CHAPTER 1

Models of Addiction and Change

A theoretical perspective provides a useful heuristic to advance our knowledge of any phenomenon and our ability to influence its existence, development, and growth.

Addictions have plagued society throughout history, as is evident from the Greco-Roman philosophers' call for moderation and condemnation of bacchanalian excesses to our 21st-century preoccupation with alcohol, drugs, food, sex, and gambling. Explanations for addiction often have consisted of blaming individuals for their excessive engagement in these behaviors. Scientific theories and models for explaining and understanding addictions have existed only for the past 100 years. Although our explanations have become more sophisticated and recent advances in neuroscience have enabled us to link addictions and brain activity, our understanding of addiction is far from complete.

WHAT IS AN ADDICTION?

Traditionally, the term *addiction* has been used to identify self-destructive behaviors that include a pharmacological component. The most stringent application would limit the term addiction and the companion label of *addict* to individuals with a *physiological* dependence on one or more *illegal* drugs. This definition usually includes a strong physiological craving, withdrawal symptoms, and the need for more of the drug to

get the same effect (American Psychiatric Association, 1980, 2013). In the strictest application of this definition, addiction would have to meet the definition of physiological dependence as in the diagnostic criteria of the DSM-III (American Psychiatric Association, 1980). However, within the last 30 years the scope of the term has expanded to include any substance use or reinforcing behavior that has an appetitive nature, has a compulsive and repetitive quality, is self-destructive, and is experienced as difficult to modify or stop (Orford, 1985). Expanded use of the term addiction has also included problematic relationships, excessive work behaviors, and even what some are calling *positive addictions* (e.g., exercise, meditation). Treatment professionals, addicts, and the public are confused by this shifting scope of meaning, and among scientists and practitioners in the field there is real concern about the continuing expansion of the term's application. If what is labeled "addiction" becomes too broad, the word will become meaningless. However, labeling a broader range of behaviors as addictions would be justified if they display common features that increase our ability to understand addictive problems and expand society's capacity to intervene.

The definition of addiction used in this volume is purposefully broad and can include an array of behaviors without making every human problem or pathology an addiction. In this book, addictions are understood as learned habits that, once established, become difficult to extinguish even in the face of dramatic and, at times, numerous negative consequences. The critical dimensions for an addiction are (1) the development of a solidly established, problematic pattern of an appetitive—that is, pleasurable and reinforcing—behavior; (2) the presence of physiological and psychological components of the behavior pattern that create dependence; and (3) the interaction of these components in the individual's life that make the behavior very important and resistant to change. Each of these aspects is critical for identifying an addiction. Addictive behavior patterns are repeated and become predictable in their regularity and excess. Powerful reinforcing effects motivate continued use, although these effects may shift from seeking pleasure to avoidance of negative consequences (Volkow, Koob, & McClellan, 2016). Dependence is the second necessary and critical dimension to define addiction. The term *dependence* indicates that there is a reliance on the behavior or its effects and that the pattern of behavior involves poor self-regulation, continues despite negative feedback, and often appears to be out of control. Moreover, reinforcers for engaging in this behavior often become prepotent in the life of the individual and an integral part of her or his way of life and coping. Reinforcers are both physiological (with a strong neurobiological component) and psychological (with a strong coping component).

They combine to create a powerful reward system that clouds awareness of problematic consequences related to the behavior and makes change difficult and, at times, seemingly impossible. In fact, failure to change, despite the outward appearance that change would be both possible and in the best interest of the individual, is considered a cardinal characteristic in defining addictions. In my view, change is the antithesis of addiction, similar to freedom being the opposite of enslavement. The polarities of change and addiction, then, can be viewed as central themes for understanding how people become addicted and how they can free themselves from an addiction.

This definition of addiction is broad but not so broad as to become meaningless. Most psychological and psychiatric problems are not appetitive in nature—that is, activities that are engaged in because of their inherent pleasurable and reinforcing effects. Moreover, most disorders do not require engaging in repetitive, intentional behaviors to become established as a problem. For example, there is nothing inherently pleasurable in a psychotic break or a depressive episode, nor do these chronic psychiatric conditions require that the individual engage in purposeful activities in order to develop these disorders. Addiction should not be used to describe most psychopathology. However, the scope of appetitive behaviors that become destructive and difficult to stop can include problematic behavior patterns related to eating, sex, drugs, and money. Habits most clearly associated with addiction include tobacco dependence, alcohol misuse and dependence, legal and illegal substance and prescription medication use disorders, a range of eating disorders (including overeating and bulimia), as well as gambling disorders (National Academy of Sciences, 1999). The clear similarities across these behaviors, which in their excessive forms are labeled addictions, include the following elements:

1. They represent habitual patterns of intentional, appetitive behaviors.
2. They can become excessive and produce serious consequences.
3. These problematic behavior patterns are stable over time.
4. They become important and salient in the life of the individual.
5. There are interrelated psychological and physiological components underlying the behavior.
6. Finally, in every case, an individual who becomes addicted to these behaviors has difficulty stopping or modifying them.

These elements represent essential components that underlie the criteria used to diagnose an addiction (American Psychiatric Association,

1994, 2013). However, categories of abuse and dependence have been abandoned in the latest version of the Diagnostic and Statistical Manual and replaced by mild, moderate, and severe use disorders (American Psychiatric Association, 2013).

The central, defining elements of addictive behaviors involve the seemingly compulsive and out-of-control nature of current behavior patterns and the level of difficulty encountered in changing them. However, most traditional models for understanding addictions have concentrated on the origins of these behaviors or on treatment options, rather than on how individuals go about changing them (McCrady & Epstein, 2013; U.S. Department of Health and Human Services, 1980). The thinking behind the emphasis on etiology reflects a belief that the best way to understand and, ultimately, to change addictions is to understand why and how they began. In most disease models, understanding etiology is critical because it often uncovers the source of the problem—a virus or a contaminated environment and a mode of transmission—which, when attacked or resolved, leads to the eradication of the problem. However, when it comes to addictions, single-cause etiological models have been woefully inadequate to explain either adoption or cessation of addictive behaviors (Donovan & Marlatt, 1988; Glantz & Pickens, 1992; Kovac, 2013; Smith et al., 2015). On the other hand, the focus on treatments and treatment programs emphasizes provider strategies and ignores the self-change efforts and process of change of the individual (DiClemente, 2006). Often the search ends up being for the best treatment for this disorder or for the *typical* individual with this addictive behavior rather than an understanding of the common elements underlying initiation or recovery.

There was a wonderful poster produced by the National Institute on Alcohol Abuse and Alcoholism in the late 1970s. The title read "The Typical Alcoholic American." Pictured were more than 20 individuals who differed by age, race, occupation, and socioeconomic status and included an American Indian, doctor, housewife, elderly female, construction worker, and many others. Clearly, the point was that there is no typical alcoholic and that stereotypes need to be discarded to adequately address alcohol problems. Understanding addiction requires complex models to explain the diversity as well as the similarities among individuals who exhibit the addictive behaviors. If complexity were required for understanding any single addictive behavior, like alcohol, it would be even more important when examining multiple addictive behaviors, wherein heterogeneity among people and types of behaviors will be even greater. Any search for similarities and commonalties must account for the diversity and heterogeneity of the individuals who become addicted and respect the distinct and specific nature of each addictive behavior.

TRADITIONAL MODELS FOR UNDERSTANDING ADDICTION

Many different theories and models of addiction have been proposed. Several broad categories can be used to summarize these models. The most prominent explanatory models include (1) social/environment models, (2) genetic/physiological models, (3) personality/intrapsychic models, (4) coping/social learning models, (5) conditioning/reinforcement behavioral models, (6) compulsive/excessive behavior models, and (7) an integrative biopsychosocial model. Each of the models proposes a way of understanding addiction or a specific addictive behavior that focuses primarily on how addictions develop. Then, based on this etiology, the models propose suggestions for prevention and cessation as well as for intervention and treatment (Leonard & Blane, 1999; McCrady & Epstein 2013; U.S. Department of Health and Human Services, 1980; Walter & Rotgers, 2012). The following review of these explanations, although brief and cursory in comparison to the more extensive discussions offered in the previously cited books and monographs, will summarize strengths and weaknesses of each type of model. Supportive facts and interesting anomalies highlighted in the review will make the case for a more integrative model based on the process of human intentional behavior change.

Social/Environment Models

The social/environment perspective emphasizes the role of societal influences, peer pressure, social policies, availability, and family systems as mechanisms responsible for developing and maintaining addictions. Certain types of drug use and individual addictive behaviors occur more frequently in some subgroups. This has encouraged researchers to examine subcultures related to drug use (Carlson, 2006) and to explore the importance of environmental-contextual influences in the search for risk and protective factors (Clayton, 1992). Patterns related to specific drug-use behavior support interesting, well-defined sociocultural connections (Connors & Tarbox, 1985; Stone, Becker, Huber, & Catalano, 2012).

Social influence and support are often evident in the social context for use. Cocaine use has spawned the "crackhouse" where cocaine addicts gather; heroin addicts have created their "shooting galleries"; inhalant abuse often is concentrated among Hispanic youth (National Survey on Drug Abuse, 2010). These phenomena, along with the fact that drug users and abusers often have more family and friends who use drugs, make a clear case for the importance of social context in the acquisition of addictive behaviors (Guerrini, Quadri, & Thomson, 2014; Jessor & Jessor, 1980). In addition, conformity to some social norms

as well as deviance from others are offered by some investigators as explanations for addictions (Kaplan & Johnson, 1992). Illegal drug use, abuse, and dependence are viewed as deviant behaviors in many sociological models (Robins, 1974, 1979). Deviance then becomes an underlying cause, while a particular addictive behavior may reflect a response to the social context of peers (Lukoff, 1980). Research with Vietnam veterans demonstrated that higher preservice deviant behavior predicted initiation of heroin use (Robins, Helzer, & Davis, 1975) and is consistent with data that show a history of delinquency prior to onset of heroin use among heroin-dependent individuals (Glantz & Pickens, 1992). However, the enormous increase in marijuana use in the 1960s demonstrated that as use spreads across the population it becomes harder and harder to use deviance as an explanation for use or dependence (Robins, 1980). Moreover, social norms and deviance explanations are more difficult to use as the sole explanation for alcohol dependence, nicotine addiction, gambling, and eating disorders. Social control depends on the strength of the social bonds and interacts with self-control (Hirschi, 2004; Wiatrowski, Griswold, & Roberts, 1981).

Additional support for the social/environment perspective comes from data indicating that availability and social policies, such as restrictions in use and taxation, influence use and abuse of certain substances. Policies restricting cigarette smoking and advertising have made important contributions to the declining rate of cigarette consumption in the United States (U.S. Department of Health and Human Services, 2014). Changing the legal age for consuming alcoholic beverages, as well as pricing and taxation, have influenced use and abuse of alcohol (Connors & Tarbox, 1985; Wagenaar, Salois, & Komro, 2009). Macroenvironmental influences also play an important role in the initiation and cessation of other addictions (Baldwin, Stogner, & Lee Miller, 2014; Connors & Tarbox, 1985; Engels, Hermans, van Baaren, Hollenstein, & Bot, 2009; Institute of Medicine, 1990). These explanations are certainly more applicable when the substances and behaviors are legal than when they are already considered illegal and banned in the society.

Some proponents of the social/environment models have concentrated on the more intimate environment of family influences as a central factor contributing to the onset of addictive behaviors. Family influences support both a genetic, nature-based pathway of influence and a nurture-based path focused on family interaction or family system (Hasin, Hatzenbuehler & Waxman, 2006; McCrady, Owens & Brovko, 2013; Sher, 1993). Advocates of family explanations point to problematic parental modeling of adult roles, which can include difficulties with relationships, conflicted and broken marriages, child maltreatment, low levels of parental monitoring, and either discouragement or excessive

use of alcohol and other drugs. These can be important influences on the child's experimenting with and continuing an addictive behavior (Brook, Brook, Zhang & Cohen, 2009; Chassin, Curran, Hussong, & Colder, 1996; Jessor & Jessor, 1977; Kandel & Davies, 1992; McGue & Irons, 2013; Stanton, 1980). Steinglass, Bennett, Wolin, and Reiss (1987) have proposed a more indirect route of transmission of alcohol problems through the child's adoption or rejection of family rituals and traditions. Stanton (Stanton, Todd, & Associates, 1982) and others (McCrady et al., 2013) have indicated that family system interactions can be responsible for one or more family members engaging in addictive behaviors because of the roles that are adopted to keep the system functioning. The idea is that family homeostasis acts as a regulatory structure in which the deviate addictive behavior plays an important role in individual and family functioning. This explanation has been used with alcohol problems, and particularly in discussions about eating disorders and anorexia (Jewell, Blessit, Stewart, Simic, & Eisler, 2016; Minuchin, 1974; Selvini-Palazzoli, 1974). Proponents of a family influence model differ dramatically on the amount of influence attributable to genetic factors as opposed to psychosocial factors (Cadoret, 1992; McGue & Irons, 2013).

The social/environment perspective has many advocates. Proponents have presented substantial evidence for the role of social and environmental factors in the adoption of various addictive behaviors. However, as Robins (1980) points out, a natural history of drug abuse can only describe the current historical perspective. His description was of the 1970s drug use era. Drug use and abuse, including alcohol consumption, were different in the 1920s and appear to have substantially changed again by the first decade of the 21st century. Marijuana use today is viewed much differently than in the 1990s, with attitudes clearly influenced by legalization and medical use of marijuana. Social influences and trends shift, as do the popularity of different types of addictive behaviors. Shifting social trends in addictions argue for an important role for social and environmental influences, while at the same time clearly offering evidence against viewing the social/environment perspective as a fixed explanation for all addiction at all historical points in time. Social and peer influences are also complicated and include both peer selection and peer influences. These effects seem age dependent: selection of deviant peers may be more influential in early adolescence and peer socialization effects more influential in late adolescence and early adulthood (Burk, van der Vorst, Kerr, & Stattin, 2012).

It is also clear that even when there are substantial trends or social influences facilitating the development or cessation of a certain behavior, many individuals do not follow those trends. Of the first two inhalant

drug abusers that I saw in treatment, one was a southern White male in his 20s, the other a Hispanic teen. The latter fit the stereotype of an inhalant abuser in Texas, the former did not. Even when a new substance is hyped by peers (bath salts, e-cigarettes, salvia), the clear majority of youth do not experiment or use. Social and environmental influences clearly contribute to both the acquisition and the cessation of addictions at a population level but often fail to explain in any comprehensive manner individual initiation or cessation.

Genetic/Physiological Models

The most convincing information concerning the role of genetics in addictions is available in alcohol use disorders. Early family studies indicated increasing risk ratios for individuals as the number of alcoholic relatives rises and as the number and severity of familial alcohol problems rise (Schuckit, 1980, 1995; Schuckit, Goodwin, & Winokur, 1972). Twin studies as well as in-depth assessments of children of alcoholics continue to support the importance of genetics as a contributing factor to alcoholism (Hasin et al., 2006; McGue & Irons, 2013). The role of genetics for other drugs of abuse varies by type of drug and whether one is focusing on initiation or progression as well as the age of the adolescent (McGue & Irons, 2013). Most scientists acknowledge a genetic influence on susceptibility to substance abuse (Hasin et al., 2006). However, the search is not for a single "alcoholism gene"; rather, the consensus is that the heritable component of addictive behavior will be polygenetic and complex (Begleiter & Porjesz, 1999; Gordis, 2000; McGue & Irons, 2013). Moreover, there seem to be many generic genetic risk factors that include inherited risk for externalizing and internalizing disorders and a common factor called behavioral disinhibition (Hicks, Kreuger, Iacono, McGue, & Patrick, 2004; Iacono, Malone, & McGue, 2008; Kendler, Myers, & Prescott, 2007; Kreuger et al., 2002; Tsuang et al., 1998).

For a long time, physical dependence and addiction were understood as synonymous. Traditional markers to define drug dependence were both tolerance—the need for more of a substance to achieve the same effect—and a clear withdrawal syndrome, which included physical reactions like nausea and a craving for the substance. The 1994 revision of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) of the American Psychiatric Association changed the definitions of drug abuse and dependence so that this distinction between abuse and dependence based solely on physiological tolerance was practically eliminated. The latest revision (DSM-5), in 2013, has eliminated the terms and the distinction between abuse and dependence, opting for a more dimensional model for understanding addiction that focuses on levels

of disordered use that can be mild, moderate, or severe based on the number of symptoms present. These symptoms include a number of indicators of neuroadaptation, like craving, withdrawal, and tolerance, as well as a number that reflect impaired self-regulation, which have both behavior and brain components. In summary, there have been enormous advances in our understanding of the neurobiology of alcohol and drug addiction (Koob & Le Moal, 2001; Koob & Volkow, 2010) that look to brain chemistry and behavioral responding as critical indicators. Even for addictive behaviors that do not involve a substance such as gambling, it appears that the "rush" or "high" produced by the behavior is an important element (National Academy of Sciences, 1999). This physiological reaction and its potential for creating and reinforcing problematic patterns of behavior is often used as a reason for the inclusion of gambling under the rubric of addiction (American Psychiatric Association, 2013; Reuter et al., 2005). However, physiological pathways are complicated and certainly not uniform in mechanism of action or type of involvement across addictive behaviors.

There are also some interesting anomalies that both support and challenge the genetic/physiological explanations of addictions. In the 1970s, researchers became quite pessimistic about the prospect of getting smokers to quit and began to focus on developing a safer cigarette, one that did not contain nicotine. They attempted to create cigarettes using cabbage leaves and other organic materials. However, no one would smoke cigarettes that did not have the active nicotine effect! Similarly, methadone-maintained patients often lament the fact that it does not produce the "heroin high" that got them addicted, although it does mimic the physiological effects of a narcotic and helps them avoid withdrawal. Clearly, physiological reactions to an active drug play an important role in creating addictions. However, research studies also have produced visible alcohol or drug effects using placebos that contain no active substance. These studies appear to contradict a completely dominant role for physiology and argue for the importance of expectations or social context in addition to the actual physical effect (Collins, Lapp, Emmons, & Isaac, 1990; Fromme & Dunn, 1992; Leigh & Stacy, 2004; Schulenberg, Wadsworth, O'Malley, Bachman, & Johnston, 1996; Southwick, Steele, Marlatt, & Lindell, 1981). In bar laboratory settings, many investigators have shown that drinkers will act as if they are intoxicated even when given nonalcoholic drinks (Collins, Parks, & Marlatt, 1985; Goldman, Del Boca, & Darkes, 1999; Larson, Overbeek, Granic, & Engels, 2012).

The physiological effects of tolerance and withdrawal as well as science and society's movement away from an explanation of addiction as morally reprehensible behavior have led to addictions being understood

within a medical model. This perspective has also been promoted in the materials describing the 12 steps and 12 traditions of AA that talk about the disease of alcoholism, which they liken to a chronic allergic reaction (Alcoholics Anonymous, 1952). Others believe alcoholism is a disease that is not completely physiologically based (Miller & Kurtz, 1994; Sheehan & Owen, 1999). The disease model has been instrumental in shifting society's view of alcohol dependence from one of moral deviance and sinful behavior to one that promotes understanding and treatment. However, there are many criticisms of this use of a disease model for understanding alcoholism (Donovan & Marlatt, 1988; Lewis, 2015; Miller & Rollnick, 1991). It is also interesting to note that proponents of the disease model for alcoholism will not always use the same explanation for drugs of abuse and have some difficulty when the concept is extended to behaviors like gambling. Even though brain regions, neurochemistry, and physiology are clearly implicated in the initiation and maintenance of addictive behaviors, these behaviors and the end state of addiction have multiple determinants. It is probably best to consider addiction as a chronic condition rather than a physical disease. However, the term "brain disease" has become a common way to describe addictions because of the neurobiological component (Volkow et al., 2016).

For all addictive behaviors, there appears to be an important role for physiological and brain mechanisms as well as genetic factors in the behavior's initiation, problematic long-term use, and disordered use. However, even among researchers who focus on genetics and the brain, there are many questions and concerns about assigning sole causality or even primacy to genetic/physiological factors for all substances and for all phases of becoming addicted (McGue & Irons, 2013; Newlin, Miles, van den Bree, Gupman, & Pickens, 2000). Because so many different individuals can become addicted to so many different types of substances or behaviors, biological or genetic differences do not account for all the cultural, situational, and intrapersonal differences among addicted individuals and addictive behaviors (Hasin et al., 2006). There seems to be a clear contribution of environment in all the heritability models, such that gene–environment interactions are the best way to consider the influence of genetic factors across the lifespan (McGue & Irons, 2013).

Personality/Intrapsychic Models

Addictive behaviors have often been conceptualized as a symptom of more historical, intrapsychic conflicts, often labeled disorders of personality. Proponents of this perspective point to the frequent correspondence between drug abuse and a diagnosis of antisocial personality

disorder or its predecessor, conduct disorder and juvenile delinquency, as evidence of drugs being a symptom of a larger psychological problem (Robins, 1980; Weiss, 1992). The search for the alcoholic or prealcoholic personality has persisted for years, with mixed and unconvincing results (Cox, 1985, 1987; Nathan, 1988; Sutker & Allain, 1988). Some prealcoholic personality characteristics seem to be related to later alcohol dependence: impulsivity, nonconformity, antisocial behavior, independence, and hyperactivity (Cox, 1985; McGue & Irons, 2013; Stone et al., 2012). However, these relationships may be true more for male than female alcoholics, and are not always present in every male alcoholic. In the related eating disorder arena, the literature on anorexia nervosa often describes a typical adolescent female with low self-esteem and an intense desire for control and autonomy (Cassin & von Ranson, 2005; Wonderlich, 1995). Psychoanalytic perspectives have characterized both alcoholics and persons with eating disorders as individuals who have had conflicts at the oral stage of psychosexual development and were fixated at this stage (Freud, 1949; Khantzian, 1980; Leeds & Morgenstern, 1995). Even the perspective of Alcoholics Anonymous describes a personality dimension when it calls alcoholism the result of a defect in character and a deficit of will (Alcoholics Anonymous, 1952; DiClemente, 1993a).

Many theorists explicitly state or imply that some internal mechanism or conflict drives what can be considered a "proneness" to addiction (Smart, 1980). Sometimes these conflicts can be the result of environmental problems, but most often they are viewed as internally derived and leading to dysphoria or a sense of meaninglessness (Greaves, 1980). Psychological dimensions, which can be conceptualized as temperaments or traits, have also been employed as predictors of addiction. Antisocial traits, low self-esteem, alienation, religiosity, high novelty seeking, activity level, and emotionality have been identified as precursors or predictors of later addiction (Kaplan & Johnson, 1992; Siegel, 2015; Stone et al., 2012; Tarter, 1988; Wills, McNamara, Vaccaro, & Hirky, 1996). Risk taking and problematic decision making are often related to addiction vulnerability as well as to pathological gambling and excessive Internet use (Balogh, Mayer, & Potenza, 2013). These traits are thought to produce the internal setting in the individual where availability or peer pressure can induce not only experimentation and use but also abuse and dependence. Many of these traits are related to self-regulation deficits and brain development, so adolescence can create a perfect storm for initiation of addictive behaviors (O'Connor & Colder, 2015).

Although it would seem logical to assume a role for internal personality dynamics in the addiction process, the evidence to date does not support the existence of an addictive personality that predictably and

reliably will result in a severe use disorder for addictive behaviors. There is a subgroup of "addicts" diagnosed with multiple drugs and other addictions who demonstrate a tendency to engage in multiple addictive behaviors (gambling, drug use, and alcohol misuse). This group would seem to be a prime location for discovering personality dynamics. Nonetheless, there are individuals who share traits or profiles with members of this group, but do not engage in any of these behaviors. As with the sociological and genetic factors described previously, personality factors appear to contribute to the development or establishment of an addictive behavior problem, but the part of addiction that personality factors or deep-seated intrapersonal conflicts can account for appears small (Nathan, 1988).

Coping/Social Learning Models

Addictions often are considered the result of poor or inadequate coping mechanisms. Unable to cope with life stresses, addicts turn to their addiction for escape or comfort. From this perspective, individuals use substances as alternative coping mechanisms and rely on their addictions to manage situations, particularly those that engender feelings of frustration, anger, anxiety, or depression (Wills, Pokhrel, Morehouse, & Fenster, 2011; Wills & Shiffman, 1985). Appraisal-focused coping, problem-focused coping, and emotion-focused coping are considered important domains of coping responses (Lazarus & Folkman, 1985; Moos, Finney, & Cronkite, 1990). One's ability to cope with stress—in particular, with anger, frustration, boredom, anxiety, and depression—has been identified as a critical deficit area in many theories or models of addiction (Pandina, Johnson, & Labouvie, 1992). Emotion-focused coping is considered an important dimension. Alcohol, for example, has been viewed as addictive because of its tension reduction (Cappell & Greeley, 1987) or stress response dampening (Sher, 1987) effects. Because alcohol's effects on stress and tension are quicker and often more effective in dealing with a stressful event than other, natural coping responses, alcohol becomes the preferred, and possibly the only, coping mechanism (Koob & Le Moal, 2000).

The social learning perspective emphasizes social cognition and not simply coping. Bandura's social cognitive theory tends to focus more on cognitive expectancies, vicarious learning, and self-regulation as explanatory mechanisms for addictions (Bandura, 1986; DiClemente, Fairhurst, & Piotrowski, 1995; Maisto, Carey, & Bradizza, 1999). There is a growing literature focused on how expectations about the effects of a specific substance or addictive behavior are related to use, abuse, or excessive engagement. Alcohol expectancies have been found to predict

initiation of use and progression to problematic use (Brown, 1985; Connors, Maisto, & Dermen, 1992; Goldman, 1999; Wood, Read, Palfai, & Stevenson, 2001). For example, individuals who believe that alcohol will make them more attractive, less inhibited, better lovers, and more fun to be around would be more prone to use alcohol and to get in trouble with alcohol, particularly in social settings (Goldman et al., 1999).

The social learning perspective also emphasizes the role of peers and significant others as models. Advertisers who use sports figures to promote a product clearly employ social influence principles. Alcohol and cigarette promotions in sports arenas offer more subtle examples of the power of modeling as an influence on substance use. The influence of expectancies is not limited to substances of abuse. The popularity of lotteries and the well-promoted jackpot for a lucky individual as well as our societal devotion to being thin play a clear role in promotion of gambling and eating disorders, respectively.

Coping and social learning perspectives have become quite popular among addiction researchers and clinicians. However, many successful businessmen and athletes who appear to have good general coping skills, or at least skills good enough to become successful in a competitive environment, get ensnared by addictive behaviors. Generalized poor coping cannot be the only reason individuals become addicted. That seems particularly true for people who engage in the behavior because of the positive enjoyment effects and not simply the relief of problematic emotions (Orford, 1985). However, even if coping defects are not the critical reason for developing addictive behaviors, one important consequence of addiction is a narrowing of the addicted individual's coping repertoire. Thus, coping responses may be even more important as a way of remediating the consequences of an addiction than as a contributor to its development (Kuntsche, Knibbe, Engels, & Gmel, 2010; Shiffman & Wills, 1985).

Conditioning/Reinforcement Models

There is a substantial body of research demonstrating the reinforcing properties of each substance of abuse (Barrett, 1985). Animal and human studies show that many of the same principles that define conventional reinforcers appear to operate in the ingestion of psychoactive drugs (O'Brien, Childress, McClellan, & Ehrman, 1992) and are clearly related to neurobiology (Volkow et al., 2016). Animals' responses to obtain psychoactive drugs seem to operate according to schedules of reinforcement (Barrett, 1985). Reinforcement theory seems an appropriate explanation for subtle physiological effects of substances as well as for the gross motor drug-seeking elements of addictive behaviors.

The classic example of the power of reinforcement has been the slot machine; its variable-ratio reinforcement schedule creates a stable, hard-to-extinguish pattern of behavior. Reinforcement models have been used to understand the initiation of addictive behaviors as well as their stability, which makes them difficult to modify. Reinforcement models focus on the direct effects of the addictive behavior, such as tolerance, withdrawal, and other physiological responses/rewards, as well as the more indirect effects described in opponent process theory (Barrett, 1985; Koob & Le Moal, 2008; Solomon & Corbit, 1974). This latter theory posits that after the initial pleasurable effect initiates use, the appearance of an effect (dysphoria and withdrawal) that is opposite to the more pleasurable effect drives the continued use of that substance. Reinforcing effects appear to play an important role when addictive behaviors are viewed as goal-directed, operant behaviors. However, even proponents of this model describe drug taking and other addictive behaviors as complex, multidetermined behaviors (Barrett, 1985).

Many theories and theorists also have used Pavlovian conditioning to understand addiction. The ability of substances to produce tolerance and withdrawal effects in laboratory animals has been at the center of basic research on substance use disorders. Demonstrating tolerance effects in animals set the stage for testing Pavlovian conditioning paradigms with these animals. It was not long before anticipatory drug-related behaviors could be linked to cues associated with the actual drug use. Situational cues could then elicit initial drug reactions and lead to "relapse," or resumption of the addictive behavior (Hinson, 1985). This process involves multiple areas and mechanisms in the brain (Carey, Carrera, & Damianopolous, 2014).

Several phenomena in the drug culture also support the important role of conditioning and cues in developing and recovering from addictive behaviors. The "needle high" of the heroin addict, who only needs to insert a needle with saline solution to get a partial replication of the actual drug-taking experience, supports a conditioning model, as does the experience of cocaine addicts who begin to sweat and get anxious at the sight of any bolus of a white substance, be it sugar or flour. In fact, many addictive behaviors seem to operate in a situation-specific manner. Until the expansion of gambling venues in many states, travel to a gambling center like Las Vegas, Reno, or Atlantic City was often critical for compulsive gamblers. Many smokers have places or settings where they do not smoke. Certain types of food ("junk") or eating settings (home vs. restaurant) seem most related to eating disorders. Drinking behavior and bars are significantly linked. Situational cues and classical conditioning have an important role to play in understanding addiction and change.

More recently, classical conditioning approaches that originally focused only on physiological responses have been expanded to include cognitions and psychological mechanisms in the repertoire of cues and responses (Adesso, 1985; Brown, 1993; Brown, Goldman, & Christiansen, 1985; Robinson & Berridge, 1993). This has led to an integration of conditioning and social learning perspectives. For example, expectancy effects can vary in strength and magnitude depending on the presence of various cues. In fact, a growing body of evidence shows that many behaviors thought to be direct effects of alcohol or drugs (e.g., increased aggression, disinhibition) can be produced by placebo doses in the right setting with the appropriate cognitive expectation (Collins et al., 1985).

The latest work in this area focuses on how repeated exposure creates implicit mechanisms, like attentional bias for alcohol and drug cues, that influence use, craving, and relapse (Field & Cox, 2008). There are also approaches being developed and tested to change implicit bias with both visual and manual manipulations (Schoenmakers et al., 2010; Weirs et al., 2006). Thus, conditioning involves physiological responses, as well as both explicit and implicit cognitive processing, which influence engagement in an addictive behavior.

There is substantive evidence for the role of conditioning and reinforcement effects in addictions. However, models that use only these two principles to explain acquisition and recovery appear to have difficulty explaining all the phenomena of addiction and change. Once addicted, even severe punishing consequences seem to be unable to suppress or extinguish the behavior. Even after long periods of abstinence, extinction appears problematic under certain conditions. For example, some women smokers stop smoking during pregnancy only to have the addiction reappear after the birth, despite 6–9 months of abstinence (Stotts, DiClemente, Carbonari, & Mullen, 1996). They appear able to suspend cigarette use at will across situations because of anticipated negative effects on the fetus. As with the previous models, the conditioning/reinforcement ones offer some insight, particularly into the development of substance use problems and into the situational cues that can promote relapse after a quit attempt, but they do not explain all initiation or successful change (Marlatt & Gordon, 1985; Orford, 1985).

Compulsive/Excessive Behavior Models

The difficulty stopping or successfully modifying addictive behaviors and the overdetermined and repetitive nature of most addictions have led some theorists and practitioners to link addiction with ritualistic, compulsive behaviors like repeated hand washing or cleaning rituals. The commonalties include the sense that the behavior is out of the

individual's control and appears to be trying to satisfy a psychological conflict or need. This same perspective can encompass both the compulsive and excessive types of models (Orford, 1985).

Those who compare addictions to compulsive behaviors most often come either from analytic perspectives, where addictions reflect deep-seated psychological conflict, or from a biologically based view that compulsive behaviors represent a biochemical imbalance reflected in brain neurotransmitters. Proponents of the first explanation would envision the solution in terms of analysis or conflict resolution. Proponents of the latter would explore psychoactive pharmacological treatments to bring the addictive/compulsive behaviors under control. Although these views are similar to ones described earlier under personality or physiological models, the compulsive behavior explanation seems to argue that the actual behavior, be it drug taking, eating, or alcohol consumption, is less important than the compulsive mechanism that somehow became attached to this behavior.

Orford (1985) has conceptualized addictions as excessive appetites where the appetitive nature of the behaviors or activities creates the potential for excess. Thus eating, sexual activity, and gambling share with alcohol and drug use not only a potential for excess but also a similar process leading to excess. This process of moving to excess is described primarily as a psychological one, wherein the appetitive activities have many interactive determinants that are important in diverse areas of functioning and that become involved in a "developmental process of increasing attachment" best understood by a "balance-of-force social learning model" (pp. 319–321). Understanding both treatment and change of excessive behaviors would require personal cost–benefit analyses and a decision-making process as well as rebuilding the balance in one's life.

Although the compulsive and excessive behavior models share common explanatory components, they can differ dramatically in their suggested cures or treatments. Once again, the connection between the addictive behavior and the individual's psychological functioning appears highlighted in this perspective as in the personality/intrapsychic models. However, the compulsive model seems to disregard the unique contribution of the various types of possible addictive behaviors. The excessive model, on the other hand, seems similar to a social learning perspective. Although it highlights the appetitive nature of the activities as a central dimension, the excessive model does not specify this appetitive process and how it can explain or underlie all addictions and, at the same time, predict unique addictions. Both compulsive and excessive behavior models appear to add a new twist to some previously described ones, adding some explanatory potential.

A Biopsychosocial Model

Discontent with the partial explanations offered by the previously described models spurred thoughtful individuals to propose an integration of these explanations (Donovan & Marlatt, 1988; Glantz & Pickens, 1992). They highlight the integration of biological, psychological, and sociological explanations by calling their model biopsychosocial (Buchman, Skinner, & Iles, 2010). This model proposes that addiction is best understood as the result of a confluence of factors representing these three broad areas of influence and that it encompasses process addictions like sex addiction (Hall, 2011; Samenow, 2010).

Donovan and Marlatt (1988) argue for the biopsychosocial model, stating that "addiction appears to be an interactive product of social learning in a situation involving physiological events as they are interpreted, labeled, and given meaning by the individual" (p. 7). The common features among addictions and the inadequacy of any single factor to explain addiction highlight the need for a more complex, multicomponent model across addictions. Thus multiple causes, systems, and levels of analyses are needed to understand the addiction process (Donovan & Chaney, 1985; Galizio & Maisto, 1985; Leonard & Blane, 1999; Volkow et al., 2016). The biopsychosocial model argues for this multiple causality in the acquisition, maintenance, and cessation of addictive behaviors. Proponents of this model often use the commonalties in the relapse process as an argument in support of it (Brownell, Marlatt, Lichtenstein, & Wilson, 1986; Davies, Elison, Ward, & Laudet, 2015; Marlatt & Gordon, 1985).

Although the proposal of an integrative model represents an important advance over the more specific, single-factor models, proponents of the biopsychosocial approach have not explained how the integration of biological, psychological, sociological, and behavioral components occur. This model does allow researchers from different traditions to agree on complexity and to use a common term. Most of the current models that explain the development of substance abuse problems emphasize risk and protective factors, identify factors from several biopsychosocial domains, and highlight an interaction of these risk and protective factors (Chassin et al., 1996; Hummel, Shelton, Heron, Moore, & Bree, 2013; Sanjuan & Langenbucher, 1999; Schulenberg, Maggs, Steinman, & Zucker, 2001; Windle & Davies, 1999). However, without a pathway that can lead to real integration, the biopsychosocial model represents only a semantic linking of terms or, at best, a partial integration. As such, it often allows individuals to use an integrative term while paying only lip service to aspects other than their primary area of interest. Biologically and physiologically oriented researchers talk about

the *bio*psychosocial model, whereas social influence advocates discuss the biopsycho*social* model, and so on. This appears particularly true when the model is used for prevention or treatment considerations. It is difficult to intervene in multiple areas at the same time, and many of the risk and protective factors are not amenable to change (family of origin, geographic location, parental absence). Often the clinician or researcher's primary interest area is highlighted, with inadequate attention given to other aspects. The biopsychosocial model clearly supports the complexity and interactive nature of the process of addiction and recovery. However, additional integrating elements are needed to make this tripartite collection of factors truly functional for explaining how individuals become addicted and how the process of recovery from addiction occurs.

CHANGE: THE INTEGRATING PRINCIPLE

This brief review of the most prevalent models of addiction and related research demonstrates several important facts. First, addiction seems to involve multiple determinants that represent very different domains of human functioning, reaching from elements deep inside the individual, like self-esteem and neurobiology, to broad-based societal influences. Second, the search for a single explanatory construct at a single point in an individual's life appears fruitless. Risk and protective factors differ with age of initiation and developmental tasks (Conrod & Nikolau, 2016). Moreover, use and misuse affect biology, social interactions, and genetic influences (McGue & Irons, 2013). Finally, integrative perspectives such as the biopsychosocial model are beginning to dominate clinical and research discussions of addiction. Unlike current iterations of the biopsychosocial model, however, a truly integrative framework should provide the glue to join the various research-supported explanatory models. Moreover, such a perspective should lead to a comprehensive view of addiction that could orchestrate the integration of the multiple determinants.

The diverse etiological perspectives for understanding addiction discussed above offer partial, often one-dimensional views of the problem of addiction. The social/environmental model envisions addiction arising mostly as a reflection of the type of social environment (poverty, lack of education and opportunity, etc.) surrounding the individual who becomes addicted or highlights the influence of labeling and other social phenomena. The genetic/physiological model searches for answers in the physiological and neurobiological dimension. The personality/intrapsychic model views addiction as a failure of character and will. The coping/social learning model sees addiction as a function of personal

coping behavior and the influence of role models, peers, and parents. Conditioning/reinforcement models search the environment for the cues and reinforcers that create an addiction. There are clear case examples that would support one or another of these elements as a critical aspect or causal influence in addiction or recovery (Fletcher, 2001; Wholey, 1984). However, it bears repeating that no single source of influence has been found that can explain any single addiction, let alone all the various types of addictions (Glantz & Pickens, 1992). There is also no single developmental model or singular historical path that can explain acquisition of and recovery from addictions (Chassin, Presson, Sherman, & Edwards, 1991; Jessor, Van Den Bos, Vanderryn, Costa, & Turbin, 1995; Schulenberg et al., 2001).

The Transtheoretical Model (TTM) of intentional behavior change attempts to bring together divergent perspectives by focusing on how individuals change behavior and by identifying key change dimensions involved in this process (DiClemente & Prochaska, 1998; Prochaska & DiClemente, 1984). It is the personal pathway, and not simply the type of person or environment, that appears to be the best way to integrate and understand the multiple influences involved in acquiring and ceasing addictions (DiClemente, 2007; DiClemente, Delahanty, & Fiedler, 2010). Beginning and quitting addictive behaviors involve the individual and his or her unique decisional considerations. A person's choices influence and are influenced by both character and social forces. There is an interaction between the individual and the risk and protective factors that influence whether the individual becomes addicted and whether he or she leaves the addiction. The transitions into and out of addictions do not occur without the participation of the addicted individual—the individual is involved in how these influences are processed and whether their impact will be strong enough to overcome contrary values and become incorporated into his or her value system. Developing an addictive behavior and recovery from addiction both require a personal journey through an intentional change process that is influenced at various points by the host of factors identified in the etiological models just reviewed.

As often occurs, conflicting models are best resolved with a "both–and" answer instead of an "either–or" type of question. The stages of change, processes of change, context of change, and markers of change identified in the TTM offer a way to integrate these diverse perspectives without losing the valid insights gained from each perspective. This is the essence of an integrative, transtheoretical perspective. The TTM of intentional human behavior change (DiClemente, 2005, 2006; DiClemente & Prochaska, 1998; Prochaska & DiClemente, 1984; Prochaska, DiClemente, & Norcross, 1992) will be the integrating framework offered in this book.

Using the process of intentional human behavior change as the

integrating construct has many additional advantages. First, implicit in the concept of human behavior change is a developmental perspective. Change in humans takes place over time, at different points in the life cycle, and most often involves a sequence of events. Addiction and recovery occur in the context of human development and of an individual's life space, which include both physiological and psychological events and transitions (Deas, Riggs, Langenbucher, Goldman, & Brown, 2000; Jessor et al., 1995; Kandel & Davies, 1992; Keyes, Iacono, & McGue, 2007; McGue & Irons, 2013). In fact, the current developmental perspective on addiction is completely consistent with a process of change view on addictions. Schulenberg and colleagues (2001) characterize a developmental–contextual framework as one that "emphasizes multidimensional and multidirectional development across the life span, with stability and change occurring as a function of the dynamic interaction between individuals and their contexts" (p. 22). Furthermore, a change-process perspective avoids static explanations for what appears to be a rather active process. Addiction and recovery are dynamic in nature, include periods of perturbation and disruption as well as of stability, and are vulnerable to acceleration and deceleration. Finally, placing addiction into the larger context of an intentional, human change process can increase our ability to identify and explore similarities across addictive behaviors and allows us to compare modifying addictive behaviors with modifying other health and mental health behaviors.

The recent shift from a symptoms-based view of recovery to a more holistic and comprehensive one also supports an individual process of change perspective. The field is moving from a view of someone as "in remission" if they are abstinent with an absence of symptoms to one that views recovery in terms of wellness and quality of life. Recently the Center for Substance Abuse Treatment defined recovery as a process of change: "Recovery from alcohol and drug problems is a process of change through which an individual achieves abstinence and improved health, wellness, and quality of life" (Sheedy & Whitter, 2009, p. 1).

In the next chapter I examine in greater depth the process of human intentional behavior change and the core dimensions of the TTM. The model has been labeled "Transtheoretical" (across theories) because, from its inception more than 30 years ago, key elements used in creating the model were derived from different theories of human behavior and diverse views of how people change (Prochaska & DiClemente, 1984). Thus the model is an eclectic and integrative one that owes a debt of gratitude to many theory builders and researchers in the behavioral sciences past and present. In the following chapters I describe how this theoretical framework can be used to better understand the process involved both in the creation of an addiction and in the recovery from addiction.