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PSYCHOACTIVE SUBSTANCE USE DISORDERS

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Throughout history and across cultures, humans have sought out various psychoactive substances, concoctions, drinks, pills, plants, herbs, and potions to alter, improve, change, and otherwise treat what ails us. The term *psychoactive* refers to a substance or drug that can alter physiology, cognition, emotion, and behavior. The part of the body most affected by psychoactive substances or drugs is the central nervous system (CNS), which comprises the brain and the spinal cord. Once inside the CNS, the drug can alter biochemical interactions both within and between brain cells or neurons. These interactions, in turn, often have a powerful and rapid impact on thinking, feeling, and behaving. Drug effects are typically classified in terms of how they alter CNS functioning. For example, alcohol is classified as a CNS depressant, given that it typically slows heart rate and respiration and leads to sedation, disinhibition, and a subjective sense of euphoric intoxication. Opiate drugs, such as heroin and prescription medications like codeine, can have dramatic effects on the CNS, such as a euphoric rush, relaxation, apathy, and impaired judgment. In contrast to opiates and depressants, CNS stimulants like cocaine and amphetamines tend to produce physiological arousal, euphoria, restlessness, diminished appetite, expansive mood, agitation, and even paranoia. However, before the biochemical processes and subsequent effects can occur, drug-seeking behavior must take place. Drug-seeking behavior, or the use of psychoactive substances, is the focus of this chapter.

Whether the substance use is characterized as *disordered* will be a major point of discussion, given that drug-seeking behavior is ubiquitous across time and cultures. Thus, it is important to distinguish between typical and expected patterns of substance use versus more problematic behaviors, up to and including substance abuse and dependence. According to the *Diagnostic and Statistical Manual of Mental Disorder*, Fourth Edition, Text Revision (*DSM-IV-TR*; American Psychiatric Association [APA], 2000), substance *abuse* is characterized by the repeated use of a drug despite evidence of recurrent and significant negative consequences of drug seeking, such as damage to bodily organs (e.g., liver, brain), occupational impairments (e.g., absenteeism, being fired), legal difficulties (e.g., multiple DUI convictions), and interpersonal problems (e.g., family estrangement, divorce). Substance *dependence* involves many of these consequences plus the added features of *tolerance*, *withdrawal*, and compulsive drug taking (APA, 2000). Tolerance means that the substance user needs to take increasingly greater amounts of the drug to achieve the desired effects, whereas withdrawal suggests there is a maladaptive pattern of behavior, physiology, and cognition when the user stops taking the substance. In addition to tolerance and withdrawal, the compulsive drug-seeking behavior becomes a major focus of a person's life (acquiring, using, recovering), which often includes several unsuccessful attempts to cut down or quit and some acknowledgment that it is causing significant problems in several areas of functioning. Indeed,

for the substance-dependent individual, drug-seeking behavior persists and even escalates despite the problems it causes the user.

Several subdisciplines in psychology, from neuroscience to learning and cognition, from developmental to health psychology, and from clinical to social psychology, have made substantial contributions in the quest to understand and ultimately treat substance use disorders. For example, experiments from learning laboratories provide support for the notion that the drug itself can serve as an unconditioned stimulus (US) in animals and humans, in much the same way that food and electric shock serve as the US in Pavlovian conditioning paradigms (Domjan, 2003). The drug (US), in turn, becomes associated with environmental cues or conditioned stimuli (e.g., drug-using friends, places) that further strengthen the relationship between the substance and the drug-seeking behavior (conditioned response, or CR). Similarly, neuroscience research has elucidated how particular parts of the brain (e.g., limbic system) respond to various drugs and create a biochemical craving when the drug levels are reduced or become depleted, which, in turn, lead to cognitive and affective states that promote drug seeking (Julien, 2005). Developmental psychologists have documented how early exposure to various substances, such as alcohol, greatly increase the risk of dependence and other adverse implications well into adulthood if the alcohol use is not curtailed soon after its initial onset. Clinical psychology has certainly made a number of contributions to the understanding of substance use disorders, particularly in the diagnosis and treatment of these conditions. Unfortunately, a complete review of these scientific and clinical discoveries exceeds the scope of this chapter. However, I have included a general discussion of the major issues to give students who are considering whether to pursue a career in substance use disorders a good overview of the field and some of the empirical and clinical possibilities therein.

A BRIEF HISTORY OF SUBSTANCE USE DISORDERS: MOTIVES AND CROSS-CULTURAL FACTORS

The reasons for drug-seeking behavior are endless. They range from a desire to treat medical ailments to assuaging psychological stress to curing simple boredom. Choosing a particular drug depends often on its ability to produce the desired effect (i.e., pharmacological properties). Substances such as alcohol are commonly used to celebrate major events such as weddings or to induce relaxation after a difficult day at work. In addition, the motives for use vary over time and across cultures. For instance, Navajo Indians in the southwestern United States have historically used peyote, a powerful hallucinogenic substance derived from a cactus plant, in their religious ceremonies. These practices are still condoned and supported by the Navajo Indians. By today's standards in the United States, taking an over-the-counter (OTC) medicine such as aspirin or acetaminophen is a common, legal, and appropriate means of seeking relief from minor aches and pains. Few would question the rationale of taking a pain reliever for pain, especially when the substance has been found to be effective. OTC medicines like aspirin and acetaminophen are classified as analgesics (derived from the Greek "an," meaning *without,* and "algia," meaning *pain*) and are therefore psychoactive, given that they alter the perception of pain. Nonetheless, because OTC pain relief preparations such as acetaminophen have an accepted and generally safe medical use along with a low potential for abuse, this type of drug-seeking behavior rarely, if ever, develops into a problematic pattern of use. Aspirin and similar substances are not classified as controlled substances by the United States government.

Governmental regulatory agencies like the Drug Enforcement Administration (DEA) and the Food and Drug Administration (FDA, part of the U.S. Department of Health and Human Services) help to determine which drugs should make the list of controlled substances, given their medical utility, safety, and potential for abuse. The overarching mission of these agencies is to protect the citizenry and assist the clinical disciplines relevant to substance use disorders in the expeditious identification and treatment of addictive illness. However, simply because a drug does not appear on the list of controlled substances does not imply that it is harmless. Neither alcohol nor nicotine is on the controlled substances list, yet these two drugs account for more substance use disorders in the United States than all of the other drugs combined (Substance Abuse and Mental Health Services Administration [SAMHSA], 2003). In 2002, alcohol was the most widely consumed intoxicating substance; approximately 80 percent of those over the age of 12 reported that they had used it, and over half of those individuals had used alcohol in the previous month (SAMHSA, 2003).

In contrast to the widespread use of alcohol today, historic attitudes toward alcohol, especially distilled liquor (e.g., whiskey), have not been nearly as supportive. For example, Benjamin Rush, a well-known Philadelphia physician who signed the Declaration of Independence, believed that heavy consumption of alcohol severely damaged a person's morality. He provided clinical descriptions of the toxic effects of heavy alcohol consumption, ranging from jaundice (i.e., liver damage) to seizures (often present during alcohol withdrawal) to "madness" (intense craving, tremors). He highlighted his views in a paper (originally written in 1784) entitled *An Inquiry Into the Effects of Ardent Spirits Upon the Human Body and Mind* (Rush, 1812). Rush was particularly admonishing about distilled spirits and was the first to describe excessive and chronic alcohol use as a *disease of addiction.* Some of these views evolved over time into variations of the temperance (consuming only wine or beer) and prohibition (banning the sale and use of alcohol) movements in the United States. During a 13-year period (1920–1933), law prohibited all sales of alcohol in the United States, which reduced alcohol consumption and the related consequences to a significant

degree, yet it did not eliminate the use of alcohol. Demand for alcoholic beverages was still high, and there were several ways to acquire it. Speakeasies (clandestine drinking clubs) and bathtub distilleries became much more commonplace, and an elaborate black market was created to satisfy the demand for alcohol.

Thus, depending on the era, cultural practices, and other circumstances, perceptions and policies about substance use have varied substantially. Suppose it was 1890 and you lived in Atlanta, Georgia. Instead of reaching for an aspirin to treat a headache, you might have walked a short distance to the local pharmacy and consumed a glass of Coca-Cola, which at the time contained an estimated 9 milligrams of cocaine. Coca-Cola was invented by pharmacist Dr. John Stith Pemberton in 1885. His original formula called for the use of fresh coca leaves and kola nuts, the sources of cocaine and caffeine, respectively. Using these ingredients, Pemberton created a flavorful, stimulant-based, and medicinal drink to treat various maladies from headaches to fatigue to morphine addiction. Pemberton's decision to use cocaine was based, in part, on the emerging evidence of cocaine's clinical utility as a local anesthetic and potential treatment for addiction.

Cocaine was originally isolated from the coca plant (*Erythroxylum coca*) in 1855 by a German chemist. Even Sigmund Freud celebrated its many uses in a scientific paper entitled *Über Coca* ("On Coca"), written in 1884. After experimenting with cocaine himself, Freud asserted that coca "wards off hunger, sleep, and fatigue and steels one to intellectual effort" (as cited in Byck, 1974, p. 60). Moreover, he downplayed the adverse side effects, including the concern expressed by some that it might have considerable addictive potential itself, even when used to treat another type of chemical dependency, morphine addiction. Freud reported "that a first dose or even repeated doses of coca produce no compulsive desire to use the stimulant further" (as cited in Byck, 1974, p. 62) and he even suggested that "the use of coca in moderation is more likely to promote health than to impair it" (as cited in Byck, 1974, p. 52). However, as the scientific evidence regarding the potential dangers of cocaine began to accumulate toward the end of the 19th century, Freud began to soften his position on the substance. Moreover, less than 20 years after Dr. Pemberton's original formula was created, attitudes toward this kind of medicinal remedy changed significantly and cocaine was removed from Coca-Cola in 1903. In 1914, cocaine use was outlawed in the United States through the Harrison Narcotics Act. The legislative act that currently prohibits the possession, use, or distribution of cocaine is the Controlled Substances Act, which was originally passed by the U.S. Congress in 1970.

Some of the early enthusiasm for using coca leaves and its derivatives originated from an awareness of ancient cultural practices of the indigenous people high in the mountains of South America, primarily in Peru and Columbia. The native coca plant was held in high esteem by local inhabitants for centuries, was used in several religious customs, and was known to alleviate fatigue and hunger, especially when working long hours at extreme altitudes. Individuals who used coca leaves in this way were often called *coqueros* ("coca chewers"). The practice involved taking a bundle of coca leaves, placing them between the cheek and gum, and adding some type of substance (e.g., lime) to extract the stimulant alkaloid (i.e., psychoactive ingredient). In fact, these patterns of use persist to this day, especially among the poor indigenous mountain farmers who rely on the crop to make a living.

Chewing coca leaves is a time-tested practice with relatively few adverse consequences or legal ramifications for the local inhabitants, even though the majority of their present agricultural efforts are utilized in the economy of the illicit cocaine trade around the globe. If you were to travel to Lima, Peru, today, perhaps one of the most cosmopolitan cities in South America, you would be able to enjoy a cup of hot coca tea without consequence, even though it contains trace amounts (approximately 4 milligrams) of psychoactive alkaloids. The practice of drinking coca tea is illegal in the United States, given that it contains cocaine. In many ways, coca tea consumption in South America is analogous to the widespread practice of caffeinated coffee consumption in the United States. In both cultures, stimulants, albeit much less powerful variants, are consumed regularly without notable adverse effects. Thus, determining whether a particular psychoactive substance is problematic depends not only on its pharmacological properties but also on the cultural context within which it is administered and used.

In summary, drug-seeking behavior has existed for centuries across the globe. The reasons for use range from attempting to relieve everyday maladies to inducing mood-altering experiences during religious rites to celebrating important life events. In perhaps the majority of instances, the occasional or legitimate medicinal use of psychoactive substances does not lead to chronic and/or severe adverse consequences. The limited number of negative consequences seen in these cases might be due to the biological properties of the substance (caffeine vs. cocaine), an individual's particular sensitivity to the substance, the context within which the use takes place, or some combination. Nevertheless, many individuals develop a pattern of drug-seeking behavior that is either abusive or dependent. Understanding the factors that lead to addiction is an important area of inquiry in the field of psychology, and several models have been proposed to help make sense of these phenomena.

THEORIES OF SUBSTANCE USE DISORDERS

Several theories have been advanced to help explain why people develop substance use disorders. Some of the prominent components of these theories include a

genetic predisposition to abuse alcohol and other drugs, a moral weakness, classical and instrumental conditioning, a stressful family environment, sensation-seeking personality traits, a preponderance of high-risk cognitions (e.g., expectancies), a desire to self-medicate, and exposure to drug-using individuals. Attempts to validate these theories through research have produced a mixture of support and controversy regarding some of the components. For example, the moral model of addiction, first proposed by Benjamin Rush, held that substance use and dependence was due to a deficit in morality or character. In fact, this model was the underlying premise to the temperance and prohibition movements in the late 19th through the early 20th centuries. However, although the moral model tended to produce a great deal of guilt in the user by insinuating that addiction was a character flaw, it essentially ignored other critical factors in addiction, such as environmental or genetic influences.

Another controversial topic that has emerged in the addictions field over the last 50 years is the idea that alcoholism in particular is a progressive and ultimately fatal medical disease. The seminal writings on the topic (Jellinek, 1960) suggested that for those who had the *disease of alcoholism*, any further consumption would invariably set off a chain of events that would ultimately result in death. Essentially, Jellinek argued that individuals with the disease of alcoholism exhibited a complete absence of control over alcohol and consuming even small amounts of alcohol would result in an irresistible craving and a return to heavy, dangerous, and ultimately fatal levels of drinking. Consequently, the only appropriate treatment for those who had the disease was total abstinence. The controversy emerged when it became virtually impossible to discern precisely who might have the progressive fatal disease versus those who exhibited less problematic drinking patterns that would improve over time, given the diversity of symptom presentation, demographics, and severity levels (Marlatt, 1983; M. B. Sobell & L. C. Sobell, 1984). Based on several outcome studies, it became apparent that some individuals were able to reduce or contain their alcohol consumption even after a period of problematic drinking that met criteria for alcohol dependence (e.g., M. B. Sobell & L. C. Sobell, 1973). Thus, more diverse treatments were prescribed, such as controlled drinking, as an alternative to the abstinence-only models.

Although the controversy regarding the disease model of addiction has not been resolved, the empirical efforts to understand how various factors contribute and interact to produce substance use disorders have grown substantially over the last several decades. The most current and well-established theories are multifactorial and include biological, psychological, and social paradigms. Thus, I will discuss three broad theories: the biological, psychological, and integrated models (also referred to as "biopsychosocial" theories of substance abuse and dependence).

Biological Models

Biological models of substance use and dependence typically focus on the influence of genetics and physiological mechanisms, such as neurotransmitters, in producing problematic patterns of drug-seeking behavior. Other biological theories of addiction hold that a person's early reaction to a substance helps determine subsequent risk for drug abuse and dependence. For example, exhibiting limited motor impairment (e.g., "body sway") when consuming moderate amounts of alcohol can influence the decision to drink more, which in turn leads to higher levels of tolerance and higher levels of subsequent alcohol consumption over time.

Another form of reactivity has to do with the substance itself and its pharmacological effects. Many of the most addictive substances, such as crack cocaine and methamphetamine, have contributed to substantial public health crises in this country over the last three decades. The current methamphetamine crisis, which originated primarily in the western United States, has crept steadily eastward across the country and impacts every geographic locale and socioeconomic stratum. In part due to the method of administration (i.e., smoking), but also due to the significant pharmacological effects of the substance, methamphetamine produces an intense euphoria for the user. That is, the "high" produced by the drug and its dramatic effect on neurotransmitters can be so compelling that any other reinforcing event (e.g., food, sex) pales by comparison. Consequently, neurological changes ensue, drug-seeking behavior intensifies, and the negative consequences mount.

Genetic Studies

It is now well established that substance abuse and dependence runs in families (e.g., Nathan, Skinstad, & Dolan, 2001). Although the exact method of transmission from one generation to the next is not clear, several genetic studies have provided support for the notion that a family history of abuse and dependence heightens the risk for subsequent generations of developing similar problems. For example, in an early twin study, Leohlin (1977) reported that monozygotic twins (who have identical genes) were significantly more likely to exhibit similarly heavy drinking patterns than dizygotic (or fraternal) twins, who share approximately 50 percent of their genes. Moreover, in a series of twin studies, concordance rates (i.e., both twins exhibiting the same behavior) for alcohol dependence were much higher for identical twins (48 to 58 percent) than for fraternal twins (29 to 32 percent; e.g., Kendler, Heath, Neale, Kessler, & Eaves, 1992; Prescott & Kendler, 1999).

In addition to twin studies, adoption studies provide a nice opportunity to study the effects of genetic transmission while holding particular environmental conditions relatively constant, such as the child-rearing environment. That is, if a child of an alcoholic parent is separated at

birth from the biological parents and raised by nonalcoholic foster parents, what are the chances the child will develop alcohol abuse or dependence? In perhaps the most often cited series of early studies, Goodwin and colleagues (1974, 1979) examined the children of Danish alcohol-dependent parents and the subsequent risk that they would themselves go on to develop alcoholism. Goodwin reported that both sons and daughters of alcohol-dependent parents were 3 to 4 times more likely to develop alcohol dependence, regardless of whether they were raised with their biological parents or with nonalcoholic adoptive parents. Thus, both twin and adoptive studies lend support to the idea that substance use and dependence are strongly influenced by genetic factors.

Reactivity to Substances

How much an individual reacts to alcohol or another drug has implications for the development of substance use and dependence. Nathan and Lipscomb (1979) presented nonalcoholic individuals with a moderate dose of alcohol and reported substantial variability in the reactivity to alcohol. Whereas some individuals exhibited little body sway after a moderate dose of alcohol, others evidenced substantial body sway to the same dose of alcohol after adjustments were made for weight and body mass indicators. Nathan and Lipscomb also reported that individuals who exhibited minimal body sway had, on average, higher levels of tolerance to alcohol after a sustained period of drinking compared to individuals with higher levels of body sway. The researchers hypothesized that this indicator of low reactivity (LR) to alcohol might predict a higher risk for alcohol dependence later in life. In other words, LR might be associated with higher average levels of consumption to get the desired effect (i.e., tolerance), which in turn would be associated with an increased risk for experiencing the adverse effects of heavy consumption (e.g., liver damage, neurological changes).

As it turned out, the prediction by Nathan and Lipscomb (1979) was supported by research conducted almost 20 years later. Schuckit and Smith (1997) conducted a longitudinal study of drinkers with a mixture of risk factors, including LR and a family history (FH) of alcoholism. They found that males who had a positive FH for alcoholism and LR were significantly more likely to develop dependency than males with a positive FH who were highly reactive to alcohol. Taken together, these data suggest that reactivity to alcohol might be a mediator of alcohol dependence, wherein those who can consume large amounts without an excessive reaction become more tolerant, go on to drink even more over time, and eventually develop more significant symptoms of abuse and addiction. Put another way, adverse reactions to substances (e.g., body sway, vomiting, passing out) might actually protect individuals from developing problematic patterns of use by preventing excessive consumption.

Another way to consider how individuals react to substances is based on the pharmacological properties of a substance and the subsequent acute and chronic physiological effects on brain chemistry. For instance, smoking methamphetamine produces an intense and almost instantaneous rush due to an excessive release of dopamine, a neurotransmitter substance in the brain that is associated with pleasure. The acute spike in dopamine levels caused by smoking methamphetamine dwarfs other events or chemicals that induce dopamine release (Rawson, Gonzalez, & Ling, 2006). Moreover, after the initial rush, the euphoria experienced from a single methamphetamine use can last for up to 12 hours. According to Rawson and colleagues, sex causes dopamine levels to rise from 100 to 200 units from baseline, whereas drugs like cocaine lead to an approximate 350-unit increase. However, methamphetamine can lead to a dopamine release that is as much as 12 times (1,200 units) greater than other dopamine-inducing events! Thus, after a user has experienced these intense effects, the ability to experience pleasure through typical methods (e.g., food, sex) becomes greatly impaired, and the desire or craving to use methamphetamine grows even stronger.

These acute effects of methamphetamine are exacerbated by some chronic implications of methamphetamine abuse as well. Even for people who remain abstinent from methamphetamine abuse for several months, the adverse cognitive and neurological effects of methamphetamine use often remain. For example, recovering methamphetamine users experience significant memory, concentration, and motor problems that can persist for several months and even years after the last use (Volkow, Chang, & Wang, 2001).

In summary, biological models have contributed a great deal to our understanding of substance use and dependence. We now know that genetics, physiological reactivity, and the pharmacological effects of the drugs themselves enhance the risk of abuse and addiction. However, other variables, many of which are the focus of inquiry in psychology, play a prominent role in the development of substance use disorders.

Psychological Models

There is a substantial body of literature that elucidates the role of psychological factors in substance use and dependence. For instance, learning theorists have described how associations are developed between a drug, its pharmacological effects, and the proximal environmental stimuli that lead to heightened drug-seeking behavior in animals and humans. Similarly, operant conditioning paradigms help explain why the reinforcing and punishing consequences of drug use can help predict the likelihood of future drug-seeking behavior. In addition, several cognitive models of substance use and addiction help us to understand these phenomena. Our expectations and beliefs about certain substances are often associated with subsequent patterns of use. For example, if we erroneously assume that most people drink excessively, it might

produce less anxiety about our own patterns of use, even if they are excessive. Two broad models of substance use disorders, learning (behavioral) and cognitive paradigms, will be reviewed here.

Learning Models

Pavlovian conditioning experiments over the last century have provided compelling evidence that certain substances such as food, shock, and drugs can elicit powerful behavioral effects such as salivation, avoidance, and craving (see Siegel, 2005, for a summary). When these so-called unconditioned stimuli are paired with the proximal contextual and environmental features, they become associated with the US and can eventually lead to these behavioral effects. The behavioral implications of Pavlovian conditioning can be appetitive or aversive. For example, in taste-aversion learning (TAL) paradigms, eating food with a distinct flavor and then getting sick shortly thereafter often produces a strong aversion to the food or flavor ingested prior to the illness, even if the food itself was not the culprit. In these cases, the association between the CS (distinct flavor) and the nausea (US) ultimately leads to aversive learning and a subsequent avoidance of the flavor or food (CR). In contrast, the appetitive implications of the association between the US (e.g., heroin) and the environmental cues (CS) that typically accompany use (e.g., people, syringes, places, and mood states) can induce drug-seeking behavior (Domjan, 2003). Once these associations are well established for the drug user, encountering these cues even after a period of extended abstinence can lead to high levels of craving, anticipation, and, ultimately, relapse (Marlatt & Witkiewitz, 2005).

Particular learning models have also been applied to important drug-related phenomena. For example, the *opponent-process theory* (Solomon & Corbit, 1973) has been used to explain why tolerance develops in users, or the fact that the primary effects of the drug attenuate after repeated administrations. Based on this theory, the attenuation or habituation to the primary effects (process "a") of a drug are due, in part, to the existence of counterbalancing opponent forces (process "b") that help to reestablish homeostasis (i.e., a state of balance) in the body. Thus, if the initial and primary effects of a heroin injection are euphoria and blissful relaxation, the opponent processes of dysphoria (distress) and agitation emerge to create balance. However, over time, the counterbalancing opponent processes, which are often experienced as extremely unpleasant withdrawal symptoms that the user seeks to ameliorate quickly by using again, typically at increasingly higher doses, can become even more prominent. Under such circumstances, the opponent process theory not only helps to understand tolerance but also provides a plausible explanation for continued drug seeking, mainly the avoidance of aversive withdrawal symptoms.

Cognitive Models

Although alcohol and other drugs have powerful pharmacological properties, our beliefs and expectations about these substances can elicit notable effects as well. For example, our thoughts about drugs are influenced over time through various pathways, both direct and indirect. Family attitudes and parental behavior can be infectious: What we hear and witness over time certainly has the potential to influence thoughts and our own subsequent behavior. Watching our mother reach for a prescription pill bottle each and every time she experiences sadness can set the stage for our own beliefs. Moreover, we are bombarded by images, slogans, and songs in the media that promote the use of psychoactive substances. For example, many of the advertisements for alcoholic beverages depict individuals who drink as happy-go-lucky souls with lots of friends, money, and free time. The scenarios are typically celebratory, not somber, and certainly not illustrative of the potential ill effects of alcohol (e.g., medical problems, vomiting). In these cases, positive perceptions of drug use tend to be associated with relatively higher levels of drug-seeking behavior (Goldman, Brown, Christiansen, & Smith, 1991). On the other hand, our beliefs and expectancies about drug use might be quite negative. Suppose a young child learns to associate the smell of alcohol on her father's breath as a harbinger of his ill temper and an increased chance that he will physically abuse her. It is possible that she will develop a entire set of expectations around alcohol that are negative, which in turn might lead to a prohibitive stance regarding her own alcohol consumption as a teenager or adult (Brown, 1993).

When applied to the phenomenon of college student drinking, some of the cognitive models of substance use are quite compelling. In fact, many college students perceive drinking, including excessive alcohol consumption, as normal. For many college students, drinking is part of a culturally sanctioned ritual or rite of passage, and approximately 80 percent of the college student population report consuming alcoholic beverages (Johnston, O'Malley, Bachman, & Schulenberg, 2004). Although as many as 40 percent of college students report a heavy drinking episode during the previous two-week period (O'Malley & Johnston, 2002), the belief that most of your peers are consuming equal or higher amounts than you (even if they are not) is often associated with relatively higher levels of drinking (Marks & Miller, 1987) and the use of other substances (Wolfson, 2000).

Substance use is a complex phenomenon, and our expectancies when we are in particular settings or contexts can influence not only our behavior but also the perceived physiological responses. Take, for instance, the myth of the magic *elixir* (i.e., cure-all) with respect to alcohol consumption. In essence, some common myths or expectancies associated with alcohol consumption are that drinking makes you feel better, more social, and more attractive (Stacy, Widaman, & Marlatt, 1990). Alan Marlatt and his

colleagues at the University of Washington conducted a series of studies with college students and reported some intriguing findings regarding these myths. In one study, students consumed what they thought was beer with several of their peers in a bar setting. Shortly after the drinking started, they began to talk and laugh more frequently and they reported some early effects of alcohol consumption (e.g., flushed face, tingling, more giggling). However, they were surprised to learn that the beer they were drinking actually did not contain alcohol! Thus, the observed substance use behavior in these college students went well beyond the physiological effects of the alcohol and included environmental (e.g., bar setting), perceptual (e.g., thoughts about alcohol effects), and contextual (e.g., social) variables. Marlatt and his colleagues concluded that our expectancies (e.g., drinking makes me more social) about alcohol consumption can have a significant impact on the consequences (e.g., talking more frequently). The overarching implications of such cognitive phenomena have been conceptualized by Marlatt and others as the *placebo* effects of alcohol (Testa et al., 2006).

In summary, psychological theories have enriched and expanded our understanding of substance use disorders. That is, Pavlovian paradigms, the opponent-process theory, and our beliefs and expectations about various substances influence whether and how substance use patterns develop and evolve over time. Essentially, the reasons for use, abuse, and addiction go well beyond genetics, individual differences in reactivity, or the pharmacological properties of the substances themselves. Indeed, more modern theories of substance use disorders do not rely exclusively on one paradigm or school of thought. Instead, the most advanced and well-articulated theories of substance use disorders are interdisciplinary and multidimensional in nature.

Integrated Models

Integrated models of substance use disorders have been developed that merge the accumulated knowledge across various disciplines to produce a more comprehensive picture of why individuals develop and maintain patterns of drug-seeking behavior. Sometimes referred to as *biopsychosocial* models of substance abuse and dependence, no single theory or perspective predominates. However, the theories that have been heretofore substantiated in the empirical literature (e.g., biological, psychological) play prominent roles in the integrated models. One such integrated model of substance use and dependence can be found within the growing field of *developmental psychopathology* (Cicchetti & Toth, 2006). Developmental psychopathologists posit that substance use disorders emerge from the dynamic interplay of genetics, temperament, physiology, social influences, cultural factors, cognitive variables, coping styles, emotional tendencies, and life events (Mayes & Suchman, 2006). According to this perspective, isolating particular variables of influence is neither desired nor possible. As unwieldy as it seems, only through a careful consideration of the variables together is it possible to appreciate fully the cumulative impact these factors have on the apparent drug-seeking behavior.

In sum, integrated models of substance use disorders honor the diverse findings to date, given that there are multiple pathways in the development of problematic drug-seeking behavior. However, given the diversity of how substance use disorders appear in their clinical form, both epidemiologically and diagnostically, our methods of evaluation and treatment need to be equally diverse but integrated. In the following sections, I describe the epidemiology and the prominent methods of assessment and diagnosis of substance use disorders.

EPIDEMIOLOGY OF SUBSTANCE USE DISORDERS

The behavior of taking psychoactive drugs is quite prevalent, as nearly 11 percent of the U.S. population currently meets *DSM* criteria for either substance abuse or dependence. This percentage is based on a national survey conducted across several types of drugs, including alcohol, nicotine, and illicit substances, such as heroin, marijuana, LSD, and cocaine (Kessler et al., 1994). Based on the same survey, lifetime prevalence of substance use disorders of any kind is approximately 27 percent. Using the *DSM-IV-TR* (APA, 2000) as a guide, substance use disorders cut across 11 drug classes, including (alphabetically): (a) alcohol, (b) amphetamines, (c) caffeine, (d) cannabis, (e) cocaine, (f) hallucinogens, (g) inhalants, (h) nicotine, (i) opioids, (j) phencyclidine (PCP), and (k) sedatives, hypnotics, and anxiolytics. As indicated above, the most commonly used psychoactive substance is alcohol. Lifetime estimates of alcohol dependence in the United States are approximately 13.3 percent, with about 4.4 percent of the population currently meeting criteria for dependence (Grant, 1997). Overall, lifetime reported illicit drug use is approximately 36 percent (SAMHSA, 2003). The most frequently sought-after illicit substance is cannabis (marijuana), with approximately 10 percent of the population reporting having used it at some point during the past year. Lifetime prevalence of cannabis use disorders is almost 5 percent, with approximately 1.2 percent meeting criteria for abuse or dependence in the past year (APA, 2000).

With respect to cocaine abuse and dependence, the lifetime prevalence estimates range between 1 and 2 percent, with approximately 0.2 percent meeting criteria during the previous 12 months. Although many of the other illicit drug categories (i.e., PCP, hallucinogens, opioids, amphetamines, inhalants) have lifetime prevalence estimates that are less than 1 percent, it should be noted that the use of these drugs has well-documented serious effects, including death (Tapert, Tate, & Brown, 2001). Moreover, with respect to methamphetamines in particular (especially the pattern of smoking "meth" made from OTC

cold medications ephedrine and pseudoephedrine), the prevalence rates are generally higher, with over 2 percent meeting criteria for abuse or dependence in the past year in some of the western states (SAMHSA, 2005). Although most prescriptions for sedatives, hypnotics, and anxiolytics (e.g., benzodiazepines such as Xanax and Valium) are taken as directed by a physician, the potential for abuse and dependence on these drugs is high (APA, 2000), and most of these medications are on the list of controlled substances regulated by the U.S. government.

Save for caffeine and nicotine, each of the 11 drug types is capable of being abused. Similarly, a diagnosis of substance dependence can be made for every drug classification with the exception of caffeine (APA, 2000). Some might find these exceptions to the diagnostic guidelines rather curious, given that they are two of the most commonly used substances from the entire list.

In the case of nicotine use, approximately 70 percent of U.S. residents over 12 years of age have smoked, and almost 30 percent described themselves as current smokers (SAMHSA, 2003). Although it is not possible to abuse nicotine according to the *DSM* guidelines (perhaps because it is not considered an intoxicating substance), cigarette smoking is addictive and the leading preventable cause of morbidity and mortality in the United States (Rivara, Ebel, & Garrison, 2004).

Consider, too, the prevalent phenomenon of flocking to coffee houses for a daily dose of caffeine. Caffeine has well-documented psychoactive effects, and it is estimated that U.S. consumers drink approximately 7 billion gallons of coffee per year. It acts as a CNS stimulant, and those who drink it often experience increased blood flow and heart rate, heightened levels of alertness, and an improved ability to focus on the task at hand. Those who consume moderate amounts of caffeinated coffee would probably defend this practice as a necessary part of their daily routine that enhances productivity. It also is widely available and generally inexpensive, and most of those who regularly consume moderate amounts of caffeine report few ill effects. Nonetheless, excessive caffeine consumption can become problematic and according to the *DSM* guidelines, there is a noted caffeine intoxication syndrome (e.g., nervousness, insomnia, muscle twitching, rapid or irregular heartbeat), especially if daily consumption is in excess of 250 mg (2–3 cups) for a person who normally does not consume this much coffee.

METHODS: DIAGNOSING SUBSTANCE USE DISORDERS

The process of diagnosing substance use disorders involves the systematic collection of data across time, incorporating a variety of instruments, observations, interviews, and sources of information. A clinician, such as a psychologist or psychiatrist, would then synthesize the data into a coherent picture and compare the findings to the currently established diagnostic guidelines for substance use disorders. As described previously, the *DSM-IV-TR* (APA, 2000) is the most widely used diagnostic classification system for psychiatric disorders, including substance use disorders. Evaluating individuals for substance use problems can be a time-consuming and demanding process. The process requires considerable training and experience in clinical interviewing, epidemiology, test administration, diagnostic formulation, case conceptualization, data management, and general clinical acumen. All graduate programs in clinical psychology that are accredited by the American Psychological Association provide the necessary background training in psychopathology, assessment, and clinical skills. However, diagnosing problematic drug-seeking behavior also requires a specialized knowledge of the substance abuse disorders in particular, including etiology, diagnosis, and treatment.

Remember, though, that simply using substances is not a sufficient criterion for diagnosing substance use disorders. Clinicians must be skilled at differentiating whether the using behavior is associated with impairment versus a normal range of use without evidence of harmful consequences. For example, it is conceivable and even common that after a long stressful week at work, a 25-year-old professional woman drinks a few martinis on Friday evening to relax a bit. When her husband returns home, he joins her in having even more drinks. She ends up drinking four more martinis, begins to slur her words, and eventually passes out for the night. She opts not to drink the rest of the weekend and resumes her work and her busy schedule on Monday without consequence. It might also seem reasonable for a 21-year-old male college student to drink a beer or two before taking a cab to the local pub. Once there, he enjoys being in the company of his friends, has interesting conversations, and has a few more beers. His prior drinking history is similar, with few episodes of excessive drinking episodes. However, on this particular night, the drinking continues and he becomes quite intoxicated. He returns home after taking a taxi and stumbles up to his bedroom to "sleep it off." The next morning, he wakes up with a serious hangover, feels sick to his stomach, and resolves not to do that again anytime soon. After a brief period of recovery, he returns to his academic work and does not miss any classes as a result of his drunken episode. In fact, he holds true to his promise to himself and no additional excessive drinking episodes occur for several months.

In these instances, there seems to be a defensible rationale for seeking out various substances to relax or to enliven conversation and the use does not seem to be part of a larger pattern of heavy alcohol consumption. Most astute clinicians would not make a diagnosis of alcohol abuse under these circumstances. In both cases, the users experience the intoxicating effects of alcohol without extended adverse consequences. In addition, although both individuals experienced some acute adverse effects of excessive alcohol use, they seem to have learned something

from the negative aspects of their experiences and continue living without further consequence. Moreover, the college student resolves not to get drunk again anytime soon and follows through with his promise.

However, there are cases when it is apparent that the substance is being misused. That is, there is evidence of recurrent drug-seeking behavior even when adverse consequences occur, such as medical or physical problems, legal difficulties (including the use of illicit drugs), family dysfunction, and problems with work or school. In such instances, a diagnosis of substance abuse is indicated, including a specification of the drug or drugs that are being used compulsively. A diagnosis of substance dependence is warranted when the collected data suggest a similar pattern of repeated use along with the associated negative consequences and evidence of tolerance and withdrawal. A diagnosis of substance dependence requires that a large portion of a person's time be spent acquiring, using, and recovering from the effects of the substance. In severe cases, the compulsive drug-seeking behavior consumes the majority of a drug user's life. It is also common for substance-dependent individuals to have several unsuccessful attempts at either reducing or stopping their use, and they often have some appreciation that their drug-seeking behavior is really wreaking havoc in their life, yet they are often reluctant to seek help voluntarily for their addiction. In cases where the diagnosis of either substance abuse or dependence is made, expeditious treatment is necessary to reduce the adverse effects and to restore the individual to a more healthful and adaptive level of functioning. Prominent models of substance abuse treatment will be reviewed in the following section.

APPLICATIONS: TREATING SUBSTANCE USE DISORDERS

Just as etiological models of substance use disorders have been developed and tested, so too have various treatments for these conditions. Many of the current treatment paradigms are based on empirically tested principles, such as behavioral and cognitive models. Other treatments are based on biological principles, such as pharmacological interventions that are designed to either block the intoxicating effects of the substance or alleviate the potentially painful symptoms of withdrawal. In addition, there are several well-established self-help paradigms, the most notable of which is Alcoholics Anonymous (AA). Unfortunately, early efforts to treat substance use disorders were not very successful, in part because the treatment models were not always based on sound scientific principles or there were few attempts to integrate the effective ingredients from several perspectives (Hunt, Barnett, & Branch, 1971). As discussed earlier in the chapter, the disease model of alcoholism and the subsequent controversy between the abstinence versus controlled drinking perspectives illustrates how passionate some people can become about treating substance use disorders within a certain paradigm. However, the controversy also highlights the potential fallacy of a clinician asserting that "one size fits all" when it comes to the treatment of addiction, because some clients respond favorably to approaches that are not strictly based on abstinence principles. One such approach is called the *harm reduction model* (Marlatt, 1998; Marlatt & Witkiewitz, 2002), which views substance abuse and dependence as a public health problem with the goal of reducing the negative consequences associated with use instead of strictly insisting on immediate abstinence as the objective.

Most current models of substance abuse treatment are integrative and honor the empirical findings in the literature. For example, given the evidence that substance users develop associations between the drug and the proximal environmental cues that accompany use, behavioral interventions have been designed whereby users are exposed to the cues that precipitate strong cravings and they are guided in developing alternative, nonusing strategies to cope with the cues and the subsequent urges. Moreover, the client can process his or her fears about relapse, risky mood states, and other events that often signal use. To illustrate, a client with a history of cocaine use and dependence might be exposed to cocaine using paraphernalia such as a mirror, a rolled-up dollar bill, and a razor blade, but without the actual cocaine. The exposures are first conducted in a controlled environment, typically with the support of a therapist or counselor, who directly teaches alternative coping strategies in the presence of cues and cravings, such as relaxation, distraction, or exercise. Once the client experiences success in coping with the cues and the cravings in a controlled setting, he or she is gradually introduced to the full range of cues in a real setting. The use of similar behavioral techniques is also evident in community reinforcement approach (CRA). In CRA, in addition to individualized behavioral treatment, other collateral strategies are incorporated, including recreational and social opportunities as well as self-help groups that foster and reinforce nonusing lifestyles (Smith & Meyers, 1995). In essence, CRA combines both substance use-specific interventions along with strategies that are designed to fit within the client's larger contextual circumstances. Thus far, empirical support for CRA approaches is promising (Tapert et al., 2001).

Cognitive techniques play a prominent role in several interventions as well. For instance, in covert sensitization, the therapist encourages the client to imagine his or her drug of choice along with some unpleasant substance mixed in (e.g., glass of wine with feces). The underlying assumption of covert sensitization is that the mental association of the drug of choice with an unpleasant feature will compete with "euphoric memories" of intoxication associated with the drug that promote further drug seeking. Other cognitive interventions are more basic, involving the development of better problem-solving and social skills techniques to cope with everyday stressors. Many relapse prevention programs incorporate cognitive interventions

where clients are encouraged to anticipate high-risk situations and modify their assumptions about how to experience pleasure without the use of psychoactive substances (Marlatt & Witkiewitz, 2005).

Several treatments use elements from multiple paradigms simultaneously, combining medication, cognitive, self-help, and behavioral interventions. For example, the treatment of heroin addiction often includes the use of a chemically similar substitute called *methadone*. Methadone is a long-acting opiate producing cross-tolerance to other opiates (like heroin), which in turn reduce the psychoactive effects of the shorter-acting heroin. Moreover, using methadone appears to lessen the withdrawal symptoms associated with heroin, and therefore the cravings for the drug. Methadone maintenance is often used in tandem with other supportive interventions, including self-help, community reinforcement, and behavioral techniques. In summary, several promising approaches have emerged to treat substance use disorders. Most of the interventions are not based on a single paradigm, but instead include elements from several approaches.

SUMMARY

Drug-seeking behavior is a prevalent and complex phenomenon that has existed around the globe for centuries. The reasons for drug use are as diverse as the number of substances used. In perhaps the majority of cases, drug-seeking behavior does not lead to serious or chronic adverse effects and can be conceptualized as a normative, culturally accepted practice. However, in a substantial number of instances, substance use does become problematic and can lead to extended and/or accelerated patterns of abuse and dependence. Several theories have been articulated to help explain why some individuals develop problematic patterns of substance use. Many of the same etiological theories of substance use disorders have been used to develop and test effective treatments for abusive and compulsive drug-seeking behavior.

Future directions in the treatment of addictive disorders are promising and will pave the way for many young scholars and clinicians who are interested in these conditions. For example, in one of the most ambitious research efforts to date, Project MATCH (Matching Alcoholism Treatments to Client Heterogeneity; Miller & Longabaugh, 2003) studied the effects of three prominently used methods of treatment for alcohol dependency—cognitive-behavioral therapy, motivational enhancement therapy, and 12-step facilitation (based on principles of Alcoholics Anonymous)— to determine whether particular treatments worked best for particular clients. Overall, the results from Project MATCH suggested that all three approaches were efficacious in reducing drinking and the associated problems with alcohol abuse and dependence (Project MATCH Research Group, 1997). Moreover, the project is an exemplar for how scholars both within and outside of psychology can collaborate to understand and ultimately address a major public health issue.

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