSONALITY

Zuckerman and Kuhlman (2000) intercorrelated six kinds of risk taking in a sample of college students: smoking, drinking, drugs, sex, reckless driving, and gambling. Smoking, drinking, drugs, and sex were all significantly intercorrelated in men and women. The correlations were low to moderate (.23–.51). Risky driving correlated only with drinking in both genders. Gambling correlated with drinking and sex in men but with none of the other behaviors in women. Averaging across all six of the risk-taking tendencies, Zuckerman and Kuhlman constructed a general risk-taking score and compared high, middle, and low risk takers on five personality traits measured by the Zuckerman–Kuhlman Personality Questionnaire (ZKPQ; Zuckerman, 2002, 2008; Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993). The groups differed on three of the five scales. High risk takers were high on impulsive sensation seeking (ImpSS), aggression–hostility, and sociability in contrast to low risk takers. The medium group was generally intermediate on these traits. All three of these traits correlated with drinking, but only ImpSS correlated with extent of drug use in men and women. As will be seen, sensation seeking and aggression but not necessarily sociability distinguish many kinds of people who abuse alcohol and drugs. The presence of sociability (extraversion) in the college sample of risk takers might be the result of the social context in which college drinking tends to occur and related to social and disinhibition motives. Although some of the extreme drinkers and drug users might have been classified as abusers or even dependent abusers, the study did not allow for such diagnoses. To summarize, Zuckerman and Kuhlman’s study demonstrated not only that risky behaviors tend to co-occur, but that these behaviors are associated with stable personality traits.

Arnett (1996, 1998) used scales for risky driving, sex, drugs, and alcohol and found not only that nearly all of these behaviors were intercorrelated but that they also correlated with the Arnett (1994) Inventory of Sensation Seeking and a scale for aggression. Bradley and Wildman (2002) found moderate and significant correlations between substance use, reckless sex, reckless driving, and risky sports. Sensation seeking predicted total risky and reckless behaviors. Other studies have also found correlations among various types of risky behaviors, including smoking, drinking, drugs, and sex, and all of these behaviors related to sensation seeking (Lejuez et al., 2002; Wagner, 2001).

A 3-year longitudinal study of U.S. adolescents ranging in age from 15 to 18 used the Disinhibition (Dis) subscale of the Sensation Seeking Scale (SSS) with the items pertaining to drug or alcohol use removed (Bates, Labourie, & White, 1985). Alcohol and drug use were related to Dis at a given age, and changes in Dis were related to subsequent changes in level, quantity, and effects of alcohol and drug use.

Thus, there is evidence of a generalized tendency toward risk taking, going beyond substance use, and evidence that sensation seeking and aggression personality traits characterize these risk takers. However, what about people addicted to a certain drug? Do symptoms of different substance use disorders interrelate and relate to personality traits in the same way as risky behaviors?

RELATIONSHIPS AMONG SUBSTANCE USE DISORDERS AND PERSONALITY

Grekin, Sher, and Wood (2006) did actual dependence symptom counts for alcohol, drug, and tobacco use in a large longitudinal study of college students. Substance dependence symptom counts across four waves of data collection were significantly correlated among the three substances within and between times of assessment.

At different times in their lives, people who use drugs may use different drugs or alcohol with different effects. Lifetime prevalence rates reflect these changes and the overlap between substance use disorders. Among opiate-dependent abusers, 65% had at some time met diagnostic criteria for cocaine dependence, and another 12% met criteria for cocaine abuse (Brooner, King, Kidorf, Schmidt, &
Bigelow, 1997). Fifty percent had qualified at some point for alcohol dependence, and another 13% had qualified for alcohol abuse. Forty-five percent met criteria for sedative dependence; 13%, for sedative abuse. In other words, between half and three quarters of those with opioid disorder qualified for another type of substance use disorder. Among a different sample of people seeking treatment for either opiate or cocaine dependence, 35% of those with a current opioid disorder and 62% of those with a current cocaine disorder had an alcohol-related disorder sometime during their lives (Rounsaville et al., 1991).

It is interesting that even more of those dependent on a stimulant drug, cocaine, had abused alcohol, a depressant drug, than had those with opioid dependence. Those who simultaneously use both cocaine and alcohol may use them to regulate their state of arousal rather than seek only arousal or suppression. Another possibility is that both cocaine and alcohol stimulate the same dopamine reward centers in the limbic brain, whereas opiates have their primary reward effects on the opiate receptors. Either way, comorbid substance use disorders appear to be the norm, not the exception, among people who qualify for drug abuse or dependence.

The most frequent personality disorder diagnosed among people who abuse opiates is antisocial personality disorder (ASPD). Half of the opiate abusers in Brooner et al.’s (1997) sample were given this diagnosis without an exclusion for drug use, and a quarter of the sample were given this diagnosis with the exclusion. In the cocaine group in Rounsaville’s (1991) study, one third were diagnosed with ASPD without the exclusion, and about 8% were diagnosed with the exclusion. The rate in the normal population is only about 2%. What accounts for the high prevalence of ASPD, particularly in people who abuse opiates? Heroin and cocaine are expensive and often require commission of crimes in men and prostitution in women to support their addiction. Perhaps the personality type most willing to become involved in a drug lifestyle is that which qualifies for ASPD. It is also true that sensation seeking, impulsivity, and aggression are strong traits in ASPD, and individuals with these traits are highly risk tolerant in pursuit of the highs of substance abuse (Harpur, Hart, & Hare, 1994; Zuckerman, 1999).

Impulsivity as a major trait category is somewhat problematic because it is defined somewhat differently by different measures. Some scales distinguish functional from dysfunctional (neurotic–impulse control) impulsivity (Dickman, 1990), some have subscales for cognitive and motoric expressions (Barratt, 1959), and some distinguish reactive impulsivity from venturesomeness or sensation seeking (Eysenck & Eysenck, 1978). Allen, Moeller, Rhoades, and Cherek (1998) used all of these scales plus a behavioral test measure of impulsivity in a comparison of participants with and without histories of substance dependence. The group with a history of dependence scored significantly higher than the control group on functional and dysfunctional impulsivity and on Barratt’s (1959) Impulsivity and Eysenck and Eysenck’s (1978) Impulsivity and Venturesomeness scales. The substance-dependent group responded more quickly and impulsively in choosing between a small immediate reward and a delayed larger reward. This last task is a good model for the type of impulsivity that makes drug abusers choose the immediate gratification in a drink or drug in contrast to the delayed rewards of abstinence or moderation.

To this point, I have examined comorbidity between substance use, substance use disorders, and personality traits, but I have not distinguished among substances. One of the clearest of those distinctions is between alcohol and illicit drugs. Alcohol is usually the first drug that individuals use. Those who are willing to cross the line between this legal and available drug and illegal drugs need to be higher risk takers in the pursuit of sensation. Is there a fundamental personality difference between those who never go beyond heavy drinking and those who are willing to flout convention in the procurement and use of illegal drugs?

SUBSTANCE-SPECIFIC PERSONALITY PROFILES

Alcohol

The first concurrent studies of alcohol and drug use and sensation seeking in college students showed that the Dis scale correlated with extent of alcohol use in both men and women (Zuckerman, Bone,
Neary, Mangelsdorf, & Brustman, 1972) and that a much higher percentage of the heavier drinkers than of the low drinking group were high sensation seekers (53% vs. 13%; Zuckerman, Neary, & Brustman, 1970).

Since these first studies of the relationship between sensation seeking and drinking, many large-scale studies using college, community, and clinical populations in different countries have been done. Some of these have been described in books on sensation seeking (Zuckerman, 1979, 1994, 2007). Hittner and Swickert (2006) did a meta-analytic review of 61 studies of the relationship between sensation seeking and alcohol use. The overall effect size for the SSS total score (mean weighted $r = .26$) was small but highly significant, but there was heterogeneity among effect sizes according to the independent variables. The effect size was highest for the Dis subscale of the SSS ($r = .40$). This subscale contains a few items with content pertaining to drinking or drugs, so there is the possibility that the higher relationship of this scale to drinking is the result of a confounding effect. However, a comparison of the effect sizes in studies that removed these items with those that did not remove them did not show a significant difference in effect. Furthermore, studies using the Impulsive Sensation Seeking scale of the ZKPQ, a measure that does not have any items with substance use content, yielded an effect size (.36) as large as that for Dis in the SSS. Effect sizes were larger in men than in women and in White than in Black populations.

Subsequent to this meta-analysis, Carlson, Johnson, and Jacobs (2010) studied sensation seeking, impulsivity, and aggression in binge-drinking college students. Only the Thrill and Adventure Seeking and Boredom Susceptibility scales from the SSS were used to avoid the confounding items in the SSS referring to drinking or drug use. Impulsivity, verbal aggression, and the two sensation-seeking subscales were all significantly related to binge-drinking frequency.

Some of the concurrent personality correlates of alcohol excess or problems may be products of the alcoholism itself, so predictive studies in which personality is assessed before the onset of heavy drinking are of great value. Jones (1968, 1971) used personality data obtained from observer ratings when children were 10 years old to predict problem drinking when they were 30. Boys who developed problem drinking by 30 years old were described at 10 years old as rebellious, undercontrolled, hostile, manipulative, self-indulgent, sensuous, negativistic, expressive, assertive, talkative, and humorous in contrast to their peers who became abstainers or moderate drinkers. These differences persisted into high school and early adulthood. As children, the problem drinkers could be described as extraverted, impulsive sensation seekers. The girls who developed problem drinking had some of these same traits as children and adolescents, but they also had other traits suggestive of neuroticism, such as depression, distrustfulness, and self-negation, that differentiated them as well as the abstainers from moderate drinkers. They were submissive when young but rebellious as adults.

Another predictive study used behavioral observations of boys from age 11 to predict alcohol problems at age 27 (Cloninger, Sigvardsson, & Bohman, 1988). High novelty seeking and low harm avoidance were strongly predictive of later alcohol abuse.

Shorter term predictive studies from adolescence to early adulthood have found similar results. In a study conducted in Israel, sensation seeking at age 14 predicted use of wine, beer, and hard liquor at ages 15 to 18 (Teichman, Barnes, & Rahav, 1989). Trait anxiety was only related to use of depressant drugs.

Finn and Hall (2004) studied a sample of high-risk adult offspring of people with alcoholism and a control group without a family history for this disorder. They obtained a measure of excitement (sensation) seeking from two of the SSS subscales, Dis and Boredom Susceptibility, removing confounding items referring to drinking or drug use. They also assessed antisocial personality in the parents and social deviance in their offspring as well as alcohol use and problems. IQ in the offspring was measured. Structural equation models showed that parental alcoholism was related to both social deviance and sensation seeking, but that parental antisocial personality was related only to social deviance in their children. Social deviance was directly related to adult alcohol problems, but the relationship between sensation seeking and alcohol problems

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was mediated by alcohol use. IQ mediated alcohol abuse only indirectly through its negative relationship with social deviance. The study showed that although sensation seeking is modestly related to social deviance, the effects of each in alcohol abuse should be considered separately.

Loukas, Krull, Chassin, and Carle (2000) used the NEO Personality Inventory (NEO-PI) to study the effects of parental alcoholism and personality traits on alcohol abuse or dependence in a high-risk sample composed of children with at least one parent with alcoholism. Both self-reports and parental descriptions of their children on the NEO-PI were used to assess children's personality. Assessments of drinking problems and personality were made when the children were of young adult age (18–26 years).

For both self- and parental descriptions of personality, three of the five NEO-PI traits were related to alcoholism in young adults: high neuroticism, low agreeableness, and low conscientiousness. The strongest effect of personality was for agreeableness. Low scores on agreeableness could be regarded as similar to the high scores on aggression found in previous studies. Extraversion and openness to experience showed little or no relationship to alcoholism. Agreeableness and neuroticism showed some mediation effects having a stronger relationship to alcoholism in children of parents with alcoholism than in those whose parents did not have alcoholism.

**Stimulants**

All addictive drugs provide an immediate euphoric effect through the release of dopamine in the reward areas of the ventral striatum, but the stimulant cocaine provides an immediate and intense experience, or high, perhaps because of the favored method of self-administration, inhalation. Whether nasally ingested or smoked as crack cocaine, the pathway to the brain is fast and intense. However, the personality associated with cocaine abuse may vary as such as that for alcohol.

Ball (2002) reported the relationships between NEO-PI, Temperament and Character Inventory, and ZKPQ scales and characteristics of addiction in a broad sample of inpatient and outpatient drug abusers. In the NEO-PI, dependence severity and polydrug use were positively related to Neuroticism (N) and negatively related to Agreeableness (A) and Conscientiousness (C). In the Temperament and Character Inventory, these two variables were positively related to Novelty Seeking (NS) and Harm Avoidance and negatively related to Self-Directiveness and Cooperativeness. In the ZKPQ, they were related to ImpSS. In addition, addiction severity was related positively to Neuroticism–Anxiety (N-Anx) and Aggression–Hostility (Agg–Host) as well as ImpSS. The results are fairly consistent across scales. ImpSS and NS are highly related measures of sensation seeking, and both are positively related to dependence severity, as is neuroticism in the form of N, Harm Avoidance, and N-Anx. Aggression in the form of low NEO-PI Agreeableness and Temperament and Character Inventory Cooperativeness as well as high scores on ZKPQ Agg–Host are also related to dependence severity. Thus, the personality profile of individuals with addiction severity is consistent across methods of assessment. This should not be surprising given the high rates of lifetime comorbidity of the various drug disorders and as indicated by the characteristic of polydrug use among the personality-related disorders. In regard to neuroticism, it should be noted that nearly all of these studies involved participants entering or in treatment. The high neuroticism scores may be more reflective of their current stressful pressures than of a basic personality trait preceding the development of addiction.

A group of prostitutes practicing their work in a particularly high-risk environment were higher on ImpSS, Agg–Host, and N-Anx from the ZKPQ than a control group of women, but only ImpSS and Agg–Host were significant when age and education were controlled (O'Sullivan, Zuckerman, & Kraft, 1996). Those prostitutes who used cocaine or were polydrug users scored higher on ImpSS than those who did not use drugs or used only one drug other than cocaine. In a study of pregnant cocaine abusers, Ball and Schottenfeld (1997) found that both ImpSS and Agg–Host were related to a history of violence, whereas N-Anx was related to anxiety and depression. Those with a history of prostitution, exchanging sex for drugs, and many sex partners were higher than others on ImpSS, Agg–Host, and N-Anx. Adding drugs to sexual risk taking is characteristic.
of those women higher in this triad of personality traits. The interaction between personality, sex, and drugs was found in a study comparing methamphetamine-dependent gay men and gay men who did not use this stimulant drug (Solomon, Kiang, Halkitis, Moeller, & Pappas, 2010). The methamphetamine users were higher on NEO-PI N and Openness and lower on A and C than the nonuser gay men. Kalichman, Heckman, and Kelly (1996) found that drug use mediated the relationship between sensation seeking and unprotected anal intercourse among gay men, a high-risk behavior for AIDS infection.

Heterosexual men and women who used methamphetamine were studied in terms of their impulsivity trait and drug and sexual behaviors (Semple, Zians, Grant, & Patterson, 2005). Users high in impulsivity consumed more methamphetamine, binged on the drug, were more depressed, reported more sexual partners, and engaged in more casual sex and unprotected oral and vaginal sex.

**Opiates**

Carter et al. (2001) gave the NEO-PI—revised (NEO-PI-R) to 230 opioid-dependent outpatients. Patients were tested on entry into treatment and about 5 months later after treatment (pharmacological or behavioral). There were no control groups, but NEO-PI-R scores were interpreted in terms of mean T scores on the basis of the normal population norms. High scores were defined as higher than 55 and low scores as lower than 45. In these terms, the mean score on N was very high (about 1 standard deviation above the population mean). The opioid group means on A and C were low, particularly on C, which was more than 1 standard deviation below the population mean. Extraversion and Openness (O) means were low but still in the defined low-normal range. After treatment, the mean N was significantly reduced and C was significantly increased, although neither change moved N or C into the defined normal range. A was not changed. The profile for opioid users resembles that found for stimulant users. However, a study comparing people who abused alcohol, cocaine, and multiple substances on the ZKPQ found that people who used multiple drugs scored higher than the group with alcoholism on the ImpSS and higher than the cocaine group on N-Anx (Patkar et al., 2004).

**Alcohol Use Versus Marijuana Use Versus Other Drug Use**

Segal, Huba, and Singer (1980) did a large-scale study of substance use in a college population and in another population of naval personnel. They divided both populations into four groups: nonusers of either alcohol or drugs, alcohol-only users, marijuana users (although alcohol users were not excluded), and polydrug users. The SSS Form IV (Zuckerman, 1971) was used along with other personality scales from the Jackson (1974) Personality Research Form. All items referring to drinking or drugs were removed from the SSS to avoid confounding with the substance use criteria. Significant differences between groups were found on all of the SSS subscales, but the strongest differences were on the Experience Seeking (ES) and Dis subscales. Little discriminative value in multivariate analyses was found on the other personality scales beyond ES and Dis.

Significant increments on ES and Dis among the college student groups separated nonusers of either class of substances (abstainers) from the alcohol-only group and separated the alcohol-only group from the marijuana users, but only small and insignificant increments separated marijuana users from polydrug users. Among the naval personnel, there were differences between abstainers and alcohol-only users similar to those in the college sample, but the other major difference in ES and Dis was between marijuana users and polydrug users; alcohol-only users and marijuana users did not differ significantly from each other. Similar differences on ES and Dis were found between nonusers of drugs, marijuana-only users, and polydrug users in another college population (Galizio, Rosenthal, & Stein, 1983).

Grekin et al. (2006) studied college students, measuring personality at entry to college, and alcohol, tobacco, and drug dependence symptoms during freshman and sophomore years. Personality measures included the short forms of the NEO-PI and the Tridimensional Personality Questionnaire, a measure that includes the NS subscale (a high correlate of sensation seeking scales). Extraversion and low openness to experience predicted alcohol symptomatology, whereas low conscientiousness predicted drug-dependent symptoms. Novelty seeking
and, to a lower degree, neuroticism predicted both alcohol and drug symptoms.

Sher, Baartholow, and Wood (2000) followed a sample of college students for a period of 7 years, measuring alcohol and drug use and dependence. During Year 1, they also administered the Tridimensional Personality Questionnaire and Eysenck Personality Questionnaire. The Eysenck Personality Questionnaire Psychoticism (P) and Tridimensional Personality Questionnaire NS were the most highly predictive scales for later substance abuse. High baseline scores on P were predictive of later alcohol dependence, and NS predicted drug disorders when baseline diagnosis was statistically controlled. The P score has been claimed to be better characterized as a measure of psychopathy and aggression than of psychoticism (Zuckerman, 1989). As previously noted, NS is highly correlated with ImpSS (Zuckerman & Cloninger, 1996).

A community sample consisting of parents of twins (McGue, Slutske, & Iacono, 1999) was classified by interview into four groups: (a) those with neither alcohol nor drug problems, (b) those with an alcohol disorder but not a drug disorder, (c) those with a drug disorder but not an alcohol disorder, and (d) those with both alcohol and drug disorders. The Multidimensional Personality Questionnaire (Tellegen & Waller, 2008) was used as the personality test. The test has 11 subscales grouped into three major factors comparable to Eysenck’s Big Three: Negative Emotionality (like N), Positive Emotionality (PE; like Extraversion [E]), and Constraint (like P reversed). The Negative Emotionality factor was primarily related to alcohol use disorders, and low scores on constraint were primarily related to drug use disorders. Neither disorder was related to PE. However, two PE subscales, High Social Potency and Low Achievement, were associated with drug use disorders. Low control and low harm avoidance were also specifically associated with drug use disorder, with or without accompanying alcohol use disorder.

Chassin, Flora, and King (2004) used an at-risk sample of children with at least one parent with alcoholism and followed them from age 13 to ages 20 and 25, measuring alcohol and drug use and dependence trajectories. The participants were divided into four consumption trajectories using latent class analysis: (a) abstaining, (b) light drinking and rare drug use, (c) moderate drinking and experimental drug use, and (d) heavy drinking and heavy drug use. The four groups were compared on parental reports of their children’s temperament traits of impulsivity and emotionality (neuroticism) at early adolescence and the participants’ scores on the NEO-PI at adult ages. Significant differences were mostly found between Group 4 and the other groups. The heavy drinking and drug use group was rated higher on impulsivity at early adolescence but did not differ from the other groups on emotionality. However, at adult ages they scored higher on neuroticism. Neuroticism was not part of their temperament before they reached adult age, but impulsivity was already apparent. They also scored significantly higher on NEO-PI O and lower on A and C than the abstainers and lighter and moderate substance use groups. These same analyses were repeated, generating five diagnostic groups: (a) no disorders, (b) alcohol only, (c) drug only, (d) comorbid, and (e) persistent. Adolescents scoring high on impulsivity and, to a lesser extent, emotionality were more likely to belong to the drug use disorder and comorbid groups than to the no-disorder group. These same differences were found for adult neuroticism; in addition, the drug use disorder and comorbid groups scored higher on neuroticism than the alcohol-only group, which scored higher than the no-disorder group. Similar patterns were observed for O, A, and C: high O, low A, and low C scores distinguished individuals with disorders from those without. The comorbid group also scored lower than the alcohol-only group on A, and the drug group also scored lower than the alcohol-only group on C.

College student and community samples may include some alcohol and drug users who have crossed the line from use to abuse, but the results on specificity of the Substance Use x Personality relationship may not be as apparent in those already diagnosed with substance abuse or dependence and those in treatment. Most of those who enter treatment do not do so because they have the sudden insight that they are drinking too much or taking too many drugs. They have usually encountered
Substance-Specific Personality Profiles in Treatment-Seeking Samples

Kilpatrick, Sutker, and Smith (1976) studied groups of young veterans from substance abuse and general medical wards of a Veterans Administration hospital. They classified subjects by primary substance disorder into four groups: (a) nonusers of either alcohol or drugs, (b) occasional users of either or both (the largest group), (c) problem drinkers (people with alcoholism), and (d) regular drug users or polydrug users who use both stimulant and depressant drugs. Problem drinkers, regular drug users, and occasional users all scored higher than nonusers on the SSS and all of its subscales. The regular drug users also scored higher than the occasional users on all subscales, but the problem drinkers only scored higher than occasional users on one subscale: Boredom Susceptibility. Both people with problem drinking and those who abused drugs scored higher on anxiety and neuroticism scales than those who did not use and those who used occasionally. However, these elevations in anxiety and neuroticism in alcohol and drug samples entering treatment are probably not a predisposing trait for most of them. These elevations are found less often in younger early users or in users in a prison population (Skolnick & Zuckerman, 1979), and in a treatment population these scores tend to be high initially but fall to normal levels in those persisting in treatment (Zuckerman, Sola, Masterson, & Angelone, 1975).

Adams et al. (2003) compared adolescents who abused substances, currently in treatment in a residential treatment center, with controls from the area. Those scoring low on NS tended to prefer alcohol and marijuana, whereas a higher proportion of the high-NS group tended to progress to stronger drugs. Among users of hard drugs, NS scores were higher for those who used stimulants than for those who preferred sedative drugs such as opiates or narcotics. However, most studies of personality and preferred drugs within drug populations have shown that sensation seeking is more strongly related to number of drugs used than to any specific class of drugs (Zuckerman, 1994, 2007). This makes sense in that the novelty motivation in sensation seeking is likely to manifest itself in the search for new kinds of sensation or experience as well as in the intensity of those experiences shown in the development of a demand for stronger dosages (to counteract the weakened high produced by habituation). This is the basis for tolerance, one of the major components of dependence.

Polysubstance users score higher than monosubstance users on impulsivity as well as sensation seeking and risk taking (Martinotti et al., 2009). They also score higher on aggression, the Eysenck Personality Questionnaire P scale, criminal histories, and violent behaviors during incarceration. Similar findings have been obtained in a study comparing prisoners with a history of substance abuse with those without a record of substance abuse: Higher aggression, impulsivity, convictions, and violence during detention distinguished the substance users (Cuomo, Sarchiapone, Di Giannantonio, Mancini, & Roy, 2008).

Jeffrey Gray developed a neuropsychological theory of personality with reinforcement sensitivity as the primary construct mediating the neuropsychological basis for personality and its trait expressions in behavior (Corr, 2008; Zuckerman, 2011). Essentially, the theory suggests that a major determinant of individual differences is the sensitivities to cues associated with reward and punishment. People sensitized to reward tend to be impulsive in the presence of cues promising reward or pleasure, whereas people sensitive to cues for punishment tend to be more fearful and inhibited in the presence of such cues. For many years, the theory was developed largely from experimental studies of rats. Research with humans was limited because unlike top-down theories developed around personality trait ratings and questionnaires, this bottom-up theory had no direct personality measures to define individual differences among humans. More recently, investigators have developed scales that more directly
attempt to assess behavior approach and inhibition in terms of self-reported behavior. One of these is Carver and White's (1994) Behavioral Inhibition System/Behavioral Activation System (BIS/BAS) questionnaire. The test contains one scale for inhibition (BIS) and three subscales for approach or activation (BAS): (a) BAS Drive is a putative measure of the persistent and energetic pursuit of rewards, (b) BAS Reward Responsiveness is the strength of the positive arousal of reward, and (c) BAS Fun Seeking is the desire for reward in new sensations and experiences. Fun Seeking strongly resembles sensation seeking even at the item level. It is highly correlated with Cloninger's (1987b) NS scale. The BIS is highly correlated with measures of anxiety and neuroticism.

Franken, Muris, and Georgieva (2006) compared patients with alcohol use disorders and patients with drug use disorders from two substance abuse inpatient treatment centers with healthy control subjects from the community in the Netherlands. The people with drug use disorders scored significantly higher than the control subjects on BAS Drive, BAS Fun Seeking, and BAS total score, whereas the people with alcohol use disorders were intermediate between control subjects and the drug use disorder group but not significantly different from either on these scales. There were no significant group differences on the BIS or the BAS Reward Responsiveness scales.

Associations Between Personality and Substance Use: Summary

There is evidence for some form of an addictive personality in the sense of relationships between different kinds of substance use, abuse, or dependence. This vulnerability in turn shows a relationship to a type of personality characterized by sensation seeking, weak conscientiousness and constraint, impulsivity, aggression, and antisocial tendencies. These traits are stronger in those who use hard drugs and have heavy alcohol use. Neuroticism and negative emotions, such as anxiety and depression, are less consistently associated with drinking and drug use and may depend on gender, age, and context. Context also affects other substance-personality relationships: For example, heavy drinking in a college setting is related to sociability because most of it occurs in social settings. However, motivations may mediate the relationships between personality and substance abuse. If this is true, different personality types may be involved in drinking or taking drugs for different reasons. I explore this possibility in more depth in the following sections of the chapter.

PERSONALITY DIFFERENCES WITHIN SUBSTANCE USERS

Cloninger (1987a) described two types of alcoholism differing on personality, motivation, age of onset, and behavioral characteristics. Type 1 is found in both men and women, with a later age of onset (after 25 years), strong dependence with a loss of control of drinking once started, and guilt and fear about the dependence. Type 2 has an early onset, is found primarily in men, and is characterized by an inability to abstain and in fact little desire to abstain. Type 2 is characterized by a lack of guilt, accompanied by fighting and arrests when drinking. Type 1 is low in novelty and sensation seeking and high in harm avoidance and reward dependence, whereas Type 2 is high in sensation seeking but low on the other two traits. In a broad sense, Type 1 is anxious and dependent, whereas Type 2 is sensation seeking and antisocial.

Cloninger's (1987a) distinction between two types of alcoholism implies a difference in motivation. The person with Type 1 alcoholism is more neurotic and depressed and presumably drinks to self-medicate or reduce negative feelings. The person with Type 2 alcoholism is more sensation seeking and somewhat antisocial and presumably drinks for the pleasure of disinhibition.

Ball, Carroll, Babor, and Rounsaville (1995) developed a two-factor typology for cocaine abuse resembling the one developed by Cloninger (1987a) for alcoholism. Their participants were diagnosed for cocaine abuse or dependence with heroin dependence excluded. The researchers' Type B classification, which included one third of the sample, was comparable to Cloninger's Type 2 alcoholism. Compared with Type A, Type B included those with a younger age of onset of drug abuse and higher scores on measures of family history of substance abuse, childhood symptoms, sensation seeking,
antisocial behavior, addiction-related social impairment, depression, higher addiction severity, and polydrug use. Type Bs scored higher on all sensation-seeking subscales and had a history of aggression, criminality, violence, and impairment of social adjustment. Type A included two thirds of the participants and was characterized by a later age of onset and lower scores on the other variables characterizing Type B. A subsequent study using participants with drug disorders, including alcohol, cocaine, marijuana, and opiates, showed that the Type A-Type B distinction was applicable in all types of substance disorders (Feingold, Ball, Kranzler, & Rounsaville, 1996).

PERSONALITY AND MOTIVATIONS FOR SUBSTANCE USE AND ABUSE

The motives for alcohol or drug abuse may vary with age and experience. The initial overuse of alcohol and drugs may represent a hedonistic tendency, reinforced by the disinhibitory effects of alcohol and drugs and a need for variety and novelty. The later phases of abuse may be motivated by the stress and problems produced by the substance abuse or other life problems. Drugs are used to suppress negative emotions or provide an escape from stress and boredom. The drugs may be a substitute for other possible behavioral coping mechanisms. In other words, some people start using drugs to increase pleasure but end up using them to avoid pain. The question is the reliability of drug and drinking motivations over time. Developmental studies of the person who abuses substances and his or her personality and motivational characteristics in childhood or early adolescence before the substance dependence in later adolescence or adulthood are needed to address this question.

Personality and Motivations for Drinking

During the past decade, addiction researchers have emphasized that different people drink for different reasons and that the relation between personality and drinking may be mediated by motivations for drinking. Cooper (1994) developed a four-factor model of drinking motives that has been widely used in subsequent research. The model was developed from interviews and self-ratings of a large, representative community sample of adolescents (13–19 years old). Factor analysis suggested four factors:

1. Social: fun in social settings such as parties or social celebrations;
2. Coping: reduction of anxiety and depression and worry about problems;
3. Enhancement: increase in positive feelings and pleasure—getting high, fun, and excitement; and
4. Conformity: fitting in with a drinking group and being accepted and liked by them.

The factors are hardly independent. Social and Enhancement factors correlate very highly, and Social and Coping and Enhancement and Coping correlate moderately. Conformity shows somewhat lower but still significant correlations with the other three motives. Older adolescents report more drinking for Social, Coping, and Enhancement motives, whereas younger adolescents say they drink more for Conformity motives. Both Coping and Enhancement are related to heavy drinking and drinking problems, but Enhancement is relatively more related to heavy drinking, whereas Coping is more associated with drinking problems.

One must note that the participants were adolescents, and most of those having problems were likely in the abuse rather than the dependence stage of alcoholism. Drinking motives might become different in later adult life when reduction of the problems engendered by alcoholism itself become the major provocation for drinking. Kuntsche, Knibble, Gimel, and Engels (2006) noted a developmental trend in the differentiation of drinking motives from one general drinking motive in childhood and adolescence to three or more later in adolescence and early adulthood. However, the drinking motives of those with advanced alcoholism might differ by context: They might drink when they are happy and when they are sad or when celebrating a positive event or mourning a negative event. They might drink to enhance positive affect when in happy situations and to reduce negative affect when in bad or stressful situations.

Summarizing the drinking motives’ relationships with personality, Kuntsche et al. (2006) described those who drink primarily for enhancement motives
as extraverted, impulsive, aggressive, and high sensation seekers. They have weak inhibitory control and low responsibility. In Cloninger's (1987a) classification, they would be Type 2 drinkers. Those who drink to cope with negative emotions tend to be neurotic and less agreeable and have low self-esteem and would be Type 1 in Cloninger's typology. Social or conformity motives for drinking relate less to personality because they are more context relevant and less stable over time.

Loukas et al. (2000) compared personality as assessed by the NEO-PI scales to drinking motivations as measured by Cooper's (1994) scale without the conformity measure. Subjects were adolescents with at least one biological and custodial parent diagnosed as having alcoholism. E and O were not related to alcohol use disorders and therefore were not used in the motivational analyses. N was positively related to coping, social, and enhancement motives, but when the other motives were statistically controlled, only the relationship with coping remained significant. A was negatively related to coping motives, as was C. C was also negatively related to enhancement motives.

Similar results were obtained in studies of nonselected college students using the NEO-PI and drinking motive scales (Kuntsche, von Ficher, & Gimel, 2008; Stewart & Devine, 2000; Stewart, Loughlin, & Rhums, 2001). In the study by Stewart et al. (2001), multiple regression analyses showed that N was related to coping motives and that coping motives partially mediated the relationship between high N and increased drinking problems. Enhancement motives primarily mediated the relation between low C and increased drinking quantity. In the Stewart and Devine (2000) study, N predicted coping and conformity motives, whereas E correlated with enhancement and social motives. Among the facets of E, gregariousness primarily accounted for social motives and excitement (sensation) seeking for the relationship to enhancement motives for drinking.

Kuntsche et al. (2008) had a very large sample of more than 2,000 Swiss college students. They used structural equation modeling to assess the relationships between three of the Big Five traits, enhancement and coping motives, and drinking quantity and frequency. C was very highly and negatively related to enhancement motives, and both were related to drinking quantity and frequency (C negatively and enhancement motives positively). C was also related negatively to coping motives, although not as highly; coping motives were in turn positively related to drinking quantity and frequency. N was highly positively related to coping motivation and E to enhancement motives; however, in contrast to other studies, the direct relationship of N to drinking indices was negative and low but significant. E, in contrast to the other two personality traits, had no direct relationship with drinking quantity and frequency.

Magid, MacLean, and Colder (2007) used path analysis to describe the relationships between sensation seeking and impulsivity, drinking motives, and alcohol use and problems. An indirect path led from sensation seeking to enhancement motives and then to extent of alcohol use. Impulsivity had both direct and indirect paths to alcohol problems. The indirect path led through coping motives to alcohol problems. A similar pathway from sensation seeking to drinking for enhancement to alcohol use was found in another adolescent sample (Cooper, Frone, Russell, & Mudar, 1995).

Colder and O'Connor (2002) used a laboratory test in addition to self-report measures of disinhibition. The laboratory test was a go/no-go task (Newman & Kosson, 1986) that involves responding or not responding to signals of reward or punishment. Responding to signals of punishment is an error of commission, and fast reaction times are considered signs of disinhibition. Errors of commission and lack of self-reported inhibitory control (impulsivity) were associated with enhancement motives for drinking but not with social or coping motives. The effect of alcohol on disinhibition in high and low sensation seekers (ImpSS scale extreme scorers) was studied in an experiment by Fillmore, Ostling, Martin, and Kelly (2009). They used a laboratory go/no-go task similar to that used in the Colder and O'Connor (2002) study. The high sensation seekers showed a poorer degree of impulse control on the task that was further impaired by alcohol. They also showed more sensitivity to the subjective positive-feeling effects of alcohol.

Sensation seekers who develop problem drinking do so through their propensity to look for enhanced
pleasure in drinking leading to excessive use and eventually abuse or dependence in some. Impulsive individuals have a problem with self-control that can lead directly to abuse of alcohol or indirectly through the use of alcohol in coping with the problems incurred by their impulsive behavior.

Anxiety is a major component of N and as with N should be expected to be related to the coping motivation for drinking. Highly anxious people may use alcohol as their drug of choice in self-medication for anxiety and depression. McNally (1996) defined a trait of anxiety sensitivity (AS) as fear of the symptoms of anxiety themselves in contrast to trait anxiety, which is fear of potential external stressors such as social rejection. AS is related to panic disorder and its symptoms, whereas ordinary trait anxiety is more related to generalized anxiety disorder characterized by worry. Because alcohol directly suppresses the autonomic symptoms associated with panic attacks such as accelerated heart rate, one might expect AS to be associated with the coping motive.

Stewart and Zeitlin (1995) used tests of AS and trait anxiety in relation to scales for drinking motivation in college students. Both AS and trait anxiety were correlated with the coping motivation for drinking but not with social or enhancement motives. However, the AS scale accounted for a much higher proportion of the variance in the multiple regression. A later study using a younger group of adolescents found that trait anxiety but not AS directly predicted coping motivation for alcohol use (Comeau, Stewart, & Loba, 2001). Instead, AS predicted conformity motives. The different results in the two studies might be a function of the younger age in the second study.

The general results of all studies indicate that N's relationship to drinking is primarily mediated by coping; C's, by enhancement or social motivations for drinking. E's relationship to drinking is mediated by social and enhancement motives. Alcohol is a suppressant drug, so it can function to reduce anxiety and depression in those with high N. However, alcohol also has a disinhibiting effect and therefore increases positive emotions in social situations for those with high E, sensation seeking and impulsivity, and low conscientiousness.

Personality and Motivations for Drug Use

Opiates and stimulants. Participants in a study of motivation for methamphetamine were drawn from a non-treatment-seeking methamphetamine-dependent population recruited from the community (Newton, De La Garza, Kalechstein, Tziortzis, & Jacobsen, 2009). Unlike those in treatment, they had no reason to hide their real motivations for using the drug. A questionnaire contained questions relevant to five major categories of motivation: (a) negative reinforcement, or pain avoidance; (b) positive reinforcement, or pleasure seeking; (c) incentive salience, or craving; (d) stimulus–response learning, or habits (conditioned response); and (e) impaired inhibitory control, or impulsivity.

By far the most frequently given motive was pleasure seeking, as in the item “Do you use drugs because you want to get high?” I most frequently encountered this response in my several years working in an inpatient treatment center. Although some initially said they used drugs to deal with anxiety and depression, as they recovered they admitted that these feelings were a response to the current stresses in their lives rather than the initial reason for starting and maintaining drug use. They admitted to often using the drug when they felt okay and enjoying many things besides drugs.

Opiates such as heroin and stimulants such as cocaine and amphetamines have somewhat different physiological and psychological effects, but as pointed out in previous sections of this chapter, the personality correlates and motivations are more similar than different. As with stimulant users, young heroin users are high on sensation seeking as both a personality trait and a motive (Craig, 1982; Kosten, Ball, & Rounsaville, 1994; Platt & Labate, 1976; Skolnick & Zuckerman, 1979). Maremmani et al. (2009) compared methadone-treated and stabilized subjects formerly addicted to heroin with control subjects using a temperament scale containing items for four major temperaments: cyclothymic, hyperthymic, dysthymic, and irritable. The subjects addicted to heroin were higher than the control subjects on cyclothymic and irritable scales but not different on depressive or hyperthymic scales. The authors described the profile as sensation seeking or
novelty seeking because the cyclothymic pattern is associated with these in a canonical analysis.

**Psychedelics.** This class of drugs includes hallucinogens such as LSD, dissociative drugs such as PCP, and amphetamine analogues like 3,4-methylenedioxymethamphetamine (MDMA, or ecstasy). LSD was popular during the 1960s and 1970s, particularly among students and others rebelling against society and looking for new internal experiences and altered emotional states. Timothy Leary, a Harvard psychology professor, began experimenting with psychedelic effects of certain mushrooms and then LSD, finally becoming a popular guru urging the younger generation to turn on with the help of LSD.

Studies of drug usage among college students during this period found that LSD was the fourth most commonly used drug (after marijuana, hashish, and amphetamines), particularly among those scoring high on sensation seeking (19%-27% of users). None of those in the low sensation-seeking range reported ever using LSD (Zuckerman, 1979).

LSD provides not only a novel and unusual high but one that is different each time. For most users, the experience—including hallucinations, perceptual distortions, what seem to be profound insights, and intense emotional experiences—is positive, but for others the experience can be negative, with frightening hallucinations and delusions and temporary psychosis (a bad trip). Many users of hard drugs such as heroin or cocaine regard the use of LSD as too risky because of the unpredictability of its mental effects. Most users of LSD are polydrug users, reflecting their willingness to take risks to have new experiences. Segal and Singer (1976) asked drug users about their motivations for using specific drugs. Nearly three quarters of those using LSD said they used it to provide a new experience, and 20% said that their motive was curiosity. Two thirds of those using other hallucinogens reported their motive as curiosity. Experience or novelty seeking is one of the central motives in sensation seeking.

MDMA fell into wide use as a party drug at raves, a kind of manic dance marathon that lasts for 6 to 8 hours at a time. The amphetamine component of the drug helps sustain the energy needed for such exertions. The drug can cause neurotoxic damage, memory problems, and emotional problems (anxiety and depression) in those who use it chronically.

In a large European study, ecstasy users were higher than nonusers on the ImpSS subscale of the ZKPQ (Benschop, Rabes, & Korf, 2003). Most ecstasy users also use marijuana. A study compared regular ecstasy users with marijuana users who did not use ecstasy and nonusers of any drug (Daumann, Pelz, Becher, Tuchtenhagen, & Gouzoulis-Mayfrank, 2001). The ecstasy users scored higher than both control groups on the nonplanning type of impulsivity and the ES subscale of the SSS. Schilt et al. (2010) contrasted people who used ecstasy with people who used polydrugs, amphetamines or cocaine, or both and controls who did not use drugs and drank socially. In this study, the ecstasy and polydrug users were higher on depression, impulsivity, and sensation seeking than those who did not use drugs, but not different from people who used many drugs but not ecstasy.

People who use ecstasy are high impulse sensation seekers compared with those who use marijuana or people who do not use drugs and drink socially but not when compared with others who used many drugs, including stimulants. The need for high levels of positive energy arousal in party settings may be common to the use of ecstasy, amphetamine, and cocaine. Sensation seeking is a personality characteristic that is based on the same rewards. The impulsivity component of impulsive sensation seeking is related to the ignoring of risk in the pursuit of intense pleasure and novelty of experience.

**PROGNOSIS FOR TREATMENT OUTCOME**

Patkar et al. (2004) also used the SSS and Impulsivity and Hostility scales to predict the outcome of a 12-week behavioral treatment for people addicted to cocaine. The SSS total score correlated negatively with days in treatment and positively with dropout rates and failed urine tests for cocaine use. The Impulsivity scale score correlated negatively with days in treatment and the Hostility scale score correlated with dropouts.

Ball (1995) studied patients addicted to cocaine in treatment using the ZKPQ. Those with high scores on ImpSS, N-Anx, and Agg-Host were more
likely to continue using drugs during treatment, with a higher percentage of "dirty" urine tests. Those high on ImpSS were less likely to keep their treatment appointments, stay in treatment for 1 month, or complete treatment. Both this and the Patkar et al. (2004) studies have shown that impulsivity, sensation seeking, and neuroticism are predictors of negative response to treatment. The first two of these are also associated with antisocial personality.

A study of personality disorders as predictors of outcome in treatment of people who abuse opiates found that ASPD was the only personality disorder predicting the outcome of treatment in this group (Haro et al., 2004). Relapse rates were high in this group, which is not surprising given the high comorbidity of opiate abuse and ASPD (Rounsaville et al., 1991).

Another study measured personality disorders using the Millon (1987) self-report inventory over a 1-year period of treatment in a residential therapeutic community (de Groot, Franken, van den Meer, & Hendriks, 2003). The patients were either cocaine or heroin users or both. Most were polydrug users. Scores on the Millon scale represent the personality disorders in the fourth edition of the DSM (American Psychiatric Association, 1994). The only scale that predicted time in treatment was the Schizotypal scale associated with early dropout. Many of the scales changed over the course of treatment such as the Avoidant and Dependent Personality (neuroticism) scale and the Schizotypal and Borderline scales. Scores on these scales showed a decrease or improvement in functioning. However, a group of scales from the Type B disorders, including Antisocial, Histrionic, and Narcissistic scales, showed no change. Another study using the Minnesota Multiphasic Personality Inventory in a therapeutic community also found that scores on neuroticism-type scales (depression, psychasthenia) tended to fall after treatment, but scores on the scale for psychopathy and mania remained at the pretreatment level (Zuckerman et al., 1975). Whatever the change in drug use achieved by such programs, it is clear that they do not change the basic antisocial personality of some of those who abuse drugs.

CONCLUSION

The first question raised in this review was whether there is an addictive personality in the broad sense of addiction as a behavioral problem, the inability to control the impulse to engage in behavior that one knows is potentially harmful even if immediately pleasurable. I first addressed this question by examining the consistency among the different types of risky behaviors. The evidence has suggested that there is some consistency in using different types of drugs, including alcohol. These substances are those referred to in the traditional medical use of the term addiction in reference to substance abuse or dependence. However, at least one type of behavioral risk taking is associated with substance-related risk taking: sexual risk taking. The common factor in drinking, drugs, and sex is disinhibition. Disinhibition is a trait of sensation seeking as well as a motive for drinking and drug use. Another factor is the sensory and pleasurable arousal that is the reward for both sex and drugs. The factor of compulsion and withdrawal discomfort implied in the use of the concept of addiction is more problematic, particularly for sexual behavior. With drugs, the motivation depends on the stage of drug use or abuse and dependence. In the use and abuse stages, the main motive seems to be simple pleasure seeking or getting high. This simple hedonic motive combined with weak inhibitory control results in the move from occasional recreational use to abuse. Dependence involves craving and pain avoidance and is closest to the concept of addiction.

Given the evidence for a risk-taking behavioral factor, is there a personality factor that is associated with the behavioral tendency? There is, although its components tend to vary somewhat with the stage of use and the methods of assessment. Sensation seeking is a trait that separates users from nonusers, particularly as one moves from legal drugs (primarily alcohol) to illegal drugs and from more commonly used illegal drugs such as marijuana to the harder drugs that are perceived as riskier. The tolerance for risk in the pursuit of pleasurable sensations or experiences is part of the definition of sensation seeking (Zuckerman, 1979, 1994, 2007). Sensation seeking is highest in people who use stimulants as well as
opiates and psychedelic drugs, which reflects the need for change and novelty that is also a defining characteristic of sensation seeking. The need for intensity of sensation is also involved because when the same drug is used repeatedly, the intensity of the arousal reaction (the rush) diminishes, and either a stronger dose or a new drug is needed to revive the sensation.

Personality tests using the NEO-PI Big Five given to those who abuse drugs who are entering or in treatment have shown a characteristic profile associated with drug abuse or dependence: high on N and low on A and C. A is highly and negatively related to aggression, and C is negatively and highly related to ImpSS in the Alternative Five, the ZKPQ scales related to drug use and abuse and severity of addiction in clinical studies of cocaine or opiate addiction. All but N distinguish drug users in unselected college or community populations, prison populations, and special community samples such as prostitutes. In contrast with sensation seeking, impulsivity, and aggression, neuroticism is not found to be high at a younger age before drug use or later after treatment for abuse. A selective effect also occurs in that only a minority of those with drug or alcohol disorders ever enter treatment programs, and those who do are probably under the greatest stress.

The general personality profile is different in those classified as Type 1 (alcoholism) or Type A (drug addiction) and Type 2-Type B. Types 1–A involve later onset of alcoholism with symptoms of anxiety and depression (neuroticism), whereas Types 2–B usually have an earlier onset and one characterized by impulsive sensation seeking and antisocial tendencies. Given the later onset of Types 1–A, the negative emotionality may be a consequence of addiction-related life stress rather than a temperament or trait predisposing the addiction.

Scores on basic personality traits are fairly stable over the adult years, but the main components of neuroticism, anxiety, and depression can change as a function of stress. People beginning treatment for drug abuse are usually under a great deal of stress as a consequence of the problems engendered by drug abuse or withdrawal, and this stress may be reflected in their neuroticism scores. There is little evidence, however, that neuroticism was part of their temperamental makeup earlier in life or later when the stress is reduced in therapy. In temperament, they are strong on BAS but neither high nor low on BIS. They are characterized by strong positive emotionality but not negative emotionality and by cyclothymic and irritable rather than dysthymic temperaments.

Personality disorders reflect basic kinds of temperament. ASPD is the one found most frequently in people who abuse drugs. There is little relationship between drug abuse and the cluster C neurotic (avoidant, dependent, obsessive–compulsive) kinds of personality disorders. Sensation seeking, disinhibition, impulsivity, and aggression are characteristic of ASPD. Most people who abuse drugs are not basically antisocial, although the need to finance their drug addictions may require antisocial behaviors. Those who are basically antisocial before addiction have the worst prognosis for treatment. The challenge of treatment is to change their behavioral expressions without likelihood of change in the basic personality. Apart from the direct pleasurable effects of the drug, most of these high sensation-seeking antisocial types will confide that they enjoyed the excitement of the drug life, including the criminal risks and challenges, as much as the drugs themselves. Knowledge of the personality of the person who abuses drugs is important in developing an effective mode of treatment.

APPLICATIONS TO THE SYNDROME MODEL OF ADDICTION

The syndrome model of addiction (see the Introduction to this handbook) suggests a broader category of disorder beyond substance addiction with some common etiology and treatment. This new category of addiction would include behavioral expressions such as sexual and gambling addiction. The criteria for addiction disorder are (a) craving or compulsion, (b) losing control, and (c) continuing the behavior despite experiencing adverse consequences. Some of these criteria rely on subjective factors. For instance, what is the distinction between craving and compulsion? Shaffer (in the Introduction to this handbook) states that distinguishing a habit that is uncontrollable from one that is simply not controlled is difficult. The stated motivation of people with addiction might not reflect their actual motivation. It might
instead reflect their perception that it is more acceptable to be in the grip of a compulsion than to voluntarily choose to use the substance or engage in the activity because it is pleasurable. Loss of a major source of reinforcement is aversive, but the state produced is not necessarily as anxiety provoking as suggested by the construct of compulsion.

Apart from the element of motivation, one can see a similarity between substance-related and behavioral expressions of addiction such as gambling or sex. All elicit positive arousal and can lead to tolerance, resulting in the need for higher dosages in the case of the drug, larger sums of money wagered in the case of gambling, and new partners in the case of sex. All can produce negative mood in the absence of reinforcement that can only be relieved by resumption of the drug or activity at the higher level. All show persistence despite negative consequences.

Recognizing the question of basic equivalence, Shaffer (in the Introduction to this handbook) suggests the need for a gold standard such as the HIV blood test for AIDS. Biological markers exist for addiction and some of its personality correlates. The presence of a long form as opposed to a short form of the dopamine 4 receptor gene (DRD4) has been associated in some studies with the personality trait of high levels of novelty or sensation seeking and heroin and alcohol use, pathological gambling, and sexual desire, function, and arousal. However, the gene form accounts for only 10% of the genetic variation in sensation seeking and only 4% of the total variance in the trait. Many other genes or gene regulators must be involved, and this must be true for any personality trait or habit. No gold standard for personality or psychopathology exists in the sense of a single gene or biological marker, except for some neurological disorders. The gene might require an additive or interactive affect with other genes or even environmental factors during fetal or childhood periods to effect a predisposition.

Understanding the relationships between alcohol, drug, and behavioral disorders (e.g., gambling and sex) might be possible in terms of common personality and behavioral trait factors. In studies of personality, it is important to assess personality before the age at which the substance and behavioral disorders develop because the demands of the disorder can influence personality traits rather than vice versa. In other words, predictive validity is needed to understand concurrent validity. Many of the substance abuse disorders, for instance, produce anxiety and depression as a reaction to the social, legal, or physical stress connected with drug abuse. If researchers are interested in the etiology of the drug use disorders, they must give priority to longitudinal studies of personality and environmental factors before development of the disorder syndromes.

Such studies from childhood or early adolescence tend to show that those who later developed an addictive substance abuse disorder were undercontrolled, sensation seeking, and impulsive before the development of addictions. The lack of constraint in children and early adolescents turns into a general risk-taking factor that plays a role in substance abuse or behavioral addictions.

Research has shown that two types of alcohol and drug abuse exist. People with the first type develop the addiction later in life and feel anxious or guilty about the addiction and their inability to control it. They are likely to experience addiction as an undesirable compulsion. People with the second type begin using the substance at an earlier age, do not feel guilty about it, and experience it as a voluntary and positive seeking of pleasure. Both types tend to be impulsive, but the first type is more neurotic and less sensation seeking. The same distinction can probably be found in pathological gambling and sexual addiction. This distinction is important in treatment because the first type is more motivated to quit and therefore has a better prognosis, whereas the second type may only seek treatment because of external pressures (e.g., legal and social). The choice for them is often treatment or jail.

The first type fits the addiction model presented by the editors of this book, but the impulsive, aggressive sensation seeker is more problematic for the model. There is a danger of overmedicalization when extremes of personality are translated as diagnoses, ignoring their continuous distribution within the population. This could result in any number of addiction diagnoses such as shopping addiction, computer addiction, or Facebook addiction. An
argument can be made for keeping the construct of addiction (or dependence) within the realm of substance abuse disorders as they are now.

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The sociological perspective helps make sense of drug use, addiction, and related phenomena by exploring the lives and social worlds of those who use drugs—the context in which they live and use drugs. This chapter brings insight into people's pathways to and from drug use and addiction—how and why people initiate drug use and why some individuals develop problematic relationships with drugs, whereas others are able to moderate or abstain from drug usage. The work highlights the role of social setting in understanding people's drug use and reveals that much more is involved than just bad decisions, unscrupulous predatory drug pushers, and uncontrollable biological addiction.

In 1984, psychiatrist Norman E. Zinberg, in his classic analysis Drug, Set, and Setting: The Basis for Controlled Intoxicant Use, presented an elegant theoretical framework for understanding the conditions that shape an individual's drug-using experience. Zinberg drew on a vast body of social and behavioral research before him, and his paradigm continues to influence wide-ranging drug research today. In this chapter, we explore the history of the sociological perspective leading up to Zinberg's framework and how it has shaped subsequent studies. Influenced by his framework, sociologists today who study drug and alcohol use, addiction, and related behaviors have generally contended that the drug experience depends on factors operating within three nested, interacting domains: drug, set, and setting.

Drug, set, and setting have very specific meanings. The action of a drug describes the properties that affect an individual's body, often manifest across the dopamine pathway. Set includes a user's psychological expectations or mindset surrounding the consumption of a drug that further influence the experience. Set includes factors such as personality and internal states of mind (e.g., depression, happiness, stress, anxiety). Setting includes the environmental, social, and cultural context in which substance use takes place. The substances that are available and the significance that society and individuals come to attach to the substances influence a person's experience (or relationship) with a substance. In this manner, the drug use experience is context dependent. This context or setting is much more than a collection of distal antecedents. The setting is an organic system with its own internal logic based in a worldview that defines the prevailing gestalt as located in time and place. In this manner, an understanding of setting greatly expands the addiction syndrome model.

The concept of setting holds strong potential to make sense of otherwise idiosyncratic addiction-related phenomena, including moderate or recreational use; the gateway phenomenon; drug epidemics; the connection of certain types of drugs with different types of music, social scenes, and lifestyles; the effectiveness of harm reduction services, why social approaches to recovery work for some people; why
natural recovery works for others; and why triggers undermine the recovery attempts for many. An understanding of the important role of setting provides a richer explanation of many drug-related experiences than an exclusively psychopharmacological or biochemical perspective, especially those that depend exclusively on the dopamine pathway.

We start this chapter by examining the historical development of sociological perspectives on addiction and the sociologists whose insights still inform this outlook. This history of ideas serves a secondary purpose of showing how people's understanding of a phenomenon such as drug use or addiction changes over time. This change could illustrate how the accumulation of knowledge helps a scientific understanding to evolve. However, sociologists have often pointed out that the distinction between the researcher and the research subject is fuzzy, especially when it comes to explaining social phenomena (Denzin, 1994; Geertz, 1973; Rose, 1990). Accordingly, how a culture understands drug use and addiction can say as much about the prevailing dominant worldview as it does about people who use drugs themselves. It is often much easier to identify the importance of this social constructionist perspective when analyzing previous views of addiction than it is when studying the broadly accepted viewpoint prevailing today. Informed by this historical perspective, we then examine how contemporary sociological theory explains the role of setting in the drug use experience. In this effort, we focus on subcultural, career, and social learning perspectives.

HISTORICAL ANTECEDENTS: THE CHICAGO SCHOOL OF SOCIOLOGY, METHODOLOGIES, AND THE STUDY OF SOCIAL PROBLEMS

Sociologists today typically refine or modify Zinberg's (1984) model of drug, set, and setting when interpreting drug use experiences; however, this was not always the case. The sociological study of drug use emerged during early 20th-century urban industrial growth, massive migration, xenophobia, and the professionalization of academic disciplines. Disciplinary growth reflected disaffection with the dominant late 19th-century and early 20th-century view of the drug user and “addict” as having inherent personality defects. For instance, Lawrence Kolb, a U.S. Public Health Service psychiatrist, regularly avowed that addicts had psychopathic personalities (Acker, 2002; Courtwright, 2001; Lindesmith, 1938; Musto, 1973).

Such personality-based perspectives came under challenge during the first half of the 20th century, in part because of the new discipline of sociology. In 1938, Alfred R. Lindesmith strongly criticized the view of addiction as based in psychopathology:

> Psychiatrists have often regarded the use of opiates as an escape from life and have viewed addicts as defective persons seeking to compensate for, or avoid, their inferiorities and mental conflicts. As would be expected, addicts have been labeled “psychopaths,” with the assumption that the attachment of this ambiguous label in some mysterious way explained the phenomenon....

This point of view contrasts the “psychopath,” who is assumed to be susceptible to addiction, with “normal” persons, who are presumed by implication to be immune. (p. 594)

A change in who had addiction also influenced the changing view of addiction. Specifically, between the 1880s and the 1930s, the profile of the typical person who used drugs shifted from one who was primarily White and of middle income to one who was primarily minority and of lower income. Drug use became more and more associated with self-indulgent behavior that many came to associate with a larger decline in traditional morality. In this regard, migration, an expanding racism, and class issues might have influenced the legal and scientific perspective on addiction, subtly or not so subtly. The late 19th- and early 20th-century population who used and were addicted to drugs consisted largely of middle-class White women who were iatrogenically addicted, aging Civil War veterans, a small underworld or sporting class, and poor urban men. By the early 1950s, contemporaries increasingly viewed the dominant addict type as a young Black or Mexican American man (A. Bennett, 2009; Courtwright, 2001; Musto, 1973). David
Courtwright (2001) contended that this shift from White middle- to upper class women to lower class Black and Mexican men "undermined the reigning psychiatric view of addicts as mentally defective individuals" (p. 152).

In light of this demographic shift, researchers increasingly questioned how addiction, thought to result from an inherent deviant pathology, could suddenly spread to Blacks and other minority groups. These demographic shifts seemed to support sociologists' interest in understanding the relationship between behaviors, social worlds, and setting for conceptualizing problematic drug practices. Simply put, as the perception of the drug user shifted to an inner-city, minority population cohort, researchers and policymakers advocated for and embraced new conceptual frameworks for understanding addiction and drug use. Sociological interpretations meshed well with this new ethos.

Chicago School of Sociology and the Emergence of Expert Knowledge

At the forefront of the disciplinary growth and professionalization of sociology was the Chicago school of sociology. During 1892, Albion Small founded the sociology department at the University of Chicago. By 1915, it was one of the premier programs. Chicago school sociologists deployed ecological and ethnographic methodologies to explore urban life and urban phenomena; they developed an ecological model to understand a range of urban social problems among diverse population groups, often immigrant and minority. Indeed, the very problems that first captivated these sociologists were the problems associated with massive immigration and urbanization, including such pressing issues as poverty, social marginalization, delinquency, race-ethnic relations, and other issues contemporaries considered the evil underbelly of urban industrial growth (Bulmer, 1984). This was playing out in the larger context of the Progressive Era in the United States, wherein new theories of drug use emerged hand in hand with the professionalization of knowledge domains (Wiebe, 1967). A cadre of newly constructed experts and professionals emerged to solve the prevailing city problems with zeal. Late 19th- and early 20th-century Progressive Era reforms included a movement toward the professionalization of academic disciplines and medical expertise; these medical experts and academic researchers would guide the direction of drug policy and shape the understanding of drug use and addiction throughout the 20th century. Chicago was the ideal setting for researchers to develop new theoretical frameworks with potential for subsequent application to pressing social problems.

Using the Voices of Participants to Solve Social Problems

Viewing the city as laboratory, early Chicago school researchers, such as Robert E. Park, Ernest W. Burgess, William I. Thomas, Roderick McKenzie, Florian Znaniecki, and Louis Wirth, focused on the relationship between urbanization, individual and group life, and human nature. These academics sought to apply research to solve social problems. The methods they deployed to capture urban life were designed to help explain the interaction between micro and macro variables and individual and group life. Their applied research attempted to capture people's lived experiences, an insider perspective, and the social context and meaning of a range of behaviors—drug users' social world (J. Bennett, 1981; Bulmer, 1984). Under the banner of objective scientific inquiry, these early pioneers embraced emergent quantitative and qualitative methods. They mined available statistics. They conducted behavioral and social surveys of city inhabitants. They coupled these objective measures with field observations, interviews with city residents, and examination of court and clinical case files to provide subjective nuance in their depictions of the complexity of city life (Bulmer, 1984).

For example, Thomas and Znaniecki, in The Polish Peasant in Europe and America (1918), analyzed letters, personal correspondence, court records, newspapers, and records of social agencies—human documents, as they called them—to capture the social experience of immigrant life in the new world. They argued that delinquency was the result of a breakdown of old world values rooted in the complex of social and cultural clashes between populations. The purposes of this seemingly eclectic use of sources was to get at the insider's point of view—to capture internal meaning—in a backdrop of what
was perceived to be a harsh urban environment. The sociologists’ gaining of an insider’s perspective—an expression that is now ubiquitous among social scientists—was thought to help the researcher to use that knowledge to help social workers and their progressive companions in crafting solutions to pressing social problems (J. Bennett, 1981).

Similarly, Clifford Shaw, in *The Jack-Roller* (1930), relied on a delinquent boy’s own story, his perspective and life history, his inner world, to interpret the dynamics of youthful delinquency. Shaw used the case study approach, a hallmark of Chicago school sociology, and coupled the use of a boy’s own story with family history documents, medical and psychiatric records, and arrest data. Shaw and his colleagues reasoned that a boy’s own story could provide an authentic point of view from the perspective of a delinquent boy himself; it could illustrate the social and cultural milieu and how a boy sequenced and prioritized past events, the setting in which life played out. This perspective could help the researcher understand a boy’s social world and the dynamics of his relations with others, which providers could subsequently incorporate into prevention and treatment programs. As Shaw (1930) put it,

> In the first place, the child’s “own story” is of particular importance in the diagnosis and treatment of cases of delinquency. The attitudes and intimate situations revealed in the life-story not only throw light upon the fundamental of behavioral difficulty, but, along with the other case material, afford a basis for devising a plan of treatment adapted to the attitudes, interests, and personality of the child. . . . Life history data have theoretical as well as therapeutic value. They not only serve as means of making preliminary explorations and orientations in relation to specific problems in the field of criminological research but afford a basis for the formulation of hypotheses. . . . The validity of these hypotheses may in turn be tested by the comparative study of other detailed case histories and by formal methods of statistical analysis. (pp. 3–6)

Clifford Shaw and his colleagues recognized the valuable contributions of statistics but filled gaps and provided nuance by joining quantitative data with interviews and other qualitative data. Ultimately, researchers suggested that the individual story, at the micro level, reflected the broader dynamics of a larger delinquent social group. Commenting on Shaw’s work, Chicago sociologist Ernest Burgess (as cited in C. Shaw, 1930) noted,

> Through communication and interaction the person acquires the language, tradition, standards and practices of his group. Thus, the relation of the person to the group is organic and hence representative on a cultural rather than biological level. . . . The study of the experiences of any individual person at the same time reveals the life activities of the group. (p. 186)

A recurrent theme of early Chicago school work is the notion that urban life and industrialization included disorienting and subsequently homogenizing forces that could foster or curtail a host of behaviors considered by contemporaries as deviant or nonnormative. The use of diverse qualitative and quantitative sources allowed these sociologists to actualize an overarching mission and ethos of Chicago school sociology. They strived for an empirical and familiar understanding of life and people in the city in relation to these larger forces of urbanization and industrialization (Bulmer, 1984). They rejected simply making data, often from the library, fit abstract theoretical models. Instead, they strove to ground theory in demonstrable and empirically observable facts. Their research involved detailed, objective examination of social life and behavior in the city. From this, they arrived at generalizations packaged into an array of evolving and changing theoretical frameworks (Bulmer, 1984; Wilson, 1997).

**Sociologists Discover Drugs**

Sociologist Bingham Dai, in *Opiate Addiction in Chicago* (1937/1970), initiated what would become a long tradition of sociological studies of addiction and drug use (Acker, 2002; Lindesmith, 1938; Musto, 1973). These studies focused on a range of populations, in diverse urban contexts, involving a
range of substances (Feldman & Aldrich, 1990). Consistent with the methods of Chicago school sociology, Dai relied on both interviews and spatial-ecological surveys of individuals with addiction to gain an understanding of the drug user's social world. Dai's work expanded on the prevailing psychoanalytic discourse by viewing drug use as a social behavior reflecting cultural norms and one's inability to adjust to them. He highlighted the ways in which the immediate social environment strongly influenced individual behavior, thus challenging prevailing individualistic interpretations of drug use that framed drug use either as willful misbehavior or as behavior underpinned by an inherent psychopathy (Acker, 2002).

Alfred Lindesmith, in his seminal works "A Sociological Theory of Drug Addiction" (1938) and Opiate Addiction (1947), drew on Dai's (1937) focus and methodologies. On the basis of analytic induction, Lindesmith and others subordinated theory to facts of observation. Lindesmith (1938) noted that one had to discard a theory if one case contradicted or challenged it. Like his predecessor Dai, Lindesmith challenged the centrality of individual, "psychopathic" character traits as the cause of drug addiction (Acker, 2002; Feldman & Aldrich, 1990; Lindesmith, 1938, 1947). He found the social world to be paramount. As he noted,

It is not the effect of the drug that produces the alleged deterioration of character in the addict but rather the social situations into which he is forced by law and by the public's conception of addiction, which does damage. (Lindesmith, 1940, p. 202)

Sociological Drug Studies on the Ascent
The 1940s through 1970s was a dynamic period in the sociological study of drug use. Research during this period shifted away from questions related to the causes of addiction to a focus on the social world of drug users (Acker, 2002; Feldman & Aldrich, 1990). However, sociological interpretations also continued to compete with older psychoanalytic and medical models. Part of this shift within sociology was associated with a new shift in the drug-using population. The drug user of concern during the early 1950s was widely an urban minority man; however, by the 1970s, drug users included a broad cross-section of all youths and young adults. This demographic shift led to analysis of the symbolic nature of drug use within the emergent youth culture, counterculture, oppositional culture, and hipster movements and to the consideration of such risk factors as community disorganization, disadvantage, and socioeconomic inequality (A. Bennett, 2009; Jonnes, 1996). Most fundamentally, sociologists sought to understand the seemingly new social world of drug users, the setting of drug use, and the ways in which the context mediated drug users' experiences.

To be sure, researchers' interpretations of drug use differed across and often within disciplines. Sociological interpretations of drug use continued to vie with traditional psychological perspectives. A 1959 report issued by the California Senate Interim Committee on Narcotics illustrated these interpretative differences and nuances. The report began with a strict psychologically rooted punitive orthodoxy:

Most narcotic addicts suffer from a basically pathological character structure. In these people, the more the drugs are used to solve their deep-rooted personality problems, the more malignant is the addiction. Here intensive treatment of a psychotherapeutic nature is necessary and must be based in each case on an analysis of the factors leading up to the drug addiction. Among the causative personality factors are frequently found an immaturity of character development, a desire to live only in the present, and a narcissistic attitude . . . drug addicts may frequently be found to suffer poor ego and superego development—a fact which also explains a certain tendency towards unreliability and untrustworthiness which can be observed among them. (Senate of the State of California, 1959, p. 13)

By the end of the report, however, the State Senate committee gave substantial attention to very different approaches to understanding and treating drug
users. Physician Joel Fort, of the Berkeley Mental Hygiene Clinic, for example, helped direct attention away from individual psychological and behavioral maladjustment to larger contextual factors embedded in setting. This redirection emphasized the potential role of the environment, peers, and family on the incidence and larger patterns of drug use: “Behavior in general is a learned thing, learned through early interrelationships with people, primarily parents, and later in schools and gangs and associates that one has” (Senate of the State of California, 1959, p. 14). Reinforcing this perspective, Dr. Frank Robertson, director of health services, San Diego College, emphasized that “drug addiction among juveniles is highest in neighborhoods where the income and education are lowest, and where there exists the greatest breakdown of normal family living arrangements” (Senate of the State of California, 1957, p. 15).

The works of Edward Preble, John Casey, and Howard Becker are characteristic of the new trends in sociological inquiry. Building on the pioneering work of Lindesmith (1938, 1947), Becker's (1963) research on marijuana users and jazz musicians led him to a “learning perspective” on drug use, whereby more experienced users cue novice users according to a socially interactive learning process about how to interpret the effects and meaning of drugs. He meshed this learning perspective with the concept of career, which provided scaffolding for interpreting transitions in individuals' drug use and addiction over the life course (Becker, 1963; Tracy & Acker, 2004). Central to Becker’s work was his understanding of deviance, which he conceptualized as a construction of social groups rather than an inherent individual or group pathology (Acker, 2002; Becker, 1963, Feldman & Aldrich, 1990). Becker contended that mainstream society created or labeled “outsiders,” as he called them, including stigmatized and marginalized populations, such as drug users, jazz musicians, or delinquents; the label given to them embodies behaviors that did not conform to the dominant, middle-class value system. In tandem and in part in response to this labeling, stigmatized populations developed a subcultural oppositional identity, often rejecting the mainstream worldview and in the process creating their own shared values, symbols, and norms as the basis of their subcultural identities. Recognizing the process by which outgroups developed and persisted, Becker and others, as did their predecessors, emphasized internal forms of organization and function in contrast to viewing nonconformity as based in disorganization and dysfunction.

A rejection of the belief that poverty invariably led to disorganization, dysfunction, and problematic behaviors was embodied in Preble and Casey's influential 1969 work, “Taking Care of Business: The Heroin Addict’s Life on the Street.” In contrast to the view outlined earlier, that drug use could be interpreted as simply an escape from responsibility and social and personal relationships (e.g., drug users are passive, dependent, withdrawn, or inadequately socially adjusted), Preble and Casey relied on interviews, participant observation, and life histories to reach their conclusion that heroin transactions among an urban poor population allowed individuals to have adventurous and satisfying lifestyles, a purposeful life in which heroin-related activities hold meaning and are highly rewarding, especially in the face of marginalization and socioeconomic exclusion (Preble & Casey, 1969). As they put it,

The career of the heroin user serves a dual purpose for the slum inhabitant; it enables him to escape, not from purposeful activity, but from the monotony of an existence severely limited by social constraints, and, at the same time, it provides a way for him to gain revenge on society for the injustices and deprivation he has experienced. (p. 23)

These researchers aimed to capture how drug users created their own social worlds with their own values, beliefs, symbols, and behaviors via shared experiences and social interaction, not just in opposition to mainstream society.

Vietnam, “H” for Heroin, and the Setting of Drug Use
Sociological studies of drug use and addiction—and drug use itself—blossomed during the late 1960s and 1970s as a result of various factors, including
(a) U.S. involvement in Vietnam and widespread concern about the return of soldiers addicted to heroin; (b) social unrest regarding racism, concentrated poverty, and the perceived impending collapse of U.S. cities; (c) a rise in the use and abuse of heroin, PCP, marijuana, psychedelics, and a range of pharmaceuticals; and (d) the inception of the National Institute on Drug Abuse (Kusmarov, 2009; Musto & Korsmeyer, 2002). In this context, researchers refined old and developed new sociological theories to examine drug-related behaviors.

During 1971, amid this widespread fear of heroin addiction among soldiers serving in Vietnam, psychiatrist Lee N. Robins of Washington University conducted a survey of service members in Vietnam and veterans who had returned to civilian life. Her findings would provide much fuel for the continued work of sociological examination of drug use, particularly highlighting the importance of setting and context as formidable factors shaping drug-using experiences. As Robins (1994) explained years later,

The results were surprising. More men had been using opiates in Vietnam than the Department of Defense supposed. Almost half (45%) had used opium or heroin, and 34% had taken heroin at least once. Eleven percent tested positive for opiates on their departure; 20% said they had been addicted and reported typical withdrawal symptoms. ... Perhaps our most remarkable finding was that only 5% of the men who became addicted in Vietnam relapsed within 10 months after return, and only 12% relapsed even briefly within three years. Treatment did not account for this high recovery rate. ... Soldiers in Vietnam had no special vulnerability to narcotics. They used heroin because it was inexpensive, unadulterated, and easily available, alternatives were few, disapproving friends and relatives were far away, and they felt that their war service was somehow not part of their real lives. When their situation changed, most of them had no difficulty giving up heroin, and that should not have been surprising. By spontaneously recovering from addiction and using heroin without becoming re-addicted, they refuted American beliefs but confirmed American experience. (Robins, 1994)

Robins's (1994; see also Robins, Davis, & Nurco, 1974) findings confirmed the importance of setting and context in understanding people's pathways to and from abusive drug use. It also called into question the prevailing disease models of addiction and illustrated that only some people who used drugs become addicted. Zinberg (1984) himself commented on Robins's findings, which would inform the development of his own perspectives on the drug-using experience:

Apparently it was the abhorrent social setting of Vietnam that led men who ordinarily would not have considered using heroin to use it and often to become addicted to it. Still, they evidently associated its use with Vietnam much as certain hospital patients who are receiving large amounts of opiates for a painful medical condition associate the drug with the condition. The returnees were very much like those patients, who usually do not crave the drug after the condition has been alleviated and they have left the hospital. ... In the case of both heroin use in Vietnam and psychedelic use in the 1960s, the setting determinant, including social sanctions and rituals, is needed for a full explanation of the appearance, magnitude, and eventual waning of drug use. (pp. 12–13)

In this context of 1970s social unrest—rife with mass media portrayals of veterans strung out on heroin and youths across the country consuming drugs at seemingly unprecedented rates—Zinberg engaged in his own research (Zinberg, 1984; Kusmarov, 2009) and concluded that the moderate and controlled use of drugs was more common than previously thought. Challenging traditional stereotyped notions that any drug use would result in devastating addiction and a pattern of unbridled consumption,
Zinberg (1984) found that the vast majority of users themselves moderated and controlled their drug intake. Such controlled intake reduced the potential harms many people thought were associated with the ingestion of psychoactive substances. Central to this finding was the integral role of drug, set, and setting in shaping the drug-using experience.

By the early 1980s, in addition to the image of returning Vietnam soldiers addicted to heroin, the image of the drug user had expanded considerably to include younger people, women, and professionals who were using a range of psychoactive substances, often in combination, in many settings that defied traditional stereotypes. With drug users no longer fitting neat typologies, sociologists expanded their focus, looking at specific substances as well as specific populations' involvement with drugs. The classic image of the heroin junkie was resilient through the 20th century but has increasingly been coupled with an assortment of images including women with needles sticking in their arms or crack pipes in their mouths, professionals snorting cocaine at cocktail parties, and youths smoking marijuana and popping pills in dorm rooms (Campbell, 2000; Jonnes, 1996).

CONTEMPORARY SOCIOLOGICAL THEORY OF DRUG USE

Sociological studies of drugs and drug-related practices during the past several decades have focused on new populations in new settings consuming a range of psychoactive substances. Following in the footsteps of Zinberg and the many scholars who informed his work, researchers have sought to understand how drug, set, and setting shape specific drug-use trajectories. We start this section with examples of the use of the drug, set, and setting perspective. We then turn to a discussion of culture and subcultures as an explanatory frame for drug-related behaviors. Afterward, we look at the individual experience of drug subcultures over the life course from the social learning and careers perspective.

Drug, Set, and Setting

Beck and Rosenbaum (1994) applied drug, set, and setting to explain the experiences of users of 3, 4-methylenedioxymethamphetamine (MDMA), or ecstasy, who included a broad mix of college students, gay men and lesbians, professionals, yuppies, New Age spiritual seekers, and music and dance enthusiasts. Beck and Rosenbaum's respondents had vastly different purposes for taking the drug. Some consumed it to help them communicate with their partner by developing a heightened sense of empathy (Beck & Rosenbaum, 1994). The use of MDMA was also integral to various recreational experiences, including the nightclub and jam band scene. Each group held different expectations of the MDMA experience. A spiritual user, for example, stated,

I think that the pre-eminent aspect of MDMA is a sense of acceptance. . . . That's why I've used it with monks, rabbis, Zen Buddhist priests, as an aid to meditation. . . . Those people who are most distant from themselves need that the most. (Beck & Rosenbaum, 1994, pp. 38–39).

In contrast, a young professional and Grateful Dead fan reported,

More than anything else, I think about the best experiences I've had with MDMA—it becomes incredibly wonderful to be with friends or to meet people you haven't known before. . . . [At the Grateful Dead concert,] I had this very powerful, overwhelmingly clear conception that we're all just like neurons. The thing about a neuron is it's no good by itself. . . . I suddenly just realized that the whole point of life is the synapse, the connection of communication and the touching of hearts. It was just amazing, I was walking around like a goddamn politician, shaking people's hands and just being completely unselfconscious about it. (Beck & Rosenbaum, 1994, p. 49)

For these respondents, the use of MDMA in a specific setting, approached with a particular set, shaped the drug use experience. Thus, whereas the drug consumed across these groupings was MDMA, people's experiences using it varied on the basis of
the set or psychological expectation of the user in a specific setting.

Dina Perrone's (2009) *The High Life: Club Kids, Harm, and Drug Policy* provided another application of Zinberg’s framework, in this case to interpret the drug-using experience of “club kids.” According to Perrone,

The drug user's access to, and utilization of, all forms of capital (human, cultural, social, and economic) affect drug access, the setting options for drug use, and the user's mindset. Moreover, the timing of the usage during the user's life shapes the choices of drug, set and setting. . . . In particular, the drug's properties, the settings in which the drug is used, the user's mood (set) and resources (capital), and the stage of the user's life trajectory (timing) shape drug-using practices and influence the relationship between drugs and harm. . . . The cultural norms of use that drug users create in the settings where they use drugs shape what to use, when to use, and how to use.

(PP. 165-166)

Club kids, Perrone (2009) argued, define themselves as much by drug use as by the clothing they choose to wear, their tendency to engage in body modification as a marker of identity, and other behavioral and body-modifying techniques. Respondents in the study engaged in such practices as using steroids to build muscles or using crystal methamphetamine to lose weight, as one respondent confessed:

I was doing crystal. I was doing everything. It was like one of those Hamptons fucking weekends. It was horrible. That's when people were like, “What made you lose so much weight?” I'm like on this great diet; diet called crystal. Best diet ever in the world. (Perrone, 2009, p. 59)

Subcultural Evolution
Culture is a very rich concept that people have understood in many different ways (Schafer, 1998).

Clifford Geertz (1973) provided a definition that is sufficiently rich to indicate the central importance of culture while remaining vague enough to allow further analysis into a range of cultural processes. He described culture as “the fabric of meaning in terms of which human beings interpret their experience and guide their action” (p. 145). Geertz went on to explain that the study of culture is not an “experimental science in search of law but an interpretive one in search of meaning” (p. 145). For exploring drug-related experiences, we emphasize two aspects of the invisible construct called culture: Contemporary U.S. culture is composed of multiple subcultures, and individual behaviors both reproduce culture and serve to produce cultural change. This perspective has led to an overarching framework we refer to as the *subcultural evolution of drug use* (Golub, Johnson, & Dunlap, 2005). One can view culture as fractionated into a variety of more personal influences rather than as a monolithic whole. The perspective provides the local subcultures that prevail greater focus as their own authentic phenomena, rather than merely as standing in opposition to a dominant mainstream culture. This subcultural focus allows that illicit drug use conforms to a subcultural norm rather than deviates from mainstream behavior. This disprivileging of mainstream norms, values, and laws allows researchers to study the insider's view of drug use instead of society's view of the drug user. In brief, our theory of subcultural evolution and drug use contends that drug use emerges from a dialectic of the prevailing culture (and especially drug subcultures) with individual identity development. Use of a drug is clearly an individual's decision, but the prevailing drug subcultures and each person's place relative to them impart a greater significance to the activity. Conversely, individual decisions to adapt or reject aspects of the prevailing drug subcultures cause the subcultures to evolve as well as lead to the emergence of new ones.

Within this overarching framework, we use the term subculture in a very limited sense, as a constellation of connected values, symbols, norms, and behavior patterns. In this regard, the individual subcultures are much less comprehensive than the fabric of meaning Geertz (1973) invoked; rather, we
view subcultures according to Swidler’s (1986) framework that each serves as a “tool kit” of habits, skills, and styles from which actors construct their strategy of actions. A subculture might be based around drug use, ethnicity, religion, region, or a variety of other affiliations. This interpretive perspective leads to the study of the meanings people accord to their behaviors, the values they bring to their actions, and the shared symbols that constitute a collective subcultural identity.

Identity can reflect a larger subcultural involvement through choices of leisure activity, modes of consumption, or fashion. In this manner, identity involves the what, when, where, and how of substance use. For example, the image of individuals wearing tuxedos and drinking wine with others on a patio on a Friday night holds vastly different connotations—both internally and externally—than a group of men passing around a bottle of wine on the front stoop on a Sunday morning. In each case, the act of consuming in a specific setting allows the participants to create or reinforce their identity through mutual participation involving shared practices and symbols of consumption. Subcultural identity manifests in decisions about self-presentation such as clothing, style, language, and use of public space (Hebdige, 1979). Our fractionated perspective allows that individuals have overlapping identification with several subcultures at a time. The extent to which a particular subculture dominates a person varies widely. A drug subculture can alternatively represent an infrequent amusement, an occasional leisure activity, a lifestyle, or even a comprehensive worldview and purpose to life (Golub et al., 2005).

Subcultural theory provides useful theoretical scaffolding for sociologists who examine drug-using behaviors among diverse populations. A drug-centered subculture consists of the related conduct norms that prescribe what people should or should not do, such as how to consume or procure a drug, and the informally sanctioned drug-related behaviors. Subcultural theory helps explain individual and group identity and norms that shape behavior. It also provides insight into the social processes that facilitate the development and evolution of drug subcultures, drug eras, and drug generations. Drug use occurs in a cultural context and emerges from a dialectic of the prevailing culture (and especially drug subcultures) with individual identity development. Use of a drug is clearly an individual’s decision, but the prevailing drug subcultures and each person’s place relative to them are what impart a greater significance to the activity. Conversely, individual decisions to adopt, adapt, or reject aspects of the prevailing drug subcultures cause the subcultures to evolve as well as lead to the emergence of new ones. Culture and identity thus engage in a dialectic of coproduction. The prevailing drug subcultures and individuals’ social position relative to them define the range of drugs readily available, the symbolic significance of their use, and the social consequences for both use and nonuse (Golub et al., 2005).

The subcultural evolution model integrates a subcultural perspective with the social learning (see Akers, 1998; Bandura, 1977) and career or life course perspectives (see Faupel, 1991). It views identity development as following an imitative and adaptive process (often un-self-consciously) that differs at successive ages and that occurs within a sociohistorical context. For example, at the earliest ages, home and family often represent the primary influence in young children’s lives; children learn from parents and other household members the expected values and norms of behavior. During adolescence, a wider range of influences can affect identity development and potential drug-using practices, including other peers or mass media (Golub et al., 2005). Drug use thus changes in light of immediate or situational influences and the life course. Any drug subculture, and its place within the larger culture, depends on the extent to which people continually adopt it and perpetuate its conduct norms. Drug subcultures can die out as people reject them. New subcultures emerge through the process of people adapting existing cultural elements to their circumstances. Hence, the prevailing drug subcultures can vary substantially over time, across locations, and with social position (Golub et al., 2005).

Drug Epidemics
We derived the subcultural evolution framework from the study of drug epidemics, also referred to as drug eras. In general, we prefer the term drug era to
drug epidemic because it emphasizes the cultural aspects of the phenomenon, and it holds a relatively neutral connotation (see Golub et al., 2005; Sifaneck, Kaplan, Dunlap, & Johnston, 2003). Politicians and the media commonly abuse the term drug epidemic to arouse concern and serve political agendas (Hartman & Golub, 1999; Jenkins 1999; Reinarman & Levine, 1997). However, we stick to the term drug epidemic because it is more common and because it does explain an important element of the social phenomenon, namely, that the behavior spreads through social networks. Prior literature has found that the use of a drug tends to rise rapidly and then fall, constituting what appears to be an epidemic of use (Becker, 1963, 1967; Golub & Johnson, 1999; Hamid, 1992; Hunt & Chambers, 1976; Johnston, 1991; Musto 1973). We divide this drug epidemic phenomenon into four distinct phases: incubation, expansion, plateau, and decline. A drug era typically starts among a highly limited subpopulation participating in a specific social context—an incubation phase. For instance, the heroin injection epidemic prevailing during the 1960s and early 1970s grew out of the jazz music scene (Johnson & Golub, 2002; Jonnes, 2002). The crack epidemic of the late 1980s and early 1990s started with inner-city drug dealers at after-hours clubs (Golub & Johnson, 1997; Hamid, 1992). During the 1990s, many youths came to smoke marijuana in a blunt, an inexpensive cigar in which users replace the tobacco with marijuana, especially in the inner city. This marijuana–blunts epidemic was based in the hip-hop movement (Golub & Johnson, 2001; Johnson, Golub, & Dunlap, 2000; Sifaneck et al., 2003).

During the expansion phase, the pioneering drug users successfully introduce the practice to the broader population. In a very broad review of the literature, Rogers (1995) identified that when ideas spread, they tend to spread with increasing rapidity, whether it involves a new consumer product, fashion, teaching method, or agricultural technique. Mathematically, many aspects of these “diffusion of innovation” processes are analogous to disease epidemics. The primary difference between social diffusions and disease epidemics is what is spreading—an idea or behavior as opposed to a bacteria or virus.

People have agency regarding whether they adopt a behavior, such as use of a new drug. Consequently, individual susceptibility to use varies greatly according to friendship networks, social position, and personal identity.

Drug epidemics reach the plateau phase when everyone most at risk of the new drug practice has either initiated use or at least had the opportunity to do so. For a time, widespread use prevails. During this period, youths first coming of age typically initiate use of the currently popular drug or drugs, if any. These users form the core of a drug generation for whom the drug has particularly symbolic significance based in their social activities and relationships (Golub et al., 2005; Golub, Johnson, Dunlap, & Sifaneck, 2004).

Eventually, the use of an illicit drug tends to go out of favor, leading to a gradual decline phase of a drug epidemic. We conceptualize that new clusters of conduct norms emerge and these norms hold that use of a drug is bad or old fashioned. The subsequent diffusion-of-innovation process then competes with the prevailing pro-use norms. During the decline phase, a decreasing proportion of youths coming of age develop into users. However, the overall use of the drug endures for many years because some members of a drug generation continue their habits.

Figure 10.1 presents a graphical representation of the dialectic of mutual causation and its relationship to subcultural evolution, illustrating three recent drug epidemics. The downward arrow at the far left shows that individuals alive during the 1960s and 1970s were influenced by the values, norms, symbols, and behaviors of the heroin injection epidemic. Some individuals became involved with the use of the drug. For some, it came to give meaning and purpose to their lives. Preble and Casey (1969) observed how for junkies, taking care of business occupied their days. These users reproduced the prevailing subculture. The upward arrow from individual identities indicates how individuals perpetuated the prevailing culture and how decisions by youths during the early 1970s to avoid heroin produced the decline of the heroin injection epidemic. Similarly, individual behaviors produced and reproduced the subsequent crack and
Prevailing Subcultures

FIGURE 10.1. A conceptual representation of the subculture–identity dialectic of mutual causation.

marijuana–blunts epidemics, and changes in behavior produced the decline of the crack epidemic. The right arrow emanating from individual identity shows that the influences on a person’s life shift over time as new drug epidemics emerge. For example, many heroin users from the 1960s and 1970s added crack cocaine to their habits as this behavior became commonplace in the 1980s (Golub & Johnson, 1999).

During the crack epidemic, people who used crack attached symbolic importance to smoking cocaine freebase (e.g., crack or rock), which became central to a new vocabulary of expectations and associated with a range of new drug-related practices and norms (see Johnson, Golub, & Fagan, 1995; Waldorf, Reinarman, & Murphy, 1991; Williams, 1992). They maintained that smoking crack yields the greatest high. They went on runs or missions, jargon derived from Star Trek. These runs involved continuously hustling for money, obtaining crack, and using it without sleep or much food. By the mid-1990s, however, inner-city youths reacted against the violence, legal consequences, and personal devastation that befell heroin and crack users, often witnessing firsthand their neighbors hustling for money or police officers arresting them and carting them off to jail. During the decline phase of the crack epidemic in inner-city New York, crackhead became a dirty word; youths avoided peers whom they suspected of crack use (Furst, Johnson, Dunlap, & Curtis, 1999). The decline of the crack epidemic paralleled the emergence of the blunt generation. For many inner-city youths, blunts became the drug of choice. Youths would often proclaim, “Crackheads are shit! Heroin injection causes AIDS” (Golub et al., 2005, p. 224).

Career and Learning Perspectives
The subcultural evolution model explains subcultural formation, evolution, and dissolution; however, within this evolutionary process individuals can follow a dynamic career trajectory involving nonuse, initiation, experimentation, escalation, habituation, problematic experiences, treatment, abstinence, relapse, maturing out, retirement, and total abstinence (V. N. Shaw, 2002; Waldorf, 1983). The career perspective—only metaphorically associated with an occupational career in the traditional sense—holds that individuals move from one position to another and experience a series of separate but related experiences as they move through time, with each stage or position presenting opportunities and obstacles that dynamically shape the direction and nature of the career (Faupel, 1991). From a
career perspective, drug use is a dynamic process that plays out over time:

In its uniqueness, it (a) emphasizes the changing character of drug use and drug users; (b) stresses the accumulative effect of use or nonuse episodes over a sequence of progression or regression; and (c) attends to the interaction of user characteristics, drug factors, and social influences through an evolutionary process. . . . [The career perspective holds that] drug use is not a static state. It is a changing condition. Change in drug use and abuse does not just feature a stochastic process. It takes place through progressive or regressive stages that reflect both the effect of accumulation and the interaction of various influences in the process of change. Most saliently, drug use and its evolutionary change make different career pathways behind drug users, drugs, and social environments or historical eras. (V. N. Shaw, 2002, pp. 28–29)

During their dynamic drug-using career, individuals learn norms and behaviors via social interactions with others in parallel career stages. From a social learning perspective, individuals acquire drug-using behaviors and practices through social interaction—both symbolic and actual—with others (Akers, 1998; Becker, 1963). From this perspective, substance use is a learned behavior and involves learning the motivations and justifications of drug use as well as methods of administration and effect management (V. N. Shaw, 2002).

Researchers use the career and learning perspectives to explain an individual’s trajectory through stages of drug use and nonuse. For example, Charles Faupel (1991) built on Becker’s (1953, 1963) studies of marijuana users to explain career patterns of heroin users. Becker found that marijuana users learn how to get high, interpret their altered state of consciousness, manage the high, and integrate into subcultural groups or peer networks to ensure access. The time and effort that go into this process is analogous to a professional career pursuit (V. N. Shaw, 2002). Faupel then looked at drug initiation and escalation among heroin users, characterizing users’ careers as following four distinct stages. An occasional user has a high level of structure and low level of access to heroin; occasional users are able to maintain a professional work schedule while at the same time beginning to learn and refine injecting-drug–using practices. An occasional user can become a stable user after acquiring basic drug–using practices and establishing connections with sources. Life structure, conditioned by such factors as work or family, often prevents one from falling into excessively problematic drug use. When this life structure is uprooted, the stable user passes into the free-wheeling junkie career stage, wherein use becomes very high and often problematic; the user experiences a loss of control because structural routines no longer limit consumption. In Faupel’s framework, the user subsequently becomes a street junkie, characterized by low drug availability, weakened life structure, loss of conventional employment, and the breakdown of social life.

Not everyone progresses in their substance use. Some people become stable users. Others desist over time, sometimes with the aid of various treatments but other times on their own. As they mature, young adults often conceive of illicit drug use as incompatible with the cultural expectations associated with their new social roles as professionals, parents, or adult community members and reduce or eliminate drug use as they enter adulthood (Bachman, Wadsworth, O’Malley, Johnston, & Schulenberg, 1997; Chen & Kandel 1995; Pierce, 1999; Winick, 1963). Even individuals who were heavily involved and perhaps dependent have been known to stop using on their own, a process called natural recovery. Granfield and Cloud (1996) examined natural recovery among middle-class people addicted to drugs and alcohol who rejected formal treatment ideologies and modalities. Instead, they underwent a very personal identity transformation without formal assistance:

They [study participants] did not adopt this [addict] identity as a “master status” nor did this identity become salient in the role identity hierarchy. Instead, the
"Addict" identity was marginalized by our respondents. Alcoholics and addicts who have participated extensively in self-help groups often engage in a long-term, self-labeling process which involves continuous reference to their addiction. Respondents in the first stage of the present study, by contrast, did not reference their previous addiction as being presently central to their lives. Their comments suggest that they had transcended their addict identity and had adopted self-concepts congruent with contemporary roles. (Granfield & Cloud, 1996, pp. 57–58)

Granfield and Cloud (1996) illustrated how individuals' careers can entail a shift away from former drug-using norms and values and adoption of more conventional behaviors. In the process, via social learning, they often come to reject the addict label or identity and instead embrace one of a parent, professional, or adult member of the community. In this sense, one's career influences subcultural identification and can shape drug-using and non-drug-using identities and the associated norms, values, and behaviors. This finding suggests that one can learn to embrace normative behaviors just as one can learn to embrace subcultural ones, both conditioned by points in the drug career and life course of the individual (see also Mullen, Hammersley, & Marriott, 2005).

**FINAL THOUGHTS**

In this chapter, we have examined the role of setting as an integral part of the addiction phenomenon. An understanding of setting can help predict and minimize multiple manifestations of addiction and related compulsive or problematic behaviors. Indeed, many of the insights provided here with regard to substance use problems also apply to problems with other behaviors that have been associated with compulsiveness such as gambling, playing video games, having sex, eating, dieting, exercising, and working. What these behavioral problems have in common is that the value one associates with the activity can come to overwhelm other values such as family or work obligations and even attention to one's health. These problematic behaviors are what lead health professionals to label an individual as addicted. However, the values involved, even when dysfunctional, are embedded within a person's worldview that is the product of the prevailing subcultures. Hence, helping an individual overcome addiction can involve helping him or her understand society and his or her relationship to it. Ultimately, reducing the incidence of addiction might require transforming the prevailing subcultures experienced by those groups most likely to become addicted. This is the central relevance of the sociological perspective for the addiction syndrome.

**References**


At its core, the addiction syndrome model seeks to answer fundamental questions about both the etiology and the treatment of addiction: Who becomes addicted and why? Once addicted, who has the most difficulty quitting, and how can they best be helped? In guiding the field toward answers, the model advances the notion that the relationship or interaction between specific features of individuals and particular features of specific objects over time in specific contexts is what creates the conditions necessary for addiction and the conditions necessary for stopping problematic use. Interactions are a critical feature of the model.

In this chapter, we examine the nature of interactions in the addiction syndrome model. We accomplish this exploration in two ways. First, we review models and perspectives within the health behavior literature that treat interactions as a core feature of their conceptualizations. Second, we review, using a case study approach, interactions in the development of nicotine dependence via cigarette smoking using the addiction syndrome model as a guiding framework. We conclude the chapter by evaluating how well the addiction syndrome model captures interactions in a way that advances understanding of the etiology and treatment of addictive behaviors.

**CORE EXPLANATORY CHALLENGE**

The number of Americans who are actively dependent on legal and illegal substances, or who engage in any one of a number of putatively behavioral addictions (e.g., gambling, shopping), varies considerably. What is striking, though, is that a far greater number of people try or experiment with these objects of addiction than actually become addicted to them. Data from the longitudinal sample of the most recent (as of this writing) Monitoring the Future survey (Johnston, O'Malley, Bachman, & Schulenberg, 2011b) suggest that perhaps as many as 80% of 50-year-olds have tried marijuana at some point in their lives, whereas only 11% have reported use in the past 12 months; up to 75% have reported using some other illicit drug at some point in their lives, whereas 11% have reported being current users. With respect to cigarette smoking, estimates have suggested that only one third of people who experiment with smoking eventually become dependent (McNeil, 1991; U.S. Department of Health and Human Services [USDHHS], 1994); smoking prevalence rates tend to be highest during young adulthood and decline significantly during the later adult years (Centers for Disease Control and Prevention [CDC], 2009).

What these prevalence data imply, then, is that exposure to a given object of addiction is in itself not sufficient for addiction to develop. These data (as well as more detailed data to be reviewed later in this chapter) suggest that certain individual differences (either innate or learned) predispose or cause some people to be more and some people to be less susceptible to the effects of exposure to objects of addiction; the data also imply that external forces play a role in regulating the development of addictive behaviors.
disorders. These ideas are certainly not novel or new. They were evident—although inaccurately so—in early lay explanations for who became addicted to drugs and why. For example, moral weaknesses, lack of character, and spiritual deficits were at one point thought to uniquely distinguish those who became addicted from those who did not (reviewed by Jarvik, Cullen, Gritz, Vogt, & West, 1977; Roiblatt & Dinis, 2004). Formal interest in discovering who is more or less apt to develop an addiction or dependence on alcohol or drugs has been a core concern since the very early days of scientific investigation of the effects of how different people respond to drugs and alcohol (e.g., Hollingworth, 1924).

INTERACTIONS IN THE ADDICTION SYNDROME MODEL

The addiction syndrome model describes, at a largely metatheoretical level, how and where interactions are theorized to occur in the development, expression, and treatment of addictive disorders. Interactions in which one or more variables have an effect on one another on the road to addiction are either explicitly described or implicitly inferred at multiple points in the model (see Figure 1 in the Introduction to this handbook). Within the distal antecedents frame, neurobiological elements composed of genetic and neurobiological risks interact with psychosocial elements that include psychological and social risks to produce an underlying vulnerability to addiction. This underlying vulnerability then interacts with exposure to one or more putative objects of addiction. If these interactions lead to immediate neurobiologically positive shifts toward a given object of addiction, repeated object interactions driven by psychosocial antecedents in a premorbid phase eventually lead to an addiction syndrome with a focus on that expression of addiction. Individual differences in the expression of addiction and capacity of individuals to quit are implicit in the latter frame of the model. As a metatheory, though, the model is less specific in describing how these interactions operate and in describing what particular variables interact with particular contextual or object-specific features to generate addictive behavior. Part of the goal of this chapter is to unpack and add specificity to the broad-based interactions proposed by the model.

INTERACTIONISM: A BRIEF REVIEW OF CORE CONCEPTS AND ILLUSTRATIVE THEORIES

In this section, we review perspectives and theories that provide a broader context for understanding how interactions play out within the addiction syndrome model. This section is not intended to represent an exhaustive review of each perspective, nor is it intended to be an exhaustive review of every theory or perspective that deals with interactions; the depth and breadth of various disciplines, and perspectives within those disciplines, that involve interactions are far too extensive and detailed to be reviewed in the context of this chapter. A point-by-point comparison of concepts from more contemporary health behavior theories to concepts described by the addiction syndrome model is also beyond the scope of this chapter. Rather, our goal is to illustrate how interactions are examined and treated more generally in the health behavior literature with an eye toward understanding and evaluating how interactions are treated in the addiction syndrome model. We deal first with philosophy-of-mind issues that have addressed interactions. Next, we review two metatheories that contributed to the development of more specific theories in the psychological sciences. Finally, we discuss two specific, but comprehensive, theories of addiction: one addresses etiology and the other addresses treatment and relapse.

Philosophy of Mind

Philosophy of mind is a branch of analytic philosophy that is concerned with the nature, quality, and function of mental events and their relationship to the body, particularly the brain (Kim, 2006). Aside from debates about the substantive composition of the mind and body (literally, what the mind and body are made of), central problems that are addressed include whether and how the mind and body interact—the so-called mind-body problem. Classic dualistic thinking viewed the mind and body as separate entities that were incapable of influencing one
another (e.g., as exemplified in the writings of Plato and Aristotle). The central issue, it seemed, was that the mind (or soul, in some versions) could not be explained or examined in physical terms—in a religious sense, the soul was seen to exist eternally, whereas the body was viewed as a temporary vessel for the soul. As such, it was impossible to conceive of them interacting or influencing one another. Hundreds of years later, Cartesian interactionist dualism (most directly attributable to Descartes) was comfortable with the notion that the mind and body were separate entities but made up of different substances; with this recognition, there was appreciation that the mind and body were capable of interacting and influencing one another. In this view, interactions took place in the pineal gland. Others dealt with this mind–body interaction issue by invoking religion (occasionalism) or by proposing that the mind and body were composed of the same material, just different parts of it (double aspect theory). Others disregarded reciprocal interactionism between the mind and body altogether by proposing that the direction of influence is one way only: The body influences the mind, but the mind is incapable of influencing the body (epiphenomenalism).

Mind–body dualism dominated much of medicine and health behavior thinking and research for centuries, and there was not much interest in or appetite for understanding how mind and body as separate entities could influence one another (Gold, 1985). Although contemporary philosophy-of-mind approaches are generally comfortable with an interacting mind and body, debate concerning where and how those interactions take place continues (see Dennett, 1991). This is similar to our current understanding of addiction: Although interactions are broadly acknowledged as being important, the exact nature of those interactions and how they influence behavior continue to be open to debate, discussion, and revision.

Biopsychosocial Models

The generic biopsychosocial model was one of the first formalized scientific attempts to account for the diverse interactions between the “person [and] the social context in which he lives” (Engel, 1977, p. 132) and how they interact to contribute to both health and illness (see also Matarazzo, 1980). Designed to bridge gaps between strictly reductionist biomedical models and more integrative, contextually sensitive psychological–psychiatric accounts of illness and health behavior, the biopsychosocial model acknowledges that biological, psychological, and social processes operate interactively to fuel both health and illness. Early forms of the biopsychosocial perspective can be thought of as metatheory that organized scientific and clinical thinking around the idea of interactions among biological, psychological, and social variables as being not just important but fundamental to understanding health and disease. Specific statements about which psychological variables interacted with which social variables were notably absent in these early perspectives. However, as this general model gained momentum, theories that explicitly articulated these interacting variables flourished. Indeed, this general biopsychosocial sensibility has been hugely influential, touching nearly every domain in the broad field of health psychology. It has also been responsible for significant advances in the conceptualization, understanding, and treatment of any number of health and illness behaviors and disease processes (Baum & Posluszny, 1999; Suls & Rothman, 2004; Williams, Holmbeck, & Greenley, 2002).

For example, chronic pain, a persistent pain lasting for longer than 3 months or intermittent periods of pain lasting for years, had primarily been conceptualized within a biomedical framework. Nociception, stimulation of nerves that communicate potential acute tissue damage, was thought to be responsible for the subjective experience of pain (reviewed by Gatchel, Peng, Peters, Fuchs, & Turk, 2007). It became clear that this relatively simple model could not account for more complex features of pain that were commonly observed, including findings that the subjective experience of pain could continue far beyond healing of the injured tissue and evidence of a sometimes variable relationship between the extent of tissue damage and experience of pain (Melzack & Wall, 1996). The biomedical model gave way to more complex biopsychosocial theories that could better account for these and other phenomena. For example, gate control theory (Melzack & Wall, 1965) introduced
the idea that emotion and cognitive evaluations could modulate the subjective experience of pain. More recently, the neuromatrix theory of pain (Melzack, 2005) proposed that cognitive–evaluative, sensory–discriminative, and motivational–affective components, all integrated within a neural network, contribute to the subjective experience of pain. Research has supported aspects of these perspectives, including finding that affective experiences such as depression, anger, and anxiety (Robinson & Riley, 1999) and cognitive factors such as fear appraisals and beliefs all modulate the subjective experience of pain (Jensen, Romano, Turner, Good, & Wald, 1999).

Diathesis–Stress Models

The diathesis–stress models (see Ingram & Price, 2001), another class of metatheory in its early forms, posit that stress plays an integral role in the development of psychological disorders: People who are more susceptible to the effects of stress are more likely to develop psychological disorders. Single, person-centered individual differences variables or multiple individual differences variables were thought to interact with stress to sufficiently tip an individual into a pathological realm (Meehl, 1977). The general model was careful to distinguish between risk factors and vulnerability factors (Ingram & Price, 2001). Risk factors, as conceptualized, are significantly correlated with symptoms of psychological disorders without necessarily being causally related to the symptoms, whereas vulnerabilities are expected to interact with and modulate an individual’s responses to stress to specifically cause a disorder.

These general concepts of the interaction between individuals and stressors have been more specifically defined to explain the development of any number of psychological disorders. For example, it has long been postulated that increased levels of stress can contribute to development of depression (Firk & Markus, 2007) but that not everyone who is exposed to stress becomes depressed. Research has identified a specific genetic polymorphism relevant to serotonin transport (i.e., two copies of the s allele of the 5-HTTLPR polymorphism vs. two l alleles) that produces an increased sensitivity to the impact of stressful events that, in turn, increases the likelihood of depression (Gotlib, Joorman, Minor, & Hallmeyer, 2008; Kendler, Kuhn, Vittum, Prescott, & Riley, 2005). In schizophrenia, current diathesis–stress models posit that preexisting dysregulation in hippocampal–prefrontal function leads to decreased inhibition of the amygdala and subsequent higher arousal levels in response to even minor environmental stressors (Preston, 2005; Walker, Mittal, & Tessner, 2008).

Theory of Triadic Influence

The theory of triadic influence (TTI) is a comprehensive model that accounts for the influence of multiple variables at different levels of analysis on the development of addictive disorders (Flay & Petraitis, 1994; Flay, Snyder, & Petraitis, 2009; Petraitis, Flay, & Miller, 1995). According to the TTI, the development of addictive behaviors can be accounted for by three classes of influences (i.e., personal, social, environmental) that operate within varying degrees of proximity to the individual (i.e., ultimate underlying causes, distal influences, proximal influences). This configuration produces a matrix in which, for example, ultimate personal causes, such as biological predispositions, have their effect on substance use through distal causal influences such as self-control, which subsequently have their effects on use through effects on proximal factors such as self-efficacy and use intentions. The TTI is careful to point out that the influence of any of these factors is likely multiply mediated through myriad other factors and that interactions between variables in the model likely occur dynamically over time. The TTI also integrates theories on adolescent development into the model, specifically discussing neurobiological (e.g., remodeling of dopaminergic circuitry; see Steinberg, 2006) and psychosocial (e.g., peers; see Kobus, 2003) influences that uniquely characterize adolescence and contribute to the upsurge in addictive behaviors (or risk behavior more generally) during this life stage. Together with trial drug use behaviors and experiences over time, these factors are expected to determine whether addiction develops (Flay et al., 2009).

The TTI, broadly a biopsychosocial model with diathesis–stress overtones, is distinguished by its
sheer comprehensiveness, complexity, and specificity in attempting to account for almost all of the known pathways that contribute to addictive behaviors. A major strength is that it provides myriad testable hypotheses about the reciprocal mediated and moderated effects of different variables on the development of substance use disorders. Indeed, reviews of the TTI have indicated much support for some of the features and single-level mediational pathways described by the model (Flay & Petraitis, 1994; Flay et al., 2009; Petraitis et al., 1995). However, a comprehensive evaluation of the interacting influence of all possible factors, variables, and pathways in the model has not yet been conducted (but see Busseri, Willoughby, Chalmers, & YLC-CURA, 2005, for an attempt to evaluate numerous TTI pathways within a single model).

**Relapse Prevention Theory**

Relapse prevention theory was advanced in the early to mid-1980s to describe the different cognitive variables that contribute to relapse after a period of abstinence from an addictive substance (see Marlatt & Gordon, 1985). The basic model posited that recently abstinent substance users are likely to encounter situations in which they are at high risk for lapsing. If they fail at coping in this high-risk situation, they are theorized to experience decreased self-efficacy and increased positive outcome expectancies for the effects of their substance of choice. These cognitive responses to that high-risk situation are seen as critical to determining whether they subsequently have a brief period of substance use (a lapse) as a consequence of exposure to that high-risk situation. Their cognitive reactions to this lapse, termed the abstinence violation effect, determine whether this lapse becomes a relapse (i.e., resumption of regular substance use).

More than 2 decades' worth of research investigating the predictive utility of variables important to the original relapse model has generally shown that these variables predict or are associated with smoking lapse and relapse (reviewed by Witkiewitz & Marlatt, 2004). However, this body of literature has also indicated that the task of predicting relapse is much more nuanced and increasingly more complex than what the original model initially suggested. Condensing this 2 decades' worth of research on relapse more broadly, Witkiewitz and Marlatt (2004; Marlatt & Donovan, 2005) introduced a dynamic model of relapse that advances the notion that the interaction of tonic (i.e., background or distal factors, e.g., family history, dependence, withdrawal) and phasic (i.e., contextually bound factors that change dynamically in the moment, e.g., self-efficacy, coping, craving) processes are critical to understanding relapse. For example, research with smokers has found that in contrast to the original relapse model (Marlatt & Gordon, 1985), self-efficacy acts as a phasic variable, whereas smoking outcome expectancies act as a tonic variable in predicting relapse after a lapse (Gwaltney, Shiffman, Balabanis, & Paty, 2005; Shiffman et al., 2000). More broadly, this new model characterizes relapse as a multidimensional, complex system in which “multiple influences trigger and operate within high-risk situations and influence the global functioning of the system” (Witkiewitz & Marlatt, 2004, p. 229). The exact features and nature of the interactions in this relatively new complex, dynamic system have yet to be clearly delineated.

**Summary**

Several conclusions can be drawn from this brief review. First, interactions are key components of understanding behavior in some of the very earliest philosophical approaches that dealt with health behavior and addictive behaviors. Second, metatheoretical perspectives that discussed interactions more generally (e.g., biopsychosocial approach) gradually gave way to more specific theories that carefully described testable interactions in particular domains, including addiction. Third, specific biopsychosocial theories that view interactions as a core feature of both the etiology of addiction and the process of stopping an addictive substance or behavior have been advanced and tested in the past decade, although comprehensive theories that describe interactions throughout an individual’s substance-abusing life—from preaddiction to treatment and recovery—have not been as well articulated. In this context, the addiction syndrome model functions as a metatheoretical framework in that it emphasizes
the importance of interactions and makes a notable contribution to the literature by providing a framework for examining addiction over the life course. In the next section, we evaluate the interactions implied by the addiction syndrome model by using cigarette smoking as the object of a case study.

CASE STUDY: CIGARETTE SMOKING AND THE DEVELOPMENT OF NICOTINE DEPENDENCE

Although individual- and contextual-level factors are expected to vary widely across individuals, some objects are more commonly associated with addiction than others. The most commonly identified drug objects of addiction are listed by the Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision; American Psychiatric Association, 2000) as dependence-producing substances: alcohol, amphetamines, cannabis, cocaine, hallucinogens, inhalants, nicotine, opioids, phencyclidines, sedatives, hypnotics, and anxiolytics. Although there is considerable debate about what objects constitute nondrug expressions of addiction and whether these should be included in the official diagnostic nomenclature (Holden, 2010; Potenza, 2006), research, clinical reports, and anecdotes have identified gambling, exercise, sex, shopping, Internet use, online game playing, and eating junk food as potentially addictive behaviors (Frascella, Potenza, Brown, & Childress, 2010).

In this diverse context, we have chosen to focus on cigarette smoking. We have taken this approach for several reasons. First, there is no debate that the nicotine in cigarettes produces dependence (Stolerman & Jarvis, 1995). Second, the public health consequences of cigarette smoking far exceed those of these other drugs (CDC, 2005; USDHHS, 1989). Third, as reviewed previously, many more Americans experiment with and regularly smoke cigarettes than almost all other drugs (with the exception of alcohol; see Johnston et al., 2011b). Finally, the state of the literature on cigarette smoking—from initiation to regular smoking to cessation—has both depth and breadth, whereas the literature for various potential objects of addiction (particularly some of the behavioral expressions of addiction, notwithstanding gambling; see Sharpe, 2002) is variable in terms of its depth and breadth.

The selection of smoking as our case study in no way implies that addiction to other drugs or other putatively behavioral expressions of addiction are not highly consequential to the overall health and well-being of people who use them. Indeed, including costs related to the use of resources to address health, crime consequences, and the loss of potential productivity from disability, death, and withdrawal from the workforce, the economic cost of drug abuse is estimated at half a trillion dollars per year (National Institute on Drug Abuse, 2008; Office of National Drug Control Policy, 2004). Rather, our goal is to evaluate the development and cessation of cigarette smoking with respect to interactions as described by the addiction syndrome model, with a broader goal of informing how other expressions of addiction might express themselves and what factors need to be considered when thinking about interactions in the context of the model.

The case study is organized in the following way. First, we review basic epidemiology about cigarette smoking. Second, we discuss how individual differences and interactions may be expressed within each of several levels of the addiction syndrome model and the manner in which interactions express themselves in those levels. The levels are presented in a hierarchical fashion and in relatively (and artificially) "pure" form so that the conceptual picture of how nicotine dependence might develop and how smokers might quit within the framework of the addiction syndrome model is clearer. This sort of hierarchical organization provides insight into how individual differences and different interactions build on those that precede them; for example, how neurobiological and psychosocial vulnerabilities set the stage for individuals with certain vulnerability profiles to be more susceptible to the effects of smoking-conducive environments and initial smoking episodes.

As we engaged in constructing this case study, though, three issues emerged. First, there are multiple implied interactions and potential points of influence within each of the "boxes" designed to represent elements of the model. For example, within the model, psychosocial elements represent
both psychological and social risk factors, which likely influence one another in a dynamically reciprocal way (see Bandura, 2006). Second, within the model more generally, there appears to be a missed opportunity to discuss how exposures to environmental stimuli and direct object exposures influence variables within the psychosocial elements box. Third, the addiction syndrome model does not account for specific genetic or neurobiological vulnerabilities to specific objects of addiction, a specificity for which our review of nicotine dependence suggests there is evidence. In our presentation, we organize our thinking about the model by including elements that are relevant to these issues.

Cigarette Smoking: Basic Epidemiology, Costs, and Treatment
More than 440,000 people in the United States die each year from tobacco-related illnesses such as lung cancer, chronic obstructive pulmonary disease, and ischemic heart disease (CDC, 2008). At present, approximately 20% of eighth graders have reported ever smoking, compared with 42% of 12th graders, and 7% of eighth graders have reported smoking at least once in the past 30 days compared with 19% of 12th graders (Johnston, O’Malley, Bachman, & Schulenberg, 2011a). Many people smoke for the first time during college (Choi, Harris, Okuyemi, & Ahluwalia, 2003), and young adults who do not attend college smoke at higher rates than college students (Johnston et al., 2011b). About 19% of adults (age 24 and older) have reported currently smoking some days or smoking every day (CDC, 2009). Although the overall prevalence of adult smokers has decreased since the early 1960s, rates of decline have stalled (CDC, 2010), and some segments of the population (e.g., those of lower income or who are chronically ill) smoke at rates that are nearly 3 times higher than national prevalence estimates (Borrelli, 2010). As a consequence, smoking continues to contribute to significant levels of morbidity and mortality in the United States (USDHHS, 1994). Each year, people who smoke cost the economy $97.6 billion in lost productivity and an additional $96.7 billion on public and private health care combined (CDC, 2005).

Community-based interventions (Sowden & Stead, 2008), school-based interventions (Thomas & Perera, 2006), and media-driven interventions (Sowden, 2008) can reduce smoking among adolescents in the short term. However, smoking prevention programs do not typically have effects on reducing smoking over the long term (e.g., Dobbins, DeCorby, Manske, & Goldblatt, 2008). Among adult smokers, only a small percentage are able to quit cold turkey (Fiore et al., 2008). A range of formal smoking cessation treatments has therefore been developed. Unfortunately, however, quit rates for such programs are low and have been static during the past 2 decades. In addition, relapse after a quit attempt remains the modal outcome for any given quit attempt (Niaura & Abrams, 2002; Piasecki & Baker, 2001). Research has begun to examine smoking cessation interventions for adolescent smokers, but more research is needed before specific treatments can be recommended (Curry, Mermelstein, & Sporer, 2009).

Neurobiological Influences
There are two broad classes of neurobiological influences: shared influences, those that can be applied to most objects of addiction; and nicotine-specific influences, those that apply to nicotine as the object of addiction and no other addiction objects.

Shared reinforcement mechanisms. Researchers have identified several biological variables that appear to be associated with vulnerability to addiction in general. For example, propensity for activation of the mesolimbic dopaminergic pathways in the ventral striatum, and in the prefrontal cortex contributes to the likelihood of developing substance dependence (Di Chiara & Imperato, 1988; Pierce & Kumaressan, 2006; Spanagel & Weiss, 1999). These areas in particular are replete with dopamine receptors (Wise & Bozarth, 1987). When these receptors are stimulated, increased levels of dopaminergic activity, and thus reward, follow. With repeated use and continued activation of dopaminergic pathways, the potential of the substance to produce dependence increases (Bozarth, 1994; Koob, 1992). Multiple objects of addiction stimulate the release of...
dopamine in the mesolimbic system, including opi­ates, alcohol, cannabinoids, and nicotine (Pierce & Kuna­resan, 2006). Behavioral objects of addiction such as gambling (Voon et al., 2007), sexual behavior (Fiorino & Phillips, 1999), and food (Hanlon, Baldo, Sadeghian, & Kelley, 2004) are also associated with a propensity for dopaminergic activation. Individual differences in the dopaminergic systems responsible for reward are likely heritable (Dreher, Kohn, Kolachana, Weinberger, & Berman, 2009). Vulnerable individuals may exhibit what has been termed a reward deficiency syndrome (Blum et al., 2000), in which their capacity to experience reward from the primary dopaminergic pathways (among others) is depleted; as a consequence, they engage in activities, such as drug use behavior, that stimulate this reward circuitry. With such vulnerability in the dopaminergic system, exposure to environments or circumstances conducive to engagement with any number of objects of addiction may be critical in moving individuals toward one or another object of addiction.

**Nicotine-specific influences.** Although individuals with vulnerabilities in their dopaminergic systems may be exposed to any one of a number of potential objects of addiction, the risk of developing addiction to one object over another may be increased by object-specific genetic or neurobiological vulnerabilities. Certainly, more specific vulnerabilities may be required to develop dependent levels of cigarette smoking. Studies have shown that genetic variations are strongly and specifically associated with the development of nicotine dependence (Bierut, 2009; Kendler, Myers, & Préstcott, 2007; Quaak, van Schayck, Knaapen, & van Schooten, 2009). The results of twin studies have suggested that the heritability of nicotine dependence is approximately 50% to 65% (Kendler et al., 2007; Lessov et al., 2004), much of which may be accounted for by heritability of variants of nicotinic receptors. Variants of nicotinic receptors have been shown to distinguish between smokers who become nicotine dependent and those who never report any symptoms of nicotine dependence; variants of nicotinic receptors are also related to how much people smoke (Amos et al., 2008; Berrettini et al., 2008; De Luca et al., 2004; Lou et al., 2006; Saccone et al., 2007). Variants of genes related to nicotine metabolism also contribute to specific increases in risk for nicotine addiction (reviewed in Quaak et al., 2009). Variation in the speed at which nicotine is metabolized is related to how long nicotine circulates in the bloodstream, how soon individuals experience symptoms of nicotine withdrawal after smoking abstinence, and the development of nicotine dependence. For example, people who metabolize nicotine quickly have more frequent dips in blood nicotine levels, feel withdrawal symptoms sooner than others, and are therefore driven to smoke more often. Consequently, these “fast metabolizers” are also more likely to become nicotine dependent than their peers who metabolize nicotine more slowly (Pianezza, Sellers, & Tyndale, 1998; Schoedel, Hoffman, Rao, Sellers, & Tyndale, 2004; Tyndale, Pianezza, & Sellers, 1999).

In short, although many genes (in conjunction with environmental risk factors; see next section) likely contribute to the development of the disorder (Bierut, 2009), at the time of this writing data suggest that nicotine dependence has a hereditary component that is nicotine specific (Bierut, 2009; Kendler et al., 2007; Quaak et al., 2009).

**Social Influences**

Social influences can be described as the processes whereby people directly or indirectly influence the thoughts, feelings, and actions of others. As an example, modeling is learning that occurs through watching others engage in behaviors that are appealing and reinforcing (see Bandura, 2006). Different social environments may provide different opportunities for modeling and hence contribute to different vulnerabilities to addiction. Among youths who have not yet tried smoking, a strong predictor of later experimentation with cigarettes is having friends or family members who smoke (Ennett et al., 2010; Huang, Unger, & Rohrbach, 2000; Kaufman et al., 2003). Also important, changes in peer networks may be associated with changes in smoking patterns. For example, nonsmoking adolescents who increasingly hang out with smokers are at higher risk for smoking (Ennett & Bauman, 1994). Adolescents who have closer relationships with smoking
friends are more likely to smoke themselves (Ennett et al., 2010). Neurobiologically vulnerable adolescents who find themselves in such situations may be particularly susceptible to these social modeling effects.

Advertising and Marketing Influences
Another external variable that influences adolescent smoking behavior is cigarette advertising and marketing and exposure to smoking in movies. The tobacco industry has for close to 30 years spent well over a billion dollars each year on advertising and marketing (Federal Trade Commission, 2009). These vast expenditures are of great concern: Studies have documented that increases in tobacco advertising budgets contributed to an increase in smoking initiation among children and adolescents (Gilpin & Pierce, 1997). Young people are more likely than adults to be influenced by cigarette advertising (Pollay et al., 1996). Meta-analytic results of 51 studies and more than 140,000 participants clearly showed that increased exposure to cigarette advertising and marketing is associated with increases in adolescent smoking; the effects of advertising exposure on initiation were significantly larger than the effects on progression to regular smoking (Wellman, Sugarman, DiFranza, & Winkoff, 2006). The thinking behind this finding is that environmental factors, such as exposure to cigarette advertising, have their most potent effects in the early parts of an individual's smoking life and that later smoking (e.g., the progression from experimental to regular smoking) is driven more heavily by biologically relevant processes relating to nicotine dependence (USDHHS, 1994).

The Master Settlement Agreement in 1998 (Koh, 1999) and the Family Smoking Prevention and Tobacco Control Act, signed into law in June 2009, changed how and where cigarettes are advertised. A primary goal was to reduce the chances that adolescents would be exposed to cigarette advertising and marketing. For example, the Master Settlement Agreement restricted how cigarette print ads can be constructed (e.g., no cartoon characters) and where advertisements can be placed (e.g., distance from schools). In this restrictive marketing climate, other potential areas of smoking exposure, for example, via movies and television, have become an increasing concern. Adolescents ages 10 to 14 are exposed to hundreds upon hundreds of smoking impressions in movies every year (Sargent, Tanski, & Gibson, 2007). Numerous studies have linked exposure to smoking in movies with adolescent smoking initiation (see Wellman et al., 2006). Emerging work has identified the way in which smoking is portrayed by characters who smoke in movies (e.g., smoking to relax) as particularly influential in orienting adolescents toward smoking (Shadel, Martino, Haviland, Setodji, & Primack, 2010).

Cognitive Influences
Social and peer influences and advertising and marketing do not operate on adolescent smoking in isolation; they seem to exert their effects through social learning processes (Bandura, 2006). Greater exposure to cigarette smoking in movies increases positive expectations about smoking and perceptions of the prevalence of smoking, each of which increases susceptibility to engage in smoking behavior (i.e., increased intentions to smoke and decreased self-efficacy to resist offers to smoke; Sargent et al., 2002; Wills, Sargent, Stoolmiller, Gibbons, & Gerrard, 2008). Greater exposure to movie smoking is also related prospectively to increased affiliation with peer smokers, which in turn raises the likelihood of smoking onset (Wills et al., 2007, 2008). The theory of reasoned action (Fishbein & Ajzen, 1975) and the newer, broader integrative model of behavioral prediction (see Fishbein, 2000) organize the diverse cognitive social learning variables that are thought to mediate the effects of social and advertising influence on adolescent risk behavior. In the integrative model, intention to perform a behavior is a key predictor of behavior. Intention is itself viewed as a function of four determinants: (a) attitudes (i.e., the relative weight of positively and negatively valenced beliefs) about the behavior; (b) perceived norms (i.e., beliefs about what others in the individual's relevant reference group are doing) about the behavior; (c) perceived control of the behavior (i.e., beliefs about one's capabilities for engaging in the behavior or not); and (d) perceived health risk
associated with engaging in the behavior (i.e., degree to which the individual's health will be jeopardized by engaging in the behavior). Indeed, a large body of research has shown that prosmoking attitudes, norms, and social models appear to increase susceptibility to smoking among non-smoking youths (Leatherdale, Brown, Cameron, & McDonald, 2005). In several large samples of adolescents, behavioral intention to smoke was found to predict the progression to regular smoking (Choi, Gilpin, Farkas, & Pierce, 2001; Wakefield et al., 2004). Attitudes and subjective norms are significant predictors of behavioral intention to smoke (Newman, Martin, & Ang, 1982; O'Callaghan, Callan, & Baglioni, 1999). Self-efficacy (Ajzen, 1985) is a strong predictor of smoking intentions and behavior (Godin, Valois, Lepage, & Desharnais, 1992; O'Callaghan et al., 1999). Perceived risk of smoking-related illness has also been shown to influence smoking intentions (Halpern-Felsher, Biehl, Kropp, & Rubenstein, 2004) and smoking behavior in adolescents (Hampson, Andrews, Barckley, Lichtenstein, & Lee, 2000). Thus, adolescents are more likely to smoke if they have a strong intention to smoke in the future, and their intentions will be strong if they have relatively more positive attitudes about smoking, perceive smoking as normative for people their age, have low self-efficacy for refusing smoking, and perceive the health risks of smoking to be low (Fishbein, 2000).

Object Interactions
What happens when a vulnerable individual (e.g., with dopaminergic and nicotinic sensitivities, with psychosocial vulnerabilities) is exposed to environments that are conducive to smoking and initiates smoking? Although the person might exhibit vulnerabilities that predispose him or her to eventual nicotine dependence, there is no evidence that these vulnerabilities lead to immediate nicotine dependence on a single episode of experimental smoking. Nonetheless, first experiences with cigarette smoking seem to be especially important to determining the propensity for continued use. Retrospective studies of adolescent ever-smokers' first experiences with smoking have suggested that positive first experiences (i.e., no aversive effects such as nausea or light-headedness) are associated with greater chances of continued smoking (Bewley, Bland, & Harris, 1974; Pomerleau, Pomerleau, & Namene, 1998). Individual differences in these initial-use episodes may reflect underlying neurobiological sensitivities, as discussed earlier (see also Pomerleau, 1995). Social context seems to moderate initial use experiences as well. First-use episodes that occur with other, more established smokers are predictive of continued use (Friedman, Lichtenstein, & Biglan, 1985; Hahn et al., 1990). In addition, adolescents may use smoking to facilitate group interaction.
(Mermelstein, 1999). Continued repetitive interactions are among the most striking characteristics of cigarette smoking. In fact, cigarette smoking develops into a behavior that is repeated with a regularity that is unequalled by other substances of abuse. Because smoking is possible throughout the day and because the effects of nicotine are rapid but short lived, smoking is frequent: The average smoker in the United States—at a pack a day—smokes more than 7,000 cigarettes a year, or more than 85,000 puffs a year. Clearly, then, multiple exposures to nicotine are easily achieved with cigarette smoking within a relatively short period of time, and interactions may occur in a variety of settings and circumstances.

Nicotine Dependence Syndrome: Characteristics and Treatment
In the mature smoker, a well-recognized nicotine dependence syndrome has been established (Shadel, Shiffman, Niaura, Nichter, & Abrams, 2000; see also Edwards & Gross, 1976). Nicotine dependence is a syndrome insofar as it represents a broad hypothetical construct designed to summarize many different, various behavioral and physiological features that appear when an individual uses substances that have abuse liabilities. The syndromal conception suggests that nicotine dependence is most profitably viewed as a construct summarized or indexed by combinations of symptoms and behaviors. Features that characterize nicotine dependence include the rewarding properties of nicotine, tolerance that develops over time, and withdrawal that occurs on cessation of smoking. These features correspond somewhat with the more formal diagnostic criteria (American Psychiatric Association, 2000). Although precise measurement of nicotine dependence has remained elusive, more recent efforts at developing questionnaires that reflect the diversity of experience and syndromal nature of dependence have been developed and validated (Shiffman, Waters, & Hickcox, 2004).

In addition to nicotine dependence, multiple individual differences are evident in the dependent smoker. For example, smoking rate, quit history, risk perceptions, motivation, withdrawal, and the situations that trigger smoking and coping can all vary considerably between individual smokers (see Cervone, Orom, Aristicco, Shadel, & Kassel, 2007; Shadel, Cervone, Niaura, & Abrams, 2004; Shadel, Niaura, & Abrams, 2000). These individual differences in the smoking habit (broadly defined) become a core concern during treatment, where they are a focus of any one of a number of different treatment strategies (e.g., self-monitoring to address variations in smoking rate and in the cues that trigger smoking; motivational interviewing to address motivation and risk perceptions; see Brown, 2003). However, despite a broad appreciation for these differences, smoking cessation programs (see Brown, 2003) are offered as “one size fits all”; the same treatment (e.g., stimulus control, coping skills training, content about the health effects of smoking and benefits of quitting) is offered to everyone. In fact, current consensus recommendations (Fiore et al., 2008) are to treat every smoker with both some form of cognitive–behavioral intervention and some form of pharmacotherapy and to offer all smokers evidence-based treatments, regardless of what the results of assessment show.

The addiction syndrome model implies, though, that treatments should be targeted to individual differences to improve treatment outcomes. However, patient–treatment matching interventions have almost never been demonstrated to be effective (Fiore et al., 2008; Kassel & Yates, 2002; Niaura & Abrams, 2002; Shadel & Shiffman, 2005; Shiffman, 1993; see also Shadel, 2010; cf. Niaura, Goldstein, & Abrams, 1994), primarily because the field lacks different treatment options that can be administered to individuals who differ on some validly assessed dimension. Formally matching different treatments to different patients is a complex endeavor because of the inherent heterogeneity of any number of characteristics between individuals within a given diagnostic label and the multiple components of a given treatment to which a given patient may respond (see Haynes, 1993). Although clinicians intuitively match treatments to patients, despite problems with clinical decision making more generally (Garb & Boyle, 2003), the search for empirically supported patient–treatment matching algorithms to improve on this clinically intuitive approach has proven unsuccessful.
CONCLUSION AND COMMENT

In this chapter, we have examined how interactions have been treated in philosophy, in exemplar metatheories, and in specific theories of substance use etiology and cessation—relapse. Broader philosophical and metatheoretical perspectives with an appreciation for interactions gave way over time to more specific theories in particular domains that attempt to more clearly describe how interactions between specific variables occur and influence behavior. The addiction syndrome model functions very much as these metatheoretical perspectives do in that it provides a blueprint for understanding the importance of interactions in the etiology and treatment of addiction. What is striking across all of the perspectives reviewed, however, is that the exact direction of these interactions and how they influence substance use behavior through other pathways and interactions are often elusive. Even in the most specific theories, the TTI and relapse prevention theory, many interactions are underspecified or untested, not (yet) supported by direct evidence.

Part of the reason may be that the field has only recently come to terms conceptually with the complexity and sheer magnitude of factors that seem to be important to the etiology, maintenance, and cessation of addiction and analytically with how to test these complex models (e.g., see Hufford, Witkiewitz, Shields, Kodya, & Caruso, 2003; MacKinnon & Lockwood, 2003). It might also be the case that the type of longitudinal studies that are needed to evaluate these more complex models of health behavior are challenging to implement within the current funding context and climate (but see Mabry, Olster, Morgan, & Abrams, 2008, for a detailed discussion of this issue). Overall, then, the addiction syndrome model is very much in line with emerging metatheoretical perspectives in the substance use literature that place a priority on the role that interactions play in the etiology and treatment of addictive behaviors.

Our review also has brought out several challenges facing the addiction syndrome model. First, in light of models such as the TTI (Flay et al., 2009), which devotes considerable time to specifying detailed hypotheses about many of the interactions and pathways on the road to addiction, the addiction syndrome model underspecifies these interactions and pathways. Second, the addiction syndrome model does not account for the interacting developmental factors that seem to play a role in the etiology of substance use behavior, particularly during adolescence (see Steinberg, 2006). Third, the model does not unpack key variables in specific domains, nor does it describe the temporal precedence for these variables clearly; for example, the reciprocal relationship between cognitive and environmental variables is underspecified in the model (Bandura, 2006). Fourth, our case study with cigarette smoking has revealed that in contrast to assertions by the model, there does appear to be genetic and biological specificity with regard to the development of nicotine dependence. Finally, the model does not offer recommendations for how to handle multiple and varied interactions. These challenges suggest that any application of the model to an understanding of interactions in the etiology and treatment of addictive behaviors needs to be much more specific before testable hypotheses can be generated.

Yet despite these challenges, the addiction syndrome model fills an important gap in the metatheoretical literature on the problem of addiction. In particular, the addiction syndrome model offers a perspective on the development, maintenance, and cessation of addictive behaviors that other models do not (e.g., the TTI is etiological, and relapse prevention is treatment oriented). This feature of the model and its appreciation for the dynamic interactions that occur over the entire history of an individual’s pre–substance-abusing and substance-abusing life—at least through the end of treatment—make it stand out among other metatheoretical perspectives. It also makes the model a viable framework for more specific theories about the etiology and life course of addiction.

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Researchers and others have typically defined addiction with reference to the compulsive-like use of psychoactive substances (including tobacco). Yet many experts now consider behaviorally expressed addictive behaviors as legitimate members of the addiction spectrum; these behavior patterns include excessive or indulgent gambling, eating, sexual behavior, shopping, and use of electronic devices (e.g., Internet, video games, and cell phones). People have characterized addiction by impairment in behavioral control, craving, inability to consistently abstain, and diminished recognition of significant problems with one’s behaviors and interpersonal relationships (Shaffer, 1999). As with other chronic diseases, addiction involves cycles of relapse and remission, and it can be progressive and result in disability or premature death (Latt et al., 2009).

Throughout history, scholars and writers have theorized about why some human beings develop addiction. These explanations have incorporated several perspectives—evolutionary, cultural, behavioral, biological, psychological, and sociological. A current and widely discussed theory in the literature is that people engage in addictive behaviors because doing so creates excitement and reward in the brain (Leshner, 1999). As with other chronic diseases, addiction involves cycles of relapse and remission, and it can be progressive and result in disability or premature death (Latt et al., 2009).

However, individuals vary considerably in the extent to which they are susceptible to developing addiction. This variability most likely has biological roots (Blum et al., 2000); however, various social factors also contribute to the onset and course of addictive behaviors. As with biogenetic factors, social factors can increase or decrease the risk for addiction (Evans & Kantrowitz, 2002; Shaffer, Vander Bilt, & Hall, 1999). To illustrate this, we turn to gambling as an example. Researchers have argued that individuals at risk for pathological gambling (as defined in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders; American Psychiatric Association, 1994) suffer from a genetically based reward deficiency syndrome in which alterations in the person's brain chemistry interfere with the brain's reward process (Petry, 2005). This perspective further holds that this neurochemical abnormality appears to supplant normal feelings of well-being with negative feelings (Petry, 2005). Thus, such individuals might be at greater risk to gamble to seek relief from chemically based emotional distress. However, personal involvement in gambling most certainly involves social factors.
The proximal and distal social rewards associated with gambling can influence innate risk-taking tendencies in humans. These social factors can improve the emotional rush from a group experience of winning when one is with friends, including the expected social rewards that might arise from a big payoff.

Another social lens through which to view the role of social factors in addiction pertains to the transitional developmental period that occurs during high school and college. This period is a critical developmental period during which many young people abruptly become part of social units that are free from the type of daily parental monitoring that occurred while they resided at home. This period is commonly associated with escalations in a range of potentially addictive related behaviors, including the use of alcohol, tobacco, illicit drugs, and gambling (Kassel, 2000; H. R. White et al., 2006; Winters, Stinchfield, Botzet, & Slutske, 2005).

In this chapter, we explore social factors associated with addiction by examining the intersection of social influences and addiction from four select perspectives: (a) social environmental factors that contribute to the onset of use and progression to a substance use disorder among adolescents, (b) impact of the sociopolitical movement to change the minimum legal drinking age, (c) social factors and college student drug use, and (d) how social factors are integral to the treatment experience of those with a substance use disorder. We chose these topics because they provide a sampling of the diverse ways in which social factors have a role in addiction. We discuss social pathways that might contribute to the development of an addiction as well as promote recovery from it. In this light, each topic represents a case study of how social factors can influence either the etiology of or recovery from addiction.

We also selected these topics because each is associated with a relatively rich and long-standing empirical research base and each provides high-profile examples of the importance of social factors in the understanding of addiction. As a group of topics, they support the view that sociological factors are part of the multiple and interacting elements or basic factors, including biological, psychological, and experiential sources, that emerge as antecedents or expressions of those who have addiction syndrome. This view rests on the relatively consistent finding in the literature that social elements combine with other factors that influence an individual’s level of risk for developing an addiction (e.g., Clayton, 1992) and contribute to a person’s course of addiction (Kendler, Myers, & Prescott, 2007).

SOCIAL ENVIRONMENTAL FACTORS AND ADOLESCENT SUBSTANCE USE DISORDERS

A wealth of research has now shown that determinants of a prominent expression of addiction observed among adolescents—substance use disorders (SUDs)—include a wide range of individual, peer, family, and community risk factors (Catalano, Haggerty, Hawkins, & Elgin, 2010; Clark & Winters, 2002; Clayton, 1992). These factors reflect both genetic and environmental influences (Kendler, Myers, & Prescott, 2007) and create either risk for (contributing to) or protection from (protecting against) the development of a SUD. No single factor is, by itself, a reliable predictor of future drug use or SUDs. Yet, a large body of research has suggested that the more risk factors associated with an adolescent, the greater the likelihood that he or she will abuse drugs, and conversely, the more protective factors present, the lower the likelihood of subsequent drug abuse (Clark & Winters, 2002; Hawkins, Catalano, & Miller, 1992). However, risk and protective factors are not destiny; these factors provide a general gauge as to the likelihood of a young person using drugs and developing a drug problem. Some youths with many risk factors will not use drugs, and some youths with many protective factors will use and abuse drugs. Table 12.1 provides a list of risk and protective factors identified by the National Institute on Drug Abuse (1997).

Multiple organizational levels (Bronfenbrenner, 1977, 1986), ranging from the prenatal environment to cultural influences, might further classify environmental factors that contribute to the development of addictive behaviors. For instance, a wealth
TABLE 12.1

Risk and Protective Factors Associated With Onset and Maintenance of Drug Use by Adolescents

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Protective factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chaotic home environment</td>
<td>Strong bonds with the family</td>
</tr>
<tr>
<td>Ineffective parenting</td>
<td>Parental monitoring with clear rules of acceptable conduct within the family</td>
</tr>
<tr>
<td>Lack of mutual attachments to family members</td>
<td>Involvement of parents in their children’s lives</td>
</tr>
<tr>
<td>Excessive shyness or aggressiveness in the classroom</td>
<td>Success in school performance</td>
</tr>
<tr>
<td>Failure in school performance</td>
<td>Strong bonds with prosocial organizations or social units (e.g., school, family, and religious organizations)</td>
</tr>
<tr>
<td>Affiliation with deviant peers</td>
<td>Adoption of beliefs that drug use is not normative</td>
</tr>
<tr>
<td>Perception of approval of drug-using behaviors in the school, peer, and community environments</td>
<td>Low availability of drugs in environments</td>
</tr>
</tbody>
</table>

Note. Information from National Institute on Drug Abuse (1997).

A substantial body of literature has pointed to the role of two major environmental influences on adolescents—parenting practices and peers—in the onset of drug use and the development of SUDs.

Parenting Practices

Numerous parenting practices associated with high-risk behaviors by children and adolescents can contribute to the development of addiction. A wealth of research evidence has pointed to the importance of the significant continuing role that parents play in shaping a child's behavior at all stages of development from childhood to young adulthood (Catalano et al., 1992). There are two areas in the adolescent literature in which parenting practices have been examined empirically as antecedents to addictive behaviors: SUDs and problem gambling (Clark & Winters, 2002; Stinchfield & Winters, 1998).

Because there is a significantly larger and more rigorous literature in the substance use area, we focus on four components of parenting commonly cited in the literature as keys to improving the likelihood that a child will be drug free during adolescence (see Bahr & Hoffmann, 2010): (a) establishing a supportive relationship, (b) healthy parent–child communication, (c) effective monitoring and supervision, and (d) appropriate role modeling.

Readers should keep in mind that none of the identified components is necessarily involved in causal relationships between the parenting behavior and adolescent behavior. Also, even the “most optimal” parenting practices cannot guarantee that a parent will raise a drug-free child who will never develop a SUD.

Establishing a supportive relationship. Parents who build a warm and supportive relationship with their child are less likely to have an adolescent who uses drugs or develops a SUD. This parent–child bonding is especially important to establish early on; there are some indications that when early parent–child social connections are very weak or there is early parental rejection, a negative cycle of parent and child behaviors can result (Brook, Brook, Zhang, & Cohen, 2009; Catalano et al., 1992; Cohen, Richardson, & LaBree, 1994; Pires & Jenkins, 2007). For older adolescents, maintaining closeness remains important for several reasons, including the benefit that a close relationship facilitates the parents’ ability to monitor the adolescent’s behavior and affiliation with peers as the child ages (Nash, McQueen, & Bray, 2005).

On one hand, parents can promote close relationships in several ways: engaging in academic or extracurricular activities together, discussing shared interests (e.g., sports, music), minimizing conflict when communicating about stressful situations, being a good listener, encouraging small and large achievements, allowing an appropriate degree of
independence, and reinforcing basic life skills to cope with stress and to problem solve (Bahr, Hoffmann, & Yang, 2005). On the other hand, several studies have shown the adverse impact of harsh discipline and family conflict on increasing the risk for drug use and SUDs (Chen, Storr, & Anthony, 2005; Tschanne et al., 2002; Wright & Fitzpatrick, 2004).

**Healthy parent–child communication.** Numerous features of parent–child communication are associated with risk for adolescent SUDs. A prominent one is that expressed disapproval of underage drinking and other drug use, as well as approval of healthy behaviors, by parents decreases the teenager's risk of drug use initiation (Catalano et al., 1992; Simons-Morton, 2004) and of transitioning to abuse or dependence (Guilamo-Ramos, Turrisi, Jaccard, Wood, & Gonzalez, 2004; Wood, Read, Mitchell, & Brand, 2004). The impact of parents' disapproval on their teenagers' risk for drug use appears to be strengthened when parents include relevant information about health and safety risks, as well as what consequences will occur if the youth violates the home rules, in the anti–drug use messages (Clark & Winters, 2002; Cleveland, Gibbons, Gerrard, Pomery, & Brody, 2005).

In addition to communication specific to drug use, research has also shown that healthy and open communication between the child and parent about the child's life in general can protect against risk for adolescent drug use (Hill, Hawkins, Catalano, Abbott, & Guo, 2005; Stronski, Ireland, Michaud, Narring, & Resnick, 2000). This finding is perhaps not surprising given that a parent who consistently connects socially to the child increases the likelihood of influencing him or her along desired paths of behavior and attitudes.

**Effective monitoring and supervision.** A robust and highly reliable finding in the adolescent drug prevention literature is the strong link between high levels of parental monitoring and supervision and decreased risk for SUDs (Barnes & Farrell, 1992; Cleveland et al., 2005; Griffin, Botvin, Scheier, Diaz, & Miller, 2000; Pilgrim, Schulenberg, O'Malley, Bachman, & Johnston, 2006; Rai et al., 2003). Given that supporting studies have typically measured the child's perceptions of parental monitoring and supervision, rather than observational data of actual parent behavior, the child's perspective of his or her parents' monitoring and supervision appears to be the critical factor. We can parsimoniously define *parental monitoring* as knowing the general whereabouts of a child at all times and being aware of his or her activities. Of course, difficulties arise when trying to monitor a child after school or when the child is in high-risk situations, such as out with friends on weekends (Richardson, Radziszewska, Dent, & Flay, 1993). A related topic is the importance of parents monitoring the child's academic performance; several studies have indicated that school engagement and academic achievement are protective factors for drug use (Guo, Hawkins, Hill, & Abbott, 2001; Simons-Morton, 2004).

*Supervision* refers to the parent being present at or maintaining awareness of the adolescent during social situations, such as recreational events or parties. A challenge for parents when supervising is to balance the need for a social presence and the teenager's need to develop social independence and autonomy in the absence of direct parental influence. This issue is particularly important for older children.

**Appropriate role modeling.** Role modeling by the parent in the context of the parent–child relationship spans a wide range of behaviors and attitudes. A key facet of role modeling is parents' drug consumption (Bahr et al., 2005). Parents have an opportunity to model healthy adult drug use behavior in several ways: consuming small amounts of alcohol with a meal or as part of a celebratory circumstance; not using alcohol to cope with stress; avoiding intoxication in the adolescents' presence; avoiding use of illicit drugs; and not abusing or misusing prescription drugs (Bahr et al., 2005). Also, parents' attitudes toward drug use might influence the children's attitudes, which might contribute to teenagers' future likelihood of drug use (Bahr et al., 2005). Parents' attitudes are evident in several verbal and nonverbal ways, ranging from their role-modeling behavior, as we noted earlier, to how they discuss topics concerning drug use, such as underage drinking or medical marijuana (National Institute on Drug Abuse, 1997).
Peers
Many have perceived peer influence to be of paramount importance with respect to risk taking by adolescents, including drug use (Harris, 1998). Studies have suggested that the more youths rely on peers over parents, the greater the risk of alcohol and other drug use (Barnes & Windle, 1987). The role of peers in shaping another adolescent’s behavior and values might occur in a direct way, through social factors that include peer pressure, or through indirect processes, in which several reasons, including risky behaviors, attract or socially pull a young person to a peer group. With respect to drug use and the development of SUDs, the primary peer construct that researchers and health providers have emphasized is peer drug use (Ary et al., 1999; Clayton, 1992; Newcomb & Bentler, 1989). Adolescents who report having drug-using friends indicate higher levels of drug use and greater likelihood of developing a SUD than those who deny having drug-using friends (Farrell & Danish, 1993; Winters, Latimer, Stinchfield, & Henly, 1999). Other peer-related factors empirically linked to adolescent drug use include peer attitudes about drug use and peer attachment (Catalano et al., 2010; Dishion, Capaldi, Spracklen, & Li, 1995; Hawkins et al., 1992; Patterson, Forgatch, Yoerger, & Stoolmiller, 1998).

Mechanisms. The processes by which peers influence adolescent drug use include the shared propensity toward behavioral dysregulation, cohesive peer groups making drugs available to each other, and drug use modeling by friends within the group (Bauman & Ennett, 1994). Other effects include peer group support and norms favoring drug use and the role of drug use in friendship selection (i.e., individuals already using drugs might tend to select friends with similar habits; Bauman & Ennett, 1994; Dawes, Clark, Moss, Kirisci, & Tarter, 1999).

These social influences are consistent with the social norm theory of behavior: If an individual thinks a given behavior (e.g., drug use) is typical, whether the perception is accurate or not, the person is more likely to engage in the behavior than others who do not share that perception. Thus, if an adolescent has an exaggerated perception of the extent of drug use among his or her peers, a situation that can be aggravated if there is heavy drug use among that teenager's peers, he or she is more likely to experiment with drugs and increase his or her likelihood of developing a SUD. Conversely, if the adolescent perceives social signals that drug use is not common or not typical among peers, then one can expect to see a reduced likelihood of drug use by that individual. Unfortunately, adolescents tend to overestimate the extent of peers' drug use (Hawkins et al., 1992; Stacy, Widaman, & Marlatt, 1990). This inflated perception of drug use might exert a greater impact on drug use than direct social pressures applied by peers. As an aside, many drug prevention efforts for adolescents have capitalized on this “perception bias” by focusing on giving youths accurate information about the extent of peers' substance use (Barnett, Far, Mauss, & Miller, 1996; Schroeder & Prentice, 1998; Winters, Fawkes, Fahnhorst, Botzet, & August, 2007).

Parent awareness of peer relationships. In addition to peer influences, a related topic is parents' awareness of their children's relationships to peers. Recent longitudinal studies have supported the view that the parent–child relationship and parental behaviors can influence the child's peer choices and affiliation with deviant peers, which in turn can alter the child's risk of drug use (Brook et al., 2009; Simons-Morton & Chen, 2005; Svensson, 2003). Furthermore, evidence has suggested that deviant peer affiliation is associated with a perception by the child that the parent is not monitoring peer relationships (Gerrard, Gibbons, Zhao, Russell, & Reis-Bergan, 1999). This problem can be the result of poor parent–child communication, including lack of (a) discussions about the child's peers (e.g., activities, personalities), (b) direct conversations with the child's friends, (c) discussions about the child's social needs, (d) attention to the importance of peer choices, and (e) establishing communication with the parents of the child's closest peers (Yu, 2003).

SOCIOCULTURAL FACTORS AND ACCESS TO DRUGS: THE MINIMUM LEGAL DRINKING AGE
Sociocultural factors include legislative policies that can shape and reflect social perceptions, community
norms, and behavior—including expressions of addiction. We focus now on how changes in the minimum legal drinking age (MLDA) in the United States have affected alcohol use and the resulting consequences among youth. The issues discussed here that focus on the United States have several parallels to other countries, particularly those in Europe.

Background
When the 21st amendment to the U.S. Constitution repealed Prohibition in 1933, it also allowed individual states to establish and govern laws regarding the purchase, sale, and consumption of alcohol. Most states set the MLDA to 21, in concordance with the legal voting age at the time. Within this social context, the MLDA-21 policy made sense. However, changes began to occur in many states that resulted in the lowering of the voting age as well as the draft age for the military, from 21 to 18 (Toomey, Nelson, & Lenk, 2009), which created an opportunity for some people to argue for a lower legal drinking age. Perhaps further bolstered by the fact that many European countries, as well as New Zealand, Australia, and Canada, had drinking ages lower than 21, several states lowered the MLDA. By the 1970s, 29 states had lowered the drinking age from 21 to between 18 and 20.

Public health researchers took notice. Available to them was the patchwork of MLDAs across various states, which allowed for an examination of drinking-related health consequences as a function of differences in MLDA. One dramatic trend in the data stood out: Higher rates of drinking-related traffic accidents among 18- to 20-year-olds occurred in states with lower MLDAs, and researchers observed the lowest rates in states with higher MLDAs (Toomey et al., 2009).

The Reagan administration saw the need for action against alcohol-related traffic accidents and during the 1980s created the Presidential Committee on Drunk Driving. This blue-ribbon committee suggested a three-pronged approach to the problem: (a) improve education, (b) enact stronger legislation, and (c) increase enforcement of existing legislation (Toomey et al., 2009). This set of recommendations yielded a marked nationwide adjustment of the MLDA as a result of the National Minimum Drinking Age Act of 1984. This law allowed, through the use of previous legislation (i.e., the Highway Safety Act of 1966), for the federal government to withhold 5% of federal highway construction funds for all states that did not have age 21 as the MLDA by 1986. By July 1988, the MLDA was 21 in all 50 states and the District of Columbia. Hence, the pathway toward the U.S. MLDA-21 policy contained numerous and occasionally diverging points of view from public health and citizen’s rights perspectives, including calls to repeal the policy and return the MLDA to 18 (Toomey et al., 2009).

Effects of MLDA on Youth Drinking
Almost all studies designed specifically to examine the impact of drinking age changes show that states with age 21 as the MLDA are associated with reduced drinking, problematic drinking, drinking and driving, and alcohol-related crashes among young people than states with a lower MLDA (McCarrt, Hellinga, & Kirley, 2010). Surveys tracking alcohol consumption among high school students and young adults have shown a consistent pattern of declines in alcohol consumption since the early 1980s, which was the period when states were establishing, or reinstating, a MLDA-21 policy. With respect to alcohol-related crashes, the changes have been dramatic. Data from the Insurance Institute for Highway Safety (Longthorne, Subramanian, & Chen, 2010) indicated that during 1982, among fatally injured drivers ages 16 to 20, 61% had positive blood alcohol contents, whereas during 2008, 31% of fatally injured drivers in this age group had positive blood alcohol contents (see Figure 12.1). Also, fatality rates were lower in 2008 than in 1982 (Longthorne et al., 2010).

Using recent national epidemiological data, Norberg, Bierut, and Grucza (2009) examined the long-term effects of MLDA on rates of a current alcohol use disorder. They compared the 12-month prevalence of an alcohol use disorder as defined by the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994) among adult subjects born in the United States from 1948 to 1970 and exposed to different MLDA laws in the 1970s and 1980s. The data were drawn from two nationally representative
cross-sectional surveys: the 1991 National Longitudinal Alcohol Epidemiological Survey and the 2001 National Epidemiological Study of Alcohol and Related Conditions. Analyses controlled for state and birth-year fixed effects, age at assessment, alcohol taxes, and other demographic and social background factors. The results supported the view that pre-age-21 MLDA is associated with poorer outcomes. Adults who were allowed to purchase alcohol legally before age 21 were more likely to meet the criteria for an alcohol use disorder (odds ratio = 1.31, 95% confidence interval [1.15, 1.46], \( p < .0001 \)) than those who were not allowed to legally drink for the first time until age 21.

**Current Debate**

A current national debate concerning the MLDA-21 issue centers on whether to lower the minimum age to 18. The arguments to lower the MLDA appear to revolve around (a) principles of freedoms and rights for youths and (b) the belief that if they are allowed to drink at a younger age, youths will gain experience and be more informed about the dangers of binge drinking; consequently, they will engage in less of this harmful activity while attending college. The current so-called “libertarian” position is reminiscent of the arguments advanced during the late 1970s and early 1980s: Age 18 is the age of majority for many basic and meaningful societal rights and points of commitment, such as voting, entering the military, getting married, and paying taxes, and thus legal access to alcohol is not only logical, it is morally justified. The education argument supports lowering the MLDA so that 18- to 20-year-olds would have more opportunity to learn how to drink responsibly in licensed establishments, rather than engaging in binge-drinking behaviors at college settings (Toomey et al., 2009).

The MLDA-21 supporters have provided several lines of argument in favor of retaining the status quo. They have contended that the cause-and-effect relationship between MLDA-21 and reductions in highway crashes is clear: If 18-year-olds were to purchase alcohol legally, an unintended consequence would be that very young teenagers or children would have easier access to alcohol, and the ongoing and persistent problems of underage drinking—such as binge drinking and drinking and driving laws—could be exacerbated by lowering the MLDA (McCartt et al., 2010).

Nonetheless, the MLDA-21 era is still characterized by the fact that many underage individuals drink and drink heavily, and alcohol remains an important risk factor in serious crashes of young drivers, especially as they progress through the teenage years. A recent study found that among 18- to 20-year-olds surveyed, 10.3% reported driving after binge drinking (Naimi, Nelson, & Brewer, 2009). Also, there is the persistent problem of heavy drinking among underage college students. The transition from high school to college is a critical developmental period,
and one health-related behavior commonly linked to this transitional period is heavy alcohol use (Grucza, Norberg, & Bierut, 2009). Whereas alcohol consumption among adolescents has decreased considerably in the United States since the early 1980s, similar declines have not been observed among college students (Grucza et al., 2009; Johnston, O’Malley, Bachman, & Schulenberg, 2008; Nelson, Xuan, Lee, Weitzman, & Wechsler, 2009). Another perspective from epidemiological data has shown that among full-time college students, an estimated 21% meet criteria for a current alcohol abuse or dependence disorder (Wu, Pilowsky, Schlenger, & Hasin, 2007), which is considerably higher than the rate among high school seniors. These trends point to the limitations of public policy on youth behavior.

Where do we stand with the debate? The highway traffic safety data pertaining to traffic deaths among youths provide compelling evidence to maintain the current MLDA at 21. Moreover, it is difficult to envision any state moving forward with a law to lower the MLDA from 21 to 18 when there is so little public health sentiment in favor of doing so and the consequence of losing federal highway funds still looms. Also, we do not support the view that lowering the MLDA will reduce alcohol use among youth.

SOCIAL FACTORS AND COLLEGE STUDENT DRUG USE

Social factors are key influences on drug use initiation in adolescence, and they most likely continue to exert a substantial role well into the college years. Most U.S. high school graduates attend college or university for some period of time, and thus the influence of the college social environment is worthy of consideration. As we describe in detail later, at least four social factors have a plausible association with college student substance use. First, shared living arrangements and limited privacy increase drug exposure opportunities. Second, limited social interactions, largely based on the behavior of the college subculture, can alter perceptions of what is normative. These perceptions might lead college students to believe that drug use is more normative in the general population than it is. Third, in college, peer drug use can have a very direct influence on drug use, especially when drug use becomes the shared activity among peers. Clearly, shared cannabis use is a form of social interaction and often co-occurs with other social activities such as watching movies, playing video games, or having sex. Fourth, student culture often portrays drug use as a social lubricant, with many individuals having high expectancies that they will become more social and socially acceptable when intoxicated or high.

Physical Environment

With regard to the physical environment, college students are often living for the first time without direct supervision from a parent or guardian. The shared nature of living space can lead to exposure opportunities, because reduced privacy results in more visible substance use. However, these same factors can also aid in intervention efforts, because college students trained to recognize potential substance use problems are likely to spot behavioral markers among students with whom they are living so closely.

Results from the College Life Study (Arria, Caldeira, O’Grady, et al., 2008), a longitudinal study of college student health behaviors, indicated high rates of drug exposure opportunity during college. Table 12.2 presents results from the College Life Study regarding

<table>
<thead>
<tr>
<th>Drug</th>
<th>Exposure opportunity (%) of students</th>
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<tbody>
<tr>
<td>Marijuana</td>
<td>89.5</td>
</tr>
<tr>
<td>Prescription stimulants (nonmedical)</td>
<td>67.7</td>
</tr>
<tr>
<td>Hallucinogens (mushrooms, PCP, etc.)</td>
<td>52.7</td>
</tr>
<tr>
<td>Prescription analgesics</td>
<td>47.0</td>
</tr>
<tr>
<td>Ecstasy</td>
<td>42.4</td>
</tr>
<tr>
<td>Cocaine</td>
<td>40.7</td>
</tr>
<tr>
<td>Prescription tranquilizers</td>
<td>27.8</td>
</tr>
<tr>
<td>Amphetamines (including methamphetamine)</td>
<td>23.0</td>
</tr>
<tr>
<td>Inhalants</td>
<td>24.9</td>
</tr>
<tr>
<td>Heroin</td>
<td>8.5</td>
</tr>
</tbody>
</table>

Note. Students were asked, “In the past 12 months, on how many days have you been offered [drug]?” All non-zero responses were counted as exposure opportunity. Data from Arria, Caldeira, O’Grady, et al. (2008).
lifetime opportunity exposure to seven illicit drugs and three types of prescription drugs (for nonmedical use) by the 4th year in college. As the table indicates, most students (89.5%) were offered the chance to try marijuana by the 4th year of college, and a substantial proportion had the opportunity to try prescription drugs nonmedically. These data suggest that opportunities arise for college students who deliberately seek drugs and for students who would not necessarily go looking for them.

These close quarters also facilitate distribution of substances, as evidenced by the repeated claims of college students regarding the ease with which they can obtain prescription drugs from fellow students for nonmedical use (DeSantis, Noar, & Webb, 2009), suggesting that a college student who is inclined to use a substance is likely able to find it.

Perceptions of What Is “Normal”
Because the college environment creates an environment in which similar-aged peers constitute the vast majority of social interactions, the subculture’s norms, values, and attitudes become somewhat homogenized and favorable toward drug use. The adoption of these attitudes and perceptions is concerning because prior research has established the significant role perceptions play in substance use (Bachman, Johnston, O’Malley, & Humphrey, 1988). For example, several studies documenting how many college students overestimate substance use among others in their cohort have demonstrated the exaggerated perception of substance use being normative in college. During the 3rd year of college, students in the College Life Study estimated that the average 3rd-year student at their university consumed approximately 6.3 drinks on a typical Saturday, yet the actual number was about four full drinks fewer than estimated. Similarly, students estimated that an average of 37.7% of 3rd-year peers had used marijuana in the past month compared with the actual value of 27.3%. These misperceptions might increase the overall risk of substance use because of the desire to fit in with most same-age peers. The degree to which the nonuser feels stigmatized in college is an empirical question that deserves research attention.

Substance Use as a Form of Social Interaction
Association with drug-using peers is a strong and consistent risk factor for substance use among college students (Brook, Whiteman, Gordon, & Brook, 1990; Dull, 1992). Research has demonstrated that college students who associate with substance-using friends and significant others are more likely to develop drug use problems, such as abuse and dependence (Taylor, 2006). This association is most likely bidirectional—associating with drug-using peers increases the risk of exposure opportunity and subsequent use, and college student drug users are choosing to affiliate with drug-using friends. Substance use can also serve as a forum for social interaction among peers. Beck et al. (2009) found that among cannabis users, the most common social context in which marijuana was used was with a small group of friends. Other research studies have found an association between illicit drug use and spending more time at parties and socializing with friends (Bell, Wechsler, & Johnson, 1997; Mustaine & Tewksbury, 2004). In short, college substance use, as with college drinking, is often a social behavior and needs to be understood as such.

Substance Use as an Instrumental Behavior
Finally, students can use substances instrumentally, for social interaction. Work by Crum and Pratt (2001) found that, among adults, having a diagnosis of social phobia increased risk for heavy drinking and an alcohol use disorder. Previous studies have detailed the use of alcohol and other drugs to self-medicate negative affective states (Bolton, Cox, Clara, & Sareen, 2006). It is possible that, for some, drug use is a form of self-medication to reduce anxiety or inhibitions (Khantzian, 1997), which in turn allows the individual to interact more easily or with more confidence in social situations. In a study examining the use of marijuana, cocaine, ecstasy, amphetamines, and LSD among young adults, Boys, Marsden, and Strang (2001) found that one of the most common reasons for using illicit substances was to relax. In addition, 67% of users reported using these drugs to lower inhibitions, and 83% reported using a drug to increase confidence in
social situations. In contrast, young adults might also engage in substance use to give themselves more energy to party with friends. The same study (Boys et al., 2001) found that the most common reason for ecstasy, amphetamine, and cocaine use was to keep going on a night out with friends. The common theme among these motivations is that young adults place themselves in a variety of social situations in which they feel that illicit drug use will enhance their social interactions.

Each of these social factors, unique to college or not, contributes to illicit drug use and is likely to play a particularly strong role in the college environment. The mechanisms through which social factors affect college student drug use interact with one another and create a “perfect storm” of exposure opportunity, misperception of norms, and developmental need to fit in socially that, in part, underlies the high prevalence of illicit and nonmedical drug use seen among college students today.

Example: Nonmedical Use of Prescription Drugs

First, the physical environment in which college students reside creates a rich environment for the sharing and selling of prescription drugs for nonmedical use. By far the most common source of prescription medication for nonmedical use is a friend or acquaintance who has a legitimate prescription (DeSantis, Webb, & Noar, 2008; Garnier-Dykstra, Caldeira, Vincent, O’Grady, & Arria, 2010; McCabe, Cranford, Boyd, & Teter, 2007; McCabe, Teter, & Boyd, 2006). Studies have shown that most college students know where they could obtain a prescription stimulant if they wanted one (B. C. Carroll, McLaughlin, & Blake, 2006), suggesting high levels of exposure opportunity and a lack of privacy (or secrecy) associated with the college student environment. One study found that 36% of college students prescribed a medication had diverted (shared or sold) the medication to someone else (Garnier et al., 2010). The most commonly diverted classes of medication were stimulants and analgesics.

The perceived normative nature of nonmedical use of prescription drugs is also evident. Perceived estimates of nonmedical prescription drug use are much higher than actual nonmedical use (McCabe, 2008), suggesting that students perceive nonmedical use to be more normative than the actual prevalence estimate. Additionally, prior research has demonstrated that the perceived harm or danger associated with nonmedical use is low (DeSantis et al., 2009) and that an association exists between nonmedical use and low perceived harm (Arria, Caldeira, Vincent, O’Grady, & Wish, 2008), indicating another mechanism through which perceptions promote nonmedical use.

Although little evidence regarding the social context of nonmedical prescription drug use exists, perceived peer use is clearly associated with nonmedical use of prescription drugs. Table 12.3 uses 3rd-year College Life Study data (Arria, Caldeira, O’Grady, et al., 2008) to illustrate the comparison between users and nonusers of each class of

<table>
<thead>
<tr>
<th>Perspective</th>
<th>Nonuser $M(SD)$</th>
<th>Nonmedical user $M(SD)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prescription analgesics: Average percentage of close friends who used analgesics nonmedically in the past 12 months*</td>
<td>1.4 (6.9)</td>
<td>18.0 (28.0)</td>
</tr>
<tr>
<td>Prescription stimulants: Average percentage of close friends who used stimulants nonmedically in the past 12 months*</td>
<td>4.8 (13.5)</td>
<td>33.5 (32.7)</td>
</tr>
<tr>
<td>Prescription tranquilizers: Average percentage of close friends who used tranquilizers nonmedically in the past 12 months*</td>
<td>1.4 (6.6)</td>
<td>22.3 (29.8)</td>
</tr>
</tbody>
</table>

Note. Data from Arria, Caldeira, O’Grady, et al. (2008).

*p < .05.
prescription drug on the average percentage of close friends who use the drug. The table clearly illustrates higher levels of peer use among nonmedical users than among nonusers for each class of prescription drug, indicating that either influence or selection is operating in this age group.

Finally, motivations for nonmedical prescription drug use, especially stimulant use, serve instrumental functions in terms of social interaction. B. P. White, Becker-Blease, and Grace-Bishop (2006) reported that 65% of nonmedical users engaged in nonmedical use for partying, suggesting that prescription stimulants, as with illicit stimulants, might be used to reduce inhibitions or to stay awake and party longer. Additional studies have linked engaging in weekly partying behavior to nonmedical use of prescription stimulants (Teter, McCabe, Boyd, & Guthrie, 2003).

SOCIAL FACTORS ASSOCIATED WITH TREATMENT

In this section, we address how select social factors might influence the accessibility and use of addiction treatment services as well as the efficacy of treatment approaches.

Accessibility and Use
We begin by discussing how three sociodemographic factors (age, gender, sexual orientation) and culture might influence access to and use of treatment for addiction.

Age. Adolescents typically do not seek treatment on their own and are often unmotivated to change when they are forced or mandated into treatment (Fishman, 2011). Youth is a period during which individuals commonly seek to acquire autonomy from adults and often reject parental influences, including parents’ concerns about the dangers of drug use (Steinberg, 2004). Elderly individuals encounter a number of unique social factors that can contribute to increased alcohol and drug use and impede the assessment and treatment process: Social isolation can be common during the later years of life when children no longer live in the home, a spouse or friends may have passed away, and mobility has decreased. In addition, retirement might engender a loss of identity and diminished self-worth. These factors, along with high rates of mental and physical illness, exacerbate seclusion, loneliness, and boredom and can increase the risk for problematic alcohol or drug use (Blazer & Wu, 2009).

Unfortunately, identifying problem drinking among older adults can be difficult and might be substantially underestimated (Campbell, 2009). Problem-use indicators such as the inability to fulfill responsibilities at work, with the family, and around the home might no longer be relevant. Interactions with peers who could help identify difficulties might have significantly decreased as a result of isolation, illness, or death. Family members might be less likely to report their concerns because of a sense of helplessness. Promising advances in substance abuse criteria for older adults have developed. For example, the treatment improvement protocol assists in the diagnosis of substance abuse or dependence in older adults by addressing issues such as diminished tolerance, lack of withdrawal symptoms, and naïveté about the causes of physical and mental health concerns (Barry, Oslin, & Blow, 2001). Special attention to the social factors associated with older adults is essential to the success of treatment as well as to aftercare and relapse prevention.

Gender. Whereas patterns of substance use among men and women have become more parallel over the past few decades (Kuczkowski, 2007), there has been a recent call for treatment programs to be designed to meet the specific needs of women (Zweben, 2009). Compared with men, women present with more adverse social, medical, and psychiatric consequences (Greenfield et al., 2007). Therefore, the need for female-oriented treatment programs that incorporate curricula to address these special issues is essential. The sparse number of women-only treatment programs that do exist have shown greater success by integrating assistance with transportation, housing, job training, and life skills training as well as options to meet the special needs of pregnant and parenting women (e.g., Grella, Polinsky, Hser, & Perry, 1999). Another issue is that the male-dominated culture common in some treatment settings (e.g., veterans programs) has been unsuccessful in meeting the needs of women (Brown, Sanchez, Zweben, & Aly, 1996). These confrontational and
somewhat aggressive formats have proven disrespectful to and even harmful for women, especially those with comorbid psychiatric conditions (Zweben, 2009). Effective treatment programs for women need to take into account societal expectations and oppressive restraints and provide a nurturing and empowering atmosphere (Schliebner, 1994).

**Sexual minorities.** Despite high rates of alcohol and other substance abuse in the lesbian, gay, bisexual, and transgender (LGBT) community, most treatment programs lack resources (e.g., staff sensitivity training, LGBT staff) to address the unique social factors relevant to LGBT individuals (Cochran & Cauce, 2006). Research has proven that openly LGBT individuals in treatment had higher rates of homelessness, were more frequently victims of domestic violence, and presented with a history of more mental health treatments than heterosexual clients (Cochran & Cauce, 2006). Social factors might also lead the LGBT population to underuse aftercare programs such as Alcoholics Anonymous (AA) and Narcotics Anonymous. The religious orientation of the programming might be somewhat responsible (e.g., Holleran & Novak, 1989). A recent study found that lesbian, gay, and bisexual individuals concerned about their drinking reported that they were more likely than heterosexual participants to attend treatment such as meditation and acupuncture than to attend AA (Dillworth, Kaysen, Montoya, & Larimer, 2009).

**Culture.** Cultural issues can also play a role in treatment. Not only can cultural factors heighten risk for substance abuse, but these factors also play a role in the client's recovery efforts and affect the therapist who is responsible for the intervention (Westermeyer, 2009). For instance, researchers found significant reduction in drinking outcomes for Hispanic men ethnically matched to their treatment provider, and outcomes were most beneficial for those born outside the United States (Field & Caetano, 2010). It important to note that the term cultural issues encompasses a variety of different culture-related terms that can be relevant to patterns of substance use and affect intervention (Westermeyer, 2009). Culture, for example, can refer to a population's way of life, including geography, family organization, and work and recreation. Ethnicity pertains to a group within a culture who might share national origin, religion, and other beliefs. Subculture applies to a group within a culture, such as a drug subculture, or to a place, such as a local bar or crack house (e.g., Dumont, 1967; Westermeyer, 2009). The way in which an individual assimilates these terms can affect his or her feelings of connectedness with others in the treatment group, the relationship with his or her therapist, the ability to achieve and maintain sobriety, and the relevance and efficacy of program format and content. Therefore, the way in which an individual interprets these terms can play a significant role in substance abuse treatment efficacy (Westermeyer, 2009).

**Interaction of demographic characteristics.** An individual often presents with multiple demographic factors that add to the complexity and efficacy of treatment use. Treatment programs do not routinely adjust treatment content and delivery on the basis of this complexity (National Institute on Alcohol Abuse and Alcoholism, 2000). However, the American Society of Addiction Medicine has provided some guidance for treatment providers; this society has developed patient placement criteria with a multidimensional approach to assessment and treatment. By using this method, health care providers can meet the individualized and multifaceted needs of a patient more successfully (Mee-Lee & Shulman, 2009). The American Society of Addiction Medicine's patient placement criteria have six criteria assessment dimensions: (a) acute intoxication or withdrawal potential; (b) biomedical conditions; (c) emotional, behavioral, or cognitive conditions; (d) readiness to change; (e) relapse, continued use, or continued problem potential; and (f) recovery environment. These dimensions facilitate a holistic, biopsychosocial approach to patient care that addresses multiple patient demographic factors throughout the entire recovery period (Mee-Lee & Shulman, 2009).

**Social Factors That Affect Treatment Effectiveness**
As we discuss in this final section, regardless of the demographic characteristics of the person seeking
treatment, all treatment programs should address the role of social factors. Although various cultures and genders might differ in their social interaction habits, the immediate environment inevitably affects a person's emotions and choices. Various theories have tried to explain the social factors underlying addiction, which might be useful to understanding the treatment process (e.g., Lettieri, Sayers, & Pearson, 1980). For example, Denise Kandel's theory states that people do not choose friends at random; rather, such choices stem from an often unconscious but selective peer group (Lettieri et al., 1980). Upholding this theory is a common practice in drug treatment programs to assist people to select a new, healthier peer group and to learn social skills to help resist temptations to use and to help cope with stress (K. M. Carroll & Onken, 2005; Winters, Botzet, Fahnhorst, & Koskey, 2009). Similarly, Caplan's (2005) theory regarding Internet addiction states that a social skills deficit paired with Internet access might predispose an individual to seek online interaction, rather than face-to-face interaction, for his or her interpersonal needs. These behaviors might then lead to compulsive Internet usage, resulting in negative outcomes. These theories exemplify how addiction treatment programs will best address the needs of the consumers if they increase social support and skills while simultaneously decreasing social risk.

Social components. Nearly all effective treatment approaches incorporate social components in their treatment approaches. The most commonly addressed social skills (Bu'dney & Higgins, 1998) include:

1. Time management: Many people struggling with addiction will stop engaging in their previous healthy activities to maintain their addiction and then allow the addictive habits to fill their time. Thus, many treatment programs address this social component by helping patients learn to plan and schedule their time so that they reduce or eliminate the idle, high-risk moments.

2. Problem solving: Often, people seeking treatment for addiction have a multitude of problems, allowing even minor problems to become overwhelming. Common socially related problems include having to deal with social pressures and personal feedback, problems with childcare, job-related issues, and family pressures. Coping skills in the past might have involved denial or impulsive decision making that can result in additional negative consequences. Monti, Abrams, Kadden, and Cooney (1989) incorporated an effective problem-solving component in their skills training for people experiencing alcohol dependence, which other programs have successfully replicated or modified (Finfgeld-Connett & Madsen, 2008; Petry, Weinstock, Ledgerwood, & Morasco, 2008).

3. Interpersonal skills: Social skills training is another important component in an effective treatment program, especially for those patients who experience difficulty interacting with others, meeting sober people, or expressing their feelings in an appropriate manner. This type of training might include anger management, assertiveness training, or reducing anxiety in social situations. Monti et al. (1989) have discussed in detail effective procedures for social skills training.

Spirituality as a social network. In addition to the social components in the preceding list, many people struggling with addiction find spirituality to be an effective resource in their quest for a healthier lifestyle, especially if they feel that doctors or other health care providers are not sufficiently considerate of the emotional burdens of the addiction (Galanter, 2009). A healthy spiritual connection can sometimes provide a level of personal support that is similar to interpersonal networks (Videbeck, 2010). However, researchers, health providers, and laypeople struggle with defining and delineating the terms spirituality, spiritual awakening, and spiritual practices (W. L. White, 2008). Because these terms represent such personal and individual experiences, some people have voiced concern that the essence of the spiritual experience would be lost or minimized in an effort to define and replicate it for research purposes (W. L. White, 2008). Similarly, W. L. White (2008) suggested that professionally organized, spiritually oriented treatment interventions might not consist of the same spiritual components that others
in long-term recovery have anecdotally reported as transformative.

The science related to the effects of spiritual guidance in treating substance-related addiction is limited. Miller, Forcehimes, O'Leary, and LaNoue (2008) evaluated the impact of spirituality on recovery from addiction in two randomized clinical trials, both of which compared a spiritually based intervention (adjunct to normal treatment plan) with a treatment-as-usual intervention. Although both trials failed to support the hypothesis that spiritual guidance would increase sobriety at posttreatment follow-ups, they noted that spiritual development is generally a lifelong process, and their attempt to measure that process in a relatively brief amount of time might have been a critical error. Thus, similar to the observations noted by W. L. White (2008, p. 443), it might be that the studies by Miller et al. (2008) did not capture the essence of the spiritual experience.

Self-help programs. The self-help phenomenon, including AA and other 12-step programs, is a primary element of most treatment programs. Started by a small group of dedicated people during the 1930s, the AA self-help phenomenon uses the powers of a higher spirit and social networking. As stated in The AA Preamble,

Alcoholics Anonymous is a fellowship of men and women who share their experience, strength, and hope with each other that they may solve their common problem and help others to recover from alcoholism. The only requirement for membership is a desire to stop drinking. . . . Our primary purpose is to stay sober and help other alcoholics achieve sobriety. (AA Grapevine, Inc., 2002)

AA currently includes more than 2 million members in 182 countries (AA, 2010). From the original fellowship grew additional self-help groups to address other expressions of addiction, such as narcotics, cocaine, eating, sex, and gambling as well as special interest groups, such as gender-specific groups, gay men and lesbians, young people, non-smokers, and older adults.

Despite the popularity of AA and other self-help groups, very few randomized controlled studies have been conducted of AA or other self-help groups as stand-alone treatments. The literature that does exist has generally concluded that AA attendance correlates positively with abstinence (McCready & Tonigan, 2009). One rigorous study conducted by Project MATCH in the 1990s (Project MATCH Research Group, 1998) compared three treatment modalities—cognitive–behavioral therapy, motivational enhancement therapy, and 12-step facilitated therapy—with respect to various personal traits and alcohol use outcomes. Results from this study indicated that 12-step facilitated therapy resulted in a greater percentage of days abstinent from alcohol at both the 1-year and 3-year follow-ups than both the cognitive–behavioral therapy and motivational enhancement therapy approaches. The greater AA social involvement with the 12-step facilitated therapy approach might, in part, explain this result (Butler Center for Research, 2010). Thus, even though rigorous 12-step research is limited, the power of this socially driven treatment approach is evident.

Additional social components of treatment. A number of alternative programs have been developed or popularized in recent years that support the social needs of people in recovery from addiction. One example of a social support system to supplement treatment is sober housing, such as that available through the Sober Living Network. These houses provide not only an alcohol- and drug-free living environment but also recovery support through 12-step groups and social networks. Research on sober housing has suggested that sober housing residents show significant improvements in alcohol and drug use, employment, psychiatric severity, and arrests (Polcin, 2009; Polcin, Korcha, Bond, & Galloway, 2010).

Similar to the sober living concept is the sober school (also called recovery school) supplemental approach to treatment. These educational programs address the social and academic needs of college and high school students who have experienced treatment for substance addiction and aim to maintain sobriety. The first recovery-based school service was developed at Brown University in 1977 (W. L. White & Finch,
2006), and the first sober high school opened in 1987 (W. L. White & Finch, 2006). People initiated recovery schools out of the recognition that college and high school environments are not always conducive to sobriety and do not provide the social support network essential to sobriety (Botzet, Winters, & Fahnhorst, 2007).

Currently, 23 recovery high schools and 16 recovery colleges exist in the United States (Association of Recovery Schools, 2010). Although no controlled research studies regarding the efficacy of recovery schools are available, two descriptive studies of a college-based recovery program found promising results (Botzet et al., 2007; Moberg & Finch, 2007). Both studies found that, among attending students, most reported abstinence from all substances and indicated that significant involvement in self-help groups was important to successful recovery (Botzet et al., 2007; Moberg & Finch, 2007).

A more recent supplemental treatment approach has addressed social support via electronic means. Web-based, or e-health, programs are largely the result of health providers trying to address financial and insurance limitations while simultaneously providing adequate care and follow-up services (Grinstead, Gorski, & Blampied, 2010). These types of programs might also ease accessibility issues and stigma concerns. However, e-health programs might foster iatrogenic effects, including a lack of personal accountability, which would otherwise be provided by a counselor or peer in a residential or outpatient program (Winters et al., 2009).

Nonetheless, health providers and patients alike might appreciate the convenience and cost-effectiveness of web-based follow-up care. E-health programs frequently occur in Internet chat rooms, in which counselors or health providers can discuss treatment- and recovery-based issues with patients who have recently completed addiction treatment either in a group format (i.e., many patients logging on at the same time providing social support through instant messaging or web-streamed video) or an individual format (i.e., telephone- or e-mail-based continuing care, instant messaging, or tracking high-risk patients with RecoveryTrack, a web-based monitor that alerts counselors when patients might relapse). Another e-health technique uses virtual 3-D worlds to merge the physical and virtual realities of addiction treatment (Gorini, Gaggioli, Vigna, & Riva, 2008). Virtual worlds are reported to convey greater feelings of connectedness, presence, and cohesiveness in the group therapy process and cultivate higher levels of trust between patients and therapists (Gorini et al., 2008). These supplemental techniques are useful especially in addiction recovery because recovery is often a lifelong process that needs continuing care and support. Web-based initiatives can provide the necessary support and educational tools for years beyond the initial treatment at a minimal cost and time commitment for both the patient and the health care provider (Grinstead et al., 2010).

Electronic communication, especially Internet usage, has become a very powerful socialization tool. Web-based recovery programs promote convenience and accessibility while maintaining the health care team and community concept. However, an area for future research is to ensure that this method of recovery does not create the unintended consequence of a co-occurring Internet addiction.

SUMMARY

Social factors contribute to people's engaging in potentially addictive behaviors as well as to the development of addiction. These influences include distal factors such as legal policies, availability of the addictive agent, and community norms, as well as proximal factors, including personal social characteristics and relations with peers and family members. Also, social factors are associated with success in curbing this same behavior. Peer-based recovery programs and environmental changes are prime examples of the potential that social factors have for helping to attenuate addictive behavior. Of course, individuals engage in and recover from addictive behaviors because of influences that represent several domains, including biology, psychology, and culture.

The commonality of social factors associated across multiple addictive disorders can further sharpen the understanding of how social factors pervasively influence the etiology and course of addictive disorders. We contend that social factors are an integral part of addiction. Addiction is an extremely
complex public health problem; many factors affect its etiology and course, including social factors. Although it is possible that the etiology and course of substance-based addiction might be influenced more heavily by nonsocial factors, such as genetics and neuroadaptation, compared with behavioral expressions of addiction (see Orford, 2001), we still view social factors as exerting meaningful influence across all expressions of addiction.

Let us illustrate with an example. Social factors are an important element of the addiction syndrome model with respect to shared social antecedents. Consider the extent to which common social risk factors have etiological significance across substance-based and behavioral expressions of addiction (Welte, Barnes, & Hoffman, 2004; Whalen, Jamner, Henker, & Delíno, 2001). One interesting and fairly large literature addresses the topic of the commonality of antecedent psychosocial risk factors associated with both adolescent drug abuse and problem gambling (Stinchfield & Winters, 1998). These two problem behavioral sets of adolescent substance use disorders (Winters, Chung, Stinchfield, Kassel, & Conrad, in press). Researchers have hypothesized that these factors influence the onset and course of adolescent substance use disorders (Clayton, 1992) and adolescent problem gambling (Stinchfield & Winters, 1998).

Whereas many studies have supported the strong connection between social variables and addiction, we caution readers that the interpretive limitations imposed by cross-sectional data hamper much of this literature. The positive link between social variables and addictive behaviors might reflect other unmeasured factors. Few studies are designed to establish how much the observed associations are the direct result of social influences. Nonetheless, explanations of why individuals develop an addiction and what contributes to a person's recovery from it require that we incorporate a social perspective. Even if we take the most biological explanation, that addiction results from a hijacking of the brain's neural reward circuitry (Leshner, 1999), social factors are most certainly relevant with respect to why a person initially seeks the addictive agent, why the addictive behavior might continue, and why a person responds to treatment.

References


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CHAPTER 13

PROXIMAL INFLUENCES ON ADDICTION

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The syndrome model provides a comprehensive conceptualization of what addiction is and how various expressions of it emerge. The syndrome model provides a unifying framework that draws on other areas of medicine (e.g., diabetes and AIDS) to clarify how different manifestations of addiction and many different types of symptoms can be understood as representing a core pathology. The central question “When is addiction addiction?” (Leshner, 1999) frames this exploration, highlighting that many of the assumptions taken for granted to answer this question do not fully hold up when exposed to counterexamples, logical scrutiny, and empirical research.

In this chapter, I address one part of the syndrome model: proximal antecedents. They are biopsychosocial factors “that influence the likelihood of further syndrome development” (Introduction to this handbook, p. xlv). Proximal antecedents are defined by their time course in relation to both earlier events (distal antecedents) and later events (subsequent addictive behavior). In general, proximal is defined as “situated close to” (“Proximal,” 2010), and “nearest to a point of reference” (“Proximal,” 2007). As Shaffer (in the Introduction to this handbook) notes, “Although distal antecedents of addiction . . . are well documented, the proximal antecedents that influence the likelihood of further syndrome development remain poorly identified” (p. xlv). The goal, then, in this chapter is threefold:

1. To identify different types of proximal antecedents that can influence addictive behavior;
2. To explore contextual factors that can increase or dampen the response to proximal antecedents; and
3. To discuss how researchers can further elaborate the proximal antecedents within the context of the syndrome model.

Much of the discussion focusing on the proximal antecedents of addiction also has implications for the development and maintenance of other mental disorders that co-occur with addiction (Kessler, Chiu, Demler, Merikangas, & Walters, 2005).

DIFFERENT TYPES OF PROXIMAL ANTECEDENTS

A virtually limitless number of proximal antecedents can increase or decrease the likelihood of addiction development. In this section, I offer a framework for identifying different types of proximal antecedents, to help categorize them in ways that are relevant for research and clinical work using the syndrome model. The key categories are traumas, stressors, physical antecedents that are not traumatic, emotional antecedents, lifestyle-related antecedents, and positive antecedents.

Traumas

Traumas are epidemic in both the United States and globally. Most Americans, for example, experience one or more traumas during their lifetime: 61% of males and 51% of females (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). The fourth edition of the Diagnostic and Statistical
**Manual of Mental Disorders (DSM–IV; American Psychiatric Association, 1994)** defined trauma as the experience, threat, or witnessing of physical harm, which evoke “intense fear, helplessness, or horror” (p. 427). Traumas are wide ranging, including natural disasters such as hurricanes and tornados, combat, severe life-threatening physical illness or injury, violent assault, physical or sexual abuse, serious accidents (automobile, industrial, toxic chemical), and terrorism incidents, among other events. Trauma-related syndromes such as posttraumatic stress disorder are, moreover, known to be associated with the development of all types of addictions; thus, traumas represent an important category of proximal antecedents (Kessler et al., 1995; Najavits & Cottler, in press; Ouimette & Brown, 2002). Numerous subgroups are at heightened risk for trauma and addiction, including veterans and active-duty military, people who are homeless, criminal justice detainees, emergency room patients, first responders, sex workers, and victims of domestic violence (Desai, Harpaz-Rotem, Najavits, & Rosenheck, 2008; Gentilello et al., 2000; Jones, Hughes, & Unterstaller, 2001; Najavits et al., 2008; Ouimette & Brown, 2002; Seal et al., 2009).

**Stressors**

There are also innumerable antecedents to addiction that are stressful but not technically traumas because they are not physical events per se. These stressors include, for example, divorce, job loss, poverty, and discrimination such as racism, homophobia, and ageism. Chronic negative stressors, such as continually working too hard, social isolation, caretaking of elderly parents, problem children, and bad marriages, can also take a toll over time that can lead to addiction. Positive events are also associated with stress (Dohrenwend & Dohrenwend, 1974): marriage, adult children leaving home, job promotions, moving to a new city, starting a new romantic relationship, beginning new projects, and so on. Stress has generally been shown to be linked to the development of addictions (Jacobsen, Southwick, & Kosten, 2001) and to addiction relapse (Sinha, 2007), although there is much less study of specific stressors in relation to addiction if they are nontraumatic in nature.

**Physical Antecedents That Are Not Traumatic**

Various normal physical events can be proximally associated with the development of addiction. These events include developmental progressions such as body changes during adolescence, menopause, aging, death of loved ones, recovery from surgery, physical illnesses and injuries, and chronic pain (Brady, Back, & Greenfield, 2009; Carinci & Mao, 2010; Najavits, 2002). Nontraumatic physical constraints, such as incarceration, lack of sufficient food, or intense physical discomfort, might also be associated with compensatory addictive behavior. The association between physical antecedents and addictive behavior can occur through different types of mechanisms. For example, having to tolerate or suffer a physical experience can lead to increased addictive behavior (e.g., numbing the pain; Najavits, 2004). In other cases, the origin might not be the original physical event per se but rather related ones, such as becoming dependent on opioid painkillers that were prescribed for physical illness. Returning veterans from Iraq and Afghanistan, for example, evidenced increased rates of dependence on painkillers that were prescribed for physical injuries resulting from their military service (Schneider, 2009).

**Emotional Antecedents**

During the course of daily life events (work, family and social relationships, traffic, leisure, sports, etc.), people experience all sorts of emotions, such as sadness, anger, anxiety, shame, guilt, happiness, joy, and excitement. Emotions of all types can be associated, for some people, with the use of psychoactive substances or other potential objects of addiction. For example, anger can trigger drinking alcohol, anxiety can lead to excessive spending, and excitement can result in gambling. The association between emotions and subsequent addictive behaviors has been observed for a long time (Marlatt & Gordon, 1985), but there is as yet no widely agreed-on, empirically based classification of emotions in relation to addiction.
Lifestyle-Related Antecedents
Numerous lifestyle-related antecedents can also accumulate to be proximally associated with addiction. These antecedents include sporting events at which fans routinely drink alcohol, college life (e.g., gaming, Internet, alcohol, parties), socializing with people who engage in addictive behaviors, and working in jobs or subcultures in which there is heightened exposure to addictive behavior (e.g., bartending, sex work, drug dealing). Although research has indicated that mere exposure to substances or gambling is not, in and of itself, associated with increased prevalence of addiction (LaPlante & Shaffer, 2007), some lifestyle factors are important to study in relation to other factors that can combine, additively, to increase the likelihood of addiction (e.g., personality traits, emotion dysregulation).

Positive Antecedents
It is important to recognize that proximal antecedents can be positive as well as negative events. Many of the situations and activities just described are negative in nature, representing challenges to the individual. However, proximal antecedents can also include positive events (e.g., acquisition of money, which can increase the likelihood of substance use or gambling; social events, which can increase exposure and social pressure to use substances; career or family advancement, which can lead to celebratory behavior that includes alcohol or other substance use). The concept of proximal antecedents is so broad that it can represent virtually any event, of any valence (emotional, physical, psychological, spiritual, social, etc.). According to the syndrome model, it will likely be most productive, however, to focus on antecedent events that contribute to a reliable and robust shift in subjective experience in a desirable way. Thus, researchers and clinicians can evaluate the proximal antecedents of addiction according to their potential to alter subjective experience in a sought-after way.

VARYING CONTEXTS OF PROXIMAL ANTECEDENTS
Beyond the complex nature of proximal antecedents, one must also consider their context. Two individuals can experience the same type of event (such as a traumatic assault) yet experience quite different impacts on the basis of the contexts of their experience. In addition, the same type of event can affect the same individual differently across time as a result of changing contexts. In this section, I address seven contextual factors: recurrence, social acceptance, level of control and preparation, beliefs, situational context, culture, and personal and family history. Each of these, either alone or combined, can strongly influence the degree to which proximal events actually become antecedents to addiction.

Recurrence
Clearly, the more frequent a negative proximal event is, the more likely it is to evoke addiction-related responses. Thus, although a single event such as a traumatic car accident can, in and of itself, be associated with the development of addiction, repeated events are more likely to result in addiction. Thus, for example, the greater the number of adverse childhood experiences, the greater the likelihood of later addiction (Felitti et al., 1998). Recurrence can also be understood as chronicity (continuance of a proximal antecedent) rather than simply number (repeated discrete events). Thus, chronic physical pain and chronic stress are associated with increased rates of addiction, for example (Carinci & Mao, 2010). Recurrent events can also be positive, however, and they would likely decrease addictive responses. Repeated job successes, health promotion events, and good friendship experiences may prevent the development of addiction.

Social Acceptance
Social acceptance can be double edged, in some cases minimizing the likelihood of developing addiction and in other cases increasing it. For example, an adolescent who is being bullied at school might be prone to developing addiction to cope with this experience, but if the adolescent obtains strong social acceptance at home from her or his family or community, this experience might mitigate the negative impact of being bullied. Conversely, a college-age man may be encouraged to drink or gamble more than he normally would as a result of peer social acceptance of such behavior (and even
encouragement or pressure to drink or gamble). The reverse of social acceptance is social stigma, which is similarly double edged. Social stigma can prevent someone from engaging in a potentially addictive behavior (e.g., conservative cultures in which it is not acceptable to use illegal drugs); alternatively, social stigma can promote potentially addictive behavior (e.g., a trauma survivor who feels alone or ostracized might try to cope by engaging in activities that are harmful, such as cutting or abusing psychoactive drugs). These attempts to cope can lead to behavior patterns that can become addiction (Gutmann, 1999).

Level of Control and Preparation
The ability to anticipate and prepare for events also plays a role (Najavits & Cottler, in press). For example, a soldier who volunteers to serve in the army and is well trained in military skills might feel more capable of managing military stressors and traumas than someone who is completely blindsided by an unexpected event. A child who is physically abused has no control over or ability to prepare for the abuse. Proximal antecedents associated with the higher levels of lack of control and lack of preparation are likely to have the strongest association with the subsequent development of addiction.

Beliefs
Cognitions play a major role in the perception and impact of events. The same event can evoke different beliefs that influence the subsequent likelihood of substance use. For example, a natural disaster such as a hurricane can elicit “I cannot cope with this; it is too overwhelming; I need to relax with a drink” versus “This is manageable; I can get help; I can cope.” There are various categorizations of cognitions, such as beliefs about self, world, and future (Beck, 1979); beliefs about trust, safety, esteem, intimacy, and control (McCann & Pearlman, 1990); beliefs about normalization versus stigmatization; and beliefs related to locus of control (Rotter, 1990), self- versus other blame, and so on. Moreover, beliefs themselves are sometimes proximal antecedents to addiction. Someone who habitually believes he or she is worthless or unlovable may drink to quiet such thoughts. It is also important to recognize the bidirectionality between beliefs and addictive behavior. Proximal antecedents can spur particular beliefs (e.g., the hurricane victim who believes he or she cannot cope), but beliefs can also lead to proximal antecedents. A young adult who believes he or she is invulnerable might be more likely to engage in risky behaviors that result in traumatic proximal antecedents (e.g., drinking and driving).

Situational Context
Some situations increase or blunt the impact of proximal events. For example, during the Vietnam era, substance use was common and encouraged during military service (Shay, 1994). With reentry to civilian life, substance use typically decreased (Najavits & Cottler, in press). The context of the Iraq and Afghanistan wars, however, has had the opposite effect: There is now a zero-tolerance policy on substance use during active duty. Postdeployment, veterans might be more likely to use psychoactive drugs because of restrictions on substance use during deployment. Similarly, restricted environments such as prisons, jails, residential treatment settings, and inpatient units might, overall, decrease the likelihood of addictive behavior in relation to proximal events, simply because many addictive behaviors are monitored and proscribed in such settings.

Other situational contexts also influence proximal antecedents. These include whether the event was experienced alone versus in a group or community, whether the event was directly experienced or just witnessed, and whether a person or neutral source caused the event. Thus, an event can have more or less impact—and therefore more or less likelihood of leading to addiction—on the basis of some of these factors. In general, events are likely to have the most negative impact when they are experienced alone, directly experienced, and caused by another human being (e.g., “betrayal of what’s right”; Shay, 1994, p. 3).

Culture
Culture is a broad concept. Culture has a profound impact on events and how people perceive and respond to them. Whereas situational context (see
preceding section) refers to actual observable events, culture is a more amorphous, but no less powerful, set of assumptions that is often unconscious. For example, culture can affect the likelihood of seeking help after traumas or other upsetting experiences; the level of emotion experienced; the level of isolation or social connection that occurs in relation to an event; attributions about who gets credit or blame for positive and negative events, respectively; the level of comfort or care received; and so on (Athey & Moody-Williams, 2003). Some subcultures are also more likely to engage in certain types of risky behaviors than others. Drinking, for example, is more common among Irish than Jewish subcultures. Drug use is heightened in gay, lesbian, bisexual, and transgender subcultures (Hughes & Eliason, 2002). Gambling is more common than drinking in some Asian cultures. Thus, both scientists and clinicians can benefit from the exploration of culture as an important class of proximal antecedents.

Personal and Family History
Observers might conceive of personal and family history as distal events; however, these events can also be proximal. For example, chronic mental illness—personal or familial—can represent both current and distal antecedents that affect addiction. All sorts of historical events can also affect the likelihood of addiction, including intergeneration trauma histories (such as those of African Americans and Native Americans) and community-wide historical events (such as the 9/11 terrorist attacks, which live on for many people in their day-to-day levels of vigilance and stress; Hudnall Stamm, Stamm, Stamm, & Higson-Smith, 2004). Such historical legacies can strongly influence how a person responds to proximal antecedents and can even affect their occurrence (e.g., people with a history of mental illness have increased trauma rates; Mueser, Rosenberg, Jankowski, Hamblen, & Descamps, 2004).

PROXIMAL ANTECEDENTS AND THE SYNDROME MODEL
The syndrome model, overall, provides a thoughtful conceptualization that integrates disparate threads to organize a formal theory of addiction. It draws on gold-standard areas of medicine, such as diabetes and AIDS, which provide exemplars of syndromes that encompass diverse symptoms yet clearly have common physiological underpinnings. Shaffer (in the Introduction to this handbook) also draws important distinctions that are often lost in the addiction field. The syndrome model provides important guidance on how common addiction concepts that appear in academic writing, clinical work, and popular culture are not sufficient to explain what actually causes addiction. Neuroadaptation, compulsion, negative consequences, the object of addiction itself, or loss of control fail to account fully for how addiction arises. Thus, key questions become all the more compelling: how does one make sense of the many different expressions of addiction (e.g., alcohol and drugs, behavioral addictions such as gambling, sex, shopping, and the Internet), how does one discern some common underlying mechanisms or patterns, and how does one identify addiction before it becomes fully manifest? As Shaffer keenly points out, the DSM-IV definition of addictions is based on tautology: The person has an addiction if he or she shows or reports an addiction. The quest to predict who will become addicted, before evidence of addiction, is the Holy Grail that researchers are far from achieving but are moving toward via advances in neuroscience, genetics, and psychosocial studies. As the syndrome model demonstrates, such research has increasingly identified similar etiological patterns for different expressions of addiction. Pathological gambling and substance dependence likely have more in common than not, for example.

As part of this broad effort, inquiry into proximal antecedents offers an important domain for greater understanding of how addiction emerges. There is both compelling research and clinical evidence at this point to buttress the strong relationship between various types of proximal antecedents and the emergence of subsequent addictive behavior. In this chapter, I have described different types of proximal antecedents, along with contextual variables that play a role in increasing or decreasing the impact of such events. However, I must also address the larger question of how proximal antecedents fit
into the syndrome model. As the syndrome model matures, several considerations warrant further elaboration.

**Proximal Is a Moving Target**
How close in time must a proximal event be to subsequent addictive behavior? For example, a trauma could be hours, days, or months away from addictive behavior, yet still hold a clear causal relationship to it. Is there some way to identify a timeframe beyond which proximal events become distal? The term *proximal* must also be defined consistently across studies.

**Some Antecedents Are Both Proximal and Distal**
As noted earlier, mental illness in oneself or one’s family can be both proximal and distal if the mental illness is diagnosed as both current and past. Thus, for example, major depression can be an important historical (distal) disorder that is associated with the development of addiction. It is also a recurring problem that can serve as a proximal event. In such cases, the distal and the proximal antecedents are not independent: The distal major depression influences how the current major depression is experienced and managed. Thus, proximal and distal events are intertwined and mutually affect each other over time. Some other mental illnesses, moreover, might not have discrete episodes the way major depression does. Dysthymia, schizophrenia, personality disorders, and other disorders are chronic and thus both distal and proximal in a continuous and synergetic fashion that probably influences the likelihood of addiction. Similarly, certain life circumstances, such as homelessness, poverty, discrimination, social isolation, or other factors, can be both distal and proximal continuous events.

**What Is an Antecedent?**
At the heart of the concept of proximal antecedents is the notion that one can define and measure them. Yet, broadly speaking, almost anything could represent proximal antecedents: In addition to external discrete events, proximal antecedents can include feeling states, physical experiences (e.g., hunger, pain, tiredness), beliefs, interactions with others, and so on. Trying to conceptualize and identify antecedents is thus difficult. Moreover, they are sometimes parallel experiences: A feeling state can be experienced physically and cognitively. One alternative is to limit the notion of proximal antecedents solely to observable, discrete events such as traumas and clearly defined stressors. Yet this would appear to limit the development of the syndrome model framework, which seeks to be inclusive and comprehensive. Another definitional quandary is that people often define a proximal antecedent only in retrospect. To illustrate, one person can go through a divorce, and this circumstance might have no connection to addiction whatsoever; for another person, the divorce is a proximal antecedent to addiction. Thus, it appears to be not just the nature of an event but the relationship of the individual to an event that meets criteria as a proximal antecedent of addiction. To keep clear terminology, then, one must distinguish proximal events from proximal antecedents: The former are a much broader class of anything a person experiences within a certain time window; the latter are actually causally related to the development of addiction.

**Causal Connections Between Proximal Antecedents and Addiction Can Be Challenging to Determine**
Assuming there are legitimate ways to define and identify proximal antecedents, another concern is how to link them rigorously to addictive behavior. The simplest framework is that the proximal event is causally associated with subsequent addictive behavior. However, there are other possibilities: The proximal event could have no direct association with the emergence of addictive behavior; instead, some earlier distal influence might account for substance use. There could also be the infamous “third variable” (i.e., perhaps an unknown factor) that influences both the proximal event and the addiction. For example, perhaps a woman is assaulted and subsequently uses alcohol, but her use of alcohol is related not to the assault but instead to a friend’s intense peer pressure. Clearly, most causal circumstances have multiple influences: To use this same example, both the assault and the peer pressure could have additively combined to influence the
woman's addictive behavior, and either influence alone might not have been sufficient to cause it.

**Addictive Behavior Can Be a Proximal Event for Other Addictive Behavior**

Risky or addictive behavior can cause other risky or addictive behavior. As the syndrome model suggests, addiction can change the risk matrix for other or revised expressions of addiction. For example, drinking can result in a drunk driving accident, which itself becomes the basis for further drinking (e.g., to cope with the trauma of the accident). Indeed, among emergency room assault victims, two thirds have been found positive for substance use on the basis of urinalysis (Zatzick et al., 2004). Substance abuse is also associated with increased rates of domestic violence (Fazzone, Holton, & Reed, 1997). Such data have indicated that substance use is associated with trauma, which in turn is associated with increased rates of substance use (Najavits, Weiss, & Shaw, 1997). Addictive behavior can also result in positive proximal antecedents for subsequent addictive behavior, such as when a gambler experiences a win, feels excitement (a proximal antecedent), and thus gambles more. Moreover, mere exposure to potentially addictive activities can increase subsequent use. For example, as gambling became legal in more states, more people gambled and ultimately, in sheer numbers (although not necessarily per capita or prevalence), more people experienced gambling addiction (Korn & Shaffer, 1999).

**Valence of Proximal Antecedents Can Be Positive, Negative, or Both**

It is tempting to try to categorize proximal antecedents as exclusively positive or negative. For example, a job promotion would generally be perceived as positive, whereas a trauma would generally be perceived as negative. However, no event is inherently and orthogonally positive or negative. A job promotion can be negative for a person who becomes very stressed or overwhelmed by the changes associated with new responsibilities, and conversely, a trauma can result in posttraumatic growth (i.e., positive meanings that develop, such as greater sense of purpose in life). Thus, as the syndrome model develops, it will be necessary to take a sophisticated view toward proximal antecedents and the manifold influences they could have on the development of subsequent addictive behavior.

**Impact of Proximal Antecedents Will Vary Depending on the Individual**

As mentioned earlier, the syndrome model cogently clarifies that a substance or activity, in and of itself, cannot cause addiction or be addictive. Rather, the relationship of the individual to these objects or activities is what results in addiction. This truth also holds for proximal antecedents. The same event—such as a trauma—can be a proximal antecedent for addiction for one person yet not influence the emergence of addiction for another. Indeed, in community-wide disasters, many people experience the same event, yet there are innumerable different types and intensities of reaction to it. Some people develop posttraumatic stress disorder and addiction, whereas others escape unscathed or even become ennobled by the trauma. Thus, clinicians and researchers cannot focus solely on proximal antecedents; rather, they must examine the whole constellation of responses a person has in relation to these antecedents to best evaluate their influence. Studying people's differential responses to the same proximal event is essential and informative.

**Treatment and Prevention Implications of Proximal Antecedents Are Unclear**

One of the major challenges of public health and clinical work is that, even if one can determine that certain proximal antecedents heighten the tendency toward addiction for the aggregate, it is notoriously difficult to determine the likelihood of addiction emerging for an individual. This is a well-known problem in other areas in which prediction is the goal, such as the prediction of suicidal or violent behavior. There are known risk factors for these types of events on a population basis, but for any given individual, even a large number of risk factors might not permit the accurate prediction of dangerous behaviors. This also occurs with addiction: Some individuals whose profile would suggest a strong tendency toward addiction do not develop it, whereas others with low likelihood do. Thus, for the treatment provider, it can be difficult to interpret the
impact of proximal antecedents. If a patient experiences major stressors, should the provider simply assume increased vulnerability to addiction and thus increase the level of care or number of treatment sessions? Patients might be highly resilient and not need an increasing level of care; from a public health perspective, more and unnecessary care can waste resources and increase treatment costs. Alternatively, a high-functioning, seemingly happy person can appear to have no proximal antecedents likely to lead to addiction yet nonetheless be highly vulnerable to it. For example, a well-known phenomenon is the upper-middle-class woman who seemingly has it all yet secretly drinks alone at home, and no one, including health care professionals, notices her vulnerability (Gomberg & Nirenberg, 1993). From a prevention standpoint, it is even more difficult to determine how to assign limited resources when the goal is to prevent rather than treat addiction. Given that most people do not develop addictive disorders, new research is necessary to help researchers and clinicians learn how to identify proximal antecedents that effectively increase the targeted focus on those who are most in need of prevention efforts.

Number and Type of Proximal Antecedents Are Seemingly Limitless

Given the myriad number and type of proximal antecedents that can occur, the question arises as to what extent a formal theoretical model can accurately account for these in a meaningful way. There are ways to cluster different types of events and determine their relative impact on the development of addiction, but such categorizations require empirical study to determine their validity, efficacy, and impact. The breadth of proximal antecedents can be, in the end, both a strength and a weakness: on one hand, spurring researchers and clinicians to think broadly about the full range of potential influences that can affect an individual's addiction potential, and on the other hand, so broad as perhaps to become confusing or ill defined. Close and careful evaluation of proximal antecedents is an important goal for clinicians and researchers, and ultimately, greater exploration can help to refine the syndrome model in this area.

Sophisticated Modeling Is Needed

The relationship among proximal antecedents, distal events, and addictive behavior is likely to require both large datasets and sophisticated statistical modeling that can account for their complex, multilayered, interactive, and recursive pathways. There will also be a need to replicate such analyses with various populations because proximal antecedents can differentially affect different groups, such as criminal justice, people who are homeless, military or veterans, community-based treatment, and adolescents.

CONCLUSION

In sum, the syndrome model holds great promise to illuminate how addiction takes root and also how it can be overcome. As scientists advance this model and refine its conceptual base with more empirical evidence, additional research will be necessary to evaluate important questions about proximal antecedents. The syndrome model provides an important statement about what addiction is and, equally as important, what it is not. It is a timely contribution now that addiction has, more than ever before, entered the mainstream of the mental health field and public consciousness. A spotlight shines on the addiction field more now than in any prior era. Even the term *addiction* is relatively new and continues to evolve in meaning over time—a reminder of how far the field has come and how far it still needs to go. Proximal antecedents are just one part of this larger picture, and as the field of addiction studies progresses, researchers will attain greater understanding of these important influences. Thus far, proximal antecedents are among the least studied risk factors for addiction, but they hold much promise in their power as tipping points, both positive and negative.

References


MD: Substance Abuse and Mental Health Services Administration.


A large proportion of the adult population consumes substances that have abuse potential, spanning alcohol, tobacco, illegal drugs, over-the-counter medications, and prescribed psychotropic drugs. These substances have the potential to cause addiction and disease. By age 28, almost 90% of the U.S. population have ingested some kind of beverage alcohol, and 80% have experienced at least one episode of intoxication (Johnston, O'Malley, Bachman, & Schulenberg, 2009). Significantly, almost 60% have used an illegal drug, illustrating that the motivation for consumption frequently surmounts prohibition against illegal behavior. Approximately 10.3% of the population that uses drugs (13.8% of men, 7.1% of women) qualify for a diagnosis of substance use disorder (SUD) at some time during their lifetime (Compton, Thomas, Stinson, & Grant, 2007). The cost exacted from society by substance use and SUD is enormous consequent to treatment and frequently associated chronic medical disorders (e.g., HIV, cirrhosis, chronic obstructive pulmonary disease), acute infectious diseases (e.g., sexually transmitted diseases), physical and neurological disability caused by traumatic injury (e.g., car and industrial accidents), crime, unemployment, poverty, homelessness, and psychiatric illness. Preventing SUD and these diverse associated outcomes is accordingly a high priority. Effective prevention of SUD, however, is contingent on comprehensively understanding etiology.

The peak prevalence of past-year alcohol and cannabis use disorders is before age 21 (Substance Abuse and Mental Health Services Administration, 2004), that is, within 5 years of completing pubertal maturation, cognitive development, and physical growth. Brain maturation, still ongoing, is not completed until approximately age 26 (National Research Council and Institute of Medicine, 2009). Accordingly, a developmental framework is essential for elucidating the etiology of SUD and for designing effective prevention strategies.

As we discuss in this chapter, suboptimal acquisition of psychological self-regulation during ontogeny, resulting largely from frontal cortex dysfunction, is the cardinal characteristic predisposing children and adolescents to SUD by early adulthood. Sometimes referred to as Type 2 (Cloninger, Bohman, & Sigvardson, 1981), this variant of SUD develops within a context of deficient or deviant socialization (Tarter, 1982). In many individuals, however, SUD manifests later in adulthood subsequent to normative socialization. In this situation, individuals engage in substance use in an attempt to relieve psychological dysregulation caused by exogenous or endogenous stressors. This variant of SUD, sometimes referred to as Type 1 (Cloninger et al., 1981), has less genetic loading and greater environmental influence on etiology than the Type 2 variant and tends to be less severe. These two variants of SUD have also been labeled essential and reactive to denote the relative salience of developmental processes and acute life stressors in SUD etiology (Tarter, 1982). Before discussing the etiology of these two types of SUD within a lifespan perspective, however, it is necessary to first specify the nature of the clinical disorder. Specifically, a
coherent body of etiology research findings can be accrued only if there is a consensus regarding the clinical phenotype. Because specifying the clinical outcome continues to hamper etiology research (see Conway, Compton, & Miller, 2006, and Conway et al., 2010, for review of the issues), it is important to address this issue at the outset of this chapter.

SPECIFYING THE CLINICAL OUTCOME OF SUBSTANCE USE

The clinical disorder ensuing from substance use is conceptualized in several overlapping ways. Viewed as a disorder of behavior excess, the defining characteristic is frequency or amount of substance consumption that deviates from cultural mores, societal norms, or laws. Alcohol and drug use disorders in this perspective are captured by the suffix *mania*, denoted respectively by *dipsomania* and *narcomania*. The problem is defined by behavior topology (quantity, frequency of consumption, amount used per consumption occasion, etc.) that deviates statistically, formally, or implicitly from consensually accepted parameters. For example, any quantity of alcohol consumption by a minor, or any quantity of marijuana use or cocaine use by a minor or an adult, is excessive because it violates the law. Consumption of any quantity of alcohol consumption by a minor, or any quantity of marijuana use or cocaine use by a minor or an adult, is excessive because it violates religious sanctions. Consuming alcohol to the point at which blood alcohol level reaches 0.1\% or higher is excessive when driving a car because it violates the law. Usually, however, the threshold distinguishing normative from excessive is vague (e.g., weekly acceptable amount of standard alcohol drinks containing 1 ounce of alcohol). The facetious refrain “an alcoholic is a person who drinks more than his or her physician” succinctly captures the difficulty of objectively defining the clinical phenotype according to excessive behavior.

Excessive behaviors remain a topic of intense interest insofar as they also encompass a large array of non–substance-using behaviors (sex, gambling, playing video games, shopping, etc.). Unfortunately, however, no clear-cut qualitative demarcation separates individuals who exhibit a particular propensity that is excessive (e.g., heliomania) from behavior that is normative (e.g., sunbathing). This dilemma calls into question the utility of designating the clinical phenotype in the context of excessive behavior. Moreover, because different standards or criteria denoting excess are applicable (e.g., legal, secular norms, cultural sanctions) and consumption patterns change over time, it is not feasible to conduct etiological research that is capable of yielding consistent findings. This shortcoming notwithstanding, *narcomania* is still the official diagnostic label used in Russia to denote the presence of problem drug use. At the descriptive behavioral level, however, it is interesting to note that the different substances having abuse potential are indicators of one underlying trait (Kirisci, Vanyukov, Dunn, & Tarter, 2002). In other words, the motivational impetus for consumption is the same for all classes of compounds.

One can also conceptualize the clinical disorder ensuing from substance use in terms of altered neurobiological functioning. Repetitive consumption results in biochemical and physiological adaptation (chronic tolerance), thereby changing the strength of drug-seeking behavior and reasons for consumption. Specifically, motivation for drug use shifts from obtaining positive or negative reinforcement to preventing punishing physiological distress and accompanying psychological misery that ensue after consumption stops. Drug use becomes a repetitive and compulsive behavior, thereby overtly reflecting the presence of addiction. In *addiction*, which is derived from the Latin term *addictus*, the person’s autonomy has transferred from self to the drug, analogous to a once-free person’s being reduced to a slave. More important, the mechanisms underlying the transition from voluntary to compulsive drug use, constituting an allostatic shift, are largely the same for all types and categories of substances having abuse potential (Koob & LeMoal, 2008). The clinical outcome of substance use is thus a circumscribed neurobiological disorder that might include co-occurring psychiatric disorders. Defining the clinical phenotype in this fashion has important advantages in etiology research, particularly the opportunity to use animal models to clarify the genetic and biological mechanisms underlying
variation in the salient predisposing psychological processes.

A broader perspective for denoting the clinical phenotype is codified in the fourth edition, text revision, of the *Diagnostic and Statistical Manual of Mental Disorders (DSM–IV–TR; American Psychiatric Association, 2000)*, the official taxonomy of the American Psychiatric Association. The clinical phenotype is any composite of health, psychological, or social problems resulting from a persistent pattern of substance consumption. Physical dependence (i.e., the neurobiological disorder described earlier) need not be present to assign a clinical diagnosis of SUD. In fact, the *DSM–IV–TR* does not require the presence of any necessary or defining characteristic to denote the presence of a disorder. Insofar as myriad negative outcomes can ensue from habitual substance consumption, a huge heterogeneous population is subsumed within the same diagnostic category. Assigning different individuals the same diagnosis despite their having vastly different and potentially nonoverlapping symptom presentations poses obvious serious challenges for etiology research. Moreover, the criteria for SUD diagnosis, and the array of problem areas making up the *DSM–IV–TR*’s criteria, have changed across successive editions. Hence, a person deemed affected at one time might be diagnostically normal at another time. In the forthcoming fifth edition of the *DSM* (the *DSM–5*), legal problems are no longer acceptable as a diagnostic criterion. Consequently, during the forthcoming period in which the *DSM–IV–TR* remains the official standard, the affected population will, on average, evince less antisociality than the population diagnosed with SUD according to previous (e.g., *DSM–IV–TR*) criteria. By changing the outcome criteria, the etiological determinants of SUD similarly change. These shortcomings notwithstanding, researchers and clinicians universally use the *DSM–IV–TR* criteria.

Paralleling the results on substance use behavior showing that different substances having abuse potential reflect one underlying trait (Kirisci et al., 2002), research has similarly shown that the different SUD categories are indicators of one underlying construct (Kirisci, Tarter, Vanyukov, Martin, et al., 2006), thus demonstrating that SUD is essentially one syndrome. This is a rather remarkable finding considering that (a) abusable compounds consist of different states of matter (solids, liquids, gases), (b) are self-administered through a variety of routes (oral, nasal, dermal, etc.), and (c) are metabolized differently. This variation among substances notwithstanding, the *DSM–IV–TR* SUD categories can be mapped onto a trait quantifying severity of externalizing disturbance (Krueger et al., 2002). The observation that the SUDs consequent to the use of legal drugs are located toward the left (less severe) pole, whereas the SUDs resulting from use of negatively sanctioned (illegal) drugs are located toward the right pole, illustrates that the main difference between the SUDs is severity of undercontrolled and norm-violating behavior.

Violation of the law, at least with respect to substance use, among youths is strikingly high. Lifetime prevalence of alcohol and nicotine use are 58.3% and 31.7%, respectively, in the most recent national survey of 10th-grade students conducted in 2008 (Johnston et al., 2009). Lifetime experience with any type of illegal drug is 34.1%. The actual prevalence may be even higher owing to the fact that high-school dropouts have higher than average rates of substance use. Moreover, truants from school on the day of the survey are more likely to consume drugs. The high prevalence of illegal behavior related to substance use is further illustrated by the finding that 16% of alcoholic beverages sold in the United States during 2001 were consumed by underage drinkers (Miller, Levy, Spicer, & Taylor, 2006). In effect, therefore, understanding the etiology of substance use leading to SUD requires elucidating the factors promoting illegal behavior. Moreover, considering that the peak prevalence of SUD resulting from alcohol and marijuana use is before the age of majority (Chen, O’Brien, & Anthony, 2005; Grant et al., 2004), etiology must be explicated within a developmental framework. The *DSM–IV–TR* taxonomy, however, is devoid of developmental criteria pertaining to SUD diagnosis. Furthermore, as discussed in the next section, the presence of two competing, irreconcilable models regarding the meaning of development additionally impedes progress in etiology research. Thus, not only is the clinical outcome of substance use (i.e., the clinical phenotype)
lacking consensual acceptance, but, as we discuss, research focusing on the etiology of substance use and SUDs has also been hampered by unresolved disagreement regarding a conceptual framework to guide systematic investigation.

COMPETING CONCEPTS OF DEVELOPMENT IN ETIOLOGY OF SUBSTANCE USE AND SUD

Substance use behavior is the necessary prodrome of SUD. Two mutually exclusive perspectives, the gateway hypothesis and common liability model, guide psychological research directed at understanding the etiology of substance use. These two perspectives not only shape the investigative agenda but also have sharply different ramifications on the practice of substance abuse prevention.

Gateway Hypothesis

The belief that consumption of one substance inexorably leads to consumption of other substances has a long history. This notion was officially advanced by Harry J. Anslinger, the first director of the Federal Bureau of Narcotics, to garner support from legislators and the public for drug policies banning cannabis sativa (colloquially, marijuana) for personal use (Booth, 2004). The assertion that cannabis use propels the ingestion of other, putatively more dangerous drugs has for decades made up the cornerstone of U.S. drug policy. Whereas habitual consumption of cannabis (and its associated by-products) is not free of risk for disease and addiction, the belief that this drug is a “gateway” to using other drugs, however, is at variance with both logic and empirical evidence.

The observation that most individuals who use hard drugs had prior experience with marijuana has mistakenly led to the conclusion that it is because of marijuana use that hard drugs are consumed. This error in logic, termed post hoc ergo propter hoc (“after this, therefore because of this”; Cathcart & Klein, 1998), results from an erroneous inference that because B (hard drugs) follows A (marijuana use), A therefore causes B. Notably, the most vigorous advocates of the gateway hypothesis have fallen victim to this logical error. For example, Kandel and Yamaguchi (2002) asserted “one licit drug is required [italics added] to make the progression to marijuana use” (p. 71). Moreover, they noted, “the identification of drug-specific risk factors is technically related to the demonstration of causal [italics added] linkages between stages” (p. 64). By this line of reasoning, drinking milk during childhood causes marijuana use.

The central tenet of the gateway hypothesis is that the order of using substances having abuse potential is invariant. Specifically, consumption begins with substances that are legal for adults (alcohol or tobacco) and is followed by the initiation of marijuana use (the putative gateway drug), which in turn leads to consumption of other so-called hard drugs. Empirical research has shown, however, that this pattern of transitions is far from invariant. Young et al. (1995) reported that in their sample, 42% of youths who violated the law—a population at very high risk for SUD—used marijuana before alcohol. Moreover, hard drugs are frequently consumed before marijuana (Golub & Johnson, 1994). Notably, the probability of developing alcohol use disorder and cannabis use disorder is unrelated to whether the order of consumption conforms to the gateway hypothesis or to the opposite pattern (i.e., marijuana use before alcohol use; Tarter, Vanyukov, Kirisci, Reynolds, & Clark, 2006).

Advocates of the gateway hypothesis (e.g., Kandel & Yamaguchi, 2002) have also claimed that each substance reflects a developmental stage. In other words, drug type maps onto developmental stage and vice versa. Considering that many studies have shown that the pattern of transition from using one drug to another is not invariant, acceptance of the gateway hypothesis requires acknowledgment that it is possible, or even conceivable, to experience reverse development. In other words, claiming that each specific substance indicates a stage of development requires acceptance of the notion that backward development is possible, which, for example, is putatively the case in youths who consume cannabis before alcohol and tobacco.

Common Liability Model

In contrast to the gateway hypothesis, which specifies that unique risk factors portend use of each type
of drug, the common liability model asserts that a complement of age-specific psychological processes is associated with risk for using all substances that could lead to SUD diagnosis. The constellation of psychological characteristics is theorized to be a latent unidimensional trait, termed liability (see Vanyukov, Tarter, et al., 2003). Severity of liability is the primary factor determining whether consumption will be confined to legal or illegal substances. The order in which drugs are consumed is considered to have little relevance to the etiology of SUD other than as a general indicator of liability severity and, accordingly, the prospect of deviant socialization. The more severe the liability at a particular age, the shorter the duration to substance use onset and proclivity to consume illegal drugs. Thus, concomitant with the findings reported by Krueger et al. (2002) showing covariation between severity of externalizing disorder and social negativity of SUD type (e.g., alcohol use disorder vs. cocaine use disorder), one can conclude that the psychological components of liability, varying only in severity, are congeners to risk for developing all categories of SUD.

The finding that 100% of genetic risk and approximately 80% of phenotypic risk are common to all SUD diagnoses provides support for the common liability model (Kendler, Jacobson, Prescott, & Neale, 2003; Tsuang et al., 1998). In addition, it is noteworthy that co-occurring SUDs, often involving drugs having opposite pharmacological properties (e.g., alcohol and nicotine), are highly prevalent. The observation that substances having stimulant and depressant properties are also frequently consumed simultaneously indicates that common reinforcement effects, not drug-specific metabolic and pharmacological mechanisms, are most integral to risk (Koob & LeMoal, 2008). Consequently, it is not surprising that when a preferred substance is not available, many others are effective substitutes.

Clues to the cardinal feature making up the psychological liability that is congeners to all SUD categories are obtained from the empirical literature showing that attention deficit/hyperactivity disorder (ADHD) and bipolar disorder are independent predictors of SUD (Biederman et al., 1997). Not only do ADHD and bipolar disorder during childhood amplify risk for SUD (Lambert & Hartsough, 1998; Molina & Pelham, 2003), but both disorders also frequently co-occur with SUD during adolescence (Wilens, Biederman, Abrantes, & Spencer, 1997). Provocative evidence indicating that overlapping bipolar disorder and ADHD symptoms make up a genetically distinct disorder (Faraone, Biederman, Menning, Wozniak, & Spencer, 1997) that is ubiquitously featured by emotion and behavior dysregulation (see Zepf, 2009, for review) may have important ramifications for understanding the biobehavioral basis of SUD liability. Specifically, the relationships between ADHD and bipolar disorder, involving disruption of neural circuits integrating the striatum, ventral frontal cortex, and amygdala (Muralidharan, Yoo, Ritschel, Simonova, & Craighead, 2010), and SUD suggests that SUD vulnerability may consist of a neurological disorder pertaining to regulation of affect and behavior.

Bipolar disorder in childhood has also been reported to increase risk for early-onset alcohol and drug addiction independently of conduct disorder (CD; Biederman, Faraone, Wozniak, & Montaix, 2000). Studies of children with hyperactivity or an ADHD diagnosis have pointed to a mixed pattern of results. Self-report (Tarter, Kirisci, Feske, & Vanyukov, 2007) and direct measurement of hyperactivity using an accelerometer (Moss, Blackson, Martin, & Tarter, 1992) have implicated a direct association between behavioral hyperactivity and elevated risk for SUD, whereas studies using a DSM-IV-TR diagnosis of ADHD have indicated that frequently concomitant conduct problems mediate or moderate the association between hyperactivity and SUD (Fergusson, Horwood, & Ridder, 2007). CD involving undercontrolled behavior predisposes individuals to substance use by facilitating low adherence to societal norms. ADHD can potentiate this risk inasmuch as poor self-regulation fosters interpersonal conflict with parents and teachers as well as marginalization by normative-behaving peers. Furthermore, substance use in children with ADHD and without CD may reflect an attempt to dampen psychological dysregulation. In effect, ADHD alone, CD alone, and co-occurring ADHD and CD may promote substance use via different, albeit overlapping, etiological
pathways. In each scenario, deficient psychological self-regulation in minors potentiates violating social norms, mores, and the law evinced as substance use. In the next section of this chapter, we review the empirical literature within a developmental perspective with the aim of showing that deficient psychological self-regulation throughout the life span predisposes to all categories of SUD.

PSYCHOLOGICAL SELF-REGULATION AND DEVELOPMENT OF SUD

All of the features associated with vulnerability to SUD reflect aspects of deficient psychological self-regulation. Considering that SUD is a developmental outcome, and self-regulation is progressively acquired during ontogeny, it is essential therefore to explicate SUD etiology in relation to self-regulation at different stages of development.

Prenatal Stage of Development

The individual's genotype is established at the moment sperm and egg unite to form the zygote. During prenatal development, spanning embryogenesis and fetal maturation, gene–environment interactions in utero determine the production of amino acids and proteins integral to morphological and functional development of the brain.

Women who use alcohol and other drugs during pregnancy frequently qualify for SUD. Accordingly, genetic risk for SUD is conferred from the mother to the fetus. Moreover, substance-abusing women are more likely to mate with men who qualify for SUD (Vanyukov, Moss, & Tarter, 1994). Hence, genetic risk is potentially also conferred to the fetus via patrilineal transmission. In view of findings demonstrating that the childhood behavioral disturbances associated with SUD vulnerability are substantially heritable (Iacono, Malone, & McGue, 2008), one can conclude that prenatal neurobiological development is the outset point of the ontogenetic trajectory to SUD.

Pregnant substance-using women potentially confer additional risk for SUD to the fetus through neurological injury induced by nutritional deficiency, exposure to toxins (e.g., lead), infections, and stress hormones. Moreover, the most commonly consumed abusable drugs are teratogens. A thorough review of the literature by Derauf, Kekatpure, Neyzi, Lester, and Kosofsky (2009) concluded that the legal and illegal drugs most frequently abused by pregnant women cause injury primarily to the frontal cerebrum and its subcortical connections in the developing fetus. Moreover, prenatal exposure to tobacco, alcohol, cocaine, and marijuana is associated with lower gray matter and parenchyma volume along with smaller head circumference in school-age children (Rivkin et al., 2008). These important findings demonstrate that in-utero events may exacerbate the genetic liability on neurodevelopment, thereby further impeding postnatal acquisition of psychological self-regulation. Significantly, one study has shown that high genetic loading for SUD, interacting with adverse events during fetal development, impairs neurological functioning that is overtly evinced as poor self-regulation during infancy (Huizink, deMedina, Mulder, Visser, & Buitelaar, 2002).

Infancy Stage of Development

The term *colic* is widely applied to infants who exhibit periods of persistent crying, fitful and irregular sleep, and low receptivity to the caregiver's attempts to ameliorate distress. Although *colic* is often transitory, disturbances in physiological regulation can have significant long-term effects. For example, sleep problems in infancy are associated with increased risk for developing ADHD in later childhood (Thunström, 2002). This latter disorder is well known to amplify the risk for SUD. Also noteworthy is that persistent crying during infancy is associated with increased risk for hyperactivity measured at ages 8 to 10 (Wolke, Rizzo, & Woods, 2002) and discipline problems measured at age 5 (Rao, Brenner, Schisterman, Vik, & Mills, 2004). Infants who are very susceptible to distress and cannot readily be soothed require more than ordinary parental investment. Hence, the baby's characteristics hamper bonding to caregivers, especially if parenting skills, motivation in child rearing, resources, and social support are lacking, or a physical or psychiatric illness impedes role performance. These latter parenting factors are commonly present among families in which youths evince behavior problems.
at a young age and subsequently use psychoactive substances.

Severity of the baby's physiological reactivity, interacting with quality of nurturing by the primary caregiver, thus determines whether secure attachment will be established. In the absence of secure attachment, management of emotional distress is less likely to shift from the caregiver to the child. Hence, for example, insecure attachment of the anxious avoidant type subsequently manifests later in childhood as labile emotion and anger (Blehar, Lieberman, & Ainsworth, 1977), which frequently presages aggressive and antisocial behavior (Renken, Egeland, Marvinney, Sroufe, & Mangelsdorf, 1989). Many studies have shown that these latter childhood characteristics amplify risk for substance abuse during adolescence and thereafter for SUD.

The capacity to exercise emotion and behavior self-regulation, especially under conditions of stress, also depends on the capacity to control attention. This capacity usually emerges in the first 2 weeks after birth when the baby is able to distinguish the face of the primary caregiver from other faces (Gopnik, 2009). Gaze at the caregiver, accompanied by smiling and appealing vocalization, are reciprocated by the caregiver in the form of positive emotional reaction and nurturing such as smiling, soothing vocalizations, and tactile stimulation. Positive caregiver response to the baby thus reinforces the baby's attention, pleasure, and experience and establishes the foundation for learning that the caregiver is a reliable source for alleviating distress. Secure emotional attachment is thus the foundation for managing emotional distress, which transitions during development from caregiver to self.

Quality of infant-caregiver bonding is partly mediated by biochemical mechanisms. Plasma oxytocin level during pregnancy and postpartum (Feldman, Weller, Zagoory-Sharon, & Levine, 2007) predicts strength of mother-infant attachment. Oxytocin level also correlates with the amount of affection the mother directs at the baby during the first several months after birth (Gordon, Zagoory-Sharon, Leckman, & Feldman, 2010). Accordingly, high oxytocin levels in infant and mother portend strong mutual engagement and affective synchrony (Feldman et al., 2007). One important consequence is effective and efficient innervation of the hypothalamic–pituitary–adrenal axis to regulate stress (Kraemer, 1992). Notably, prolonged release of cortisol (and other glucocorticoids) in response to chronic stress causes lasting neurological injury (Sapolsky, 1996). Hence, persisting stress associated with insecure attachment can have an injurious impact on neurological development. Significantly, high cortisol levels in 6-month-old babies are associated with neurophysiological disturbances in the frontal cortex (Buss et al., 2003), which, as noted earlier, encompasses neural circuitry that is integral to psychological self-regulation. In effect, neurogenetic liability is exacerbated by stress-induced neurological injury so as to further impede acquisition of psychological self-regulation. Hence, chronic stress concomitant to insecure attachment via stress-induced injury to the brain is likely to further impede acquisition of psychological self-regulation.

Complicating the situation, however, is the finding that chronic stress appears to produce a down-regulation of hypothalamic–pituitary–adrenal axis activity. Consequently, research has shown that attenuated release of cortisol is related to low socioeconomic status (Lupie, King, Meaney, & McEwen, 2001), child maltreatment (Hart & Brassard, 1991), and emotional trauma (Yehuda, Halligen, & Grossman, 2001). These factors are well known to be more prevalent among youths who develop SUD. Furthermore, offspring of fathers who have early-onset SUD are at especially high genetic and environmental risk to develop SUD before age 25 (Cloninger et al., 1981) and to exhibit attenuated cortisol response (Vanyukov et al., 1993). Last, it is noteworthy that youths with externalizing disorders, the most ubiquitous behavioral characteristic associated with SUD risk, exhibit a diminished cortisol response (McBurnett, Lahey, Rathouz, & Loeber, 2000; Snoek, Van Goozen, Matthys, Buttelaar, & Van Engeland, 2004). These findings suggest that psychological dysregulation, conjoint with attenuated hypothalamic–pituitary–adrenal axis activation in response to threat, might underlie risky behavior, including substance use, presaging SUD.

In summary, the cumulative effects of genetic and intrauterine influences, in conjunction with
parenting characteristics, predispose some babies to interactions with caregivers that hinder acquisition of emotional and behavioral self-regulation. In addition, low levels of the neuropeptide hormone oxytocin in the caregiver might undermine, at least in part, parental motivation for bonding. One consequence of the insecure attachment that results from these interactions is stress, which, via chronic activation of the hypothalamic-pituitary-adrenal axis, impedes development of neural systems integral to acquiring psychological self-regulation. Hence, suboptimal bonding or attachment is the first measurable developmental outcome biasing the ontogenetic trajectory toward SUD. Interventions directed at potentiating parent-child bonding most likely diminish the risk of psychological dysregulation in childhood, substance use in adolescence, and subsequently SUD.

**Early Childhood Stage of Development**

By age 3, dispositional patterns of behavior and a predictable style of reacting to novel and stressful situations are reasonably well established. These childhood psychological characteristics, consisting of temperament traits, have a strong heritable basis. Notably, deviations on temperament traits reflecting poor self-regulation heighten risk for SUD. Caspi, Moffitt, Newman, and Silva (1996) observed that low attention, high behavioral activity, and negative emotionality in 3-year-old children predicted SUD in young adulthood. Sleep dysrhythmicity, a dimension of temperament within the framework advanced by Thomas and Chess (1977), measured in children ages 3 to 5 is a predictor of alcohol and drug use at age 14 (Wong, Bower, Fitzgerald, & Zucker, 2004). The constellation of characteristics constituting difficult temperament (manifested as slow adaptation to new situations, high mood intensity, high emotional negativity, low rhythmicity in everyday routines, and avoidance or withdrawal from new situations) frequently presages CD in middle and late childhood (Maziade et al., 1990), which is well known to increase risk for SUD.

Children with difficult temperaments are also more strongly influenced by quality of rearing experience than children who have more adaptable temperaments (Belsky, 2005), thereby underscoring the importance of the quality of interactions between the individual and the social environment on risk for developing SUD.

However, it is also important to note that the child's rearing environment is affected by the caregiver's adverse experiences during development. For instance, a history of physical abuse in the mother has been observed to be related to negative emotionality in offspring and inability to manage stress (Lang, Gartstein, Rodgers, & Lebeck, 2010). Negative emotionality in the mother also covaries with child's anger (Kochanska, Clark, & Goldman, 1997). These latter childhood characteristics are well known to augment risk for SUD. In view of findings showing that poor regulatory functioning in the child results from interactions with parents having low sensitivity to the child's needs (Gartstein, Crawford, & Robertson, 2008), one can conclude that parenting quality exercises a lasting influence on children during the critical period in which psychological self-regulation is established.

The 3-year-old child possesses to a degree the attention resources required to self-monitor behavior, appraise the environment, and suppress impulses (Rothbart & Sheese, 2007). Subsumed within the term effortful control, these processes are integral to adaptive behavior. Significantly, low effortful control is associated with both internalizing (e.g., negative affect, anxiety, depression) and externalizing (e.g., aggression) psychological self-regulation disturbances (Muris, Van der Pennen, Sigmond, & Mayer, 2008). The observation that low effortful control in 33-month-old children portends intense expression of both negative (e.g., anger) and positive (e.g., joy) emotions (Kochanska, Murray, & Harlan, 2000) illustrates that the cardinal facet of SUD liability relates to regulatory capacity and not merely magnitude of expression (i.e., “too high” or “too low”) on psychological traits. Notably, almost 80% of variance in effortful control is heritable (Lemery-Chalfant, Doelger, & Goldsmith, 2008). Low effortful control is also associated with a smaller volume of orbitofrontal cortex in adolescents (Rothbart, Sheese, & Posner, 2007; Whittle et al., 2008). Thus, although systematic research has not yet been directed at elucidating the association between effortful control and development of SUD,
it is noteworthy that sons of fathers with alcoholism score lower than peers on this trait (Eiden, Edwards, & Leonard, 2004). Children who have low effortful control are more likely than peers to receive suboptimal parenting (Eisenberg et al., 2005). In addition, adaptation to stress is suboptimal (Valiente, Lemery-Chalfant, & Swanson, 2009). Thus, similar to other potential risk-enhancing traits, low effortful control affects SUD risk via contextual influences.

**Middle Childhood Stage of Development**

Children with severe psychological dysregulation, evinced as either difficult temperament or psychopathology (e.g., ADHD), are at high risk for long-term social maladjustment. As many as 90% of youths with ADHD qualify for another psychiatric disorder. In particular, 47% of youths with ADHD have co-occurring CD, and 65% also qualify for a diagnosis of oppositional defiant disorder (Willcutt, Pennington, Chhabildas, Friedman, & Alexander, 1999). These externalizing disorders promoting deviant socialization are well-known precursors of substance use and SUD (Krueger et al., 2002).

ADHD alone and ADHD co-occurring with CD are not distinct disorders but rather are variants of one disorder in which the comorbid condition has stronger genetic loading (Rhee, Willcutt, Hartman, Pennington, & DeFries, 2008). As discussed previously in this chapter, risk for SUD is increased when ADHD co-occurs with CD. In this more severe variant (ADHD and CD), the child is unable to meet age-appropriate role expectations, has persisting conflict with parents and teachers, and is marginalized from socially normative peers. Friendships are formed with peers who are similarly dysregulated and have low motivation to adhere to societal norms. In effect, a core disorder of impaired self-regulation (ADHD) in conjunction with failure to comply with age-appropriate norms of childhood behavior manifests as conflictual (e.g., CD, oppositional defiant disorder) interaction with adults and peers. This circumstance can bias development toward deviant socialization. Accordingly, the association between ADHD and substance abuse is mediated by antisocial behavior (Marshal & Molina, 2006).

**Late Childhood Stage of Development**

The discussion up to this point has shown that young children at high risk for SUD evince psychological dysregulation encompassing impaired executive cognitive capacities, behavioral undercontrol, and poor modulation of emotions. These characteristics have been shown in very young children; however, by age 10 they have been found to comprise a unidimensional trait (Tarter et al., 2003). The score on this trait, termed neurobehavioral disinhibition (ND), captures diverse cognitive, behavioral, and emotional processes that differentiate sons of affected (with SUD) and nonaffected (without SUD) fathers (Tarter, Kirisci, Habeych, Reynolds, & Vanyukov, 2004). Moreover, the score on this psychological trait is a significant childhood predictor of SUD manifested by early adulthood (Tarter et al., 2004). To date, research has not been conducted to determine whether the same psychological processes aggregate into one trait among girls.

It is also interesting to note that the ND score correlates with activation of the frontal cortex but no other cortical region while performing a task requiring inhibition of an oculomotor response to a prepotent stimulus (McNamee et al., 2008). In effect, low physiological activation is associated with a more severe ND score. Moreover, the total score on the ND trait predicts SUD beyond the contribution of any of its constituent cognitive, affective, and behavioral indicators of self-regulation (Mezzich et al., 2007). In effect, the ND trait appears to capture, in holistic fashion, the individual’s quality of self-regulation linked to the neurological substrate. Research has yet to be conducted to ascertain whether the cognitive, behavior, and emotion components of dysregulation make up a unidimensional trait among children younger than age 10. Determining the age during development when these disparate processes cohere into a unidimensional psychological trait is important for advancing research from a current emphasis on manifold particular elements of SUD risk to a thematically and empirically holistic framework anchored to neurological functioning. In view of a large empirical literature pertaining to gender differences in overt manifestation of psychological characteristics, the ND trait can also be informative for investigations.

Adolescent Stage of Development
Adolescence, essentially encompassing the teen years, is the developmental period in which adult behaviors are assumed, such as working for pay, driving a car, and forming intimate relationships. Acceptance by society that adolescence is the preparation phase for assuming adult roles and responsibilities does not, however, extend to allowing consumption of legal drugs such as alcohol and nicotine. Thus, whereas adolescents can make major health decisions without parental consent (e.g., obtaining an abortion), perform complex and dangerous technical tasks (e.g., obtaining a license to fly an airplane), enlist in the military, and be prosecuted as adults in court, it is illegal for them to drink alcoholic beverages or smoke tobacco. This prohibition notwithstanding, consumption of alcohol and tobacco by adolescents is strikingly high. Results of the most recent national survey in 2008 revealed that lifetime prevalence of alcohol and tobacco use among high school seniors is 71.9% and 44.7%, respectively (Johnston et al., 2009). Moreover, the amount consumed is often excessive, as indicated by a lifetime intoxication prevalence of 54.7%. Indeed, 45% of high school seniors endorse the statement that they drink to get “moderately high” or “very high.” Significantly, the highest past-year prevalence of alcohol use disorder diagnosis peaks before age 21—that is, before the age at which legal purchase and consumption of alcoholic beverages is allowed (Grant et al., 2004). Thus, for most of the adolescent population, the motivation to emulate adults is stronger than the motivation to adhere to the law.

The high prevalence of alcohol consumption among adolescents is due to numerous factors, including but not limited to disengagement from parental oversight, transportation mobility, fiscal resources obtained from working, and increased access to drugs and alcohol from friends at work, school, and parties. Simultaneously assuming adult roles and rebelling against authority, adolescents thus have ample opportunity to initiate substance consumption. Notably, 92% of high school seniors report that it is “fairly easy” or “very easy” to obtain alcohol. Furthermore, marijuana, cocaine, heroin, and amphetamines are reported to be “fairly easy” or “very easy” to obtain by 83.9%, 42.4%, 25.4%, and 47.8% of high school seniors, respectively (Johnston et al., 2009). Adolescence is also a particularly stressful period owing to multiple adaptational challenges concomitant to striving for acceptance by peers, starting and ending romantic relationships, maintaining academic performance, managing a work schedule, and fulfilling obligations at home. Analogous to adults, adolescents who harbor beliefs about the benefits of substance use for alleviating stress are prone to initiate consumption.

Hyposensitivity to the neuropharmacological effects of alcohol and drugs during adolescence may lead to consumption of dangerous quantities (Spear, 2000). Because a relatively larger amount needs to be consumed to obtain the desired subjective effect, risk for neurotoxic injury is augmented during a developmental period when the neural circuitry is undergoing extensive reorganization. It is thus noteworthy that the results of one study revealed that substance use in adolescents causes neurologic injury, localized primarily in frontal cortex (Squeglia, Jacobus, & Tapert, 2009). This finding suggests that the toxic effects of abusable drugs impede neuromaturation, thereby further exacerbating genetically determined psychological dysregulation underlying risk for SUD. In addition, consumption of large quantities potentiates rapid development of tolerance, thereby establishing the motivation for compulsive drug seeking to prevent onset of withdrawal. In addition to neurodevelopmental determinants of hyposensitivity, it is noteworthy that Schuckit and Smith (2000) reported that in young adults heritable factors account for 40% of the variance in subjective response to alcohol. Accordingly, a positive family history of alcoholism is associated with lower sensitivity to the effects of alcohol than is a negative history (Schuckit, 1994). Moreover, the magnitude of subjective response to alcohol is a predictor of alcoholism as many as 15 years later.
These findings suggest that heritable low sensitivity to the pharmacological effects of abusable drugs amplified by neurodevelopmental susceptibility during adolescence predisposes some individuals to consumption of large quantities, thereby potentiating rapid progression to physical dependence.

Timing and rate of sexual maturation also affect risk for SUD. Onset of puberty triggers profound biochemical and physiological changes promoting physical development (increase in height and body mass), resulting in adultlike appearance (e.g., facial hair in boys, breast development in girls). Many studies have shown that precocious maturation amplifies risk for serious long-term problems and maladjustment, including unplanned pregnancy, school dropout, substance abuse, and antisocial behavior (Dawes et al., 1999; Magnusson, Stattin, & Allen, 1985; Patton et al., 2004). One mechanism responsible for this heightened risk appears to involve a propensity of early-maturing youths (who appear older than peers of the same chronological age) to affiliate with more mature youths who engage in risky behaviors such as sex, drinking alcohol, and smoking. Initiation into substance use facilitated by peers thus occurs at a time before social skills are consolidated to ensure desistence. This propensity to engage in substance use is likely further amplified among youths who have had low psychological self-regulation since childhood.

Evidence has also accrued demonstrating that sex hormones, particularly testosterone, potentiate expression of psychological characteristics that are associated with SUD risk. In a thorough review of the literature, Archer (2006, p. 332) concluded that nonpathological elevation of testosterone level promotes “leadership, toughness, personalized power and aggressive dominance.” Notably, testosterone level has been reported to be higher among male adolescent substance users than among age-matched abstainers (Udry, 1990). Moreover, testosterone level has been reported to be elevated in early-onset alcoholism (Dabbs, Hopper, & Jurkovic, 1990). This variant of alcoholism is primarily distinguished by high genetic loading and adverse environment early in life presaging deviant socialization (Cloninger et al., 1981). Furthermore, results of a prospective study have indicated that testosterone level during late childhood is a significant, direct predictor of SUD in adulthood (Reynolds et al., 2007). In addition, this study found that disruptive behavior in early adolescence and affiliation with deviant peers, as well as non-normative attitudes and delinquency in mid-adolescence, mediate the association between testosterone level and SUD in adulthood. Youths with high testosterone levels who live in a disadvantaged neighborhood appear to display aggressive behavior as a strategy to assert social dominance (Schaal, Tremblay, Soussignan, & Susman, 1996). Neighborhood quality has also been shown to correlate inversely with testosterone level in 10- to 12-year-old boys, which in turn predicts aggressive behavior 2 years later and an adjustment style at age 16 marked by dominance striving, low adherence to social norms, and affiliation with deviant peers, leading to SUD in early adulthood (Tarter et al., 2009).

Neuromaturational changes occurring during adolescence further amplify risk for substance use initiation (Ivanov, Schulz, London, & Newcorn, 2008; Spear, 2000; Tarter et al., 1999). The neural circuitry that modulates emotions, behavior control, and the executive cognitive capacities completes maturation in early adulthood. As many as 40% of synapses involving neural circuits that are not used on an ongoing basis are eliminated during adolescence. At the same time, myelination of neurons is taking place to increase functional efficiency of neural circuits that are integral to ongoing environment interactions (Rakic, 1996). These dynamic changes involving reorganization of neural circuitry underlie risky, undercontrolled behaviors and poor modulation of emotions during adolescence (Spear, 2000), a time when access to alcohol and drugs and motivation for commencing substance consumption are increased. Significantly, negative emotionality and poor behavior control during late adolescence predicts SUD outcome within the ensuing 5 years (Elkins, King, McGue, & Iacono, 2006).

In the face of the profound biological changes occurring during adolescence, in conjunction with manifold opportunities to engage in risky behaviors, it is not surprising that youths scoring high on the ND trait are at elevated risk for SUD. Not only has it
been shown that this trait measured during midadolescence predicts subsequent development of SUD, but emerging research has also pointed to several key factors during this life stage that mediate the relation between this facet of vulnerability and clinical outcome. Besides the capacity of the ND trait to directly predict SUD in boys, several factors during adolescence have also been shown to mediate the relation between ND in childhood and SUD in adulthood. These factors include distorted social cognition (Kirisci, Tarter, Vanyukov, Reynolds, & Habeych, 2004), affiliation with socially nonnormative peers (Tarter et al., 2004), and residing in a disadvantaged neighborhood (Ridenour et al., 2009). A high ND score, in conjunction with early-onset substance use, nonnormative peers, and SUD in parents, has been reported to confer a .93 probability of developing SUD by early adulthood (Kirisci, Vanyukov, & Tarter, 2005) as well as to predict nonresponsiveness to brief prevention intervention (Kirisci, Tarter, Vanyukov, & Reynolds, 2006). Although research remains to be conducted to fully explicate the variety of pathways to SUD emanating from poor psychological self-regulation, the emerging findings have suggested that multiple biological systems might be involved in the development of ND. Moreover, the influence of this trait on developing SUD is intricately bound with multiple environment contexts (i.e., phenotype-environment correlation), thereby establishing opportunity for contagion and selection influences potentiate initiation during adolescence and subsequently habitual consumption leading to SUD.

Neurodevelopmental Mechanisms
Underlying Psychological Dysregulation
Poor postural control during infancy has been reported to significantly predict alcohol dependence 3 decades later (Manzardo et al., 2005). In addition, stationary ataxia (upper body sway while standing) has been reported among youths who are at high risk for SUD (Hegedus, Tarter, Hill, Jacob, & Winsten, 1984). Direct measurement of motor activity using an accelerometer has also revealed that children at high risk for SUD exhibit more movement while performing tasks requiring focused attention (Moss et al., 1992).

Neurophysiological studies have indicated that in addition to deficient inhibitory motor control, disrupted arousal regulation might also make up a component of the risk for SUD. Several aspects of the central and peripheral nervous systems control arousal regulation. Research related to the association between arousal and risk for SUD has, for the most part, focused on cortical arousal, measured by electroencephalograph waveform frequency. The paucity of systematic research notwithstanding, the available results point to decreased alpha activity (9 Hz–12 Hz) among offspring of parents with alcoholism (Finn & Justus, 1999; Propping, Kruger, & Mark, 1981). In addition, increased beta activity (13 Hz–18 Hz) has been reported in unaffected first-degree adult relatives of people with alcoholism (Bauer & Hesselbrock, 1993; Gabrielli et al., 1982; Pollock, Earleywine, & Gabrielli, 1995) as well as in children of parents with alcoholism (Rangaswamy et al., 2004). Notably, heritability accounts for most of the variance in electroencephalograph activity (Propping et al., 1981). Hence, genetically predisposed low alpha-wave and high beta-wave activity might not only be related to poor attention control but also account for poor emotion modulation and low behavior control among youths who are at high risk for SUD.

As we discussed previously, ADHD amplifies risk for SUD and shares significant genetic variance with SUD. Undercontrolled behavior, poor modulation of emotions, and low executive cognitive capacities are present in youths who are at high risk for SUD as well as children with ADHD. Moreover, many studies have pointed to a frontal subcortical dysfunction in children with ADHD (Castellanos et al., 2001). Studies of youths at high risk for SUD have similarly revealed a frontal subcortical dysfunction on the basis of neurophysiological and neuroimaging findings. Notably, diminution of the P300 wave of the event-related potential is most pronounced over the frontal cortex for high-risk individuals (Bauer, 1997; Bauer & Hesselbrock, 2001). It is significant that low P300 amplitude in childhood and adolescence has been shown to predict SUD (Habeych, Sclabassi, Charles, Kirisci, & Tarter, 2005; Viana-Wackermann, Furtado, Esser, Schmidt, & Laucht, 2007). Moreover, neurobehavioral
disinhibition—encompassing cognitive, emotional, and behavioral correlates of psychological dysregulation—mediates the relation between P300 amplitude in childhood and SUD manifested by early adulthood (Habeych, Charles, Sclabassi, Kirisci, & Tarter, 2005). The results of functional MRI have revealed that high-risk youths evince hypoactivation in the frontal cortex when required to perform challenging cognitive tasks (McNamee et al., 2008; Schweinsburg et al., 2004). Research has also shown that the executive cognitive functions subserved largely by dorsolateral prefrontal cortex are critical for transitioning from substance use to SUD (see George & Koob, 2010, for review). These executive cognitive capacities encompass strategic planning, self-monitoring, goal-directed behavior, attention control, and flexible problem solving. On a moment-to-moment basis, the executive cognitive capacities are integral to exercising regulatory control of emotional and behavioral responses that are appropriate to the particular circumstance. Accordingly, these capacities are integral to inhibiting the impulsive behavior known to predispose individuals to SUD. Through the use of internal language (thought), the executive capacities mediate motivation and decision making during the interval between stimulus and behavioral response. Considering that variation in executive cognitive abilities is large part accounted for by heritability (Friedman et al., 2008) and that these cognitive abilities are related to the DRD4 receptor, which has particularly high density in the anterior cortex (Mulcane & Kerwin, 1997), we conclude that higher order mental capacities having a neurogenetic basis are integral to SUD risk. Consequently, improving executive cognitive function capacities using neurorehabilitation strategies might be an effective strategy to prevent SUD by inculcating control over behavior and emotions along with avoidance of poor choices and decisions.

It is noteworthy that heritability accounts for more than half the variance in P300 amplitude among 18-year-olds (Perlman, Johnson, & Iacono, 2009). These findings point to the possibility that attenuated amplitude of this wave in the frontal cortical region among high-risk individuals reflects a neurogenetic disturbance. Furthermore, it has been reported that up to 100% of variance in executive cognitive capacity is heritable (Friedman et al., 2008). Low executive cognitive function capacity has been documented in many studies of high-risk youths (Tarter et al., 2009). A component of executive cognitive function, effortful control, has heritability of 79% (Lemery-Chalfant et al., 2008), and smaller orbitofrontal cortex volume has been demonstrated in children who score low on this trait (Whittle et al., 2008). Low effortful control is also related to poor regulation of emotion and aggression (Muris et al., 2008), which frequently presages substance use and SUD.

The physiological disturbances underlying deficient psychological self-regulation might emanate from a morphological abnormality of the brain. Children of parents with alcoholism have been reported to have smaller intracranial volume (Gilman, Bjork, & Hommer, 2007). This potentially important finding clearly awaits replication; however, it is noteworthy that whole brain mass, as well as white and gray matter mass, are largely genetically determined (Baañé, Hulshoff, Boomsma, & Posthuma, 2001). Research has also revealed that the 7-repeat polymorphism of the DRD4 receptor gene influences the size of the dorsolateral prefrontal cortex (Shaw et al., 2007). In another study, density of the DRD4 receptor was found to be related to smaller volume of the cingulate gyrus, hippocampus, amygdala, and cerebellum in 8- to 24-year-old individuals who were at high risk for SUD (Benegal, Anthony, Venkatasubramanian, & Jsyakumar, 2007). Significantly, the DRD4 polymorphism has been related to both executive cognitive processes and risk for alcoholism (McGeary, 2009). Although there is a strong need to replicate and extend these latter findings, the results at this juncture point to a disturbance predisposing individuals to SUD that spans all levels of biological organization. Specifically, genetic, particularly dopaminergic, mechanisms underlie variation in frontal subcortical morphology, which in turn accounts for the physiological and psychological manifestations of low psychological self-regulation. Clearly, systematic research is required to flesh out the details pertinent to the mechanisms influencing each level of biological functioning to comprehensively characterize the diathesis for SUD.
Adult-Onset SUD After Normative Development

A large segment of the population develops SUD during adulthood despite normal childhood and adolescent development. In this circumstance, substance use is not a result of deficient acquisition of self-regulation and suboptimal socialization. Rather, adult-onset SUD is the culmination of habitual substance use that is motivated, at least initially, by the desire to alleviate discomfort. Substance use behavior is a highly ingrained coping strategy in Western society. Caffeine is consumed to combat fatigue. Alcohol and nicotine are used to alleviate tension even though they exert opposite effects of sedation and stimulation. Habitual consumption of over-the-counter drugs, herbal preparations, energy beverages, and prescribed medications underscores the acceptability and prevalence of consuming chemicals to alleviate aversive subjective states. However, because habitual consumption of psychoactive compounds can lead to serious health, psychiatric, interpersonal, and legal problems, some users will qualify for SUD diagnosis. In this context, SUD is an epiphenomenon, that is, a secondary condition accompanying and caused by another primary condition. In the DSM-5 taxonomy, only two of 11 criteria need to be present to assign a diagnosis. Considering the almost limitless endogenous (e.g., psychiatric illness, pain, insomnia, fatigue) and exogenous (e.g., job loss, trauma, divorce, death of spouse) stressors, combined with cultural beliefs regarding the positive benefits of consumption and acceptability of substance use as a coping tactic, it is to be expected that a subset of consumers will develop at least two problems and thus qualify for SUD diagnosis. In effect, the presence of a disorder (i.e., the state of affectedness) in this population is directly related to the rather arbitrary diagnostic criteria making up the DSM-IV-TR.

EPGENETIC MODEL OF SUD ETIOLOGY

The discussion up to this juncture has focused on demonstrating that either deficient acquisition or stress-induced diminution of psychological self-regulation is the cardinal phenotype predisposing to substance use leading to SUD. Risk for SUD, however, is not fixed; rather, it changes as a result of interactions with the social environment during the lifespan. Accordingly, the trajectory from childhood to SUD diagnosis is rarely, if ever, linear. Figure 14.1 depicts a template, adapted from Vanyukov, Tarter, et al. (2003), for delineating the etiology of SUD within a developmental framework. At the top of the figure, the outset point of the ontogenetic trajectory, the magnitude of risk for SUD is the result of gene–environment and phenotype–environment interactions. As can be seen, risk is normally distributed in the population commensurate with the central limit theorem.

The multifactorial model has for many years guided research into SUD etiology. The key premise of this model is that many genes interacting with multiple facets of the social and physical environment determine individual SUD risk. Commensurate with the multifactorial model, the individual and contextual variables, denoted by \( v_1 \) – \( v_N \), in aggregate determine risk for SUD. These manifold factors vary according to magnitude and direction of influence on overall SUD risk. For example, an individual can have high impulsivity (risk enhancing) along with high intelligence (risk attenuating). Moreover, magnitude of expression is not the same on the variables associated with increased or

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![Figure 14.1](image-url)
decreased risk. Accordingly, a wide spectrum of severity for each trait is associated with SUD risk. Consequently, overall risk for SUD encompasses a configuration of factors varying in severity that is unique to each individual. Furthermore, each variable or psychological characteristic is a vector (v), that is, a quantity that has both magnitude and direction biasing the person's future development toward or away from SUD. The combination of all vectors, taking into account their severity and valence, is the resultant vector (R), that is, the quantum designating overall risk for SUD at a given time point. Joining each resultant vector (R) across time graphs the epigenetic trajectory culminating in either SUD+ or SUD- outcome. As shown in Figure 14.1, the hypothetical epigenetic trajectory results in SUD.

It is thus readily apparent why it is difficult, if even possible, to accurately predict whether a child or adolescent will succumb to SUD. Because overall risk (R) fluctuates throughout the lifespan concomitant with the changing quality of interactions with the environment, the direction of the trajectory accordingly shifts direction and changes slope. Moving to a neighborhood in which drugs are easily available, forming a friendship with a drug-using peer, attending a party at which drugs are freely accessible, and depression after the death of a spouse are examples of events that can sharply deflect the slope of the trajectory toward SUD. Converting to a religion that negatively sanctions use of any psychoactive substance, regular urine drug screening, and a committed intimate relationship with a substance-abstaining partner can likewise deflect the trajectory to a non-SUD outcome.

Whereas prediction of SUD may not be feasible, monitoring risk level is nonetheless important so that timely intervention can be implemented once the trajectory begins to orient toward SUD outcome.

Practical Application of the Epigenetic Model of SUD Etiology

Prevention of SUD will remain ineffective unless the capacity to identify youths who are at high risk for SUD can be improved. Two approaches, each having serious shortcomings, are currently used to designate individual risk. The most common method of estimating whether a person is at elevated risk for SUD involves determining whether a biological parent has SUD. The rationale underlying this approach is based on findings from family studies indicating that children of parents with SUD have, on average, a four- to sevenfold increased risk for developing SUD (Vanyukov & Tarter, 2000). This approach, however, does not enable estimating risk status at the individual level. As a group, at least half of the children of parents with SUD do not develop SUD; conversely, many youths who develop SUD do not have a parent with SUD. Thus, although it is heuristic in research on SUD etiology to compare offspring of parents with and without SUD to detect the group-level characteristics associated with risk, parental SUD status is not informative for determining whether risk is elevated in a particular child.

Another method of estimating risk for SUD uses scores on psychological scales that measure characteristics known to presage SUD or co-occur with substance abuse. Dozens of psychological traits reflecting different aspects of self-regulation (sensation seeking, poor attention control, hyperactivity, negative affect, etc.) have been shown to be related to substance use. Using either standardized or experimental measures to estimate risk for SUD, however, has serious limitations because of their poor to mediocre discriminative and predictive utility. Currently, no scale captures the range of factors encompassing cognitive, emotional, and behavioral domains of psychological functioning that are associated with SUD risk. Because most scales were not designed to specifically quantify the components of risk for SUD, it is to be expected that many items constituting a particular scale are not specifically related to SUD risk. Hence, using scores on these scales leads to suboptimal accuracy.

Researchers at the Center for Education and Drug Abuse Research at the University of Pittsburgh have addressed the need to develop instrumentation specifically for measuring individual risk for SUD. Working with research findings showing that genetic and phenotypic risk is largely congenerous to developing all categories of SUD, Vanyukov and colleagues described the rationale (Vanyukov, Tarter, et al., 2003) and method (Vanyukov, Kirisci, et al., 2003) of deriving a scale that is useful for
quantifying individual liability in a developmentally appropriate fashion. A key focus of this effort involved detecting the elemental characteristics specifying risk for SUD. As we discuss next, these elemental characteristics (i.e., nonreducible variables) are indicators of two dimensions of SUD risk: transmissible and nontransmissible.

Transmissible risk. Transmissible risk denotes the portion of phenotypic variance associated with SUD that has intergenerational continuity. Using item response theory methods, Vanyukov et al. (2009) developed the transmissible liability index (TLI) using a multistage procedure. First, items were selected from a large database containing responses to many psychological questionnaires and psychiatric interviews. These items were provisionally assigned into constructs (e.g., aggression, depression, coping skills) that have been implicated in the empirical literature to influence risk for SUD. Exploratory factor analysis was performed to identify the best items in each construct, after which confirmatory factor analysis was conducted to confirm unidimensionality of the trait. Next, each construct was tested to ascertain whether it discriminated youths according to the presence or absence of SUD in biological fathers. Constructs that did not discriminate youths in these two groups were eliminated from further analysis. The remaining constructs were then subjected to another round of exploratory factor analysis and confirmatory factor analysis to derive a second-order latent factor. Last, item response theory methods were applied to determine the threshold and discrimination parameters of each variable. The resulting TLI, with an internal consistency exceeding .90, has been shown to predict SUD between late childhood and young adulthood (Vanyukov et al., 2009). To date, the TLI has been developed for children (ages 10–12), adolescents (ages 12–14 and 16), and young adults (ages 19 and 22). As expected on the basis of the discussion up to this point, the items constituting the TLI align with the general construct of psychological dysregulation. Examples of these items include “hot temper,” “talking excessively,” “stealing,” “assault,” and “trouble concentrating.” In addition, several TLI items measure general well-being, mood quality, and feelings of self-control. Hence, paralleling the measurement of other complex psychological processes such as intelligence, in which a variety of different cognitive processes are aggregated into an IQ, the variety of psychological processes associated with intergenerational risk for SUD make up a unidimensional trait, the TLI.

The results of a twin study revealed that as much as 85% of variance on TLI is due to heritability, thereby documenting construct validity (Vanyukov et al., 2009). In addition, a version of the TLI derived from the variables measured in the National Epidemiological Survey of Alcohol and Related Conditions showed that TLI is a significant predictor of all categories of SUD (Ridenour, Tarter, Kirisci, & Vanyukov, 2011). The childhood TLI scores of 22-year-old men who developed cannabis use disorder have also been shown to be higher than the scores of men who did not develop this outcome (Kirisci, Tarter, Reynolds, & Vanyukov, in press). More interesting, however, was the observation that, among the men who developed cannabis use disorder, transmissible liability continued to increase after their first exposure to cannabis until they qualified for a diagnosis of cannabis use disorder. In contrast, the magnitude of transmissible risk remained unchanged throughout child and adolescent development among cannabis-using boys who did not develop cannabis use disorder. To date, the factors underlying the increase in transmissible risk after initial exposure have not been explained. It may be that substances having abuse potential are especially reinforcing for youths at high transmissible risk. Substance use might also amplify the psychological characteristics making up transmissible risk, which in turn promotes social interactions leading to substance use and SUD. Other possible reasons for the increase in transmissible risk after initial exposure among youths who develop SUD cannot be discounted; however, the key point is that this aspect of the diathesis for SUD is not static. Rather, transmissible risk is changeable concomitant to maturation and experience.

Nonnormative socialization mediates the relation between TLI score in childhood and SUD manifested by young adulthood (Tarter et al., 2011). In this recent study, TLI scores of 10- to 12-year-old boys
negatively correlated with level of cooperative behavior in the home, which in turn predicted affiliation with deviant peers during adolescence, leading to cannabis use disorder by young adulthood. TLI scores were also observed to be higher in children with low parental involvement. Furthermore, severity of transmissible risk in children correlated with severity of SUD in parents. In another investigation of the same cohort, TLI score was shown to be a predictor of number of different types of violent offense committed up to age 22 (Reynolds, Tarter, Kirisci, & Clark, 2011). This association was mediated by rate and acceleration of cannabis use, but not alcohol use, during adolescence. Last, a prospective investigation demonstrated that TLI is moderately accurate at identifying college freshmen who transition to SUD during the next 4 years (Arria, Vincent, & Caldeira, 2009). Taken together, the available findings indicate that TLI captures the psychological characteristics associated with transmissible risk for all categories of SUD. Moreover, TLI is informative for elucidating the etiological trajectory to SUD. On the basis of these findings, Kirisci, Tarter, Reynolds, et al. (in press) developed computer-adaptive test versions of the TLI for practical use on the Web platform to assist detection of high-risk youths who require preventive intervention.

Nontransmissible risk. The second dimension contributing to overall risk for SUD encompasses characteristics that do not have intergenerational continuity. Research devoted to developing an instrument to measure this component of SUD risk has yielded a scale termed the nontransmissible index (NTI) that has discriminative and predictive validity (Kirisci et al., 2009). Notably, none of the items in the NTI correlate with the TLI. Thus, the TLI and NTI ideally encompass all of the variance associated with risk for SUD. Items making up the NTI include measures of environmental adversity, quality of relationship between parents and siblings, and availability of parental and social support. Figure 14.2, depicting TLI and NTI scores in a sample of boys, illustrates that there is marked variability with respect to severity on these two components of risk.

Deconstructing overall risk for SUD into transmissible and nontransmissible components informs both the modality and intensity of prevention that

![Image](image-url)
are appropriate for the individual. Moreover, the Cartesian matrix, shown in Figure 14.2, is a useful framework for measuring the magnitude of change resulting from intervention. Ideally, intervention should lead to a repositioning of the individual that is closer to the vertex, reflecting reduction of risk to at least the mean level of the general population.

It is interesting to note that transmissible and nontransmissible risks contribute equally to developing SUD. T. Ridenour (personal communication, 2010) found that among 22-year-old men who qualified for cannabis use disorder, 19% had below-average transmissible risk and above-average nontransmissible risk at ages 10 to 12. Among youths having the opposite pattern, 20% qualified for cannabis use disorder. As expected, youths who scored below average and above average on both dimensions of risk for SUD had the lowest (12%) and highest (34%) rates of cannabis use disorder a decade or more later. These findings also underline the point that high risk does not inevitably portend SUD. Many intervening and unforeseen events between childhood and adulthood can diminish risk. Similarly, the observation that 12% of youths who score below the mean on both dimensions of risk for SUD had the lowest (12%) and highest (34%) rates of cannabis use disorder by age 22 illustrates that unforeseen events can likewise shift the etiological trajectory to this adverse outcome.

In summary, the TLI and NTI quantify the two components making up the risk for SUD. These summary indices, however, obscure the role of each component in the psychological processes and environmental factors of the developmental trajectory to SUD. Not only can the salience of each component change during development, but the interplay among the components likely affects SUD risk in complex and currently unknown ways. Understanding the changing salience of each variable and its interaction with all other variables is essential for designing effective SUD preventions (Ridenour et al., 2009). As can be seen in Figure 14.3, the severity of scores on three variables integral to SUD risk have sharply different curves spanning the period between childhood and mid-adolescence. The fact that the multifaceted components of SUD do not develop at the same rate, and do not progress in the same fashion, illustrates the importance of profiling risk status using developmentally sensitive measurements. Hence, at any particular age, intervention intensity (“dose”) is aligned to type and severity of disturbance for each trait.

Heterogeneity in the population with respect to developmental patterning poses another complicated challenge. Figure 14.4 depicts developmental change in severity score on a trait, sensation seeking, that is widely acknowledged to be associated with risk for adolescent substance abuse and SUD. Whereas mean score of the whole sample increases linearly during ontogeny, there is enormous individual variability with respect to pattern of scores on this trait over time. As can be seen, the sample mean completely obscures inter- and intraindividual variability. Accordingly, prevention tactics directed at a
group (e.g., a class in school) may not be relevant, or at least equally relevant, for all individuals. It is thus important to monitor risk status at the individual level so that interventions can be appropriately initiated when a designated threshold of severity on particular SUD risk-enhancing characteristics is surpassed (Molenaar & Campbell, 2009).

UNITARY NATURE OF THE CLINICAL SYNDROME

At the outset of this chapter, we pointed out that a serious barrier for etiology research pertains to the lack of consensus regarding the nature of the clinical disorder. The clinical phenotype is variously conceptualized in terms of behavior excess (e.g., dipsomania or narcomania), neurobiological adaptation (addiction), or adverse biopsychosocial consequences of consumption (DSM–IV–TR criteria of substance use disorder). Within each conceptual system, however, the various disorders are manifestations of one syndrome. The marked differences between alcohol and other drugs with respect to molecular structure, biochemical effects, and metabolic mechanisms notwithstanding, the SUDs in the DSM–IV–TR make up a unidimensional trait (Kirisci, Tarter, Vanyukov, Martin, et al., 2006) and can be ordered on a scale according to severity of underlying externalizing behavior (Krueger et al., 2002). In effect, the disorders consequent to consumption of particular compounds are not discrete conditions but rather variants of one syndrome. Evidence accrued from diverse lines of etiology research reviewed herein has accordingly documented the presence of a common liability. The core feature of the liability for all SUDs is deficient psychological self-regulation, consisting of behavioral undercontrol, poor modulation of emotion, and suboptimal executive cognitive capacities. Emerging findings have also suggested that genetically influenced morphological, biochemical, and physiological disturbance of frontal cortex and its connections to subcortical nuclei underlie the low psychological regulation predisposing individuals to all categories of SUD.
Genetic, biological, and psychological characteristics unique to only one category of SUD have not been documented. Although not ruling out the possibility that etiological factors specific to a particular SUD category may yet be discovered, the available evidence at this juncture indicates that the factors contributing to etiology are congenerous to all SUD categories. In the event that SUD category-specific etiological factors are identified, it is of utmost importance to ascertain the amount of variance they account for beyond the contribution of common liability.

Last, it is important to emphasize that a strong liability does not inevitably portend SUD outcome. An environment that contains substances that are readily available and affordable is necessary for the liability to ultimately manifest in SUD. Accordingly, socialization, as the outcome of the interaction between the individual and the social environment, is the primary factor biasing the person toward initiation of or desistance from use of alcohol, tobacco, and other drugs. As discussed in this chapter, psychological dysregulation beginning early in life via interaction with multiple facets of the environment during development determine whether youths engage in illegal behavior. Poor psychological self-regulation is more likely to be present among children exposed to adverse home and neighborhood environments. Moreover, low psychological self-regulation is the ubiquitous feature of youths who are at high transmissible risk for SUD. Thus, as the result of both genetic and contextual influences, opportunity for normative socialization is impeded among youths who are at high risk for SUD. Poor self-regulation biases development toward non-normative socialization, thereby predisposing youths to early-age onset of substance use and eventually SUD diagnosis. Effective prevention of SUD thus requires interventions that consolidate psychological self-regulation during ontogeny in conjunction with inculcating adherence to societal mores and laws.

**SUMMARY**

SUD is an outcome of suboptimal acquisition of psychological self-regulation during childhood and adolescence or results from diminution of self-regulation concomitant to endogenous or exogenous stressors in adulthood. Although the empirical literature is still relatively sparse, the early-age onset variant of SUD appears to have a neurogenetic basis linked to structural and functional disturbance of frontal cortex and subcortical connections. The manifest psychological dysregulation during ontogeny hampers normative socialization, resulting in disruptive and conflictual interpersonal interactions accompanied by weak adherence to societal norms and laws. One manifestation is substance use initiation at a young age, which in turn results in an increase in the range and severity of problems so as to qualify the person for diagnosis of substance use disorder.

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PART III

EXPRESSIONS OF ADDICTION
In this chapter, we provide an overview of the characteristics of the addiction syndrome that are shared by all, or at least by most, of the many different expressions of addiction. These expressions range from substance misuse (e.g., alcohol) to excessive behavior patterns (e.g., pathological gambling [PG]). In the second part of this chapter, we describe the shared consequences of the addiction syndrome.

Some observers might regard our effort to distinguish between the characteristics and consequences of addiction as strange or at least unnecessary. However, this differentiation is not only advantageous but also indispensable: Years ago, in two seminal papers, Edwards (1986; Edwards & Gross, 1976) not only laid down the foundation for the modern understanding of addiction as a syndrome of disorders, he also argued for a strict separation of "primary symptoms" (of the syndrome) and "secondary damage" (Edwards & Gross, 1976). The patterns and severity of addiction-related consequences are highly individual and also strongly related to environmental conditions (e.g., the availability of medical and social services, social support or stigmatization, and economic resources). For example, many people who are alcohol or heroin dependent or who have PG often lose their place in the workforce; however, this type of consequence differs in terms of probability of and time to occurrence, as well as impact on life among individuals with the same expression of addiction and for those with different expressions of the syndrome. Hepatitis or HIV infection is a frequent consequence among those who are sexually active and injecting heroin, but the occurrence and severity of these diseases are influenced by environmental conditions such as health education; availability of opioid maintenance treatment, condoms, and clean syringes; and the quality and availability of treatment options for infectious diseases. Many factors, including genetic vulnerability and quality of medical help, moderate the expression, severity, and course of direct health consequences (e.g., intoxication for alcohol or apnea for opioids) and the long-term consequences (e.g., increased risk of cancer) of addiction. The severity of the addiction syndrome and the related consequences do correlate, but many individual and environmental factors influence this correlation. It would be misleading to determine the severity of the addiction syndrome by its consequences and even useless to treat only the consequences without tackling the syndrome itself. Unfortunately the current diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision [DSM-IV-TR]; American Psychiatric Association, 2000) and International Classification of Diseases—10 (World Health Organization, 1992) handle characteristics, such as tolerance or withdrawal, and consequences, such as progressive neglect of social interests or work activities, as interchangeable features of the addiction syndrome (by merely counting the number of applicable criteria without differentiation). Consequently, we consider these two aspects separately.

We should also note that it is difficult to make an exact separation of the addiction syndrome and its
consequences. For example, impaired cognitive control and related neurobiological correlates in the prefrontal cortex are a candidate symptom of the addiction syndrome, but these features might also occur as a consequence of addiction. In terms of temporal sequences, it is difficult in general to differentiate between symptom development and onset of negative consequences (e.g., reduced work attendance). Given our actual knowledge of the addiction syndrome, we have to concede that we do not yet know the exact pattern and degree of symptom severity to determine the onset of the addiction syndrome. We do not even know whether we have to deal with one pattern of the addiction syndrome or with several alternative patterns. We are not even sure whether we observe a continuum of severity or a dichotomous structure. A further challenge for separating characteristics and consequences is the recursive development and progression of addiction: Core consequences, such as loss of work or social contacts, might function as a trigger for continued heavy substance use or gambling; in turn, these patterns can lead to the development of other expressions of addiction and related characteristics.

CHARACTERISTICS OF THE ADDICTION SYNDROME

We can describe signs and symptoms of the addiction syndrome on many different levels—for example, as gene variations (polymorphisms); protein or neuronal impairments; brain circuit dysfunctions; or planning, learning, and decision-making impairments—or as disorders of attitudes, thoughts, feelings, and motor behavior (Lesch, 2007). Our current knowledge does not allow a preference for one core aspect over other aspects of the addiction syndrome. Therefore, when compiling universal characteristics of addiction, we take into consideration as many levels as possible.

The DSM–IV–TR and International Classification of Diseases—10 separate substance use disorders (SUDs) from other—possibly nosologically similar—excessive behavior patterns (e.g., PG, which is currently classified as an impulse control disorder). Discussions about the integration or separation of these disorders remain controversial (Bühringer, 2004; Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2006; Grüsser-Sinopoli, Böning, Watzl, & Rist, 2008; Petry, 2006; Potenza, 2006; Shaffer, Hall, & Bilt, 1999). The working group for the American Psychiatric Association's fifth edition of the Diagnostic and Statistical Manual of Mental Disorders, the DSM–5, has suggested integrating SUDs and nonsubstance patterns of excessive behavior (currently only PG) into one new category. For the purpose of this chapter, we assume as a working hypothesis a common core syndrome and analyze the universal characteristics and consequences of the syndrome according to the present state of scientific knowledge. Many authors have contributed to the syndrome model of addiction in recent years, and our following proposal is based on the work of Bühringer, Wittchen, Gotzlebe, Kufeld, and Goschke (2008); Redish, Jensen, and Johnson (2008); Robinson and Berridge (2003); Shaffer et al. (2004); van Holst, van den Brink, Veltman, and Goudriaan (2010); Volkow, Fowler, and Wang (2003); Volkow, Fowler, Wang, Baler, and Telang (2009); and West (2006).

Genetic and Neurochemical Characteristics

Recent reviews have indicated that, among people with addiction, there are abnormal receptor, neurotransmitter, and other regulating activities related to transmitter and drug metabolism (J. E. Grant, Brewer, & Potenza, 2006; Ibañez, Blanco, Perez, Fernandez-Piqueras, & Saiz-Ruiz, 2003; Potenza, 2008). There may be a genetic and neurochemical vulnerability to developing an addiction (e.g., a better metabolism of ethanol leading to heavy drinking and a higher risk of developing an alcohol use disorder). However, psychotropic substances and permanent changes in homeostasis might influence gene transcription, the protein products, and neurochemical mechanisms (see the Neuroadaptation section later in this chapter). Additionally, some genes are associated with interindividual characteristics that are strongly linked to addiction (e.g., impulsivity, dysphoria, stress responsivity; Kreek, Nielsen, Butelman, & LaForge, 2005). Finally, there is the question of specificity of these genetic and neurochemical findings for each expression of addiction.
Specific genetic characteristics are often linked to substance metabolism or receptors mediating the specific effect of a substance in the brain transmitter system. Regarding drug metabolism, research reports have shown that alleles for the gene encoding the enzyme alcohol dehydrogenases (ADI2 and ALDH2) are related to alcohol dependence (Vanyukov & Tarter, 2000). Specific genes in cerebral neurotransmission thought to be involved in alcohol dependence are the GABA Type A receptor genes GABRB2 and GABRA2 (Volkow & Li, 2004). Similarly, variants in GABBR2, which encodes the GABA Type B receptor, have been associated with nicotine dependence (Li & Burmeister, 2009). Specific for PG and other behavioral expressions of addiction are noradrenergic system abnormalities that have been hypothesized to be linked to arousal and excitement. Studies focusing on norepinephrine and noradrenaline have indicated noradrenergic dysfunctions and higher levels of norepinephrine during gambling (Goudriaan et al., 2004; Potenza, 2008). On the genetic level, an association has been found between low functional monoamine oxidase (MAO) inhibitors (MAO-A) gene promoter and PG in men (Ibáñez et al., 2003).

In reviewing common features of different SUDs and PG, the evidence has indicated that overlapping genetic characteristics are linked with abnormal activity in receptors, neurotransmitters, and other regulating elements related to dopamine (DA), serotonin, noradrenergic, and opioid metabolism; investigators have hypothesized that these neurotransmitters and neurobiological processes are involved in addiction, for example, as part of brain reward systems or mood regulation (J. E. Grant et al., 2006; Ibáñez et al., 2003; Potenza, 2008). The reactivity of the dopaminergic system influences reward-seeking behavior and can therefore influence addiction (see also Dysfunctional Learning Processes section). Associations of addiction have been reported to polymorphisms at DA receptor genes (DRD1–DRD4) for nicotine, alcohol, cocaine, heroin, methamphetamine, and PG (J. E. Grant et al., 2006; Ibáñez et al., 2003; Kreek et al., 2005; Volkow & Li, 2004). Studies of the serotonin transporter gene (5-HTTLPR) have yielded associations of the less functional short allele with nicotine, alcohol, cocaine, heroin, and methamphetamine use or abuse as well as with PG and excessive Internet use (Ibáñez et al., 2003; Koob, 2003; Lee et al., 2008; Li & Burmeister, 2009). Studies of MAO-B activity, considered as a possible peripheral marker of 5-HT function, have found decreased levels in individuals with both PG and SUDs (J. E. Grant et al., 2006). Endogenous opioids mediate reward and pleasurable effects of substances and behaviors alike; genetic factors influence these opioid functions. Individuals with a genetic predisposition to alcohol use disorders demonstrate enhanced β-endorphin release and euphoria after alcohol administration. Opioidergic involvement in both behavioral and substance addictions has been further substantiated by clinical studies demonstrating the efficacy of the opioid antagonists naltrexone and nalmefene in the treatment of impulse control disorders and SUDs (J. E. Grant et al., 2006; Potenza, 2008). Catechol-O-methyltransferase (COMT) is involved with the inactivation of the catecholamine neurotransmitters (DA, epinephrine, and norepinephrine). The high-activity Val variant of the Val158Met polymorphism of the COMT gene leading to higher enzymatic activity is related to polysubstance use, alcohol, and heroin dependence (Kreek et al., 2005).

A critical objection to common genetic characteristics is the observation that they are also characteristics of other mental disorders and personality traits. For example, COMT activity is also related to stress reactivity; COMT, MAO, and DA-related polymorphisms are also related to impulsivity and risk taking, and serotonin-linked polymorphisms are related to mood disorders (Kreek et al., 2005). To summarize, there is evidence of common genetic and neurochemical characteristics of different expressions of addiction (Buckland, 2008; Shaffer et al., 2004), but they are not specific for particular expressions of addiction. Nevertheless, evidence exists for specific genetic and neurochemical characteristics of different objects of addiction that primarily relate to substance-specific metabolism.

**Neuroadaptation**

There is solid evidence that regular substance use and other types of rewarding behavior are changing homeostasis as well as homeostatic set-points.
(equaling change of allostasis) on a cellular, neurotransmitter, and brain circuit level (Redish et al., 2003; Volkow & Li, 2004). These changes reflect the process known as neuroadaptation. The neuroadaptive changes in brain areas responsible for reward and learning, inhibitory control, and decision making as well as motivation lead to a shift from pleasant and voluntary use of psychoactive substances and activities to a compulsive use that avoids unpleasant withdrawal symptoms triggered by developing tolerance (Robinson & Berridge, 2003; Volkow et al., 2009).

Tolerance. At the cellular level, chronic substance use has been reported to change transcription factors and protein synthesis in reward-associated brain regions regulated by DA; substance use also changes regions innervated by glutamate, GABA, opiates, and serotonin. These consequential alterations include, for example, a reduction of D2 receptor density among chronic cocaine users, a higher density of cholinergic receptors among chronic nicotine users, changes in function and expression of GABA-A and N-methyl-D-aspartate receptors among chronic alcohol users, or changes in opioid receptor expression and opioid release through repeated alcohol, cocaine, and opiate use (Redish et al., 2003; Volkow et al., 2003). Changes at the cellular level contribute to alterations at the neurotransmitter level and thus in the specific brain circuits. For example, reduction in DAD2 receptor density is associated with abnormal brain activity in the orbitofrontal cortex and the anterior cingulate cortex (Volkow & Fowler, 2000). Both regions are related to cognitive control functions such as attention control, inhibitory control, or error and conflict monitoring (see also the Dysfunctional Cognitive Processes and Decision Making and the Impaired Motivational Processes sections). Adaptation also seems to occur in mesolimbic reward circuits, including the nucleus accumbens, and in the nigrostriatal circuit, including the dorsal striatum. Adaptation in the reward circuits might be a possible cause of enhanced saliency of addiction-related cues, whereas adaptation in the dorsal striatum might result in an automatic habit such as addiction-related behavior (Volkow & Li, 2004). Additionally, a more rapid neuroadaptation to psychotropic substances during adolescence might explain the higher risk for the onset of a SUD early in life (Volkow & Li, 2004).

It is evident that for SUDs, tolerance through neuroadaptation results in higher levels of use (i.e., dosages) to obtain the same subjective effects as were achieved previously at lower dosages. However, for behavioral expressions of addiction, it is difficult to argue that the mere increase in frequency and duration of a behavior is a sign of neuroadaptation and tolerance. Charlton’s (2002) results revealed that playing on a computer with increasing “dosage” might be not solely a characteristic of addiction but also an attribute of higher engagement and pleasure, which are milder criteria more relevant at the onset of excessive computer use. Whether neuroadaptation proceeds for rewarding nonsubstance behaviors in the same way as for psychotropic substance use is a question that requires additional study.

Withdrawal. Deviation from homeostasis and allostasis and the resulting tolerance leads to general and substance-specific withdrawal symptoms when stopping or cutting down consumption after long-term use; withdrawal symptoms can be present even after a single use (Redish et al., 2003). In general, a negative affective stage is often followed by further substance use to terminate mental (e.g., dysphoria, anxiety, or hyperactivity) and physical (e.g., pain, hypertension, or fever) symptoms of withdrawal. Mental symptoms of withdrawal also seem to be associated with behavioral expressions of addictions (Cunningham-Williams, Gattis, Dore, Shi, & Spitznagel, 2009; Wray & Dickerson, 1981). However, physical withdrawal effects are not common in behavioral expressions of addiction but are common in SUDs. For example, alcohol and benzodiazepines have very strong withdrawal effects both after acute intake (hangover) and after long-term use. Withdrawal from these substances (i.e., sedatives or hypnotics) can produce physical symptoms that include sweating, hallucinations, or nausea; in addition, life-threatening withdrawal consequences such as delirium and convulsions may occur. Cannabis withdrawal symptoms do not provoke similarly
severe manifestations. Therefore, the existence of a cannabis withdrawal syndrome was, after a long period of discussion, not included in DSM-IV-TR. In DSM-5, cannabis withdrawal will be represented because of the growing body of scientific evidence for it. In addition to psychological symptoms, cannabis withdrawal will include sleep disturbances, stomach pain, shakiness or tremors, sweating, fever, chills, and headache (according to Budney, Hughes, Moore, & Vandrey, 2004).

To summarize current knowledge, the tolerance and withdrawal symptoms associated with neuroadaptation are common for all substances and behavioral expressions of addiction. However, the phenotypes of the syndrome vary between substances and between SUDs and excessive addictive behaviors. Although gamblers are nervous and aggressive during withdrawal, alcohol-dependent people can evidence severe physical reactions, including death; alternatively, cannabis-dependent people often experience mild physical withdrawal symptoms. However, withdrawal per se cannot explain continuous and progressive addictive behavior or relapse (Robinson & Berridge, 2003). Other mechanisms such as learning or motivation have to be taken into account.

Dysfunctional Learning Processes
Imaging studies with humans have revealed that psychoactive substances increase DA concentration in striatal regions, including the nucleus accumbens, which is associated with the rewarding effect of drugs and a heightened salience of associated stimuli (Volkow et al., 2009). The DA release caused by drugs thus mimics the effect coding for saliency and reward of a stimulus (Schultz, Tremblay, & Hollerman, 2000); drug-induced DA-level increases are much higher than when triggered by natural reinforcers (Wise, 2002). The greater the magnitude of DA release and the more abrupt it is, the higher is the likelihood that the user will experience subjective feelings of reinforcement, such as a high or euphoria (Volkow et al., 2003). The strong reward potential of psychoactive substances and their repeated use lead to a special learning environment that can result in addiction memory, which reflects strongly wired neuronal pathways related to drug taking and drug use. Activation of regions linked to memory functions has been reported during drug intoxication and during craving induced by drug exposure, video, or recall (Volkow et al., 2003).

It is critical to mention that rewarding effects have been studied mostly for stimulants and nicotine (Volkow et al., 2009). The rewarding effects of other substances and excessive behaviors such as PG should be examined further. To date, evidence that gambling and other rewarding excessive activities lead to reward system effects that are similar to those produced by psychoactive substances is increasing (Breiter, Aharon, Kahneman, Dale, & Shizgal, 2001). However, it is not clear whether (a) such behaviors have the potential to release DA-level increases that are comparable to psychotropic substances and (b) the involvement of the same reward pathways is necessarily an indicator of a linkage between the two disorder groups (Holden, 2001).

Because of the special rewarding characteristics of drugs, we hypothesize that drugs produce much stronger neurobiological “wiring” associated with reward learning than do natural reinforcers. Classical conditioning (stimulus–stimulus/stimulus–outcome learning) and operant conditioning (action–outcome learning) as well as habit learning (stimulus–response learning) seem to be implicitly or explicitly fostered by dopaminergic mechanisms (Robinson & Berridge, 2003): Via classical conditioning, a neutral stimulus, coupled with a psychoactive drug or behavior, acquires reinforcing properties and motivational salience, even in the absence of the drug. Operant conditioning processes reflect much stronger associations between the drug or behavior and the subjective experience of reward than do natural reinforcements. Thus, drugs and drug use–related behavior are overevaluated in their reward potential, and natural reinforcers become less attractive (Redish et al., 2008).

During the course of permanent regular drug use and ongoing disturbance of learning processes, a transition occurs from planned and explicit guided expectations about the action–outcome relationships (i.e., the memory of drug pleasure) to more automatic behavior consisting primarily of stimulus–response habits (Redish et al., 2008). Researchers
have hypothesized that changed learning processes (e.g., fostered conditioned learning and strong habit learning and the associated functional neuronal changes) play an important role in all SUDs (Everitt, Dickinson, & Robbins, 2001; Redish et al., 2008; Volkow et al., 2003) as well as in behavioral expressions of addiction (Blaszczynski & Nower, 2002; Sharpe, 2002). These changes influence both the maintenance and the relapse features commonly observed among the variety of expressions of addiction. Besides conditioned learning (mediated in part by the nucleus accumbens and amygdala) and habit learning (mediated in part by the caudate and the putamen), declarative memory systems (mediated in part by the hippocampus) are involved in drug addiction (White, 1996). Aside from changes in reward learning toward positive learning and better memory about the substance, people with addiction seem to learn less from negative consequences (Park et al., 2010; Torregrossa, Corlett, & Taylor, 2011), which might be one characteristic of low D2 receptor density (Klein et al., 2007). It might be one explanation of continued drug intake despite adverse consequences of action (e.g., family or health problems). This phenomenon can additionally be the result of dysfunctional error and conflict monitoring, as we discuss in the next section of this chapter.

In summary, dysfunctional learning and disturbed associated brain circuits are core aspects of addiction. But we have to deal with two problems: First, we cannot regard the maintenance of addiction as solely a consequence of aberrant learning per se; we must also consider cognitive, motivational, and emotional aspects. Second, theoretical concepts provide the basis of the learning approach, and whether the proposed mechanisms are really relevant working mechanisms is not clear. In the sections that follow we will examine studies that have demonstrated functional changes in the reward system as well as the motivational system and in which patients have shown cue reactivity as conditioned behavior and automatic habits such as substance intake or behavioral excesses. However, whether structural changes in the brain and the consequent dysfunctional learning processes really account for these behavioral changes is not yet clear.

Dysfunctional Cognitive Processes and Decision Making

Cognitive control impairments. As we mentioned earlier, alteration of neural reward circuits and dysfunctional learning alone cannot account for the complex mechanisms of addiction. Another central aspect of addictive behavior is the person's lack of self-control despite awareness of the adverse consequences (Lubman, Yücel, & Pantelis, 2004). Recent neuroimaging studies and behavioral data underline the important role of control processes during the development, maintenance, and cessation of addiction (Bühringer et al., 2008). For different SUDs as well as PG, research has consistently shown that cognitive control functions (e.g., conflict monitoring, anticipation of long-term consequences) are impaired among people with addiction compared with control subjects (Finn, 2002; Garavan & Stout, 2005; Petry, 2006). Additionally, imaging studies have revealed structural and functional changes in the prefrontal cortex that might reflect underlying core mechanisms associated with functional abnormalities (Garavan & Stout, 2005; Kalivas & Volkow, 2005; Lubman et al., 2004). Next, we summarize the research evidence for impaired impulse control and decision making as well as for error and conflict monitoring.

Impulsivity and dysfunctional decision making. Observers have defined impulsivity as a multidimensional concept: an inability to wait, a tendency to act without forethought to future consequences, and an inability to inhibit automatic behaviors (Bachorowski & Newman, 1985; Barratt & Patton, 1983; Eysenck, 1987). Accordingly, various measures have been developed to assess impulsivity, including personality questionnaires, which rely on individuals' self-perceptions of their behavior, and neurocognitive tasks that provide objective measures of impulsive behavior.

Studies addressing impulsivity have demonstrated that SUDs and problem gambling are associated with increased self-reported impulsivity as well as deficient performance on impulsivity-related neurocognitive tests. For example, clinicians and researchers have used self-report measures such as the Barratt Impulsiveness Scale (Patton, Stanford,
Barratt, 1995) to assess different aspects of impulsive behavior. People with SUDs as well as PG show heightened self-reported impulsivity on different questionnaires compared with their non-drug-using or nongambling counterparts (Moeller & Dougherty, 2002; Reynolds et al., 2007; Whiteside, Lynam, Miller, & Reynolds, 2005). Additionally, longitudinal studies have revealed that this increased impulsivity might not be just a symptom of problem substance use or excessive behavior; it might also reflect an underlying vulnerability (Vitaro, Arseneault, & Tremblay, 1997; Wong et al., 2006).

Because there are various problems with using self-report measures for impulsivity (Verdejo-García, Lawrence, & Clark, 2008), using neurocognitive tasks to assess impulsive behavior is also important. These measures can be classified into three broad types (van Holst et al., 2010): (a) measures of response inhibition such as the stop-signal task, the Stroop task, or go/no-go tasks; (b) measures of delay discounting, which define impulsivity in terms of choosing an immediate small reward over a larger but delayed reward; and (c) measures of cognitive impulsivity using decision-making tasks such as the Iowa Gambling Task (Bechara & Damasio, 2002) or the Cambridge Gambling Task (Rogers, Everitt, et al., 1999; Rogers, Owen, et al., 1999).

To date, studies have revealed heightened impulsivity among people with SUDs and PG using measures for response inhibition (for SUD, see Forman et al., 2004; Mitchell, 2004; Reynolds et al., 2007; for PG, see Kertzman et al., 2006, 2008; MacKillop, Anderson, Castella, Mattisson, & Donovich, 2006), delay discounting (for SUD, see Heil, Johnson, Higgin, & Bickel, 2006; Kirby, Petry, & Bickel, 2004; Odum & Baumann, 2007; for PG, see Dixon, Marley, & Jacobs, 2003; Holt, Green, & Myerson, 2003; MacKillop et al., 2006), and cognitive impulsivity (for SUD, see Bechara & Damasio, 2002; Ersche et al., 2005; Rogers, Everitt, et al., 1999; for PG, see Brand et al., 2005; Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009; Roca et al., 2008). Regarding predictive validity, one study has shown that poor response inhibition predicts problem drinking and illicit drug use among a high-risk population (Nigg et al., 2006). However, more longitudinal studies are needed to clarify these associations.

Neuroimaging studies have revealed frontocortical and frontostriatal dysfunctions as neurobiological bases of decision-making deficits and the inability to inhibit automatic responses (Bechara, Damasio, & Damasio, 2000; Davidson, Jackson, & Kalin, 2000). Until now, various studies have shown that people with addiction have lower activations than normal control subjects within frontal areas for tasks requiring control or planning; in addition, people with addiction show the same deficits as patients with frontal lesions (Bechara & Damasio, 2002; Potenza, Leung, et al., 2003; Rogers, Everitt, et al., 1999). However, using cross-sectional studies, we cannot determine whether these deficits are a symptom of addiction or a vulnerability that increases the likelihood of excessive drug use or other addiction-associated behavior patterns.

To summarize, higher levels of impulsivity, which are possibly based on a less efficient neurobiological impulse control system, are a core characteristic of all SUDs (e.g., research that has focused on alcohol, nicotine, cannabis, opioids, and stimulants) and PG. Some comparable evidence has also been found for other excessive behaviors such as Internet addiction (Cao, Su, Liu, & Gao, 2007), but these studies are too sparse to draw confident conclusions. We want to emphasize that heightened impulsivity is also a characteristic of other mental disorders, such as bipolar disorder (Rogers, Moeller, Swann, & Clark, 2010), impulse control disorders and attention-deficit/hyperactivity disorder, and personality or conduct disorders (Swann, Bjork, Moeller, & Dougherty, 2002). Consequently, scientists have questioned the specificity of this characteristic. Unfortunately, only a few studies have compared different impulsivity facets. Thus, little evidence is available to clarify the shared or unique nature of specific patterns of impulse control impairments associated with each disorder or each expression of addiction. For example, impaired decision making among people who gamble is characterized by chasing losses, whereas other expressions of addiction do not show this type of impairment (Rogers et al., 2010). To date, it is not clear whether dysfunctional cognitive control
processes are a distal vulnerability factor, a characteristic symptom, or a consequence of addiction (de Wit, 2009).

**Impaired monitoring and anterior cingulate cortex dysfunction.** The mobilization of cognitive control processes is strongly associated with error and conflict monitoring for an adequate contextual activation of control resources (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Botvinick, Cohen, & Carter, 2004). It is important to study monitoring networks to understand whether cognitive control functions are impaired or whether control functions are intact but cannot be mobilized adequately because of impaired monitoring processes. Regarding neuronal networks of error and conflict monitoring, there is evidence that the anterior cingulate cortex is an alarm system that is involved with monitoring errors and response conflicts during task performance (Kerns et al., 2004; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Yeung & Sanfey, 2004). Using behavioral tasks with alcohol- and opiate-dependent individuals, recent studies have shown inadequate error and conflict monitoring with reduced neural activity in the anterior cingulate cortex (Forman et al., 2004; Franken, van Strien, Franzek, & van de Wetering, 2007; Hester & Garavan, 2004; for an overview, interested readers should see Garavan & Stout, 2005). However, more research is needed to determine whether inadequate error and conflict monitoring is one characteristic symptom of the addiction syndrome or a vulnerability factor. Also, functional magnetic resonance imaging studies for network and connectivity analyses and the inclusion of subjects with other SUDs and PG are essential.

**Erroneous beliefs.** There is evidence of cognitive distortions among individuals with SUDs and PG. These distortions (i.e., erroneous beliefs) can take several forms. Among the substance use expressions of addiction, erroneous beliefs about the substance's effects and permission-giving beliefs are often observable and might be one cause of urges and relapse (Beck, Wright, Newman, & Liese, 1993). Addiction-related beliefs develop over time through influences of the social environment (social influence model; e.g., for smoking, see Vries, Backbier, Kok, & Dijkstra, 1995) and through the special rewarding characteristics of the drug (e.g., easy administration that leads to immediate negative reinforcement; see also the Dysfunctional Learning Processes section earlier in this chapter). Thus, erroneous beliefs can function as a cause or a consequence of SUDs. People who gamble typically show two types of erroneous beliefs: (a) the belief that they can directly or indirectly influence the outcome of a game (e.g., illusion of control: The gambler knows that certain cues can predict wins and other cues predict losses) and (b) the belief that they can predict the outcome of a chance event correctly (e.g., gambler's fallacy: belief that the chance for a result with fixed probability increases or decreases depending on recent occurrences; Raylu & Oei, 2002). Irrational cognitions can play an important role in the development and maintenance of PG, for example, by chasing or the rewarding effect of near-miss outcomes (Chase & Clark, 2010; L. Clark, Lawrence, Astley-Jones, & Gray, 2009). Whether these erroneous beliefs are a cause or a characteristic symptom of PG is not clear (Delfabbro, Lahn, & Grabosky, 2006; Joukhador, Blaszczynski, & Maccallum, 2004). Moreover, emotional (e.g., hot cognition) and motivational (e.g., trying to chase losses) aspects of gambling are important to include in research targeting erroneous beliefs and PG (Delfabbro et al., 2006). For Internet-associated expressions of addiction, dysfunctional beliefs might also be important to consider (e.g., distinct positive outcome expectancies about Internet use; Grüsser, Thalemann, & Griffiths, 2007).

In sum, erroneous beliefs play an important role during the development and maintenance of addiction. However, the qualities associated with these beliefs seem to be different for substance-related and behavioral expressions of addiction (e.g., beliefs concerning the prediction of gambling outcome vs. beliefs concerning substance-related effects).

**Impaired Motivational Processes**

As we mentioned earlier, regular drug-induced DA release leads to fundamental changes in the reward-associated transmitter systems. Imaging studies have shown a decrease in the amount of DAD2 receptors among drug-dependent subjects compared with
control subjects (Volkow et al., 2003). This decrease is correlated with drug craving (Heinz et al., 2004) and a variety of impaired motivational processes, which are described in the sections that follow.

**Incentive salience.** Together with other changes in the dopaminergic system (Kalivas & Volkow, 2005), this reduction could be a core mechanism associated with the decrease in sensitivity for natural rewards and an enhanced seeking and importance of drug-related stimuli; readers interested in this phenomenon should note that the incentive-sensitization theory of addiction summarizes this relationship (Robinson & Berridge, 1993, 2000). The main idea of this theory is that drugs are more salient than other rewarding stimuli because of their high reward potential; this circumstance provokes a hypersensitivity (sensitization) of the user's neural circuits to drug-related cues (see also Neuroadaptation section earlier in this chapter). Psychologically, the individual perceives this incentive salience as “wanting” to take drugs. This wanting to take the target drug or drugs increases over time, whereas the “liking” of the target drug decreases through the development of tolerance. Some authors have suggested that this might also be a mechanism that influences excessive behaviors triggered through highly rewarding activities (Thalemann, Wölling, & Grüsser, 2007). For example, some studies have shown that even behaviors such as gambling and videogaming lead to DA release in the reward system (Koepp et al., 1998; Zack & Poulos, 2009), which is in turn associated with the subjective excitement related to playing the game (Linnet, Møller, Peterson, Gjedde, & Doudet, 2011). However, whether excessive behaviors can result in an extensive DA burst comparable to the DA release associated with heroin or cocaine use remains unclear (Holden, 2001). Therefore, the question of whether DA release plays the same role in substance-related and activity-related addiction remains to be elucidated.

**Addiction-related cues: Attentional bias, cue reactivity, and craving.** The high salience of addiction-related cues is reflected by an increased attention to addiction-related stimuli and a higher physiological, neurological, and subjective cue reactivity. Some authors have suggested that these attentional and motivational processes are core mechanisms for relapse (Franken, 2003). **Cue reactivity** refers to the enhanced responsiveness to addiction-related signals; researchers measure this reactivity using addiction-related versus nonrelated cues and assess their impact on different neurological, physiological, and subjective reactions. At the subjective level, self-reported craving provides an index of cue reactivity. In a meta-analysis by Carter and Tiffany (1999), subjective cue-induced craving had an overall effect size of .92. The evidence for cue reactivity among people with PG is less available. However, as with substances, the available studies have shown higher urges toward simulated gambling or gambling pictures (Kushner et al., 2008; Sodano & Wulfert, 2009). The effect sizes for physiological responses to substance-related cues were, across all groups, .26 for increased heart rate, .40 for sweat gland activity, and .24 for decreased skin temperature when comparing people with addiction to control subjects (Carter & Tiffany, 1999). Studies also revealed higher physiological reactions after gambling cues (Blanchard, Wulfert, Freidenberg, & Malta, 2000; Sodano & Wulfert, 2009).

Using neuroimaging techniques, cue reactivity showed increased activation of the ventral stream, limbic circuits, and prefrontal cortex among people with SUDs (Grüsser et al., 2004; Heinz et al., 2004; Tapert, Brown, Baratta, & Brown, 2004; Wrase et al., 2002); this activation points to attentional- and craving-related motivational systems. In contrast, only three imaging studies are available for gambling (Crockford, Goodyear, Edwards, Quickfall, & el-Guebaly, 2005; Goudriaan, de Ruiter, van den Brink, Oosterlaan, & Veltman, 2010; Potenza, Steinberg, et al., 2003), and these studies have provided conflicting results. Goudriaan et al. (2010) revealed similar activations for people with PG and SUDs, and the other two studies did not. It is not clear whether this difference is the result of different processing of gambling cues among pathological gamblers compared with their substance-abusing counterparts or of methodological differences between the studies. Regarding excessive Internet use, research has revealed similar brain processing in reaction to computer-related cues as with SUD to substance-related cues (Ko et al., 2009; Thalemann et al., 2007).
SUD and PG are also associated with a heightened attention to disorder-related cues (Cox, Fadardi, & Pothis, 2006; Hester, Dixon, & Garavan, 2006). This biased attention might play an important role in the maintenance of addictive behavior and in the relapse process by turning individuals’ attention toward these cues and by inducing craving (Franken, 2003). Attentional bias has mainly been studied using neurocognitive tasks. Within these tasks, attentional bias has been defined by higher attention to addiction-related stimuli than to nonrelated stimuli. Researchers have operationalized this relationship as an increase or decrease in reaction time, depending on the paradigm. Both people who use substances and people who have PG showed less attention control and higher attentional bias for the tasks regularly used in this field: the addiction-related Stroop task (Boyer & Dickerson, 2003; Cox et al., 2006), the dot-probe task (Bradley, Mogg, Wright, & Field, 2003; Ehrman et al., 2002; Field, Mogg, & Bradley, 2004), and the visual search task (Bonitz & Gordon, 2008; Bradley, Garner, Hudson, & Mogg, 2007).

In sum, the extant evidence permits us to conclude that cue reactivity and attentional bias are associated with various SUDs, PG, and excessive computer use. Neuroimaging studies have shown some cue reactivity differences, but whether these conflicting findings are due to methodological problems is not clear. Although we presented study results separately, we want to stress that cue reactivity, with all its facets, and attentional bias are overlapping concepts. For example, an increased neuronal cue reactivity is related to a pronounced attentional bias and elevated subjective craving (Goudriaan et al., 2010); all of these features are risk factors for relapse (Grüsser et al., 2004, Heinz, Beck, Grüsser, Grace, & Wrase, 2009).

**Impaired attention to non-addiction-related cues.** For subjects with SUD or PG, although attention and reactivity to disorder-related cues is increased, non-drug-related cues seem to be of lower value compared with the time before the onset of excessive drug use or behavioral expression of addiction. For example, studies have revealed lower neuronal cue reactivity to monetary or verbal rewards compared with drug-related rewards (Martin-Söelch et al., 2001; Volkow et al., 2003; Wrase et al., 2007). This relationship has also been found for money as reinforcement among people with PG (Reuter et al., 2005). Observing reduced neuronal cue activity among people with PG is counterintuitive because money should function as a reward for those who evidence this expression of addiction. Nevertheless, this finding could be the result of methodological issues (e.g., the monetary reward was very low in the study) or of other mechanisms in the reward system of behavioral expressions of addiction compared with substance-related expressions of addiction.

**Impaired Emotional Processing**

The effect of substances on emotion regulation and emotional processes seems obvious, but the mechanisms behind this phenomenon are currently not sufficiently understood. Several pathways are possible considering emotional disturbance as the cause, consequence, or symptom of SUDs (Brady & Sinha, 2007). First, substance use can function as an emotion regulation strategy (i.e., self-medication) among individuals with psychological disorders or emotional vulnerabilities or as a dysfunctional coping strategy for everyday stress. Using substances to relieve or manage adverse emotional states can preclude the learning of other adaptive emotion regulation strategies; it can also lead to a higher reward effect of the drug as a short-term, easy-to-administer negative reinforcement. This circumstance complicates life without substances because relapse is highly probable in the absence of alternative coping strategies for managing problems in stressful situations. According to this view, the impaired handling of emotional problems is a possible cause as well as an expression of addiction (e.g., Blaszczynski & Nower, 2002; Markou, Kosten, & Koob, 1998). This view reflects the recursive aspect of the syndrome model of addiction (Shaffer et al., 2004).

Another view considers emotional dysregulation as a consequence of addictive behavior. From this perspective, substance use might lead to structural changes that disturb the neuronal basis of emotion regulation, such as conflict monitoring or verbal skills. Up until now, researchers have studied this
assumption only among those with alcohol dependence. Through neurotransmitter system neuroadaptation, functional brain changes might also affect emotion regulation as a short- or long-term consequence (Koob & Le Moal, 1997; McEwen, 2000). This effect is evident for substance use and might occur with other expressions of addiction. Among the evidence supporting the hypothesized relationship is the high comorbidity of SUDs and PG as well as other behavioral addictions with secondary affective disorders and anxiety disorders. Finally, we can view both the many expressions of addiction and comorbid mood disorders as two sets of indicators reflecting one underlying emotional and cognitive control vulnerability (McCue & Iacono, 2008).

In sum, there are associations of addiction and dysfunctional emotion regulation leading to shifts in mood. These shifts can extend to mood disorders (e.g., depression). However, whether mood disorders are an expression or consequence of addiction is not clear. There is evidence for SUDs and PG being secondary to mood disorders (Kessler et al., 1996, 2008) but also for mood disorders being secondary to addiction (e.g., Chaiton, Cohen, O'Loughlin, & Rehm, 2009, for smoking; see also Mental Health Consequences section later in this chapter). Additionally, impaired emotion regulation could be one vulnerability factor for addiction as well as for mood disorders.

**Distinctive Progression of Severity**

Conventional wisdom and clinicians alike suggest that SUDs or PG progress as a downward spiral, characterized by growing severity of mental, health, and social problems, sometimes until death. However, the stability and progression of substance use and related disorders vary considerably. Even for the early stages, clinicians and scientists have noted that trajectory aspects, such as risk and speed of critical transitions (e.g., from first use to disorder), vary by substance and gender (Behrendt, Wittchen, Höller, Liev, & Beesdo, 2009; Wagner & Anthony, 2002, 2007). Some specific symptoms of the addiction syndrome might typically occur as first symptoms, but so far, the knowledge to permit generalization across substance classes and age groups is limited (Chung, Martin, Armstrong, & Labouvie, 2002; Harford, Grant, Yi, & Chen, 2005; Nocon, Wittchen, Pfister, Zimmermann, & Lieb, 2006; Young et al., 2002). For example, certain first symptoms and a subclinical status might be associated with the risk of progression to a full addiction syndrome (Behrendt et al., 2008; Hasin & Paykin, 1999; McBride & Adamson, 2010), but a subclinical status can also remit or remain stable (McBride & Adamson, 2010).

Also, many scientific studies have revealed that addiction is more variable and that most people with substance addiction and PG (episodically) recover from addiction or at least become more or less symptom free. Natural recovery might occur, for example, because young people will grow up and have familial and working responsibilities or because their problems are less severe than those with a late-life onset (Sobell, Ellingstad, & Sobell, 2000). But many other causes are also conceivable.

Progression, recovery, and relapse patterns differ between expressions of addictions. Regarding alcohol use disorders, Jellinek's (1946) view of alcohol dependence as a progressive disease ending in death or abstinence has been refuted by the literature. Progression is visible among a small proportion of those who are alcohol dependent, mostly in treatment environment studies, but there are also people with alcohol addiction who are abstinent or who have returned to nonproblematic drinking (Bühringer, 2008; Vaillant, 1996). Adolescents who use cannabis regularly have a low remission rate until midlife; the number of experiences using cannabis triggers their progression (Perkonigg et al., 2008). Studies focusing on nicotine have consistently shown that a large majority of smokers stop without any formal assistance (D. B. Clark, 2004). A long-term study on heroin, cocaine, and methamphetamine demonstrated the persistence of use over time for all three drugs, with heroin having the highest use level (Hser, Huang, Brecht, Li, & Evans, 2008). Patterns of use identified were consistently high use (30%), increasing use (14%), decreasing use (14%), moderate use (36%), and low use (6%).

The studies available regarding the natural course of PG are scarce. Results from those studies that are available, however, have revealed no stable course of PG but rather episodic changes from excessive gambling and spontaneous remission to
relapse in previous behavioral patterns (Benschop & Korf, 2009; Shaffer & Hall, 2002; Shaffer & Martin, 2011; Slutske, 2006). In these processes, treated gamblers have more chances to stop the PG behavior than untreated gamblers (Nathan, 2003).

**CONSEQUENCES OF THE ADDICTION SYNDROME**

In this section, we describe common health, mental health, and social consequences of various substance- and activity-related expressions of addiction. We apply the generic term use (as in substance use) to cover the broad range of use behaviors with potentially negative consequences. For some substances (e.g., tobacco), this includes any form of use; for others, it includes patterns of excessive use that may occur in the context of addiction or use in critical situations (e.g., road traffic). By excessive behavior, we imply predominantly addictive behavior such as PG or pathological Internet use, because there is little evidence for negative consequences of nonaddictive patterns of this class of behaviors.

**Physical Health Consequences**

There is little doubt that psychoactive substance use is associated with an elevated risk of significant adverse health consequences (Rehm, Taylor, & Room, 2006). For some substances (e.g., alcohol), this holds especially true for higher amounts (e.g., more than 25 g daily for men or 15 g daily for women) or problematic use patterns and behaviors (e.g., binge drinking, drunk driving).

**Morbidity.** Alcohol use is associated with diseases of the central nervous system, different types of cancer, digestive diseases (e.g., pancreatitis), liver disease, cardiovascular diseases, diabetes, tuberculosis, and a variety of accidents and self-harm (Rehm et al., 2010; Rehm, Taylor, & Room, 2006). Alcohol use can also interfere with prescription medication. In contrast to nicotine, the detrimental effects of alcohol use occur relatively early in life (Rehm, Taylor, & Patra, 2006), and for most diseases associated with alcohol use, there is evidence for a dose-response relationship between level of alcohol use and disease risk (Rehm et al., 2010).

Tobacco use is associated with different types of cancer, cardiovascular disease, and respiratory disease. It is also associated with the indirect health risk of fire (Rehm, Taylor, & Room, 2006). The use of illicit drugs, as a more general category, is associated with infectious diseases (e.g., HIV/AIDS, hepatitis C), unintentional injuries, and self-harm (Rehm, Taylor, & Room, 2006). For heavy and prolonged cannabis use, there is evidence for an increased risk of respiratory problems and traffic accidents (Roffman & Stephens, 2006).

However, with regard to the specific health consequences of substance use, with few exceptions (e.g., Levy & Brink, 2005), the lack of longitudinal research focusing on the risk of adverse health consequences secondary to the onset of SUD is striking. Also, most studies that have examined adverse consequences have addressed the association between substance use—not SUDs—and health consequences, which makes sense given the observed detrimental health effects of substance use but precludes the identification of differences in health effects between users who fulfill SUD diagnostic criteria and those who do not. SUDs and especially substance dependence might have a specific impact on health consequences. Tolerance and withdrawal could contribute to more frequent and more intense consumption associated with greater harm. The changed homoeostasis (Redish et al., 2008) behind these two symptoms could interfere with medical treatment. Loss of control can contribute to binges and risky substance use behaviors that can be more detrimental to health than other forms of use. The prioritization of problem substance use behavior in substance dependence (i.e., the DSM–IV–TR dependence criteria include spending much time, neglect of other activities, continuation of use in spite of a mental or physical problem; American Psychiatric Association, 2000) might have a negative effect on health via a variety of factors (e.g., nutrition, lack of physical activity, lack of attention to the need for medical care, lack of compliance or continued compliance with medical care). Finally, some research support exists for the severity of SUDs affecting health consequences: Evidence from a treatment population has suggested that an increased number of
alcohol dependence symptoms is associated with a greater risk for health problems (Caetano, 1993).

In contrast to substance use, gambling behavior does not directly involve consumption of potentially health-damaging substances. Still, it is possible that PG could lead to adverse health consequences via neglect of, for example, nutrition, need for medical treatment, or physical activity. There is evidence of poorer physical health among people with problem gambling (Griffiths, Wardle, Orford, Sproston, & Erens, 2010). Also, report of poorer mental health, physical health, or both is associated with problem gambling as well as probable PG and its frequency (Mason & Arnold, 2007; Petry & Weinstock, 2007). More important, problem gambling is associated with smoking, higher amounts of alcohol use, and problem alcohol use (Griffiths et al., 2010; Mason & Arnold, 2007). Although the direction of this relationship remains to be elucidated, these behaviors might contribute to the health risks among subjects with problem gambling behavior. Finally, it is important to acknowledge that, other than for alcohol use, little is known about the relation between (low-level) substance use and possible positive health consequences.

To conclude, health risks are common consequences of substance use, but specific consequences differ by substance and use behavior. Evidence exists that health risks differ by the use level associated with various substances; evidence has also shown that the incidence periods of such consequences might vary by expression of addiction. Problem gambling and PG are also associated with poorer health. However, longitudinal data focusing on the specific health consequences that occur secondary to onset of SUDs or PG are distinctly lacking. Thus, it remains difficult to identify with precision the specific and shared health consequences across the different expressions of addiction. Another methodological problem associated with the identification of health consequences of addiction is the challenge of disentangling the often commingled effects of multiple-substance use and SUDs and of co-occurring gambling and substance use behaviors. Because of these entanglements, it is important to remember that the use of different substances can have additive negative health effects.

Mortality. Use of different substances such as heroin, tobacco, alcohol, and nicotine is not only associated with impaired physical health but also with the risk of premature death. In fact, drug use-related mortality accounts for a considerable proportion of mortality among young individuals (European Monitoring Centre for Drugs and Drug Addiction [EMCDDA], 2010; Rehm, Taylor, & Room, 2006; Rostron, 2011). Drug-related mortality can be classified as drug-induced death (i.e., by overdose) and as drug-related death that occurs as a consequence of illnesses contracted within the context of substance use or caused by substance use (e.g., HIV, lung cancer) or of accidents and suicides. Some reasons for drug-related mortality (e.g., contracted illnesses) may play a greater role among chronic and problem users, and others (e.g., accidents and fatal poisoning) may even occur among occasional users. Drug-induced death can occur in the context of both single-drug and polydrug use, and concomitant use of other drugs can play a role in the risk of fatal poisoning and of premature death in general (EMCDDA, 2010; Hart, Smith, Gruer, & Watt, 2010). There is evidence that drug-related mortality rates vary by country, substance, use pattern, gender, and age group (EMCDDA, 2010; Patra et al., 2010; Rehm, Taylor, & Room, 2006; Rehm, Zatonksi, Taylor, & Anderson, 2011; Singleton, Degenhardt, Hall, & Zabransky, 2009; United Nations Office on Drugs and Crime [UNODC], 2010), which indicates yet again that there are many known and unknown factors influencing the probability and severity of addiction-related consequences. For example, heroin and opiates are in general associated with a high risk of substance-induced and substance-related mortality. In dependent heroin users, mortality risk is 6 to 20 times higher than in age- and gender-matched groups from the general population (UNODC, 2010). Overdose of heroin or other opiates is the main cause of drug-induced deaths in Europe (EMCDDA, 2010). Factors that contribute to the risk of both fatal and nonfatal overdoses are the small difference between a tolerable dose and an overdose of opiates, difficulties drug
users have in estimating the drug concentration in substances obtained from street dealers, binge drug use, and previous overdose (EMCDDA, 2010; UNODC, 2010).

There is evidence for an elevated mortality in individuals with cocaine dependence (Degenhardt et al., 2011). Moreover, acute cocaine intoxication can lead to death even in occasional users. The risk of fatal cocaine intoxication can be higher in individuals with preexisting health problems, some of which can be a consequence of cocaine use in long-term and problem consumers (EMCDDA, 2010). Although cannabis is the most common illegal drug used in Western societies, cannabis-related deaths are rare (UNODC, 2010). Also, for another relatively common substance of use, ecstasy, cases of death are infrequent, and the relationship between ecstasy use and mortality might be indirect and partially explained by other factors (EMCDDA, 2010).

Few studies have systematically investigated mortality in users of amphetamines other than ecstasy, and it is still difficult to differentiate between the specific contribution of amphetamine use and the contribution of contextual factors—for example, poor infrastructure of the drug treatment system—to amphetamine-related mortality (Singleton et al., 2009). Still, some evidence exists for elevated mortality among problem and dependent amphetamine users. Mortality may be especially high among subjects who inject amphetamines and those with a longer history of use (Singleton et al., 2009).

Overall, the elevated mortality rates associated with substance use and disorders related to many substances underline the significance of addiction as a serious, health-threatening problem. Although elevated mortality can be found for use of almost all psychoactive substances, this risk and its acuity vary by substance; type and pattern of use; age group; gender; preexisting health problems; and other substance-, person-, and context-related factors.

**Mental Health Consequences**

As we mentioned earlier, SUDs are highly comorbid with other mental disorders, and most of these disorders are not substance induced (B. F. Grant et al., 2004). As for the temporal sequence in comorbidity, evidence has shown that most addiction is preceded by a mental disorder (Kessler et al., 1996), but an important proportion of cases of mental disorder still occur secondary to addiction. However, this proportion varies with the incidence periods of the respective disorders (Behrendt et al., 2011; de Graaf, Bijl, Spijker, Beekman, & Vollebergh, 2003; Kessler et al., 2005). For PG, evidence exists of a range of comorbid mental disorders and that PG is secondary in the vast majority of cases (Kessler et al., 2008). Still, the observation that comorbid disorders also appear secondary to SUDs gives reason to speculate whether these comorbid disorders appear as a consequence of an existing SUD. Some prospective evidence has indicated that prior substance use and SUDs can be risk factors for other mental disorders. For example, regular smoking and nicotine dependence predict panic attacks as well as suicide ideation and suicide attempts (Bronisch, Höller, & Lieb, 2008; Isensee, Wittchen, Stein, Höfler, & Lieb, 2003). Smoking is also associated with an elevated risk of secondary depression (Chatton et al., 2009). Cannabis use is associated with the risk of subsequent psychotic symptoms as well as symptoms of anxiety and depression (Hayatbakhsh et al., 2007; Henquet et al., 2005). Also, for alcohol dependence there is evidence of an elevated risk of secondary depression (Gilman & Abraham, 2001).

Problem gambling is predictive of many mental disorders: posttraumatic stress disorder, anxiety disorder, and bipolar disorder. It is interesting that individuals with gambling disorders also have an elevated risk for secondary alcohol use disorders and nicotine dependence (Kessler et al., 2008). Gambling disorders are also associated with self-report of poorer overall quality of mental and physical health (Mason & Arnold, 2007; Petry & Weinstock, 2007).

Although there is overall evidence that different forms of substance use and SUDs are related to an elevated risk of various secondary mental disorders and mental health outcomes, the restricted number and topical range of available studies does not permit us to conclude whether the consequences are specific to a certain SUD or to a specific level of use. For problem gambling and especially PG, longitudinal
studies with classificatory diagnoses of mental disorders as outcomes are distinctly lacking. The mechanisms behind the observed associations remain widely unclear, and putative confounding variables are not always taken into consideration. For example, the relationship between smoking and suicidality can possibly be explained by comorbid mental disorders (Kessler et al., 2007). As for physical health consequences, SUDs are less frequently investigated as predictor variables for other mental disorders than is substance use, which makes it difficult to identify the specific and shared effects of SUDs as risk factors for secondary mental disorders. The same can be said for gambling behavior and the diagnosis of PG.

The observed relationship between substance use (disorders) and secondary mental disorders might be the result of shared genetic factors or underlying deficiencies in affect regulation and impulse control (McGue & Iacono, 2008). It might also be the case that the sequelae of SUDs and PG (e.g., isolation, financial, occupational, and health problems) leads to an elevated risk of mental disorders.

Social Consequences
Substance use and SUDs are associated with a range of adverse social consequences, for example, impaired social functioning (Hasin, Stinson, Ogburn, & Grant, 2007), high societal costs (Konopka & Konig, 2007), and a risk of SUDs among offspring (Lieb et al., 2002; Merikangas et al., 2009). However, prospective studies focusing on the social consequences of specific SUDs are rare. Research has documented the negative social correlates in the greatest detail for alcohol use, which is associated, for example, with legal problems, unemployment, homicide, and personal relationship problems (Rehm & Gmel, 1999; Rehm, Taylor, & Room, 2006). There is some evidence that individuals with greater severity of alcohol dependence have a higher risk of experiencing adverse social consequences such as family and occupational problems (Caetano, 1993). Adolescent-onset alcohol use disorders are associated with lower educational achievement (Hicks, Iacono, & McGue, 2010), and alcohol use disorders are associated with social stigma (Keyes et al., 2010).

For cannabis, probably the most widely documented negative social consequence is lower educational attainment. However, these results are mainly the result of other influential factors and are not necessarily a specific consequence of cannabis use compared with alcohol or nicotine use (Macleod et al., 2004). A substantial proportion of those with cannabis dependence report social problems (Coffey et al., 2002). There is to our knowledge no scientific report focusing specifically on the social consequences of tobacco use, which might be the result of the legal status of smoking and its cultural acceptance in many countries. It might also be because most nicotine-dependent smokers continue to fulfill role obligations.

Gambling is associated with financial problems, family conflicts, and mental problems experienced by relatives (Wenzel, Oren, & Balken, 2008). As with SUDs, the prioritization of the gambling behavior over other important activities and relationships, acknowledged as a symptom of PG in the DSM-IV-TR (American Psychiatric Association, 2000), might lead to these social consequences. Although detailed prospective information about the social consequences of specific SUDs and PG is lacking, substances apparently differ by their associated social consequences. In general, among all characteristics, the prioritization of the object of addiction might be the most important factor contributing to the adverse social consequences of addiction. One can also assume that the negative mental and health consequences of addiction contribute to their adverse social consequences, for example, by hindering social and occupational participation. However, financial status and social networks can act as both risk and protective factors for the social consequences of addictions.

CONCLUSIONS AND CONSEQUENCES FOR RESEARCH AND PRACTICE
In this chapter, we provided an overview of universal characteristics and consequences of the addiction syndrome. We are faced with two major challenges: first, the separation of these two aspects, and second, the integration of different expressions of the addiction syndrome, especially of excessive
behavioral patterns (e.g., PG), into the present concept of substance dependence.

Is the Distinction Between Characteristics and Consequences of the Addiction Syndrome Possible and Useful?

One might argue that behavioral characteristics, such as impaired reactivity of the DA system, reduced cognitive control, and impaired motivational processes, serve as risk factors, symptoms, and consequences of the addiction syndrome. In addition, the recursive development and progression of the addiction syndrome, in which the first negative consequences might function as triggers for further problems and other expressions of addiction (e.g., comorbid substance dependence), make it impossible to discount this complex pattern according to the characteristics and consequences of the addiction syndrome.

In spite of the present difficulties, a better conceptual and empirical differentiation between the antecedents (i.e., the mediators and moderators of addiction onset that we did not cover in our chapter), the characteristics, and the consequences of addiction would have many advantages. First, a separation of these factors would enhance diagnostic procedures: It would be easier to determine whether a person is addicted or not and to separately determine the type and severity of negative consequences, including a better characterization of nonaddicted but harmful use or excessive behavior. Second, the existence of one or more expressions of addiction determines the probability of successful change and therefore holds the potential to improve treatment outcome prognoses. Third, treatment concepts would be easier to explain to patients (e.g., "We have to tackle the addiction syndrome and handle the negative consequences") and easier to structure, with interventions for the addiction syndrome (e.g., cognitive training to shift from impulsive drug taking to planned decision making) and for the negative consequences (e.g., family therapy to improve impaired social interaction).

Given these potential advantages of the addiction syndrome, what has to be done? Primarily, we need a better conceptual and empirical understanding of the addiction syndrome to separate core characteristics from possible consequences. On the basis of the conceptual and empirical work of many colleagues, we suggest as core features of the syndrome impaired brain reward systems, dysfunctional learning processes, neuroadaptation, cognitive control impairments, and impaired motivational processes. To possibly reduce or modify the core features and to test our assumption about the dichotomous character of the syndrome, further research is needed to test the relevance of our hypothetical characteristics of the addiction syndrome. We also need to obtain better empirical support to determine critical thresholds, the point of no return, for the addiction syndrome.

Consequently, we have to modify diagnostic concepts, criteria, and instruments. For example, one might measure the diagnosis of the addiction syndrome on a dichotomous scale and the related consequences on continuous severity scales, using more than one dimension (e.g., physical illnesses, mental impairments, and social problems). This alternative diagnostic concept would also allow more precise measurements of natural or treatment-related changes and the allocation of interventions for the addiction syndrome as well as for the related consequences.

Is It Feasible and Useful to Integrate Substance Dependence and Excessive Behaviors Not Associated With Drug Dependence Within One Common Addiction Syndrome?

The discussion of this topic overlaps with a current controversy about terminology: Should the term dependence be replaced by addiction (e.g., O'Brien, Volkow, & Li, 2006)? O'Brien et al. (2006) argued that dependence should be seen and handled as a normal consequence of adaptation to drug withdrawal after regular use of psychotropic medication (e.g., among patients with pain medication); alternatively, addiction is characterized by adaptations that result in loss of control over strong urges, leading to compulsive drug-seeking behavior in spite of severe negative consequences. The assumptions of O'Brien et al. are questionable because many patients are not able to stop medication use after termination of their previous disease and show symptoms of compulsive...
drug seeking. From our perspective, we do not see the relevance of this discussion. The definition of the syndrome is paramount. The selection of an appropriate term is secondary. We must also consider the different linguistic and cultural connotations of the terms.

Because of similar symptoms, the inclusion of maladaptive excessive behaviors, such as PG, characterized by loss of control over strong urges to repeatedly exercise that behavior despite negative consequences has some practical advantages for prevention, treatment, and treatment service systems. For example, universal prevention could be based on common risks, and selective and indicative prevention would screen target populations according to common individual addiction-related vulnerabilities and risk factors. Treatment could integrate patients with different expressions of addiction into common treatment programs and centers.

An essential precondition for such an integration is to establish a common set of symptoms that characterize an integrated addiction or dependence syndrome. On the basis of our suggestions at the beginning of this section for a core set of such symptoms, scientists and clinicians need more research to better theoretically and empirically define this syndrome and then to decide which maladaptive excessive behaviors should be integrated.

Nevertheless, a common addiction syndrome risks covering all excessive behaviors without any clinical relevance. As such, a diagnosis of addiction will be associated with not being responsible for the onset and negative consequences of the expression of addiction, but with maintaining responsibility for its successful reduction or termination; under these circumstances, there will likely be social pressure to integrate more and more excessive behaviors under the umbrella of the addiction syndrome. Therefore, we have to strive for a clear list of symptoms that can be checked easily in single cases or for classes of excessive behaviors.

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People use drugs and engage in some behaviors to feel good (e.g., euphoria, novel sensations, social interactions) or to feel better (e.g., to self-medicate, reduce anxiety or pain, induce sedation). Repeated use of certain drugs and participation in some behaviors can result in addiction, which can be defined as compulsive, uncontrollable engagement with drugs or behaviors in the face of adverse consequences. Addiction to drugs, including nicotine, alcohol, prescription opioids, cocaine, and heroin, is among the leading causes of chronic illness and preventable death. Addiction is also among the most prevalent, costly, and deadly of neuropsychiatric disorders (Uhl & Grow, 2004).

The consequences of addiction-related drugs permeate every sector of human existence: brain, body, behavior, family, social networks, community, the economy, education, transportation, the workplace, criminal justice system, nations, and public health. Some consequences are unique to specific drugs; others are shared across drugs and some addiction-related behaviors. Each sector bears a heavy burden of drug consequences. In the acute state, drugs (other than nicotine) can engender cognitive impairment or psychotic reactions, which can result in accidents, trauma, unplanned pregnancy, compromised educational achievement, workplace errors or accidents, inappropriate or violent behavior, overdoses, or death. Research surveys have estimated that more than 22 million people (i.e., nearly 9% of the population over 12 years) harbor a medical diagnosis of alcohol or illicit drug abuse or addiction (according to the Diagnostic and Statistical Manual of Mental Disorders, 4th ed., text revision; DSM-IV-TR; American Psychiatric Association, 2000). More than double this number of people engage in risky, problematic alcohol and other drug use and are therefore at increased risk for addiction (Substance Abuse and Mental Health Services Administration, 2010a). Within health care settings, more than 22% of those presenting for medical reasons screen positive for problematic substance use and are eligible for an intervention (Madras et al., 2009). Although the Drug Enforcement Administration has drawn clear distinctions between illegal (Schedule I) and legal psychoactive drugs (Schedule II and higher), these boundaries are eroding with opioid prescription drug abuse. Opioid prescription drug abuse now ranks second, after marijuana, in number of users and number of new initiates of drug use (Substance Abuse and Mental Health Services Administration, 2010a); second among those dependent on illicit drugs (Substance Abuse and Mental Health Services Administration, 2010b); and first among drug-related deaths, far in excess of heroin- or cocaine-related deaths (Paulozzi, Weisler, & Patkar, 2011). Overdose deaths involving opioid analgesics correlate directly with per capita sales of opioid analgesics. It stands to reason that public health, public safety, and the disease of addiction are major drivers of public policy for drugs. These concerns also drive research on drug mechanisms in the brain and guide discovery of new medications and clinical care in addiction, relapse prevention, withdrawal, and overdose crises. Such research provides the public with information on the long-term consequences of use.
The objective of this chapter is to provide an overview of the neuroadaptive and neurotoxic effects that are unique to the use of addiction-related drugs and apparently not shared with behavioral expressions of addiction. The uniqueness does not reside in generalized neuroadaptive processes because many therapeutic drugs (e.g., antidepressants, antipsychotics, analgesics) engender neuroadaptive changes, yet little reported toxicity. Instead, and in contrast to most therapeutic drugs, drugs associated with addiction produce changes at the molecular level that are associated with maladaptive and dysfunctional behaviors that accrue adverse consequences.

Neurobiological and pathological research findings attributed to drugs require interpretive caution. Research using human subjects is largely limited to brain imaging techniques and analyses of peripheral body fluids or to serendipitous acquisition of post-mortem tissues. Unlike clinical trials to assess novel medications, justifiable limitations prevent research that focuses on the drugs associated with addiction from randomization procedures in drug-naive subjects. Instead, such research relies heavily on self-reported drug use behavior to form comparative groups. This methodological approach raises questions about whether (a) the findings result directly from drug consumption, polysubstance abuse, or adulterants; (b) users accurately report doses, exposure time, and route of administration; and (c) the lifestyle of the user (e.g., nutritional status, infections, sleep patterns, other diseases) contributes to the findings. In animal studies, researchers can carefully control the drug, dose, environmental status, genetic factors, and dosing regimen; however, one drawback is that it is not possible to accurately model myriad factors unique to humans that affect use (e.g., human genetics, environment, physical or psychological abuse, psychiatric comorbidity, depression, or various forms of stress). The final challenge is whether an objective distinction can be drawn between drug-induced neuroadaptive changes and neuropathology either preceding or consequent to drug use.

Notwithstanding these limitations, neuroscience research has revealed ample evidence for the creation of working models of addiction neurobiology. Not surprisingly, drugs engender convergent and divergent effects in the brain. They modulate common neurochemical pathways and neural circuits; however, they differ to the extent that they (a) are associated with addiction and (b) are steered toward unique immediate targets in the brain that conceivably trigger unique adaptive mechanisms in specific neurons. This multifarious array of neurotoxic, neurochemical, neuroadaptive, neural circuitry, and cognitive and behavioral changes has shaped the designation of addiction as a disease.

RISK FACTORS

Researchers have identified numerous pro- and anti-addiction risk factors. Individuals respond to drugs in a unique way, determined by their current and previous environmental experiences, their unique biology (e.g., genetics, personality, psychiatric state), and the interplay of a specific drug with the biology and psychology of the individual (see the Introduction to this handbook). Family, twin, and adoption studies have indicated that genetic and environmental factors contribute equally to the development of addiction. The genetics of individuals influence several dimensions of drug response and even success in treatment. Siblings of abusers are more likely to use drugs, and adopted children with substance abuse histories in their biological families are more likely to abuse drugs, even if their adopted environment is devoid of drugs. Identical twins have a higher propensity to share drug histories than fraternal twins, with a genetic component greater for drugs with higher addiction potential (e.g., heroin). It is now feasible to scan multiple genes at the same time—a million or so single nucleotide polymorphisms in genes simultaneously—to seek genetic differences between nonaddicted and addicted populations and between treatment success or failure. Addiction susceptibility is associated with more than 100 candidate genes, reflecting complex genetic influences converging from polymorphism in multiple genes. Yet susceptibility genes overlap for different drugs (Drgon et al., 2011), even though the starting point of addiction, diverse brain receptor activation, is unique to each drug class. These findings imply that addictive processes converge on common molecular events and neural
networks in the brain. Susceptibility genes encode cell adhesion molecules and genes involved in learning, memory, and cognition, implying that memory and salience function critically in compulsive drug seeking. The genetic approach portends genetically based personalized prevention and treatment approaches in the future (Drgon et al., 2011; Uhl et al., 2010).

Drugs with addictive potential can elicit powerful, unique, and subjective responses, shaped by their chemistry and formulation (e.g., salt, type of salt, additives, free base), dose, dosing regimen, route of administration (i.e., intravenous, inhalation or smoking, insufflation, subcutaneous, oral), rate of transport and metabolism by the intestinal tract and liver, penetration of the blood–brain barrier, access to brain targets, and user response. At equimolar doses, the intravenous route or inhalation results in faster brain entry and a more robust high than the oral route. This principle is common to heroin, cocaine, nicotine, methamphetamine, and other drugs (Samaha & Robinson, 2005). Drugs that directly target brain dopamine, opioid systems, or both (e.g., cocaine, heroin) are associated with a higher prevalence of addiction, on the basis of the number of users who transition from initial use to addiction (Anthony, Warner, & Kessler, 1994; O'Brien & Anthony, 2005; Wagner & Anthony, 2002). The prevalence of addiction among users of cocaine, heroin, amphetamines, or marijuana is higher than for 3,4-methylenedioxymethamphetamine (MDMA, or ecstasy) or inhalants. Higher yet is the percentage of smokers who manifest addiction symptoms—a questionable comparison, because nicotine-containing tobacco products are legal, inexpensive, socially acceptable in certain domains, and widely available. Predicting the relative addiction potential of drugs among individuals is daunting because the convergence of their unique individual history, environment, genetics, epigenetic and psychiatric status, metabolic function, and drug response.

Neurobiology has clarified specific drug-induced events that can compromise brain function. Drugs such as cocaine, methamphetamine, ecstasy, inhalants, and alcohol can be toxic to the brain by destroying neurons (e.g., alcohol and inhalants) or their axons (e.g., amphetamines), disrupting normal blood supply (e.g., cocaine), or altering gross brain morphology (Buttner, 2011; Kaufman et al., 1998). Even without inducing conventional neurotoxic responses, drugs can promulgate adaptive changes in cell structure, metabolism, and brain signals and circuitry, leading to aberrant behavior and impaired cognition. The drug-centered existence can be interrupted by those motivated to sustain abstinence and by medications that attenuate craving (e.g., methadone), interfere with the subjective effects of the drugs (e.g., naltrexone), or engender adverse responses during consumption (e.g., disulfiram [Antabuse]). Nonetheless, drug cravings can surface months or years after withdrawal symptoms have ceased; these urges reflect a subterranean persistence of the drug experience memory and conditioned responses. Drug cues, paraphernalia, familiar drug-associated environments, or stress can trigger cravings during vulnerable periods that result in relapse to use. Relapse prevention strategies target drug cues and craving with cognitive-behavioral therapy and medication-assisted therapy to surmount the biological triggers that lead to relapse.

**DRUG-INDUCED BRAIN CHANGES**

Addiction results from complex biological adaptations in the brain that are reflected by progressive behaviors: initial use; escalation of dose and frequency; transition to loss of control; and compulsive use, withdrawal, craving, and relapse. The biology of addiction begins with unique initial responses elicited by a simple chemical. The initial processes (e.g., activation of receptors or blockade of transporters) trigger a cascade of biological events manifest as self-gratifying sensations. These sensations can eventually imprint as dominant, persuasive memories, leading to changes in behavior, personality, and values. During the transition from drug use to addiction, behavior becomes focused on seeking the drug, even though people with addiction report diminished euphoria compared with interoceptive effects during initiation. Addiction usually results in a decreased response to normal biological stimuli (e.g., food, sex, social interaction, responsible behavior in school or at work).
Ethical limitations in clinical research and imprecise animal models challenge researchers’ ability to unravel the biological substrates that influence the progression from initiation to addiction. Nonetheless, studies of these processes are yielding exciting insights into this vast and largely uncharted route from molecule to mind. Drugs can produce epigenetic modifications (e.g., persistent changes in DNA that alter its capacity to be expressed) and can change the expression of signaling systems secondary to receptors, transcription factors, genes and encoded proteins, cell biology, morphology, synaptic strength, neural circuitry, and behavior (Bahi & Dreyer, 2005; Koob, 2009; LaPlant & Nestler, 2011; Maze & Nestler, 2011; Robinson & Kolb, 2004; Russo et al., 2010; Saka, Goodrich, Harlan, Madras, & Graybiel, 2004). These changes ultimately affect memory, cognition, judgment, executive function, the value of natural rewards, and behavior. Although rudimentary, current models proffer insights crucial for infusing prevention programs with sound scientific discoveries and for providing biochemical leads to develop effective medications and other treatment strategies to alleviate withdrawal symptoms or block the rewarding effects of drugs, prevent relapse, and treat comorbid disorders.

Initiation of drug use is largely an adolescent phenomenon, with at least 60% of new initiates younger than age 18 and an even higher percentage initiating tobacco and alcohol use (National Survey on Drug Use and Health, 2010). Onset of prescription drug abuse is more likely to emerge during the 3rd decade of life, often in association with earlier use of alcohol and other drugs. Onset of drug use before age 18 for marijuana, cocaine, other psycho-stimulants (e.g., amphetamines), hallucinogens, opioids, inhalants, alcohol, smoking, and prescription drugs (stimulants, opioid analgesics, sedatives, tranquilizers, anxiolytics) is associated with higher prevalence of addiction among adults (Anthony & Petronis, 1995; Chen, Storr, & Anthony, 2009; B. F. Grant & Dawson, 1998; McCabe, West, Morales, Cranford, & Boyd, 2007; Palmer et al., 2009; Stone, Storr, & Anthony, 2006; Storr, Westergaard, & Anthony, 2005). At the other end of the spectrum, drug use is rising rapidly among older adults (National Survey on Drug Use and Health, 2010), but whether drug-induced adaptation or compromised cognition and judgment differ for this cohort compared with their youthful drug-using counterparts is not known.

Drugs can modulate the expression of proteins responsible for neurodevelopment (Bahi & Dreyer, 2005; Jassen, Yang, Miller, Calder, & Madras, 2006), conceivably altering the normal trajectory of adolescent brain development and raising addiction vulnerability. A single cell gives rise to 100 billion neurons and an estimated 1 trillion “supporting” glial cells. These brain neurons can extend thousands of connections, in local circuits or in long-distance connections that command every complex action, sensory interpretation, or thought. The risk for the adolescent brain arises when drugs introduced during this phase of neurodevelopment interfere with the trajectory of normal brain development.

Animal Models: Biological Effects of Drugs

Intriguingly, drugs elicit hedonic or psychoactive responses in all mammalian species examined, and even in pigeons and flies (Bergman, Madras, Johnson, & Spealman, 1989; Winsauer & Thompson, 1991; Wolf & Heberlein, 2003). Mammalian species, from mice to monkeys, learn to self-administer the same drugs that humans compulsively seek: marijuana, cocaine, heroin, ecstasy, amphetamine, methamphetamine, PCP, nicotine, and alcohol. Behavioral and neurobiological research has yielded an array of brain changes in animals injected with or self-administering drugs. Critical to understanding these changes is brain communication.

Cell–cell communication is a key to survival, with at least 15% of the human genome devoted to encoding proteins involved in communication. All addictive drugs target the complex communication system of the brain, triggering a prodigious array of molecular and cellular changes. Neurons communicate with their neighboring cells by releasing quantal amounts of neurotransmitters into a synapse, and adjacent neurons bind to the neurotransmitters and decipher the encoded chemical signal, which can be propagated to other brain regions. This
elegant coordinated sequence is terminated rapidly, so as to maintain homeostasis. Specific neurons synthesize specific transmitters that partner with other neurons expressing their corresponding receptors. The transmitter-receptor signaling partners can initiate movement (dopamine), suppress pain (opioids), engender tranquility or fear (serotonin), imprint or erase memories (dopamine, glutamate, acetylcholine, anandamide), produce arousal and pleasurable or unpleasant sensations (dopamine, endorphins, norepinephrine, serotonin, dynorphin), induce paranoia, regulate heart rate (catcholamines) and respiration (opioids), and myriad other functions. Specific signals and circuits alert the brain to natural rewards that are necessary for human survival: food, water, safety, social bonding, and sex.

Drugs resemble but are not identical to the structures of endogenous transmitters. Cocaine, amphetamine, and ecstasy have common core features of dopamine, serotonin, and norepinephrine. Heroin shares some elements of the brain’s opioids (endorphins, enkephalins). LSD resembles the neurotransmitter serotonin. The structure of Δ9-tetrahydrocannabinol produced by the marijuana plant overlaps structurally with the brain’s cannabinoids (anandamide, 2-arachidonylglycerol). The transmitter “imposters” are recognized by receptors and transporters and embed themselves in the communication system, but drugs do not duplicate natural communication with fidelity. Communication is an exquisitely orchestrated series of events designed for brain-produced neurotransmitters. Brain transmitters are produced selectively in specific brain regions by specific neurons, stored in discrete amounts by vesicles, and released in carefully controlled amounts to local neurons. In contrast, drugs are delivered to all brain regions at concentrations that are unregulated. Drugs might activate multiple receptor targets simultaneously, and this activity is not natural. They might generate signals of unusually long duration, trigger abnormal signal transduction, and change the rhythm of normal tonic or phasic signals, which maintain homeostasis or alert the brain to a natural reward. An essential step in normal communication, resetting the system and maintaining homeostasis, is termination of signals. A rapid transport removes and returns the transmitter to its original neuron to be stored in vesicles to cycle once again. Most transporters cannot accommodate the unusual structures, shapes, and charges of psychoactive drugs (e.g., LSD, morphine, cocaine, marijuana), enabling drugs to trigger persistent signals at abnormally high strength for abnormally prolonged periods of time and in inappropriate brain regions.

Unlike alcohol or inhalants, drugs that resemble transmitters produce unique effects, with each binding to a spectrum of receptors normally activated by natural transmitters. LSD produces hallucinations via two serotonin receptor subtypes. Marijuana activates CB1 cannabinoid receptors to magnify sensory perception, distort time, produce relaxation, reduce coordination, and interfere with working memory. By blocking dopamine transporters and raising extracellular levels of dopamine, cocaine indirectly activates dopamine receptor subtypes to produce powerful stimulant and euphoriant sensations. Heroin induces a measure of tranquility combined with euphoria by activating opioid receptors. Alcohol acts on a range of receptors, including excitatory glutamate and inhibitory GABA receptor subtypes, monoamine receptors, and others. All drugs have highly distinctive sensory effects on the brain that contribute to their emotional value. With repeated use, the signaling system of the brain adapts to these abnormal signals. Adaptation is manifested dramatically during withdrawal, with withdrawal apparently involving regional and neurochemical processes distinct from adaptations that contribute to drug-seeking behaviors. Yet as complex, unique, and diverse as these acute and adaptive responses may be, addictive drugs also activate convergent neuronal circuits, circuits that signal changes in homeostasis.

Drugs propagate one common biological response, the release of dopamine in the nucleus accumbens. Dopamine is not a hedonic signal but a signal for learning, for recording the salience of an experience and contributing to motivated behavior. Dopamine can be released in anticipation of, or during, natural rewards (e.g., food) and in response to aversive stimuli, serving as the brain’s “adrenaline” to alert the body of novel, meaningful rewarding or aversive stimuli and of a pending stimulus associated with a hedonic reward. Signals that alert the
brain to natural rewards differ from drug-induced reward signals. Dopamine is released in the nucleus accumbens by drugs at levels and within a time frame grossly exceeding release and clearance generated by natural rewards. An unregulated dopamine signal proliferates outward, alerting other brain regions of a novel, motivating, positive experience and recruiting other transmitter systems, primarily glutamate, along the way (Kalivas, 2009). Glutamate, one of the transmitters implicated in mediating short- and long-term memory, most likely encodes the specific details of the drug experience and stores these details. These complex signals (a) carry the message to specialized circuits and brain regions that interpret and consolidate positive memories and regulate conditioned responding (hippocampus, amygdala), (b) learn cues associated with the experience (hippocampus, amygdala), (c) learn that repetition of the behaviors will reacquire these rewards (dorsal striatum, nucleus accumbens), (d) assign a priority and value for response to these awards (orbital prefrontal cortex), and (e) imprint cognitive control over rewarding behaviors (prefrontal cortex, striatum, thalamus).

Natural rewards and drugs share some common alerting circuits and memory processes, but the natural rewards benefit the individual by rousing survival behaviors (Kalivas & O’Brien, 2008; Koob & Volkow, 2010; Nestler, 2004; Volkow et al., 2010). In contrast, the preternatural drug-induced hedonic signals overwhelm signals of natural rewards, suppress the salience (value) of essential rewards, and eventually supplant the drive for essential, natural, rewarding behavior, drawing the person into a deleterious state. Drug seeking evolves into a compulsion that can persist for months or years after the drug was withdrawn. The transcription factor deltaFosB is one candidate for a molecular switch. Transcription factors turn genes on or off. DeltaFosB accumulates in the nucleus accumbens and dorsal striatum after repeated administration of many kinds of drugs of abuse. After induction, it is stable and persists for prolonged periods, possibly maintaining adaptive changes that persist long after the drug has cleared the body. Accordingly, Nestler, Barrot, and Self (2001) have designated deltaFosB as a sustained molecular switch that functions as a transducer to convert sustained drug exposure into complex adaptive responses (Nestler, 2008; Nestler et al., 2001). Yet deltaFosB might not be the critical or only molecular switch responsible for addictive processes.

Another potential consequence of prolonged drug exposure is interference with the development of new neurons in the adult hippocampus (Eisch & Harburg, 2006). The adult brain produces new neurons in two regions: the subgranular zone of the dentate gyrus of the hippocampus and the subventricular zone of the lateral ventricles. Research has suggested that the hippocampus is critical for learning and memory; newly born neurons in this brain region relate to spatial-navigation learning, long-term spatial memory retention, spatial pattern discrimination, contextual fear conditioning, clearance of hippocampal memory traces, and reorganization of memory to extrahippocampal locales (Ming & Song, 2011). Although controversial, adult hippocampal neurogenesis has also been implicated in certain behavioral responses to antidepressants.

Repeated drug exposure leads to brain adaptation and compensation for abnormal reward signals. Adjustments in numbers of gene copies and proteins lead to profound changes in every dimension of brain biology and function. Neuroadaptation begins at the level of receptors, is amplified in signal transduction cascades within neurons, and leads to changes in transcription factors that regulate gene expression. These changes ultimately modify synaptic strength, the density and morphology of dendrites, the configuration of neural circuitry, and behavior.

Do these changes represent a molecular switch that drives compulsive drug seeking and loss of control? To fulfill this role, a switch would persist for months or years after the drug was withdrawn. The transcription factor deltaFosB is one candidate for a molecular switch. Transcription factors turn genes on or off. DeltaFosB accumulates in the nucleus accumbens and dorsal striatum after repeated administration of many kinds of drugs of abuse. After induction, it is stable and persists for prolonged periods, possibly maintaining adaptive changes that persist long after the drug has cleared the body. Accordingly, Nestler, Barrot, and Self (2001) have designated deltaFosB as a sustained molecular switch that functions as a transducer to convert sustained drug exposure into complex adaptive responses (Nestler, 2008; Nestler et al., 2001). Yet deltaFosB might not be the critical or only molecular switch responsible for addictive processes.

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Repeated use of opioids, psychostimulants, nicotine, and alcohol decrease neurogenesis in rodents; this circumstance raises the possibility that drugs might compromise learning and memory ability by interfering with neurogenesis. Drug-induced brain changes documented by preclinical studies have provided important leads for clarifying human drug response, but these observations and models can have practical applications and validity if replicated in humans.

Brain Changes in Human Drug Users: Research in the Postmortem Brain

With human subjects, the challenges of investigating the molecular and cellular changes readily obtained in drug-exposed animals are daunting. Systematic assessments of drug consequences in the postmortem human brain are not available, but there is sufficient evidence indicating that drug effects are profound. At the cellular level, axonal damage, neuronal loss, microglial activation, depletion of astrocytes, and vasculopathy have been reported (Büttner, 2011). The brains of opioid abusers display depletion of neurons in the hippocampus and other brain regions, along with ischemic lesions, which might be attributed to recurring hypoxia during opioid intoxication. Although opioid receptors remain stable after prolonged use, other receptors, enzymes, and signal transduction proteins are altered in postmortem tissue. Cocaine acts by blocking monoamine transporters (sympathomimetic) and elevating brain levels of dopamine, norepinephrine, and serotonin. It is the most frequent drug of abuse associated with fatal or nonfatal vascular ischemia or hemorrhage in various brain regions, attributable to vasospasm, cardiac arrhythmia with secondary cerebral ischemia, or enhanced platelet aggregation (Büttner, 2011). The brains of opioid abusers display depletion of neurons in the hippocampus and other brain regions, along with ischemic lesions, which might be attributed to recurring hypoxia during opioid intoxication. Although opioid receptors remain stable after prolonged use, other receptors, enzymes, and signal transduction proteins are altered in postmortem tissue. Cocaine acts by blocking monoamine transporters (sympathomimetic) and elevating brain levels of dopamine, norepinephrine, and serotonin. It is the most frequent drug of abuse associated with fatal or nonfatal vascular ischemia or hemorrhage in various brain regions, attributable to vasospasm, cardiac arrhythmia with secondary cerebral ischemia, or enhanced platelet aggregation (Büttner, 2011).

Cocaine has robust effects on components of neurotransmission. Signaling systems that engender positive sensations—enkephalin mRNA—μ opioid receptor binding and dopamine D1 and D2–3 dopamine receptors—are reduced, possibly resulting in dysphoria. In contrast, the signaling systems implicated in dysphoric or unpleasant sensations—dynorphin mRNA and kappa opioid receptor binding—are increased. This imbalance and dysregulated glutamate signaling in the amygdala might account for the profound dysphoria long-term users report on cessation of cocaine use (Hurd & Herkenham, 1993; Okvist et al., 2011).

Amphetamine and methamphetamine also target monoamine transporters and the trace amine receptor (Bunzow et al., 2001; Miller et al., 2005; Verrico, Miller, & Madras, 2007). Not surprisingly, these drugs can rapidly elevate blood pressure and cause strokes in young people. Both drugs are neurotoxic to human brain dopamine systems, reducing dopamine levels and markers for dopamine and serotonin with unknown reversibility (Büttner, 2011; Quinton & Yamamoto, 2006; Yamamoto, Moszczynska, & Gudelsky, 2010). Similarly, MDMA, a derivative of amphetamine, is neurotoxic to human serotonin systems, as detected by a loss of markers for serotonin axons and serotonin in the postmortem brain of frequent MDMA users and with brain imaging techniques.

Brain Changes in Human Drug Users: Imaging Research of Living Brain

At a different level of analysis, adaptive and toxic changes have been visualized in living brains of human drug users, either during drug use or during abstinence. Neuroimaging procedures ranging from positron emission tomography to single-photon-emission computed tomography can reveal abnormal receptors, transporters, neurotransmitters, other relevant proteins, blood flow, and oxygen and glucose use. Magnetic resonance imaging (MRI), functional MRI, and proton magnetic resonance spectroscopy are powerful imaging tools that can reveal changes in gross brain anatomy, brain activity (by detecting changes in arterial blood hemoglobin oxygenation during a cognitive task), and levels of specific chemicals in the brain. Diffusion tensor imaging MRI monitors microstructural changes, and computed tomography scans can gauge changes in brain size.

Functional brain imaging has identified brain circuitry that might account for recalibration of the urge to seek natural rewards and substitute drug-induced rewards. Increased metabolic activity in the anterior cingulate and orbitofrontal cortex, areas of the prefrontal cortex, become overactive in people addicted to cocaine (Bonson et al., 2002; S. Grant et al., 1996; Koob & Volkow, 2010; London,
The heightened activity is thought to reflect greater motivation to seek a drug instead of a natural reward and a decreased ability to overcome this urge. Subjects addicted to cocaine also show decreased activity, compared with those who are not addicted, in the prefrontal cortex when presented with stimuli associated with natural rewards. One brain region, the insula, has recently been designated the “hidden island of addiction,” because it may play a crucial role in the conscious urge to take drugs (Naqvi & Bechara, 2009; Naqvi, Rudrauf, Damasio, & Bechara, 2007). This brain region is uniquely activated in human subjects during urges to use cocaine or heroin, or alcohol or cigarettes. Intriguingly, addicted smokers with lesioned or destroyed insula (but not populations with other damaged brain regions) readily quit smoking without relapse and lose the urge to smoke. This brain region is viewed as a key location for integrating stimuli arising from the body, arousing conscious awareness of these feelings, attributing a value to them, and integrating a response to them (Naqvi & Bechara, 2009; Naqvi et al., 2007). Accordingly, signals from the insula may override the prefrontal cortex, subverting its function as a reasoning center for impulse control. Conversely, the impaired insula might permit the prefrontal cortex to reassume impulse control, executive function, and judgment. It is conceivable that medications that interrupt insula function may dampen the conscious urge to seek and consume drugs during the abstinence phase.

Clarifying addiction-related altered neural circuitry that localizes cue-induced craving or suppression of natural rewards is a critical pursuit; however, other effects of drugs, beyond addiction states, also require scrutiny. For example, heavy marijuana, cocaine, ecstasy, amphetamine, and heroin users acutely display, after repeated exposure or during abstinence, deficits in attention, memory, and executive function as a function of cumulative dose and period of exposure. Imaging techniques have revealed widespread changes in the brains of drug users; these changes include brain atrophy, decreased gray- and white-matter volume, ischemic lesions, depressed glucose metabolism, reduced receptors and transporters, altered neural circuitry, and others (Barrós-Loscertales et al., 2011; Cosgrove, 2010; Lane et al., 2010; Licata & Renshaw, 2010; Tomasi et al., 2010).

The relevance of these brain abnormalities to impaired cognition or behavior is unsettled. Marijuana induces deficits in attention, memory, and executive function in the acute phase and in working memory and attention after the acute phase (i.e., 24 hours–28 days). Researchers have detected persistent deficits with prolonged marijuana use, even after the drug clears. Lengthy exposure to marijuana is associated with subnormal blood flow in various brain regions, lower cerebellum metabolism, and reduced activity in the prefrontal cortex and cerebellum. Heavy use is associated with gross anatomical abnormalities: reduced volumes of hippocampus and amygdala and brain regions with high densities of cannabinoid receptors, with left hemisphere hippocampal volume inversely associated with cumulative exposure to the drug (Lundqvist, 2010). Evidence is accumulating that the adolescent brain is particularly vulnerable to the effects of marijuana: Earlier age of onset of use and cumulative dose are related to lower activity in the prefrontal region, the attention network, and medial cerebellum compared with nonusers. These aberrant responses might portend neuroadaptation or changes in the trajectory of normal brain development in youthful heavy marijuana users. Heavy cocaine use is associated with impaired cognitive function, as a function of cumulative dose. Functional MRI studies in people who abuse cocaine have revealed abnormalities in thalamo-cortical responses that conceivably contribute to impaired sensory processing, inattention, and executive function. Chronic methamphetamine or amphetamine users display alterations in frontal, temporal, and subcortical brain metabolism and metabolites and reduced density of dopamine neurons in dopamine-rich brain regions. Although dopamine neurons apparently recover after a lengthy period of abstinence from methamphetamine, residual effects persist because neuropsychological test scores do not recover in parallel (Volkow, Chang, Wang, Fowler, Franceschi, et al., 2001; Volkow, Chang, Wang, Fowler, Leonido-Yee, et al., 2001; Wang et al., 2004).
CONCLUSION

Current research is at an early phase in developing a comprehensive view of how the brain assigns value for competing rewards; how it stores reward-related experiences and cues to motivate behavior and supplement natural rewards; how age, environment, and other factors heighten susceptibility for addiction; whether a biological switch to addictive behavior is quantal or gradual; and whether biochemical adaptive, structural, or functional changes viewed in living brain or postmortem tissue are linked to cognitive and behavioral impairment and whether these changes are reversible. The limitations of imaging research are dissolving with the emergence of creative new technologies to monitor changes with increasing resolution, even at the molecular level. As an example of exciting progress, regulation of gene expression is pivotal to addiction-related processes or their reversal, stress response, drug-induced pathology, psychiatric comorbidities, and neuronal repair, yet until recently researchers have not been able to monitor gene expression or surrogates for genes in the living brain. During 2011, a method for noninvasive MRI imaging of gene transcription profiles in living rodent brain was reported (Liu & Liu, 2011). This approach will eventually permit analyses of critical molecular living parameters within the human brain that are currently accessible only in postmortem animal tissues.

Progress in clarifying the neurobiology and pathology of addiction has accelerated rapidly with new imaging, molecular, and cell biology techniques. Evidence for drug-induced neuroadaptation at the molecular, cellular, and anatomical levels is robust in the animal brain, but the relevance of some findings to a human disease of addiction is indeterminate because corresponding studies with the human brain are meager. All drugs with addiction potential, including alcohol, nicotine, marijuana, opioids, cocaine, amphetamines, inhalants, and MDMA, cause profound adaptive and possibly toxic changes in the human brain. Abnormalities of brain anatomy, biochemistry, metabolism, or function detected by brain imaging are increasingly amalgamated with manifestations of addiction and cognitive and other functional impairments to support the designation of addiction as a disease state. Neurotoxic consequences of long-term exposure to drugs in the living or postmortem human brain reinforce the disease model, notwithstanding that a single initial dose of inhalants, cocaine, amphetamines, or opioids can also be toxic.

It is intriguing that current (DSM-IV-TR) criteria for addiction minimize symptoms of biological adaptation, namely drug tolerance and withdrawal, highlighted by older definitions. With alcohol and heroin use as paradigms, biological hallmarks of addiction were assumed to be manifest by diminished pharmacological effects of a fixed drug dose (i.e., tolerance) and physical withdrawal. In DSM-IV-TR, these traditional criteria were retained but diluted by the preponderance of other criteria emphasizing loss of behavioral control and adverse consequences. Although accumulating evidence of brain changes is robust, no consensus exists about valid biological markers for the disease of addiction. The behavioral terminology and criteria prevail. Nonetheless, imaging and postmortem findings are increasingly revealing whether drug-induced changes are reversible, suggesting new targets for medication development, determining the association between adaptive or pathological changes and cognitive or behavioral impairment, and assessing the effectiveness of treatment and clinical care to reverse addiction-related behavior and pathological findings. Finally, positioning addiction as a biological disease also offers a quantifiable framework to accelerate treatment research, reduce stigmatization by professionals, improve medications assistance, and medicalize this public health challenge.

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Researchers, treatment providers, and others have expressed increasing interest in the concept of behavioral addiction as a way to classify behaviors that mirror the symptoms and consequences of classic alcohol- and drug-related expressions of addiction (Shaffer et al., 2004). Many clinicians and researchers have embraced the term because it offers a way to reconceptualize the relationships among many neglected disorders and their treatment options. The concept of behavioral addiction includes disorders that the National Institute on Drug Abuse considers relatively pure models of addiction because the presence of an exogenous substance does not contaminate their processes (Holden, 2001).

The concept of a group of behavioral disorders related to substance addiction remains of great theoretical interest to many people. Relatedness is a fundamental idea underlying any classification system. The question of relatedness to substance addiction is of singular interest at present because of ongoing discussion regarding the creation of a category for behavioral addictions within the general class of addictions and related disorders in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), scheduled for release in 2013 (http://www.dsm5.org). Thus far, pathological gambling (PG) is the only proposed member of the category, but other disorders could be added, such as kleptomania, compulsive buying (CB), or Internet addiction. The current proposal is to keep some of these disorders where they are now—for example, maintaining kleptomania in the impulse control disorder category—and to continue to exclude others (CB, Internet addiction). Apart from the confines of the proposed behavioral addiction category in DSM-5, addiction’s boundaries as discussed by researchers and others are relatively fluid, expanding or contracting depending on the individual’s own particular views. Some experts have chosen to include several of the impulse control disorders (PG, kleptomania, pyromania) currently listed in the fourth edition, text revision, of the DSM (DSM-IV-TR; American Psychiatric Association, 2000) as members of the behavioral addiction category, but they have also included several disorders not currently recognized in the DSM system (CB, Internet addiction, compulsive sexual behavior [CSB]; Grant, Brewer, & Potenza, 2006; Holden, 2001).

Although there is some controversy regarding the optimal categorization of behaviorally expressed addiction, growing evidence has supported the linkage between these disorders and substance addiction. This evidence strengthens the rationale to include both behavioral and substance addictions in the same general class. Furthermore, recognizing behavioral addiction could contribute to improved classification, enabling a more accurate description of endophenotype and biological markers that characterize these conditions. More precise classification could lead to specific treatments.

Evidence in support of a relationship among proposed behavioral addictions to the substance addictions is accumulating and includes phenomenological, genetic, and neurobiological data (Grant,
Blach, Kuzma, and Shaw

Brewer, et al., 2006; Potenza, 2006). An alternative classification system that has received wide attention has placed the behavioral addictions in a class related to obsessive–compulsive disorder. Hollander (1993a, 1993b) and others (Koran, 1999) have long promoted the concept of an obsessive–compulsive spectrum, although supporting evidence has been limited (Dell'Osso, Altamura, Allen, Marazitti, & Hollander, 2006; Tavares & Gentil, 2007).

The proposed behavioral (or process) addictions and substance addictions have many commonalities. They share common core clinical features; for example, both involve the performance of repetitive or compulsive behaviors despite negative consequences, diminished control over the behaviors, craving before engaging in the behavior (Grant, Brewer, et al., 2006). They appear to share features of tolerance, withdrawal, repeated attempts to cut back, and impairment in multiple life domains (Blanco, Moreyra, Nunes, Siz-Ruiz, & Ibanez, 2001; Grant, Brewer, & Potenza, 2006; Grant & Potenza, 2005). Phenomenological data have also suggested a relationship between the behavioral and substance addictions. These addictions often begin in the late teens or early 20s, and although several of the behavioral addictions, such as CB and kleptomania, are more common in women (Black, 2007; M. J. Goldman, 1991), other behaviorally expressed addictions appear to have a male preponderance (PG, CSB) similar to that seen in substance addictions (Black et al., 1998). Neuroimaging studies have suggested that similar brain circuits are activated in both PG and substance addictions (Potenza et al., 2003). Finally, researchers have reported that people with several behavioral addictions benefit from cognitive–behavioral therapy (CBT; PG, CB) or 12-step programs (PG, CSB), similar to improvement seen in the substance addictions (Black, 2007; Briken et al., 2010; Grant & Odlaug, 2010). Research has suggested that the opioid antagonists help dampen the drive to engage in addictive behavior in several of the behavioral addictions. Naltrexone, for example, which has an indication from the U.S. Food and Drug Administration to treat alcohol dependence, reduces the drive to engage in addictive behavior in both PG and kleptomania (Grant, Kim, & Odlaug, 2009; Kim, Grant, Adson, & Shin, 2001).

With this background in mind, in this chapter we focus on PG, CB, kleptomania, CSB, and Internet addiction. We have selected these behaviorally expressed conditions because scientists and others writing about behavioral addictions have described common elements that link them with substance addiction. In the following sections, we describe the disorders in turn as well as both their common and unique distinguishing features.

**PATHOLOGICAL GAMBLING**

Ms. A, a 42-year-old accountant, had gambled recreationally for years. At age 38, for reasons she could not explain, she became hooked on casino slot machines. Her interest in gambling gradually escalated, and within a year Ms. A was gambling during most business days. She also gambled most weekends, telling her husband she was at work. To acquire money for gambling, Ms. A created a fake company to which she transferred nearly $300,000 from her accounting firm. Authorities eventually detected the embezzlement and arrested her.
Following her arrest, and the associated public humiliation, she became severely depressed and attempted suicide by drug overdose. After a brief hospital stay, she entered counseling and her doctor prescribed the serotonin reuptake inhibitor (SSRI) antidepressant paroxetine to treat the depression. In a plea bargain related to her legal proceedings, she agreed to perform 400 hours of community service. (Argo & Black, 2004, p. 39)

Clinicians and laypeople have frequently observed similarities between PG and substance addiction, and in fact, the term addiction has long been used to describe PG. The existence of Gamblers Anonymous, a 12-step program patterned after Alcoholics Anonymous, also contributes to the common perception that PG is related to classic alcohol and drug addiction, lacking only a substance of abuse. These observations were made long before scientists were able to demonstrate shared neurobiology and genetics, findings that now are driving discussions about the appropriate categorization of PG.

Regardless of discussions on classification, people have increasingly recognized PG as a public health concern that is costly and potentially disabling (Grinols, 2004). A commission formed by President Bill Clinton during the late 1990s focused attention on PG and documented its negative impact on individuals and society (National Opinion Research Center at the University of Chicago [NORC], 1999). One consequence was that in 1998, the National Institutes of Health actively began to seek and fund research grants related to gambling (National Institutes of Health, 1998). On the basis of the findings of the presidential commission and other published work, it became clear that PG substantially impairs quality of life, is associated with comorbid psychiatric disorders, and is linked to many suicides (Argo & Black, 2004; Black, Moyer, & Schlosser, 2003; Crockford & el-Guebaly, 1998; Petry & Kiluk, 2002). Other documented problems include financial distress, child and spousal abuse, and divorce and separation (Shaw, Forbush, Schlinder, Rosenman, & Black, 2007).

Yet recognition of PG as a mental health disorder came relatively late. Only in 1980, with the publication of the third edition of the DSM (DSM-III) by the American Psychiatric Association, were operational diagnostic criteria first enumerated. Committee members patterned the criteria after those for substance dependencies, reflecting their views that PG and substance addictions were related, and the committee emphasized features of tolerance and withdrawal. Minor changes were made in the criteria for subsequent editions of the DSM. The criteria in the present version, DSM–IV–TR (American Psychiatric Association, 2000, p. 671), define PG as “persistent and recurrent maladaptive gambling behavior (criterion A) that disrupts personal, family, or vocational pursuits,” in the absence of mania (criterion B). To reconcile nomenclature and measurement methods, Shaffer and Hall (1996) developed a generic multilevel classification scheme that gambling researchers have widely embraced. The draft criteria for DSM–5 remain unchanged from DSM–IV–TR except for the omission of one item regarding committing illegal acts to finance gambling because of its relatively poor specificity (http://www.dsm5.org). However, as noted previously, a major change includes the proposal to relocate PG from impulse control disorders to a new addiction category, which includes substance-related addiction. In response to concerns with the pejorative connotations of the word pathological, some have further proposed that PG be renamed disordered gambling.

PG and other forms of problematic gambling are surprisingly common. Estimates of lifetime prevalence for PG vary from 0.6% to 3.4% in the general population (Kessler et al., 2008; Volberg, 1996). Problem or at-risk gambling, considered a lesser form of problematic gambling, might be more frequent, with lifetime prevalence estimates ranging from 3.5% to 5.1% in the general population (Volberg, 1996). Researchers have linked prevalence to gambling availability (Jacques, Ladouceur, & Gerland, 2000). For example, the presence of a casino within 50 miles has been associated with a nearly twofold increase in PG prevalence (NORC, 1999). These and other studies have suggested that the development of PG is strongly related to the ease
with which people can access gambling, not unlike substance addictions whereby locales that restrict or prohibit alcohol sales, such as predominantly Muslim countries, have low rates of alcoholism (Özgür Ilhan, Yildiram, Demirbas, & Dogan, 2008).

As with the substance addictions, gambling behavior typically begins in adolescence, with PG developing by the late 20s or early 30s, although it can begin at any age including senescence, and it affects more men than women (Argo & Black, 2004). PG has a later onset in women yet progresses more rapidly than among men, similar to alcohol disorders. This phenomenon has been referred to as telescoping because it truncates, or shortens, the time from onset of gambling behavior to the development of problematic gambling (Tavares et al., 2001). This same phenomenon has been described in people with an alcohol addiction, providing yet another link between PG and substance addictions. Populations that are at risk for PG include adults with mental health or substance use disorders, people who have been incarcerated, African Americans, and people with low socioeconomic status (Potenza, Kosten, & Rounsaville, 2001). Family and twin data have suggested that PG is familial and has a heritable component shared with the substance addictions (Black et al., 2006; Slutske et al., 2000).

The most widely discussed clinical distinction among gamblers is that between escape seekers and sensation seekers (Blaszczynski & McConaghy, 1989). Escape seekers tend to include older people who gamble out of boredom, to alleviate depression, or to fill time. Such individuals tend to choose passive forms of gambling, such as slot machines. Sensation seekers tend to include people who are younger and prefer the excitement of card games or table games that involve active input. Also well known is the pathways model (Blaszczynski & Nower, 2002), which integrates biological, developmental, cognitive, and other determinants of disordered gambling. The model suggests the existence of three subgroups of pathological gamblers: (a) behaviorally conditioned gamblers; (b) emotionally vulnerable gamblers; and (c) antisocial, impulsive gamblers. Behaviorally conditioned gamblers have no specific predisposing psychopathology but make bad judgments regarding gambling. Emotionally vulnerable gamblers have premorbid depression or anxiety and have a history of poor coping. Finally, antisocial, impulsive gamblers are highly disturbed and have features of antisocial personality disorder and impulsivity that suggest neurobiological dysfunction. Although these proposed subtypes are helpful to researchers and clinicians in considering pathological gamblers, data are needed to validate these distinctions.

Community and clinic-based studies have shown that substance addictions, mood disorders, personality disorders, and other behavioral addictions are common in people with PG (Black & Shaw, 2008). For example, in clinical samples, from 25% to 63% of pathological gamblers meet lifetime criteria for a substance use disorder (Black & Shaw, 2008). Personality disorders are relatively common among individuals with PG (Argo & Black, 2004; Black & Moyer, 1998). Researchers and clinicians have singled out antisocial personality disorder as having a close relationship with PG, with rates ranging from 15% to 40% (Black & Shaw, 2008).

The DSM–IV–TR holds that PG is chronic and progressive (American Psychiatric Association, 2000) and that the essential feature of PG is “persistent and recurrent maladaptive gambling behavior . . . that disrupts personal, family, or vocational pursuits” (p. 671). Historically, and perhaps because of the pioneering observations of Custer (1985), PG is widely considered a progressive, multistage illness. Custer described PG as beginning with a winning phase, followed in turn by a losing phase and a desperation phase. The final phase, giving up, represented feelings of hopelessness. These four phases have gained acceptance despite the lack of empirical data. Recent work (LaPlante, Nelson, LaBrie, & Shaffer, 2008; Slutske, Jackson, & Sher, 2003) has challenged the notion that PG is intractable and has suggested that many gamblers spontaneously improve, just as do many people with substance addictions.

It has become clear through functional MRI and other technologies that brain circuitry mediating PG, particularly those areas involved in reward pathways, is similar to those seen in the substance addictions (Goudriaan, Oosterlaan, deBeurs, & van den Brink, 2004; Potenza et al., 2003). The
involvement of reward circuitry has also been strongly suggested by research showing that dopamine agonist medications for Parkinson’s disease (e.g., pramipexol) have led to the development or exacerbation of PG in some people (Lader, 2008). Dopamine is widely considered the neurotransmitter most involved in reward-based neurocircuitry (Grant, Brewer, et al., 2006).

Not unlike people with substance addictions, few people with PG seek treatment (Cunningham, 2005), and until recently the treatment mainstay has been participation in Gamblers Anonymous. Patterned after Alcoholics Anonymous, the group provides a nonjudgmental atmosphere of mutual support. Health care providers have also developed inpatient treatment and rehabilitation programs similar to those for substance addictions (Russo, Taber, McCormack, & Ramirez, 1984; Taber, McCormick, Russo, Adkins, & Ramirez, 1987), but they are not widely available. Influenced by treatment research for the classic alcohol and drug addictions, CBT and motivational interviewing have become established treatment modalities (Petry, 2005). Self-exclusion programs, in which gamblers agree not to enter a casino, appear beneficial for some people (Ladouceur, Sylvain, & Gosselin, 2007) and have gained acceptance. Finally, family and marital (or couples) counseling might be beneficial to those for whom gambling has disrupted family life or marriage (Shaw et al., 2007).

Researchers actively are pursuing medication trials. Thus far, opioid antagonists, such as naltrexone, have achieved the best results. Two randomized controlled trials of opioid antagonists (one naltrexone, the other nalmefene) have shown efficacy in alleviating gambling-related problems, and specifically reducing gambling urges (Kim et al., 2001; Grant, Potenza, et al., 2006). Researchers have also studied antidepressants. Randomized controlled trials of the SSRIs paroxetine and bupropion showed that the drugs were no more effective than a placebo (Black, Arndt, et al., 2007; Black, Shaw, Forbush, & Allen, 2007; Grant et al., 2003), and open-label studies of nefazodone, citalopram, carbamazepine, and escitalopram have been encouraging but need to be followed up with controlled studies (Black, Arndt, et al., 2007; Black, Shaw, & Allen, 2008; Black, Shaw, et al., 2007; Pallanti, Rossi, Sood, & Hollander, 2002; Zimmerman, Breen, & Posternak, 2002).

**COMPULSIVE BUYING**

Ms. B, a 47-year-old divorced woman, had shopped compulsively since she first obtained credit cards at age 19 years. She knew her shopping behavior was excessive, and reported that it had been continuous for nearly its entire duration. She only had controlled her shopping briefly for two short periods, both coinciding with bankruptcy proceedings. Ms. B currently was in debt on four credit cards. Her life revolved around shopping and spending, even though she worked full time. Spending gave her a “rush,” despite the guilt she experienced afterwards. She would either return items or give them away. She enjoyed shopping—mostly alone—at expensive department stores and would spend her money mainly on clothing and shoes. She was unhappy with her inability to control her spending, and was ashamed that her behavior had contributed to her divorce, as well as to her serious financial problems. She described the shopping behavior as relatively spontaneous and impulsive, and generally not planned. Despite her problems, she was not depressed and had never sought psychiatric care. (Black, 2012, p. 196)

CB is another example of a behavioral addiction that is common and problematic yet, compared with PG, has been less well investigated. Like PG, clinicians and laypeople have long recognized its addictive qualities, and perhaps this is why it is widely referred to as a shopping addiction (Black, 2007). In several respects, CB is similar to substance addictions because both involve excessive or poorly controlled behaviors, cravings, and preoccupation (R. Goldman, 2000). Of course, the unique behavioral aspect of CB is the object of the craving or preoccupations: shopping.
Although many people assume that CB is a relatively recent phenomenon that has resulted from our modern consumer-oriented society, clinicians have in fact described it for nearly 100 years. In the early 20th century, Swiss psychiatrist Bleuler (1911/1930) wrote,

As a last category, Kraepelin mentions the buying maniacs (onomaniacs) in whom even buying is compulsive and leads to senseless contraction of debts. . . . The particular element is impulsiveness; they cannot help it, which sometimes even expresses itself in the fact that not withstanding a good school intelligence, the patients are absolutely incapable to think differently and to conceive the senseless consequences of their act, and the possibilities of not doing it. (p. 540)

CB has experienced somewhat of a renaissance since the early 1990s, at which time empirical and clinical interest in the disorder increased. Consumer behaviorists showed CB to be widespread (Faber & O’Guinn, 1992), and three independent research groups published descriptive studies that had remarkably similar findings (Christenson et al., 1994; McElroy, Keck, Harrison, Smith, & Strakowski, 1994; Schlosser, Black, Repertinger, & Freet, 1994). At the same time, McElroy et al. (1994) published operational diagnostic criteria that have since become standard in the research community. They require the presence of cognitive and behavioral aspects of CB and impairment from both subjective distress and interference in social or occupational functioning or from financial or legal problems. The criteria also require that the disorder not co-occur with mania or hypomania. CB does not appear in DSM-IV-TR (American Psychiatric Association, 2000), and there are no current plans to include CB in DSM-5 (http://www.dsm5.org). For classification purposes, clinicians can use the DSM-IV-TR’s category “impulse control disorder not otherwise specified.”

Many people would be surprised to learn that CB is common in the general population and clinical settings. The lifetime prevalence of CB has been estimated to fall between 0.8% and 8.1% in the general adult population. These figures are based on results from a mail survey, in which researchers administered the Compulsive Buying Scale to 292 individuals selected to approximate the demographic makeup of the general population of Illinois (Faber & O’Guinn, 1992). The high and low prevalence estimates reflect different score thresholds set for CB. (The higher figure is based on a probability level of .70 [i.e., 2 standard deviations above the mean], whereas the lower figure is based on a more conservative probability level of .95 [i.e., 3 standard deviations above the mean]). More recently, Koran, Faber, Aboujaoude, Large, and Serpe (2006) used the Compulsive Buying Scale to identify compulsive buyers in a random telephone survey of 2,513 U.S. adults, and they estimated the point prevalence at 5.8% of respondents. Not surprising, a 9.3% estimated lifetime prevalence has been reported in an adult psychiatry inpatient sample (Grant, Levine, Kim, & Potenza, 2005).

Similar to other addictions, CB has an onset during the late teens and early 20s. Conventional wisdom has suggested that onset might correspond to emancipation from the nuclear family as well as with the age at which people first can establish credit (Black, 2007). Although most epidemiological and clinical research has suggested that the disorder has a female preponderance (Black, 2012), the survey reported by Koran et al. (2006) found nearly equal rates in men and women. Some people have suggested that the reported difference between genders might be an artifact and stem from the fact that women more readily acknowledge CB than do men, who are more likely to characterize their excessive shopping behavior as “collecting” (Kuzma & Black, 2006). Another link to the substance addictions is its overlap with co-occurring disorders, particularly substance use, mood, anxiety, personality, eating disorders, and other behavioral addictions such as PG (Black, 2007). The genetic roots of CB are largely unknown, but research has suggested that the disorder is familial and coaggregates with mood and anxiety disorders and with substance addictions (Black et al., 1998; McElroy et al., 1994).

Not unlike the substance addictions, the disorder is thought to be chronic or recurrent for most people with the condition but fluctuating in severity.
and intensity (Black, 2001). Aboujaoude, Gamel, and Koran (2003) found that people with CB who responded to treatment with citalopram were likely to remain in remission during a 1-year follow-up. Their study suggested that treatment can alter the natural history of the disorder so that its course is not always chronic. Assessing the long-term course of CB raises questions about suicide as an ultimate outcome. Data from Lejoyeux, Tassian, Solomon, and Ades (1997) have addressed this issue and found that whereas CB is associated with suicide attempts, it does not appear to lead to completed suicide.

The hallmark of CB is preoccupation with shopping and spending. This characteristic provides a strong link with the other behavioral and substance addictions wherein preoccupation is a defining feature, whether the object of the preoccupation is a substance or a specific behavior. For example, people with CB might spend many hours each week engaged in these shopping and spending behaviors, and many will describe increasing tension or anxiety relieved by a purchase (Christenson et al., 1994; Schlosser et al., 1994). People with CB might try to resist their urges, as will the substance abuser or person with PG, but often with little success (Schlosser et al., 1994).

Compulsive buyers are mainly interested in consumer goods such as clothing, shoes, crafts, jewelry, gifts, makeup, and compact discs or DVDs (Christenson et al., 1994; Schlosser et al., 1994). CB has little to do with intellect or educational level, and research has documented the disorder occurring in people with mental retardation (Otter & Black, 2007). In addition, income has relatively little to do with CB, although finances might determine the shopping venue, for example, shopping at a discount store rather than at a department store (Dittmar, 2007). The impact of the Internet on CB is unknown; however, because shopping is available around the clock, its impact—like that of catalog and television shopping—may be considerable.

Undoubtedly, cultural and social factors contribute to the onset or maintenance of the disorder because CB mainly occurs in developed countries. Related factors include the presence of a market-based economy, the availability of goods, easily obtained credit, and disposable income (Black, 2001). The findings of Neuner, Raab, and Reisch (2005) in Germany illustrate the influence of these factors on the development of CB. After Germany reunified in 1989, the prevalence rate increased, presumably because of the influx of goods into the former East Germany combined with increased income, which fueled consumer spending.

There are no standard treatments for CB, but most recent work has emphasized the use of CBT models, several using a group setting. Mitchell, Burgard, Faber, and Crosby (2006) were able to show that group CBT produced significant improvement compared with a wait list in a 12-week pilot study; participants maintained improvement through a 6-month follow-up. Benson (2006) has developed a comprehensive self-help program that both individuals and groups can use but has not reported on its efficacy.

Early reports suggested the use of antidepressant medication (McElroy et al., 1994), and because some considered CB to fall within an obsessive-compulsive spectrum, treatment studies have tended to use SSRI antidepressants, drugs known to be effective in treating obsessive-compulsive disorder (Koran, 1999). For example, Black, Monahan, and Gabel (1997) reported the results of an open-label trial in which subjects given the SSRI fluvoxamine improved. Yet two subsequent randomized controlled trials found fluvoxamine treatment no better than placebo (Black, Gabel, Hansen, & Schlosser, 2000; Ninan et al., 2000), although people in both treatment cells improved. Koran, Chuang, Bullock, and Smith (2003) reported that subjects with CB improved with open-label citalopram, another SSRI. Later, these researchers randomized subjects who responded to citalopram or placebo. Compulsive shopping symptoms returned in five of eight subjects assigned to placebo but not in subjects who continued taking citalopram (Koran et al., 2003). In an identically designed discontinuation trial, the SSRI escitalopram did not separate from placebo (Koran, Aboujaoude, Solvason, Gamel, & Smith, 2007).

Grant (2003a) and Kim (1998) have described cases in which individuals with CB have improved after taking naltrexone, suggesting that opiate antagonists might have a role in its treatment either through their action on opioid receptors or their...
dopaminergic activity. As with other behavioral addictions, the neurotransmitter dopamine has been theorized to play a role in “reward dependence” (Holden, 2001).

Other recommendations include self-help books, 12-step programs (Debtors Anonymous), and financial counseling (Black, 2007), although researchers have not yet evaluated these approaches. Recommendations to avoid carrying credit cards or shopping alone might help if the person with CB complies with these recommendations (Kuzma & Black, 2006).

KLEPTOMANIA

Ms. C, a 78-year-old widow, had sought care for her recurrent depression. She disclosed a 62-year history of near chronic and compulsive stealing, typically stealing unneeded objects, mainly from discount or department stores (e.g., jewelry, toiletries)—items that she otherwise could afford. She would also steal objects from friends’ homes or even doctors’ offices. She had not told any clinician about the stealing because of the shame and embarrassment it had caused her.

She grew up in a large but poor family in a small Midwestern community and was the youngest of five siblings. She reported having been sexually abused by one of her brothers but said her mother did not believe her and told her never to speak about it. She moved out at age 16 to escape the abuse, running away to a larger city. She eventually returned to her small town, trained to be a nurse, married, and had three children. She later divorced, after many years of emotional and physical abuse by her husband.

Ms. C says that her stealing began after she left home at 16 and was briefly homeless. She shoplifted food and other items she needed to survive on the streets. This gradually led her to begin stealing other items in response to what she described as an overwhelming and uncontrollable drive that was only satisfied by stealing. She often kept the items, but would sometimes give them away, sell them, or return them to the store hoping to receive money for returned merchandise. She was ashamed of her behavior and knew it was wrong.

Although adept at stealing, Ms. C had been apprehended several times over the years, only to suffer the humiliation of having the crime reported in local newspapers. She was generally able to control herself after an arrest and refrain from stealing for a few months, but the control never lasted. She had taken many different antidepressant medications over the years, including SSRIs, and although they helped treat her depression, they had never curbed her stealing behavior. Ms. C declined referral for CBT for the stealing but was willing to take medication for her depressive symptoms.

In 1930, despite the condition having been clinically recognized for nearly 200 years (M. J. Goldman, 1991), Swiss psychiatrist Bleuler (1911/1930) provided one of the first formal descriptions of kleptomania, which he considered an example of an “impulsive insanity.” He wrote,

The kleptomaniacs in the old sense cannot even otherwise resist the impulse of appropriating things, and here again it is done regardless of whether they can make use of the things or not, they hoard them, give them away, destroy them, and under conditions even return them to the owners. (p. 539)

The DSM-IV-TR (American Psychiatric Association, 2000) defines the disorder as the “recurrent failure to resist impulses to steal objects not needed for personal use or for their monetary value” (p. 667). In addition, there is an increasing sense of tension immediately before committing the theft followed by pleasure, gratification, or relief at the time of the theft. More important, individuals do not steal to express anger or vengeance or in response to hallucinations or delusions. Last, clinicians need to rule
out an antisocial personality disorder, conduct disorder, or mania as a cause of the stealing behavior (American Psychiatric Association, 2000). Kleptomania is classified in DSM-IV-TR as a disorder of impulse control, where it likely will remain in DSM-5 (http://www.dsm5.org).

Although kleptomania is presently—and likely will remain—categorized as a disorder of impulse control, several investigators have considered it a behavioral addiction (Grant, Brewer, et al., 2006; Potenza, 2006). As with substance addictions, kleptomania involves repetitive behaviors that lead to negative consequences, a feeling of one's inability to control the behaviors, cravings (or urges) before engaging in the behavior, and experiencing a pleasurable response while engaged in the behavior. Unique to kleptomania is the focus on stealing unneeded objects and the consequences that ensue, for example, the possibility of arrest and incarceration, and the humiliation that results.

The prevalence of kleptomania is unknown, perhaps because individuals rarely report their symptoms. A recent survey of nearly 800 college students reported a 0.4% current prevalence for kleptomania (Odlag & Grant, 2010). The rate might be higher in psychiatric samples. For example, Grant et al. (2005) reported a 9% lifetime prevalence rate for kleptomania among psychiatric inpatients. The shoplifting literature has provided another source of data to make inferences about kleptomania prevalence because these two conditions overlap. A survey of the U.S. adult general population found shoplifting to have a lifetime prevalence of 11% (Blanco et al., 2008).

Kleptomania occurs more often in women than in men (M. J. Goldman, 1991; Grant & Potenza, 2008). However, gender differences might reflect differences in help-seeking behavior by women (e.g., seeking mental health care) or the fact that men are more likely to be apprehended for stealing (M. J. Goldman, 1991). As with other behavioral or process addictions, kleptomania can begin at any stage of life, but it usually starts during the late teens to early 20s (Grant & Potenza, 2008). By the time patients seek treatment, women are typically in their mid- to late 30s and men are in their 50s. Researchers have described the course of the disorder as continuous or episodic and generally chronic and nonremitting (Koran, Bodnik, & Dannon, 2010).

People with kleptomania usually steal items that they could otherwise afford, such as toiletries, makeup, or jewelry. The stealing usually occurs with planning or forethought (McElroy, Pope, Hudson, Keck, & White, 1991). Triggers for the behavior include feelings of depression, anxiety, or boredom or sometimes the particular sights, sounds, and objects found in a store (Aboujaoude, Gamel, & Koran, 2004). The stealing provides momentary tension relief, often followed by intense feelings of remorse, anxiety, embarrassment, or guilt (Aboujaoude et al., 2004; Grant & Kim, 2002a; McElroy et al., 1991). As mentioned by Bleuler (1911/1930) and others (Grant & Kim, 2002a), the individual might hoard the stolen items, give them away, furtively return them, or dispose of them in some other way.

Psychiatric comorbidity is common, especially mood disorders, eating disorders, anxiety disorders, and substance misuse (Grant & Potenza, 2008). Other behavioral addictions are common as well, but more so in men than in women (Grant & Potenza, 2008). Men are more likely to have an intermittent explosive disorder and CSB, whereas women are more likely to have an eating disorder or bipolar disorder.

A study of 12 patients with kleptomania and no other Axis I disorder revealed significantly higher traits of novelty seeking and harm avoidance and lower reward dependence than control patients (Grant & Kim, 2002a). In addition, these patients reported lower levels of paternal and maternal affection and decreased maternal encouragement to explore their environment (Grant & Kim, 2002b).

Family study data are limited, but the pattern in two studies suggests a connection with the substance addictions (Grant et al., 2003; McElroy et al., 1991). McElroy et al. (1991) found depression, bipolar disorder, alcohol disorders, and anxiety disorders among 103 first-degree relatives of 20 people with kleptomania; 2% had kleptomania themselves. Grant (2003b) reported significantly higher rates of alcohol use disorders and any psychiatric disorder in the first-degree relatives of 31 people with kleptomania compared with control relatives. Dannon, Lowengrub, Lancu, and Kottler (2004) reported high
rates of depression, bipolar disorder, and obsessive-compulsive disorder as well as higher dimensional ratings of depression, anxiety, and obsessionality.

Not unlike the substance addictions, there is general agreement that the goal of treatment should be sustained control of the problematic behavior (in this case, stealing) and not just a reduction in the behavior (Koran et al., 2010). Medication studies are ongoing, and to date there have been several case reports and open-label studies that have tested SSRIs, mood stabilizers, or opioid antagonists. For example, in a case series of 20 patients with kleptomania, 10 of 18 patients given various antidepressants, mood stabilizers, or both improved after several weeks of treatment. Stealing behavior resumed for two patients when the study discontinued their medication (McElroy et al., 1991). However, there have been case reports of people treated for depression with SSRIs who subsequently developed kleptomania (Kindler, Dannon, Iancu, Sasson, & Zohar, 1997). Dannon (2003) reported the successful treatment of kleptomania in three patients using topiramate, a mood stabilizer, alone or in conjunction with SSRIs. In a recent randomized controlled trial, naltrexone (50-150 mg/day) produced significant reductions in stealing urges and behaviors compared with placebo (Grant et al., 2009). The latter study is particularly interesting because naltrexone is known to reduce cravings for alcohol and U.S. Food and Drug Administration has indicated that it can be used to treat alcohol dependence.

Clinical experts have recommended various forms of behavioral therapy, including covert sensitization, systematic desensitization, and aversion therapy, but researchers have not yet systematically evaluated these treatments (Koran et al., 2010). Family therapy might be helpful when the kleptomania disrupts the family unit, for example, by the shame and embarrassment caused when a parent is arrested (Koran et al., 2010). Likewise, marital (or couples) counseling might be helpful when the kleptomania in one member of the dyad disrupts the relationship (Koran et al., 2010).

Many people with kleptomania are arrested for shoplifting and face legal consequences. The embarrassment and shame they experience might keep some from acting on their urges to steal, but this improvement tends not to last, as for example in the case of Ms. C described earlier (Grant & Kim, 2002a). Common wisdom suggests that a self-imposed ban on shopping in an attempt to head off potential thefts might help curb stealing, but this is probably not sustainable.

**COMPULSIVE SEXUAL BEHAVIOR**

Mr. D, a 37-year-old married man, sought psychiatric evaluation after being suspended from work for viewing Internet pornography. Monitoring of his Internet use showed that he spent as many as 6 hours each day viewing pornographic websites, and masturbating as many as six times daily. Mr. D had a long history of compulsive masturbation starting at age 12, which he described as a way to comfort himself during periods of depression and anxiety. He described a troubled childhood with emotionally distant and verbally abusive parents, but there was no history of physical or sexual abuse.

Poor productivity and frequent absenteeism characterized Mr. D's work performance. He had been married twice and had three children. His first wife sought a divorce because she could not tolerate his sexually compulsive behavior, and his second wife had recently left for the same reason.

He had seen several therapists over the years, usually in the context of marital discord, and had sought psychiatric treatment for depression and anxiety. A psychiatrist once prescribed the SSRI fluoxetine for depression, but Mr. D discontinued the drug after taking it less than a week, and dropped out of therapy after two sessions. He never disclosed his sexual compulsion to any clinician. (Kuzma & Black, 2008)

Clinicians and laypeople have long recognized the addictive quality of CSB. Although German psychiatrist Krafft-Ebing (1886/1927) described
hypersexuality nearly 100 years ago, many people credit Orford (1978, p. 299), who observed that “a theory of dependence must take into account forms of excessive appetitive behavior which do not have psychoactive drugs as their object,” for being one of the first investigators to recognize sexual addiction. Carnes (1983) introduced the concept of sexual addiction as a behavioral disorder similar to a chemical addiction to a lay audience in Out of the Shadows: Understanding Sexual Addiction.

Despite the lack of consensus on its definition, operational criteria for CSB have been proposed by several investigators, all of which incorporate the concepts of inappropriate or excessive sexual cognitions or behaviors, subjective distress, and impaired functioning in one or more important life domains (Black, 1998; Goodman, 1993; Kafka, 1997; Stein, Black, & Pienaar, 2000; Stein, Black, Shapira, & Spitzer, 2001). As currently conceptualized, CSB encompasses various problematic sexual behaviors that scientists and researchers have divided into paraphilic and nonparaphilic subtypes (Coleman, Granzer, & Nesvacil, 2000). The former involve pathological sexual behaviors (e.g., exhibitionism, voyeurism); the latter involve conventional sexual behaviors taken to extremes (e.g., compulsive masturbation, promiscuity, pornography dependence). Eight specific paraphilias are listed in DSM-IV-TR (American Psychiatric Association, 2000), but nonparaphilic forms of CSB are excluded. For classification purposes, nonparaphilic disorders could be diagnosed in DSM-IV-TR as either an impulse control disorder not otherwise specified or as a sexual disorder not otherwise specified. Hypersexual disorder, a term that corresponds with the concept of nonparaphilic CSB, has been proposed for inclusion in DSM-5 within the category for sexual and gender identity disorders (http://www.dsm5.org), although it should arguably instead be classified as a behavioral addiction (Briken, Hill, & Berner, 2010).

There have been no careful studies of prevalence of CSB, but on the basis of clinical surmise, its lifetime prevalence has been estimated to range from 3% to 6% in the general U.S. adult population (Carnes, 1991; Coleman, 1992). In one of the few clinical studies to report prevalence, Grant et al. (2005) found a 4.4% current and 4.9% lifetime prevalence of CSB in adult psychiatric inpatients.

Frequency of sexual outlets (i.e., orgasm) is only one aspect of CSB. However, Kafka (1997) suggested that data on total sexual outlet (total number of orgasms achieved through any means during a designated week) might more accurately reflect CSB prevalence in men. Kinsey, Pomeroy, and Martin (1948) originally developed the concept of the total sexual outlet and reported that 7.6% of men 30 years and younger had an average total sexual outlet of seven or more for at least 5 years, primarily through masturbation. The median total sexual outlet was 2.14 for that age group and 1.99 for all men. Atwood and Gagnon (1987) reported that 5% of high school age boys and 3% of college age White men masturbated at least once daily. Laumann (1994) reported that 34% of men between 18 and 25 years old masturbated weekly, 15% masturbated two to six times weekly, 2% daily, and 1% at least once daily in the past year. Examining these and other data, Kafka (1997) suggested that seven or more weekly orgasms over 6 consecutive months could be used to define hypersexual behavior, a figure that characterizes 3% to 15% of the adult male population in the United States.

Some people have criticized the concept that high-frequency sexual behavior is inherently pathologic. A study from Sweden (Långstrom & Hanson, 2006) suggested that simple frequency of sexual activity is an insufficient metric for CSB. Ironically, they noted that high-frequency sexual behavior with a stable partner is associated with better psychological functioning; frequent solitary or impersonal sexual behavior correlated with comorbid psychiatric disorder and psychosocial dysfunction.

Clinical data have shown a male preponderance for CSB (Kuzma & Black, 2008). Some studies have included only men, representing a clear selection bias, yet centers that treat sexual addictions also find a male preponderance. For example, Carnes and Delmonico (1996) reported that 80% of 290 respondents at treatment centers for sexual addiction were men. When CSB is examined dimensionally, men have more symptoms as well (Dodge, Reece, Cole, & Sandfort, 2004). There might be gender differences in the way CSB manifests. Data have suggested that men are more likely to report compulsive masturbation, to engage in paraphilias, to pay for sex, or

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to engage in anonymous sex (Bancroft & Vukadinovic, 2004). Women are more likely to engage in fantasy sex (e.g., seductive behavior leading to multiple affairs or relationships) or sadomasochism or to use sex as a business (Bancroft & Vukadinovic, 2004). Women with CSB are more likely to experience emotional attachments, for example, presenting as a series of multiple failed or dangerous relationships, and might refer to themselves as “love addicts” (Black, 2000).

CSB appears to have an onset during adolescence, with paraphilic behaviors frequently occurring earlier than nonparaphilic behaviors (Black, Kehrberg, Flumerfelt, & Schlosser, 1997; Kafka & Prentky, 1992). Like substance addictions, for most people the disorder is chronic or episodic but not remitting. On the basis of his clinical impressions, Carnes (1991, 1996) has characterized CSB as a progressive, multi-phase disorder that grows more intense the longer it is untreated. Carnes described preoccupation as the first phase, in which the person develops sexual thoughts and urges. This is followed by ritualization, or the development of a routine that prompts the sexual behavior. The third phase, gratification, involves the sexual behavior. The final phase, despair, is characterized by feelings of guilt, powerlessness, and isolation, all of which fuel the tension underlying CSB and prompt the person to repeat the cycle.

In their sample of 36 people with CSB, Black, Kehrberg, et al. (1997) reported that 92% of respondents reported having sexual urges and fantasies or that they were overly sexually active. These investigators also found that three quarters of the sample abused drugs or alcohol while engaging in their CSB. Black, Kehrberg, et al. surmised that the concurrent substance misuse might disinhibit the individual sufficiently to promote the sexual activity, to enhance his or her pleasure, or to numb his or her sense of shame. When asked what they disliked about the CSB, respondents reported the lack of control and its time-consuming nature. Nearly two thirds of the sample reported being subjectively distressed by their sexual thoughts or behaviors, and nearly half felt that it caused impairment in important life domains, such as their marriage or significant relationships, or that it had affected their work (e.g., through intrusive thoughts or from frequent lateness). Similarly, Raymond, Coleman, and Miner (2003) reported that nearly one third of a sample described their sexual thoughts as intrusive, and more than two thirds had attempted to resist thoughts, and 87% to resist urges.

Researchers have observed psychiatric comorbidity in clinical CSB samples, particularly for mood, anxiety, substance use, and personality disorders (Black, Kehrberg, et al., 1997; Kafka & Prentky, 1992; Raymond et al., 2003). Grant and Kim (2003) reported that of 96 people with pathological gambling, 9.4% reported a lifetime history of CSB, suggesting that there might be a special relationship between the disorders. Kafka (1994) has reported an increased prevalence of attention deficit/hyperactivity disorder among people with CSB.

Axis II disorders are common among people with CSB as well. Black, Kehrberg, et al. (1997) reported that 44% of their sample met criteria for a personality disorder, most commonly the histrionic, paranoid, and obsessive–compulsive types. Raymond et al. (2003) reported that 46% of 25 people with CSB had a personality disorder, but they identified no particular type. These investigators made the point that although one might expect that people with CSB have a tendency to act out, many are instead anxious individuals who likely have difficulty establishing intimacy.

Early theories regarding the development of CSB focused on the psychodynamics of uncontrolled sexual behavior and the relationship with intrapsychic conflicts and environmental traumas (Black, 1998). Both Coleman (1992) and Carnes (1991) have suggested that CSB begins with childhood sexual abuse, leading to low self-esteem, feelings of shame, and chronic anxiety. In their view, CSB develops as a means of coping with uncomfortable affects and feelings. Carnes (1996) pointed out that in a survey of self-identified people with CSB, nearly 80% endorsed having been sexually abused in childhood, but other researchers have reported much lower rates (Black, Kehrberg, et al., 1997; Kafka & Prentky, 1992). Some people have used learning theory to explain the development of CSB (Konopacki & Oei, 1988; McConaghy, Armstrong, & Blaszczyński, 1985), but Kafka (1997) has focused on the possible contribution of disturbed neurotransmission, noting
that norepinephrine, serotonin, and dopamine all serve to modulate sexual behavior and other dimensions of human and animal pathophysiology. Although androgenic hormones play an important role in modulating sexual drive and response, results from studies of sexual offenders and men with paraphilias do not support an association of excess testosterone leading to hypersexual behavior (Briken et al., 2010).

Treatment approaches vary widely and tend to be driven by the theoretical orientation and interests of the clinician rather than by empirical data (Allen & Hollander, 2006). Psychotherapy is commonly recommended, and clinicians have used a variety of therapeutic modalities, including imaginal desensitization, aversion therapy, group therapy, and both psychodynamic and cognitive–behavioral approaches (Briken, Hill, & Berner, 2010; Coleman, 1992; Goodman, 1992; Mick & Hollander, 2006). In its simplest form, psychotherapy begins with providing education about sexual behavior to help patients understand their disorder (Coleman, 1992). The goal is then to assist patients in learning more appropriate ways to express their sexuality and to meet their intimacy needs. CBT can be used to help restructure faulty cognitions that people with CSB use to justify their behavior. This can include relaxation exercises to help reduce the anxiety and stress that they frequently report as well as relapse prevention methods to forestall future CSB (Mick & Hollander, 2006).

Group therapy might be beneficial in confronting the patient's defensive lies as well as to sanction one another's acceptance of more appropriate sexual behavior (Carnes, 1991). Twelve-step programs (e.g., Sex Addicts Anonymous) patterned after Alcoholics Anonymous are available in some areas and might be helpful (Briken et al., 2010). Because CSB can damage marital relationships and family ties, treatment might need to include the spouse or partner, and sometimes the entire family (Schneider & Schneider, 1996). Self-help books are available and can be helpful (Carnes, 1983, 1991).

Medication studies have mainly focused on antidepressants and antiandrogenic agents. In uncontrolled studies reported by Kafka (2000) and Kafka and Prentky (1992), patients have generally tolerated SSRIs well and have experienced a reduction in sexual preoccupations and impulsivity. In a retrospective observational study, the antidepressant nefazodone appeared helpful in treating people with CSB (Coleman et al., 2000). In one of the few controlled studies, Wainberg et al. (2006) compared the SSRI citalopram to placebo in 28 gay or bisexual men with symptoms of CSB. Citalopram was associated with decrease in sexual drive, frequency of masturbation, and pornography use.

Kafka (2000) has argued that psychostimulants might be useful adjuncts to SSRIs because many men with CSB have comorbid attention deficit/hyperactivity disorder. He has described a series of men with comorbid attention deficit/hyperactivity disorder and has reported improvement in their CSB after the combination of an SSRI and the stimulant drug methylphenidate (Kafka, 2000). In one case, a patient with CSB was treated successfully with naltrexone, an opiate antagonist (Grant & Kim, 2003). The article noted that the patient had been refractory to fluoxetine, behavioral therapy, and individual psychotherapy but that high-dose naltrexone (150 mg daily) led to remission of urges for excessive sexual behavior as well as concomitant stealing.

Practitioners have used antiandrogens to treat CSB, although their use should probably be limited to control sexually aggressive forms of CSB (Black, 1998; Briken et al., 2010). Medroxyprogesterone, an analog of progesterone, and cyproterone (not available in the United States), an androgen-receptor blocker, are the two most commonly used agents (Allen & Hollander, 2006). They can reduce sexual desire and associated fantasies, penile erections, and other sexual behavior within 2 to 4 weeks after initiation of therapy. One advantage is that physicians can administer the drugs as long-term injections weekly or biweekly. However, there are no controlled studies to evaluate their dosing, safety, or efficacy. Nonetheless, a retrospective study of 275 sexual offenders who received medroxyprogesterone injections over a 4-year period had no new offenses compared with one third of men judged to need the medication who did not receive it (Maletzky, Tolan, & McFarland, 2006). A placebo controlled trial of the gonadotropin-releasing hormone analog triptorelin in men with severe paraphilias suggested that it is effective (Röslcr & Witztum, 1998). A
Bladl, Kuzma, and Shaw's meta-analysis of 118 patients suggested that luteinizing hormone-releasing agonists were effective in treating patients with severe paraphilias (Briken, Hill, & Berner, 2003).

**INTERNET ADDICTION**

Mr. E, a 47-year-old married computer consultant, spent nearly 12 “recreational” hours on the computer on weekdays and up to 18 hours daily on weekends. His time was spent answering and sending electronic mail, using chat web sites, and “surfing” the Internet. He reported having developed several romantic relationships and would exchange sexually explicit photographs with the women. He owned three personal computers and incurred significant debt purchasing computer paraphernalia. He had been arrested several times for computer hacking, and had lost several jobs due to inappropriate computer usage at work and admitted spending little time with his wife and three children. He admitted a preference for an on-line social life and rarely socialized in other settings. He acknowledged feeling powerless over his computer usage. (Black, Belsare, & Schlosser, 1999, p. 842)

Personal computer use and Internet access have become universal, and as they have spread, reports of their misuse have gradually increased (Aboujaoude, 2010). To illustrate, Aboujaoude, Koran, Gamel, Large, and Serpe (2006) conducted a random telephone survey of 2,513 adults in the United States and reported that 69% of the respondents were regular Internet users. Of regular Internet users, 5.9% felt their relationships suffered as a result of their excessive use, 8.7% attempted to conceal nonessential Internet use, and 3.7% felt preoccupied by the Internet when offline.

The many names given the phenomenon of excessive computer use recognize the various ways in which it has been regarded: compulsive computer use (Black et al., 1999), pathological Internet use (Davis, 2001), problematic Internet use (Caplan, 2003), Internet dependency (Scherer, 1997), and Internet addiction (Shaw & Black, 2008). These terms suggest a tension between those who view the disorder as involving any abnormal or pathological computer use and those who focus specifically on Internet usage. Because most investigators have acknowledged that this phenomenon involves a variety of computer use behaviors, Shaw and Black (2008) suggested that consideration of the phenomenon should acknowledge all forms of inappropriate and excessive computer use, even when it does not involve Internet access. This broader conceptualization should also take into account rapidly evolving electronic means of communication, including popular social networks such as Facebook. Thus, it is important to consider the vehicle (computer), connection (Internet), and content (e.g., pornography, gambling) as important facets of the syndrome.

There are no agreed-on definitions of Internet addiction. In the psychiatric literature, Black et al. (1999) defined compulsive computer users as those research participants who endorsed “compulsive computer use that had contributed to personal distress, or social, occupational, financial, or legal consequences” (p. 840). Shapira, Goldsmith, Keck, Khosla, and McElroy (2000) defined problematic Internet use by enumerating operational criteria that emphasize cognitive and behavioral aspects of the disorder as well as impairment characterized by subjective distress and interference in social or occupational functioning, but ruling out mania and hypomania. Stein (1997) suggested that whereas the terms addiction and compulsion used to describe the phenomenon do not fully capture essence of the disorder, the “intense attachment to computers seems to be a real one” (p. 890). Internet addiction is not included in DSM-IV-TR, nor has it been proposed for inclusion in DSM-5 (http://www.dsm5.org).

Internet addiction is an example of a new behavioral addiction possible only in contemporary society because of the presence of new technology. For this reason, it shows the clear influence of cultural and technological change on socially sanctioned behavior taken to its extreme. Despite its newness, it captures the same common features that distinguish the other behavioral addictions and connect them to...
substance addictions: repetitive and driven behaviors despite negative consequences, diminished control over the behaviors, cravings for the behavior, and experiencing a pleasurable response while engaged in the behavior.

Young (1998b) proposed criteria for Internet addiction patterned after the DSM-IV criteria for PG. According to her criteria, only nonessential computer or Internet usage (e.g., nonbusiness or nonacademic use) is counted, and Internet addiction is present when five or more symptom criteria are present during the past 6 months and mania plays no causal role. According to Young, Pistner, O’Mara, and Buchanan (1999), Internet addiction is a broad term covering a wide variety of behaviors including pornography or adult fantasy; role-playing chat rooms; online relationships; gambling, shopping, or stock trading; web surfing and database searches; and preprogrammed computer games. People have developed several screening instruments to identify Internet addiction, although none has emerged as the gold standard (Brenner, 1997; Egger & Rauterberg, 1996; Morahan-Martin & Schumacher, 2000; Widyanto & McMurrain, 2004; Young, 1998a).

Surveys have suggested that Internet addiction is widespread. In studies that have focused on younger people, prevalence estimates have ranged from 0.9% (Yoo et al., 2004) to 38% (Leung, 2004). Online surveys have produced estimates ranging from 3.5% (Whang, Lee, & Chang, 2003) to 18% (Niemz, Griffiths, & Banyard, 2005). In their random telephone survey of U.S. adults, Aboujaoude et al. (2006) reported prevalence rates ranging from 0.3% to 0.7%. An additional 4% to 13% of respondents endorsed one or more markers consistent with problematic Internet use, such as being preoccupied when offline or concealing one’s Internet use. Clearly, much work is needed to clarify the prevalence of Internet addiction. The findings reported here vary widely, perhaps because there was little uniformity among the studies in either the definitions used or in the assessment methods. Although research has suggested the disorder is widespread, no study indicates its severity, for example, whether the person with Internet addiction is subjectively distressed or is disabled in important life domains.

The data are inconclusive as to whether there is a gender difference for Internet addiction. Of the 13 published surveys, six found a male preponderance but five did not, and two did not report a gender distribution (Aboujaoude et al., 2006; Egger & Rauterberg, 1996). Clinical reports have suggested a male preponderance, but this could be the result of biased ascertainment (Black et al., 1999; Shapira et al., 2000). The gender distribution might possibly be explained by the fact that men are more likely to have an interest in games, pornography, and gambling, activities that have all been associated with problematic Internet use (Morahan-Martin & Schumacher, 2000). Popular social networking websites (e.g., Facebook) are of great interest to men and women and have the potential to alter the gender distribution of Internet addiction. Nearly all relevant data predate the explosion of interest in these websites.

The age of Internet addiction onset is not known. Although two clinical studies have suggested an onset during the late 20s or early 30s (Black et al., 1999; Shapira et al., 2000), many studies of Internet addiction have involved youths, suggesting a much earlier onset. Young (1998b) suggested that use often becomes problematic 6 to 12 months after exposure to the Internet.

Psychiatric comorbidity appears common, particularly for mood, anxiety, and substance use disorders as well as other behavioral addictions (Shaw & Black, 2008). Using a dimensional approach to assess psychological status, Kraut et al. (1998) reported that increased use of the Internet was associated with higher ratings on measures of depression, loneliness, and social isolation. These findings were compatible with those of Nie and Erbring (2002), who concluded that the Internet is an isolating technology, even more so than television. Young and Rodgers (1998) administered the Beck Depression Inventory (Beck, Steer, & Brown, 1996) to 259 “addicted users” and reported a mean score of 11.2, which suggests that the group had mildly elevated levels of depression. These investigators suggested that the low self-esteem, poor motivation, fear of rejection, and need for approval associated with depression might contribute to increased Internet use, presumably as a way of emotional coping.
Bladl, Kuzma, and Shaw (1998) concluded from a study of 445 individuals, 46% of whom self-identified as having addiction, that there was a significant relationship between frequent Internet use and both depression and introversion.

Research has not yet identified particular personality traits associated with Internet addiction. Black et al. (1999) reported that subjects often meet criteria for at least one personality disorder, with borderline personality disorder being the most frequent (24%), followed by the narcissistic (19%) and antisocial (19%) types. In terms of dimensional characteristics, Young and Rodgers (1998) found that people dependent on the Internet rank high in self-reliance, have a strong preference for solitary activities, and tend to restrict their social outlets.

Family history data are limited. In their study of 20 problematic Internet users, Shapira et al. (2000) observed that all but one problematic Internet user had positive family histories of psychiatric disorder. Thirteen (65%) problematic Internet users had at least one first- or second-degree relative with a depressive disorder, 10 (50%) with a bipolar disorder, and 12 (60%) with a substance use disorder. These investigators, however, did not ask respondents whether their relatives had an Internet addiction.

Davis (2001) has used cognitive–behavioral theory to explain the onset and maintenance of Internet addiction. In this model, maladaptive cognitions such as self-doubt and low self-efficacy combine with compulsive Internet use, and over time, cognitions and behaviors intensify and continue to produce negative outcomes, producing a diminished sense of self-worth and increased social withdrawal. Caplan (2003) expressed the belief that deficient social skills are important contributing factors and that computer-mediated communications are particularly attractive to people who see themselves as low in social competence. Whereas these theories are helpful in understanding and conceptualizing the disorder, neither is supported by empirical evidence.

Neurobiological theories of the development of Internet addiction center on disturbed neurotransmission, particularly serotonin and dopamine (Shaw & Black, 2008), but there is no direct evidence to support the role of these or other neurotransmitter systems in Internet addiction.

Pallanti, Bernardi, and Leonardo (2006) observed that most work on Internet addiction has involved adolescent samples. They hypothesized that immaturity of the frontal cortical and subcortical monoaminergic system during normal neurodevelopment underlies the adolescent impulsivity considered the “foundation of disorders marked by disturbance of reward motivation” and that underdeveloped brain circuitry might underlie Internet addiction (p. 968).

Shotton (1991), in perhaps the earliest systematic study of 106 computer “dependents,” found that compared with two normative groups, dependents were less likely to be married and most were first-born children. People who were dependent tended to buy computers as soon as they were available and owned more computer paraphernalia and computers than others, and most admitted to becoming addicted from their first “hands-on” experiences with computers. Additionally, they spent significantly more time using their computers at home and at work than did others and found it difficult to stop “computing” when at the keyboard, often losing all sense of time. Shotton wrote, “Old hobbies disappeared and family activities were no longer undertaken” (p. 223).

Black et al. (1999) systematically assessed the experiences of 21 compulsive computer users. Subjects admitted that their computer use led them to feel excited (52%), happy (48%), or powerful (19%), yet that sometimes they used their computer to assuage feelings of sadness (38%), frustration (10%), or irritability (14%). The subjects reported positive aspects from their computer use as well; 52% reported that computer use distracted them from their problems or concerns, and 29% reported that they enjoyed obtaining new information on the Internet. Most admitted that their computer usage had caused problems with family or friends or with work or school. Nearly one third had tried to cut back but observed that doing so made them more anxious. None felt the disorder was sufficiently problematic to seek treatment. Another aspect of the disorder, as captured in the case reported by Belsare, Gaffney, and Black (1997), is the sense of tension or arousal before successfully logging on to the Internet and the sense of relief obtained through that act.

Black et al. (1998) reported that their 21 subjects spent a mean of 27 hours per week in nonessential
computer use. Shapira et al. (2000) reported a similar figure (28 hours per week) for their 20 subjects. In contrast, Morahan-Martin and Schumacher's (2000) pathological Internet users spent a mean of 8.5 hours online weekly. It might be that the actual amount of time spent online is less important than the resulting distress or impairment. Nonessential activities recorded in these studies included web surfing, chat rooms, e-mail, games, designing web pages, pornography, newsgroups, and shopping. These activities frequently intertwine; for example, people interested in pornography might spend hours searching websites for particular images or spend many hours in chat rooms (Stein et al., 2001).

There are no established treatments for Internet addiction. Hall and Parsons (2001) modified CBT to treat Internet addiction and observed that these techniques are familiar to many mental health treatment providers, and can apply to treating not only substance misuse but also process addictions. Young (2010) has developed a guide using cognitive-behavioral techniques for therapists working with Internet addicts.

Clinicians have used the SSRIs to treat Internet addiction, probably because of their efficacy in treating obsessive-compulsive disorder. Hadley, Baker, and Hollander (2006) reported the results of a small open-label study of 19 people with a "compulsive-impulsive computer usage disorder" who received escitalopram for 10 weeks, followed by a 9-week double-blind discontinuation phase. In the first phase, subjects experienced significant improvement in hours spent in nonessential computer activity and other measures of response. Improvement persisted throughout the second phase, although there were no significant differences between the escitalopram and placebo groups. These results suggest that the improvement experienced by the subjects could have been due to the placebo effect. Sattar and Ramaswamy (2004) had earlier reported in a single case that escitalopram reduced the subject's urges for online gaming.

Self-help books and tapes are available online and might be helpful to some people with Internet addiction (Young, 1998b). Support groups are available in some areas and are available online as well. Family therapy might be helpful when the person with addiction disrupts the family unit; marriage (or couples) counseling might be helpful when the behaviors have disrupted the relationship (Shaw & Black, 2008). A halfway house for adolescents with Internet addiction has opened in China, and treatment includes group therapy, medication, acupuncture, and sports (Ang, 2005).

Common sense suggests that at least for some, a self-imposed ban on computer access outside of work situations might be the best solution (Shaw & Black, 2008). This could include getting rid of home personal computers and canceling Internet service. A less harsh strategy might be to place the PC in a central location, such as the kitchen or family room, where excessive or inappropriate use can be monitored by family members. For young people, software is available to restrict Internet access and can provide an external control when a person has trouble controlling his or her behavior (Liu & Potenza, 2010).

**SUMMARY**

Interest in the behavioral addictions has grown in the past decade, leading to a better understanding of these conditions. Yet there is little agreement regarding the breadth of this new category or the definitions of several proposed members such as CB, CSB, and Internet addiction. The lack of agreement has complicated attempts to study their prevalence, gender distribution, and natural history, but research has suggested that behavioral addictions are relatively common. Whether the prevalence of these disorders is increasing needs further study. For example, it would come as no surprise that the prevalence of Internet addiction has continued to grow as computer availability and Internet access expands.

Important gaps remain in the understanding of behaviorally expressed addictions. Psychometrically sound instruments for screening and diagnosis are available for some of the disorders (PG, CB, Internet addiction), but researchers have devoted little scientific attention to other disorders (CSB, kleptomania). Little is known about the natural history of any of these conditions, yet nearly all are assumed to be chronic but fluctuating in severity and intensity,
mainly on the basis of clinical impression. Follow-up studies are necessary to chart their course, track their emergence and subsidence, and determine their relationship with co-occurring disorders, including other behavioral addictions. Neurobiological and genetic research has been sparse, and although early work with PG suggests commonalities with substance addiction (Potenza et al., 2003; Slutske et al., 2000), there are no brain imaging or genetic studies of the other behavioral addictions.

Whether these disorders make up a single, unified category related to the substance addictions, are independent entities, or are better grouped alongside obsessive-compulsive disorder remains to be determined. Family history and genetic studies might help answer these questions, and thus far they suggest a connection between substance addictions and PG, and perhaps CB, but data are limited as to the familial links with kleptomania, CSB, and Internet addiction. Finally, although behavioral addictions are widespread, with the exception of PG there have been no systematic studies of proposed treatments. Also, it is not clear which patients might benefit from CBT or other psychotherapeutic methods or whether medication has any role in treating these disorders.

Additional research needs to explore the connections among these disorders and with the substance addictions. The lack of federal funding has further hampered researchers’ progress, and many have had to turn to industry or private foundations for support. Until the federal government chooses to broaden its concept of addictions, funding will probably continue to be limited and require that scientists be creative in conducting research. The creation of the proposed behavioral addictions category in DSM-5 might help push this process along by recognizing that scientific and clinical evidence supports the connection among these disorders. To obtain a fully informed understanding of the disorders, these disorders should be viewed as different and unique behavioral expressions of addictions.

References


